Gait impairment in cervical spondylotic myelopathy: Analysis, impact on function, and effect of surgical intervention

Volume 1 of 1

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Declaration

I declare that this thesis, which I submit to RCSI for examination in consideration of the award of a higher degree of PhD, is my own personal effort. Where any of the content presented is the result of input or data from a related collaborative research programme this is duly acknowledged in the text such that it is possible to ascertain how much of the work is my own. I have not already obtained a degree in RCSI or elsewhere on the basis of this work. Furthermore, I took reasonable care to ensure that the work is original, and, to the best of my knowledge, does not breach copyright law, and has not been taken from other sources except where such work has been cited and acknowledged within the text.

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Date _______________________________________________________

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<td>A2</td>
<td>Peak concentric ankle plantarflexor power at terminal stance</td>
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<td>ACCF</td>
<td>Anterior cervical corpectomy and fusion</td>
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<tr>
<td>ACDF</td>
<td>Anterior cervical discectomy and fusion</td>
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<tr>
<td>ADL</td>
<td>Activities of daily living</td>
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<tr>
<td>AGLR</td>
<td>Approximated generalised likelihood ratio method</td>
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<td>AIDS</td>
<td>Acquired immunodeficiency syndrome</td>
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<td>ANOVA</td>
<td>Analysis of variance</td>
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<td>AP</td>
<td>Anterior-posterior</td>
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<td>ARV</td>
<td>Average rectified value</td>
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<td>ASIA</td>
<td>American Spinal Injury Association</td>
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<td>ASIS</td>
<td>Anterior superior iliac spine</td>
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<td>ASCII</td>
<td>American standard code for information interchange</td>
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<tr>
<td>BF</td>
<td>Biceps femoris</td>
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<td>BWSTT</td>
<td>Body weight supported treadmill training</td>
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<td>CINAHL</td>
<td>Cumulative index of nursing and allied health literature</td>
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<td>cm</td>
<td>Centimetres</td>
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<td>CMC</td>
<td>Coefficient of multiple correlation</td>
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<td>CMD</td>
<td>Coefficient of multiple determination</td>
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<td>CMRR</td>
<td>Common mode rejection ratio</td>
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<td>CNS</td>
<td>Central nervous system</td>
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<td>CP</td>
<td>Cerebral palsy</td>
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<td>CSM</td>
<td>Cervical spondylotic myelopathy</td>
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<td>CSV</td>
<td>Comma separated values</td>
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<td>CT</td>
<td>Computed tomography</td>
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<td>dB</td>
<td>Decibels</td>
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<td>DTM</td>
<td>Double threshold method</td>
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<td>EMS</td>
<td>European Myelopathy Score</td>
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<td>FIM</td>
<td>Functional independence measure</td>
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<td>GORD</td>
<td>Gastro-oesophageal reflux disorder</td>
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<td>GP</td>
<td>General practitioner</td>
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<td>GRF</td>
<td>Ground reaction force</td>
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<td>H reflex</td>
<td>Hoffmann reflex</td>
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H1  Peak concentric hip extensor power during loading response
H2  Peak eccentric hip flexor power during mid-stance
H3  Peak concentric hip flexor power during terminal stance
HC  Healthy control
HRQOL  Health-related quality of life
Hz  Hertz
ICC  Intraclass correlation coefficient
ICF  International Classification of Functioning, Disability and Health
ISEK  International Society for Electromyography and Kinesiology
JOA  Japanese Orthopaedic Association scale
K1  Peak eccentric knee extensor power during loading response
K2  Peak concentric knee extensor power during mid-stance
K3  Peak eccentric knee extensor power in terminal stance
K4  Peak eccentric knee flexor power, terminal swing
KAD  Knee alignment device
kg  Kilograms
km/h  Kilometres per hour
l0/s  Normalised muscle lengths per second, units of lengthening velocity
LBP  Low back pain
LLMP  Laminectomy and lateral mass plating
LOA  Limits of agreement (Bland and Altman)
LSMS  Locomotor specific measure of spasticity
LVT  Lengthening velocity threshold
m  Metres
MAS  Modified Ashworth Scale
MDM  Mean dynamic method
MEP  Motor evoked potentials
MeSH  Medical subject headings
MG  Medial gastrocnemius
MHH  Modified Helen Hayes model
MI  Myocardial infarction
mJOA  Modified Japanese Orthopaedic Association scale
mm  Millimetres
MΩ  Mega Ohms
m/s  Metres per second
MRI  Magnetic resonance imaging
MRMI  Modified Rivermead Mobility Index
MSE  Mean standard error
mV  Millivolts
µV  Microvolts
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<td>Maximum voluntary contraction</td>
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<tr>
<td>MVIC</td>
<td>Maximum voluntary isometric contraction</td>
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<td>N</td>
<td>Newtons</td>
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<td>NIDDM</td>
<td>Non insulin dependent diabetes mellitus</td>
</tr>
<tr>
<td>Nm</td>
<td>Newton metres</td>
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<tr>
<td>NOF</td>
<td>Neck of femur</td>
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<tr>
<td>Ω</td>
<td>Ohms</td>
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<tr>
<td>PASW</td>
<td>Predictive Analytics Software</td>
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<td>PC</td>
<td>Principal component</td>
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<td>Principal components analysis</td>
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<td>Peak dynamic method</td>
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<td>PEDro</td>
<td>Physiotherapy evidence database</td>
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<td>PI</td>
<td>Principal investigator</td>
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<td>PIG</td>
<td>Plug-in Gait ®</td>
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<td>PMH</td>
<td>Past medical history</td>
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<td>PWS</td>
<td>Preferred walking speed</td>
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<td>QQ plot</td>
<td>Quantile–quantile plots</td>
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<td>RA</td>
<td>Rheumatoid arthritis</td>
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<td>RCSi</td>
<td>Royal College of Surgeons in Ireland</td>
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<td>RCT</td>
<td>Randomised controlled trial</td>
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<td>RF</td>
<td>Rectus femoris</td>
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<td>ROM</td>
<td>Range of motion</td>
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<td>RMS</td>
<td>Root mean square</td>
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<td>RMS_MAX</td>
<td>Maximum root mean square amplitude of a signal</td>
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<td>RR</td>
<td>Relative risk</td>
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<td>RTM</td>
<td>Resting threshold method</td>
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<td>s</td>
<td>Seconds</td>
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<td>SCI</td>
<td>Spinal cord injury</td>
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<td>SD</td>
<td>Standard deviation</td>
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<td>SEM</td>
<td>Standard error of measurement</td>
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<td>SEMG</td>
<td>Surface electromyography</td>
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<td>SENIAM</td>
<td>Surface Electromyography for Non-Invasive Assessment of Muscles</td>
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<td>SEP</td>
<td>Somatosensory evoked potentials</td>
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<td>SF36</td>
<td>RAND Medical Outcomes Study 36-item Short Form Health Survey</td>
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<td>SI</td>
<td>Signal intensity</td>
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<td>SNR</td>
<td>Signal to noise ratio</td>
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<td>TA</td>
<td>Tibialis anterior</td>
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<td>TBI</td>
<td>Traumatic brain injury</td>
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<td>TKEO</td>
<td>Teager-Kaiser energy operator</td>
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<td>TSP</td>
<td>Temporal-spatial parameters</td>
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<td>UMNL</td>
<td>Upper motor neurone lesion</td>
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<td>V</td>
<td>Volts</td>
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<td>W</td>
<td>Watts</td>
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<td>WISCI</td>
<td>Walking index for spinal cord injury</td>
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## Glossary of terms

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<td><strong>Cervical spondylotic myelopathy</strong></td>
<td>Compression of the spinal cord in the cervical canal due to narrowing of the spinal canal as a result of cervical spondylosis</td>
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<tr>
<td><strong>Kinematics</strong></td>
<td>Angular movement of joints</td>
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<tr>
<td><strong>Kinetics</strong></td>
<td>Forces, moments and powers acting on joints</td>
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<tr>
<td><strong>Joint moment</strong></td>
<td>The product of a force applied and its distance from the pivot point or fulcrum. In gait, a joint moment represents the body’s internal response to an external load</td>
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<td><strong>Joint power</strong></td>
<td>Scalar product of the joint moment and the angular velocity of the moving segment. Quantifies the net energy absorbed or generated by the working muscles</td>
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<td><strong>Gait analysis</strong></td>
<td>The systematic measurement, description, and assessment of quantities that characterise a person’s walking pattern</td>
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<tr>
<td><strong>Three-dimensional gait analysis</strong></td>
<td>Analysis of gait using motion analysis cameras, force plates and sophisticated computer algorithms to determine joint biomechanics from the trajectories of reflective markers placed over skeletal landmarks</td>
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<td><strong>Electromyography</strong></td>
<td>Measurement and interpretation of the electrical signal associated with muscle contraction</td>
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<tr>
<td><strong>Gait cycle</strong></td>
<td>Time during gait from initial contact of one lower limb to initial contact of the ipsilateral lower limb</td>
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Summary

Gait impairment is a primary symptom of cervical spondylotic myelopathy (CSM), yet it is poorly understood due to a lack of quantitative data on kinematics, kinetics, and electromyography (EMG) of muscle activity during gait. Furthermore, the effect of surgical decompression of the spinal cord on gait impairment is not well established. The aims of this study were to analyse and describe the gait patterns of people with CSM compared to age- and gender-matched healthy controls (HCs), and to determine the effect of surgery on gait impairment. A secondary objective was to measure changes in CSM severity, functional mobility, health-related quality of life (HRQOL) and spasticity following surgery. The thesis was divided into three studies: a reliability study of three-dimensional gait analysis (3DGA) and EMG parameters in the CSM population, a cross-sectional study to compare gait in CSM with HCs, and an experimental study to evaluate changes in gait following surgery.

A test-retest reliability study of 12 participants with CSM determined that most temporal-spatial, kinematic and kinetic parameters showed sufficient repeatability for use in clinical and research practice. EMG parameters were more variable. A range of measures of the timing and amplitude of muscle activation and its response to lengthening were chosen based on the results of the reliability study.

The cross-sectional study involving CSM participants (N = 16) and age- and gender-matched HCs (N = 16), found that CSM participants had a significantly slower comfortable gait speed, with reductions in multiple peak kinematic and kinetic parameters in the sagittal plane. At matched speed, differences persisted in several parameters pertaining to propulsion and momentum generation, and this was confirmed by principal components analysis. EMG analysis showed prolonged duration of activation of rectus femoris, biceps femoris and tibialis anterior, and excessive co-activation between rectus and biceps femoris, in the CSM cohort. Analysis of the 3DGA and EMG data indicated that paresis was a significant underlying factor to the CSM gait. Spasticity and proprioception appeared to have less effect.

Thirteen participants underwent decompressive surgery and participated in the experimental study. At last post-operative follow-up, there were significant improvements in CSM severity, functional mobility and HRQOL. No changes were detected in temporal-spatial or kinematic gait parameters, however kinetic parameters showed an improvement in the absorption and generation of power at the knee and ankle at key points in the gait cycle. EMG analysis showed compensatory responses in muscle activation patterns of tibialis anterior, and an increase in lengthening velocity threshold, indicating reduced spasticity, of the rectus femoris. The results confirmed that surgery had achieved its aim in preventing further deterioration of function. Kinetic and EMG changes indicated that the locomotor system showed potential for improvement following surgery, a novel finding given that recovery of function is not generally expected. It was recommended that this potential should be explored through rehabilitation strategies aimed at maximising the locomotor system’s ability to recover following spinal cord injury through neuroplasticity and compensatory mechanisms.
Chapter 1: Introduction

1.1 Purpose of the thesis

The purpose of this thesis was to identify and describe the changes in gait in people with cervical spondylotic myelopathy, and to determine the impact of decompressive surgery to the spinal cord on gait impairment.

1.2 Background to the development of the thesis

Cervical spondylotic myelopathy (CSM) is a degenerative disorder of the cervical spine characterised by narrowing of the cervical spinal canal and compression of the spinal cord (Montgomery and Brower, 1992). It is the most common cause of spinal cord dysfunction in individuals over the age of 55 (Fehlings and Skaf, 1998). The clinical presentation may include symptoms such as clumsy hands (Olindo et al., 2008), hyperreflexia, paraesthesia in one or both upper limbs, neck pain and gait disturbance (Nurick, 1972). The diagnosis relies heavily on clinical examination with support from radiological findings (Cook et al., 2009).

Gait impairment is one of the primary symptoms of CSM. The condition often manifests initially through subtle changes in gait and balance, progressing to weakness, stiffness and sensory loss and the feeling of an awkward, clumsy gait pattern (Rao, 2002). Some patients may present with spastic paraparesis or tetraparesis (Dvorak et al., 2003). There is little evidence to date in the literature to describe the gait patterns of people with CSM. Many published studies are limited to temporal-spatial and kinematic analysis (Kuhtz-Buschbeck et al., 1999, Maezawa et al., 2001, Kim et al., 2010, Lee et al., 2011), without analysis of kinetics or electromyography (EMG). The rehabilitation of people with CSM is limited by this lack of evidence, as without a clear description of the problem, therapists cannot design rehabilitation interventions to address it.

CSM can be managed conservatively, through the use of traction (Yoshimatsu et al., 2001) or soft cervical collars (Phillips, 1973, LaRocca, 1988), or surgically, by decompression of the spinal cord (Furlan et al., 2011). The aim of surgery in most cases is to arrest the progression of neurological signs and symptoms by stabilising the degenerative cervical levels. Improvement in function is not generally expected (Rao et al., 2006). Due to its uncertain natural history, the possibility of rapid irreversible loss of function, and unsatisfactory outcomes with conservative management, surgery has emerged as the gold standard of treatment (Jankowitz and Gerszten, 2006). However, prediction of outcome following surgery is limited by the lack of robust evidence from published research. Many studies have been criticised for methodological flaws such as inadequate randomisation, retrospective designs, and small sample sizes (Jankowitz and
Gerszten, 2006). In particular, many of the scales commonly used to assess outcome following surgery are ordinal in nature, and may be insensitive to change. One category can cover a range of severity and non-quantitative measures may be prone to bias (Singh and Crockard, 1999). There is a need for more sensitive, quantitative and reliable measures to ensure that outcomes for people with CSM are accurately measured, and to provide greater certainty in clinical decision-making (Jankowitz and Gerszten, 2006).

### 1.3 Justification for the thesis

It was evident that several gaps existed in the literature on CSM. Firstly, the lack of quantitative data on gait impairment in this condition was a significant barrier for rehabilitation professionals and patients alike. A disorder cannot be effectively treated if it is not fully understood. It has been stated that a detailed analysis of gait, such as that provided by three-dimensional analysis of joint motion, moments and powers, coupled with electromyographic analysis of muscle activity, can significantly enhance the understanding of a deficit (Patrick, 2003). Such analysis forms the mainstay of the assessment and classification of gait in cerebral palsy (CP) (Gough and Shortland, 2008). The use of three-dimensional gait analysis (3DGA) and EMG to compare gait in people with CSM to healthy matched controls has potential to address the gaps in knowledge on gait in CSM, and to direct rehabilitation protocols towards the key deficits.

The second major gap in the literature concerned the lack of quantitative, sensitive data on outcomes following decompressive surgery to the spinal cord. In determining the best approach to management, the surgeon and patient must weigh up the risks of intra-operative complications against the possibility of further irreversible neurological deterioration if the cord is not decompressed. Previous studies have been hampered by the use of ordinal scales as outlined above (Jankowitz and Gerszten, 2006). The introduction of a standardised timed walk test has provided more quantitative data in the evaluation of CSM (Singh and Crockard, 1999) however gait speed alone provides no indication of the quality of a gait pattern. There may be changes in gait following surgery that are not apparent from an ordinal scale or a timed walk test. Analysis of gait using 3DGA and EMG would allow greater precision and sensitivity in the evaluation of post-operative outcomes, and could reveal changes in the quality of gait that were not previously documented.

### 1.4 Structure of the thesis

The thesis is divided into ten chapters, including the current chapter. The literature review is contained in Chapters 2, 3 and 4. The aims and objectives of the thesis are outlined in detail at the end of Chapter 4. To achieve these aims and objectives, the project is divided into three distinct but related studies: 1) a reliability study of the primary outcome measure, 3DGA and EMG analysis of gait, 2) a cross-sectional study to compare gait in
people with CSM to age- and gender-matched healthy controls, and 3) an experimental study to evaluate gait impairment in people with CSM before and after surgical decompression of the spinal cord. Chapter 5 discusses the development of methodology common to the three studies, and Chapter 6 describes the implementation of this methodology. The results of the three studies are detailed and discussed in Chapters 7, 8 and 9, respectively. The concluding chapter, Chapter 10, draws together the findings of the three studies and discusses their implications for patients, surgeons and physiotherapists in the understanding of CSM and its management. Figure 1.1 illustrates the structure of the thesis.
Figure 1.1: Structure of the thesis
Chapter 2: Pathophysiology, Clinical Presentation and Natural History of Cervical Spondylotic Myelopathy

2.1 Introduction

This chapter will examine the pathophysiology and aetiological mechanisms underlying the development of CSM. The clinical presentation and diagnosis of the disorder will be discussed. Finally, current understanding of the natural history of CSM, and its response to surgical and conservative management, will be considered. The aim of this chapter is to outline the aspects of pathophysiology, prognosis and current management of CSM that led to the development of the hypotheses, aims and objectives of this thesis.

2.1.1 Terminology

Many of the terms used in this chapter describe a pathophysiological process involving some related but distinct entities. To ensure clarity, the terminology used will be as follows.

- Cervical spondylosis: Progressive, degenerative changes within the cervical spine.
- Cervical myelopathy: Compression of the spinal cord in the cervical canal due to any cause.
- Myelopathy: Compression of the spinal cord at any spinal level or levels.
- Cervical spondylotic myelopathy (CSM): Compression of the spinal cord in the cervical canal due to narrowing of the spinal canal as a result of cervical spondylosis.

2.2 Pathophysiology of Cervical Spondylotic Myelopathy

2.2.1 Prevalence

Cervical spondylosis is a common progressive disorder of the ageing cervical spine and has been reported to affect up to 95% of people over the age of 65 (Garfin, 2000). It is characterised by degenerative changes affecting the intervertebral discs, vertebrae, facet joints, and ligamentous structures around the cervical spinal canal (Tracy and Bartleson, 2010). The most common symptoms of cervical spondylosis are neck pain and radiculopathy (Shedid and Benzel, 2007). In a small number of cases, spondylosis can progress to the point where it affects the function of the spinal cord due to narrowing of the central spinal canal, resulting in the condition known as CSM (Asgari, 1996).
The exact prevalence of CSM is unknown (Dvorak et al., 2003). In many cases, the symptoms are incorrectly attributed to age or other neurological conditions (Rao, 2002). Research has generally been confined to a select population of patients who have a definite diagnosis and well-established disease, resulting in a lack of data on the true epidemiology of CSM. Given its degenerative aetiology, it is almost certain that the true incidence is significantly higher than the number of people who present to the health service with symptoms and signs.

2.2.2 Aetiology

Risk factors for the development of CSM have not been fully established, but are believed to include a combination of genetic and environmental factors. CSM is also associated with other medical conditions.

2.2.2.1 Genetic factors

Racial factors have been implicated in the genetic aspects of CSM aetiology. Asian populations in particular have a higher incidence of CSM than other races. This has been attributed to an increased prevalence of defects in the nucleotide pyrophosphatase gene, which is associated with ossification of the posterior longitudinal ligament (Nakamura et al., 1999).

CSM is also more prevalent in Down syndrome. In this population, it tends to present at a young age with progressive deterioration in gait and weakness of all four limbs (Bosma et al., 1999). Down syndrome is associated with ligamentous laxity. This could be a risk factor for spondyloarthrosis of the cervical spine, in addition to its association with instability of the atlanto-axial joint (Tyrrell et al., 1998).

Gender has also been considered a genetic risk factor. Salvi et al. (2006) reported that males are more frequently affected by CSM than females, with a ratio of 2.4 to 1. A radiographic analysis of the canal to vertebral body ratio of the cervical spine in young healthy adults found a smaller ratio in males, and this could explain the higher prevalence of CSM in men (Hukuda and Kojima, 2002).

2.2.2.2 Environmental factors

Environmental factors implicated in CSM include repeated occupational trauma, such as the carrying of heavy axial loads (Baron and Young, 2007) and the repeated cervical extension strains sustained in occupations with a high prevalence of overhead activities, such as fruit farming (Takamiya et al., 2006). These factors might also account for the prevalence of the disorder among males, who may be more likely than females to engage in occupations involving load carrying or other cervical stresses.
Smoking has been associated with disc degeneration, and some authors have considered it a risk factor for the development of spondylosis (Hadley and Reddy, 1997). However, there is no conclusive evidence of a greater prevalence of CSM among smokers compared to non-smokers.

2.2.2.3 Medical and other causes of CSM

CSM has been linked to co-existing medical conditions. One of the most common associated conditions is rheumatoid arthritis (RA), where progressive joint destruction can affect the cervical spine, leading to cord compression (Ranawat et al., 1979, Iizuka et al., 2009). Acquired immunodeficiency syndrome (AIDS) has been suggested as a possible factor in vascular CSM, however histological studies of spinal cord pathology in 90 AIDS patients did not support the hypothesis that AIDS was directly involved in the pathogenesis of CSM (Shepherd et al., 1999).

Many other causes of cervical myelopathy have been described in the literature, however some of these presentations do not have a clear spondylotic aetiology. The potential adverse effects of spinal manipulation were described in a case series involving 18 patients, nine of which had symptoms of spinal cord injury (SCI) (Oppenheim et al., 2005). Two cases of delayed radiation myelopathy following radiotherapy treatment for cancer have been documented (Koehler et al., 1996). A case study described the course of cervical myelopathy due to acute disc protrusion during maternal labour (Tsai et al., 2006). A complete picture of the risk factors for these rare causes of myelopathy has not been established, but risk factors may have included diabetes and hypertension in the radiation group (Koehler et al., 1996). Three of 18 patients with complications following manipulation had pre-existing spinal pathology such as syringomyelia and atlanto-axial instability (Oppenheim et al., 2005). Some of these presentations may have been more consistent with acute traumatic SCI than CSM, though it is difficult to separate traumatic cord injury from degenerative cord compression on retrospective analysis. In this review, focus will be maintained on CSM as a degenerative process, though it is acknowledged that the symptoms and signs of this degenerative process may be accelerated by acute trauma (Matsunaga et al., 2002, Bednarik et al., 2011).

2.2.3 Pathophysiological processes

2.2.3.1 Overview

Stookey (1928) published the first reports of spinal cord compression due to degenerative changes, describing compression of the cord by cartilaginous nodules of degenerated disc material. A landmark paper later attributed the clinical signs of neurological deterioration to cervical spondylosis (Brain et al., 1952). This section will now consider the pathophysiology of cervical spondylosis, and will then discuss its progression to CSM.
2.2.3.2 Pathophysiology of cervical spondylosis

The understanding of the pathophysiology of cervical spondylosis has evolved over time. It is now generally accepted that spondylosis results from degenerative processes that alter the mechanics of the spinal column in bearing axial load (Baptiste and Fehlings, 2006). White and Panjabi (1988) divided the factors involved in the pathogenesis into static and dynamic factors. The static factors relate to primary degenerative processes, precipitated by non-inflammatory disc degeneration in the ageing cervical spine. The process of disc degeneration is complex. It involves alterations of normal physiology as well as the process of ageing (Shedid and Benzel, 2007). The chemical composition of the disc deteriorates with age (Rao, 2002). This leads to a gradual loss of water within the discs, causing fragmentation and collapse of the inner nucleus pulposus (Baron and Young, 2007). The discs lose their effectiveness in bearing and transferring load, and begin to split medially (Baptiste and Fehlings, 2006). The load on the uncovertebral processes increases, causing them to become flattened (Kumaresan et al., 2001). Greater stress is then placed on the articular cartilage of the vertebrae and their end plates, leading to the formation of osteophytic spurs at the margins of the end plates to increase the weight-bearing surface of the vertebrae (Baptiste and Fehlings, 2006). The ligamentum flavum also thickens and may buckle posteriorly (Muthukumar, 2005). Degeneration or calcification of the posterior longitudinal ligaments may occur (White and Panjabi, 1988). The net effect of these changes is a reduction in the sagittal spinal canal diameter (Shedid and Benzel, 2007).

Dynamic pathological factors also contribute to the development of cervical spondylosis. With progression of the static changes outlined above, abnormal forces on the spinal cord and spinal column are generated during movement (White and Panjabi, 1988). Changes in the dynamics of neck flexion and extension cause narrowing of the spinal canal during movement, increasing the strain and shear forces on the spinal cord (Baptiste and Fehlings, 2006). This further contributes to osteophytosis. Mihara et al. (2000) found that elderly patients with CSM had increased segmental mobility at C3/4, the level at which their myelopathy developed, than at C4/5 and caudal segments. This provided evidence for the role of dynamic factors in the pathophysiology of CSM. Dynamic factors explain why occupational activities such as the carrying of heavy loads contribute to the development of CSM. Further abnormal forces will be generated during these activities, compounding the development of osteophytosis.

2.2.3.3 Development of myelopathy as a result of cervical spondylosis

In the majority of people, spondylotic changes have no adverse effects on the spinal cord. Individuals who become symptomatic with cervical spondylosis will usually present with symptoms of axial neck pain or radiculopathy, rather than with spinal cord signs (Shedid
and Benzel, 2007). The exact degree of canal stenosis that leads to the development of symptoms and signs of cord compression is not fully known.

Attempts to correlate the size of the cervical spinal canal with neurological signs have shown variable results (Rao and Fehlings, 1999). On plain radiographs, clinical signs of cord compression have been associated with sagittal anterior-posterior (AP) canal diameters of varying sizes including less than 13 millimetres (mm) (Arnold, 1955), 11 mm during extremes of flexion and extension (Penning, 1962), 9 mm (Fukui et al., 1990), and 17 mm (Edwards and LaRocca, 1985). The sensitivities and specificities of these cut-off points varied from below 50% in some studies to over 80% in others (Rao and Fehlings, 1999).

Computed tomography (CT) myelography has been used to measure the cross-sectional area of the spinal cord. One study found that the onset of long tract neurological signs was associated with a 30% reduction in the cross-sectional area of the spinal canal (Penning et al., 1986), a finding reported to have more than 80% sensitivity and 80% specificity for clinical cord compression (Rao and Fehlings, 1999). A 60% encroachment into the spinal canal from an ossified posterior longitudinal ligament has been linked with the development of cord symptoms and signs (Matsunaga et al., 2004). In a further study using CT myelography, Hukuda et al. (1996) found that, when compared to controls, CSM was associated with a larger vertebral body size as well as a narrowed spinal canal. The authors suggested that a high vertebral body to spinal canal size ratio, known as the Torg ratio, could be indicative of a greater degree of osteophyte formation and disc protrusion. A review paper found few studies that provided objective, quantifiable, and reliable radiographic measurements of cervical spinal canal compromise or cord compression (Rao and Fehlings, 1999).

Magnetic resonance imaging (MRI) may provide a greater degree of accuracy. The use of T1- and T2-weighted images to evaluate mid-sagittal AP cervical canal diameter found that a reduction in diameter of 25% or more showed 76% sensitivity and 98% specificity for clinical cord compression (Fehlings et al., 1999). However, this study was conducted in acute cervical SCI, and the findings may not generalise to a degenerative aetiology such as CSM. Further studies are needed to establish a more accurate measurement of canal stenosis at which neurological deterioration can be predicted.

2)2):)D! ;'-054#-59!'//'-,1!./!5!45&&.?'<!-'&7#-59!1A# 459!-5459!

From a mechanical point of view, a narrowed spinal canal is thought to cause compression of the spinal cord, leading to local tissue ischaemia and injury to the neural cells (Baptiste and Fehlings, 2006). The spinal cord can also be damaged dynamically by tensile stretch during extremes of flexion and extension, as demonstrated by Shi and Pryor (2002). The cord initially responds well to stretch, but then becomes progressively
stiffer as the fibres straighten out and begin to bear tensile load. The effects of tensile stretch are limited initially to temporary ionic imbalances across the nerve cell membrane, but this can progress to more permanent conduction loss as the membranes suffer more profound anatomical damage (Shi and Pryor, 2002). As a result, damage to the spinal cord occurs from both a narrowed spinal canal diameter and the resulting pathological changes in spinal cord biomechanics.

2.2.3.5 Vascular changes following narrowing of the spinal canal

Mechanical factors alone, however, do not adequately explain the spectrum of clinical findings in CSM. While a narrowed spinal canal may be an essential prerequisite for the development of myelopathy, the degree of canal stenosis does not always correlate with the neurological deficit, and indeed many patients with a spinal canal diameter of less than 13 mm have a normal neurological examination (Hayashi et al., 1987). Nurick (1972) first put forward this consideration, stating: “CSM is associated with a narrow canal, although a narrow canal does not always lead to CSM”. Other processes are therefore implicated in the pathophysiology.

There is considerable evidence that vascular ischaemia may be an important factor. Cadaveric studies have shown that compression of the spinal cord anteriorly will reduce the perfusion through the transverse arterioles arising from the anterior sulcal arteries, while posterior compression will compromise perfusion through the intramedullary branches of the central gray matter (Baptiste and Fehlings, 2006). Furthermore, angiographic studies have shown a reduction in perfusion to the spinal cord in animal models of myelopathy (Hukuda and Wilson, 1972). Shedid and Benzel (2007) stated that although the degeneration of cervical spinal elements is the primary pathological lesion in cervical spondylosis, it is the secondary vascular sequelae that lead to myelopathy.

Ischaemic or traumatic injury to the central nervous system (CNS) can result in programmed cell death, namely apoptosis. Studies in traumatic brain injury (TBI) and SCI have found that oligodendrocytes are particularly vulnerable to the effects of ischaemia. Post-mortem studies of both human and animal spinal cord compression have found evidence of oligodendroglial death alongside intact but demyelinated axons (Bunge et al., 1993). Oligodendrocytes are critical to the CNS in the promotion of neurological development and the formation and maintenance of myelin sheaths. Their apoptosis may explain the demyelination associated with chronic myelopathy (Pfeiffer et al., 1993). Kim et al. (2003) also suggested that early apoptosis of oligodendrocytes may contribute to the profound and irreversible neurological deficits found in chronic CSM.

The effects of mechanical compression and ischaemic cell death do not manifest uniformly throughout the spinal cord. Histological analyses of the cord in affected people have found that the central grey matter and medial portions of the white matter are the
most severely affected areas, showing evidence of gliosis, cystic cavitation and demyelination (Fehlings and Skaf, 1998). By contrast, the anterior columns are usually less severely damaged. Wallerian degeneration tends to affect the posterior columns cephalad to the site of compression, and the corticospinal tracts below the site of compression. The progression of these pathological changes depends on the extent of cord compression. The lateral corticospinal tracts are the most vulnerable, while severe compression can lead to anterior horn cell loss or localised infarction of grey matter (Fehlings and Skaf, 1998). The extent and distribution of these changes within the cord may in part account for variations in the clinical presentation of CSM. The clinical features of CSM will now be considered.

2.3 Clinical presentation

2.3.1 Symptoms and signs

CSM presents with variable combinations of symptoms and signs depending on the relative involvement of corticospinal tracts, posterior columns and spinothalamic tracts. Most people with CSM will present with involvement of more than one of these structures (Takayama et al., 2005a). Early symptoms include subtle changes in gait and balance (Emery, 2001). This can progress to weakness, stiffness, or sensory loss in the lower limbs, often in an asymmetrical distribution (Baron and Young, 2007). Patients may complain of increasing difficulty with balance and an “awkward” gait, though they frequently attribute these symptoms to old age or arthritis (Rao, 2002). In the upper limbs, a feeling of “numb, clumsy” hands may be described, manifesting as a loss of dexterity, worsening of handwriting, or difficulty with fine motor tasks such as fastening zippers and buttons (Rao, 2002). Pain can also be a feature. Stabbing pain in a non-dermatomal pattern has been reported (Young, 2000). Pain may spread in a cape-like distribution across the shoulders (Rao, 2002). A positive Lhermitte’s phenomenon may be present with cervical flexion (Salvi et al., 2006).

Clinical signs feature a mix of upper and lower motor neuron findings, as the exiting nerve root may also be compressed at the spondylotic level, causing lower motor neuron signs at this level and upper motor neuron signs below this level (Connell and Wiesel, 1992). Physical examination may reveal limitation in the range of motion (ROM) of the cervical spine, particularly of lateral flexion and rotation to the side of greatest degenerative changes (Salvi et al., 2006). Muscle weakness and wasting can be evident on assessment of power. This is often bilateral and affects multiple muscle groups, rather than one cervical root (Salvi et al., 2006). Some people will present with spastic paraparesis or tetraparesis (Montgomery and Brower, 1992, Dvorak et al., 2003). Findings on sensory examination depend on the area of the cord that is involved, and the extent of compression of the nerve root at the exit foramen of the affected level (Rao,
Bilateral loss of sensation to vibration and joint position sense in the lower limbs has been described as a characteristic pattern in CSM (Brain et al., 1952, Dvorak et al., 2003). Clinical signs that pinpoint upper motor neurone pathology include hyperreflexia, an extensor plantar response, ankle clonus, a positive Hoffman’s sign, and an inverted supinator response (Cook et al., 2007). The term “myelopathy hand” has been used to describe a pattern of dysfunction of the intrinsic hand muscles associated with CSM, which is characterised by positive finger escape and grip-and-release tests (Ono et al., 1987, Ebara et al., 1988). A summary of these signs is provided in Table 2.1.
Table 2.1: Description of clinical signs used in the diagnosis of CSM

<table>
<thead>
<tr>
<th>Clinical Sign</th>
<th>Description</th>
<th>Positive Finding for CSM</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inverted supinator sign</td>
<td>Brachioradialis reflex elicited in the normal way</td>
<td>Reactive flexion of the ipsilateral fingers</td>
<td>(Kiely et al., 2010)</td>
</tr>
<tr>
<td>Grip and release test</td>
<td>Hand outstretched. Patient grips and releases with the fingers as rapidly as possible, and the number of complete cycles in 10 seconds is counted</td>
<td>Less than 20 complete cycles in 10 seconds</td>
<td>(Ono et al., 1987)</td>
</tr>
<tr>
<td>Finger escape sign</td>
<td>Hand held outstretched with palms down, fingers extended and adducted for 30-60 seconds</td>
<td>Ring and little fingers drift into abduction and flexion</td>
<td>(Ono et al., 1987)</td>
</tr>
<tr>
<td>Hoffman’s sign</td>
<td>Proximal interphalangeal joint of the middle finger is stabilised, distal phalanx then “flicked” into a flexed position</td>
<td>Adduction of the thumb and flexion of the fingers</td>
<td>(Sung and Wang, 2001)</td>
</tr>
<tr>
<td>Suprapatellar quadriceps test</td>
<td>Sitting with feet unsupported, suprapatellar tendon tapped quickly with reflex hammer</td>
<td>Hyperreflexive knee extension</td>
<td>(Cook et al., 2009)</td>
</tr>
</tbody>
</table>

2.3.2 Clinical diagnosis

There is no definitive diagnostic test for CSM. Accurate identification of the pathology presents a clinical challenge. The vague nature of the early symptoms of myelopathy, particularly with respect to lower limb weakness and gait abnormality, often leads to a delay in diagnosis (Salvi et al., 2006). In clinical practice, neurological signs are often used to identify possible myelopathy, and MRI evidence is then sought to confirm the diagnosis (Cook et al., 2009, Harrop et al., 2010).

2.3.2.1 Neurological signs

The neurological signs commonly used to diagnose CSM include Hoffman’s sign, the Babinski sign, clonus, the hand withdrawal reflex, deep tendon reflexes, the suprapatellar quadriceps test, and the inverted supinator sign (Cook et al., 2009). No single test has shown sufficiently high sensitivity, nor sufficiently low negative likelihood ratio for effective screening of patients with suspected myelopathy (Cook et al., 2009). In fact, many tests used in isolation show inconsistent results. One study found that the inverted supinator sign demonstrated moderate sensitivity (61%) and high specificity (75%) in tests of diagnostic accuracy for myelopathy (Cook et al., 2009). However, the same sign was present in 27.6% of healthy individuals, of whom 10% also had a positive Hoffman’s sign
but no other signs of myelopathy (Kiely et al., 2010). This indicated a high likelihood of false positives. Similarly, two further studies (Houten and Noce, 2008, Chikuda et al., 2010) found that Hoffman’s sign was more sensitive than the Babinski sign in myelopathy with mild neurological deficit. Another study found that the Babinski sign was the most accurate finding to confirm the presence of myelopathy on MRI, with a positive likelihood ratio of 4.0 (Cook et al., 2009). Combinations of tests did not increase the negative likelihood ratios compared with tests used in isolation (Cook et al., 2007). The prevalence of positive long tract signs correlated with severity of myelopathy, and therefore these signs may be of limited utility in the diagnosis of early or mild CSM (Chikuda et al., 2010). A recent systematic review concluded that most clinical tests for CSM demonstrated high specificity and low sensitivity, and supported the conclusions of previous studies that the tests are of limited utility in screening for myelopathy or in early diagnosis (Cook et al., 2011).

2.3.2.2 Role of MRI

In the absence of a definitive diagnostic test, the clinician must look to MRI to provide further clarity. Spinal cord compression can be indicated on MRI by an indentation on the spinal cord parenchyma that changes the contour of the perimeter of the cord, or by signal intensity (SI) changes within the cord itself (Harrop et al., 2010). SI changes manifest as a high SI on T2-weighted images, a low SI on T1-weighted images, or both (Uchida et al., 2005). A scale, presented in Table 2.2, was developed by Mehalic et al. (1990) to classify these MRI abnormalities into levels of severity.

**Table 2.2: Classification of intramedullary signal intensity**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade I</td>
<td>Normal intensity on both T1 and T2 weighted images</td>
</tr>
<tr>
<td>(N/N)</td>
<td></td>
</tr>
<tr>
<td>Grade II</td>
<td>No intramedullary signal intensity abnormality on T1 weighted image, with high intramedullary signal on T2 weighted image</td>
</tr>
<tr>
<td>(N/Hi)</td>
<td></td>
</tr>
<tr>
<td>Grade III</td>
<td>Low intramedullary signal intensity on T1 weighted image and high intramedullary signal intensity on T2 weighted image</td>
</tr>
<tr>
<td>(Lo/Hi)</td>
<td></td>
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</tbody>
</table>

Changes in SI on either T1 or T2 weighted images are usually sufficient to confirm the diagnosis of CSM. Increased intramedullary SI on T2 weighted images represents diffuse neuronal cell loss, gliosis, oedema, demyelination, and axonal and spongy degeneration in the white matter, and is a sign of advanced spinal cord damage (Uchida et al., 2009, Chikuda et al., 2010). SI changes on MRI are associated with poorer neurological outcomes following surgery for CSM (Morio et al., 2001, Uchida et al., 2005). Therefore, although this finding clarifies the diagnosis, it appears that the pathophysiological mechanisms underlying SI change are indicative of more severe, perhaps irreversible
cord damage, and therefore an earlier diagnosis would be desirable. MRI evidence of spinal cord compression by indentation into the cord surface without SI changes is associated with a better outcome (Uchida et al., 2005), however it can be present in the absence of spinal cord pathology. Spinal cord compression without signal change was noted in 16% of cervical spine MRI scans in asymptomatic individuals under the age of 64 years, and in 26% of scans of those aged 65 years and over (Teresi et al., 1987). It is clear therefore that MRI cannot be relied upon in isolation. Findings on imaging must be correlated to the patient's clinical presentation.

2.3.2.3 Role of electrophysiological tests

As outlined above, definitive MRI evidence of cord damage, manifesting as altered signal intensity, and the presence of long-tract signs on examination, tend to be associated with more severe myelopathic presentations. Early diagnosis in the case of mild or inconclusive symptoms and signs can be aided by the use of electrophysiological tests, including somatosensory evoked potentials (SEP) and motor evoked potentials (MEP). Unlike conventional nerve conduction studies and electromyography, these tests evaluate CNS integrity (Dvorak et al., 2003). One prospective study evaluated 199 patients who had MRI evidence of cord compression but no signs or symptoms of myelopathy on entry to the study. During a two-year follow-up period, 22.6% of these patients developed clinical evidence of CSM, and this correlated significantly with abnormal MEP and SEP findings (Bednářík et al., 1998). A further prospective study examined the correlation between transcranial magnetic stimulation and MRI in people with CSM. It found that prolonged central motor conduction time correlated strongly with classifications of disease severity on MRI (Spearman's rho, 0.73–0.75), and showed 98% sensitivity and 98% specificity for spinal cord abnormality (Lo et al., 2004). The evidence for electrophysiological tests is based, however, on a relatively small cohort of studies, and therefore their use cannot be universally recommended in the management of CSM. Future studies may point to a more conclusive role for this technology (Lo, 2008).

2.4 Natural history and management

2.4.1 Progression of CSM

Several authors have acknowledged that the course of CSM varies greatly among individuals. Detailed cohort studies on its natural history are lacking. Much current knowledge comes from clinical observation or case series studies, with poor standardisation of interventions and variable follow-up. A stepwise progression of symptoms was described by authors of early publications, who observed periods of quiescent stability followed by intervals of marked neurological deterioration in their populations (Clarke and Robinson, 1956, Nurick, 1972). Some studies reported an inevitable decline in neurological function, while others showed static symptoms (Matz et
al., 2009). A number of studies pointed to poorer prognosis in patients who were older (Morio et al., 2001, Harrop et al., 2010), had a longer history of symptoms (Morio et al., 2001), and more severe symptoms (Matz et al., 2009). MRI indicators of poorer prognosis included low SI on T1 weighted MRI (Uchida et al., 2009, Avadhani et al., 2010), high SI on T2 weighted MRI (Mehalic et al., 1990, Wada et al., 1999, Chen et al., 2001, Suri et al., 2003, Uchida et al., 2009, Harrop et al., 2010), and increased transverse area of the spinal cord (Wada et al., 1999, Uchida et al., 2009). There was significant variability in the management and follow-up time of the individuals with CSM who participated in these studies. For this reason, the timescale for these prognostic indicators remains unknown.

It is not known whether patients with sub-clinical canal stenosis or very mild symptoms will inevitably progress to clinical myelopathy. Some risk factors for such progression have been identified in a prospective study. Bednarik et al. (2008) followed a cohort of 199 patients who had radiological but no clinical evidence of myelopathy. During a follow-up period ranging from two to 12 years, 22.6% of the cohort developed signs and symptoms of myelopathy. Using a univariate Cox proportional hazards model, progression to clinical myelopathy was predicted from initial symptoms and signs by symptomatic cervical radiculopathy (relative risk (RR) 3.68), abnormal SEP (RR 3.21) and MEP (RR 2.91), and high SI on T2 weighted images (RR 1.6). The 25th percentile time from sub-clinical to clinical myelopathy was 48.4 months. However, some patients were followed up for just 24 months, so the true conversion rate may be higher.

### 2.4.2 Management

There are no definitive guidelines underpinning the medical or surgical management of CSM (Dvorak et al., 2003). Two options are available, namely conservative management and surgical decompression. There is no consistent approach to conservative management in the literature. Protocols involving immobilisation of the cervical spine in a collar (LaRocca, 1988) or the application of cervical traction (Yoshimatsu et al., 2001) have been described. Similarly, a variety of surgical techniques to decompress the spinal cord have emerged, including anterior cervical discectomy, anterior cervical discectomy and fusion, anterior cervical corpectomy and fusion, laminectomy, laminoplasty, and laminectomy and fusion (Mummaneni et al., 2009). These techniques are often broadly divided into two groups, determined by an anterior or posterior surgical approach.

Most reports have found unsatisfactory results with conservative management. There is some evidence that intensive conservative management, including cervical traction for three to four hours daily for periods of at least a month, may be effective in patients with milder symptoms (Yoshimatsu et al., 2001). However, there are no high quality randomised controlled trials (RCTs) comparing surgical and conservative management. A Cochrane review in 2010 found just two trials. Both showed inadequate randomisation
and had small sample sizes. One included patients with mild symptoms only. The reviewers found low quality evidence that patients with mild myelopathy felt subjectively better shortly after surgery, but no evidence of a long-term benefit (Nikolaidis et al., 2010).

In the absence of definitive evidence for or against surgery or conservative management, a number of arguments in favour of surgical intervention have emerged (Jankowitz and Gerszten, 2006). Firstly, degenerative changes within the cervical spine are widely considered to be irreversible and progressive. It is thought that surgical decompression may arrest the progression of further degenerative changes, leading to stabilisation of symptoms and prevention of further deterioration (Jankowitz and Gerszten, 2006, Rao et al., 2006). However, this argument is based on assumption that untreated individuals will inevitably experience progressive neurological deterioration. There is no convincing evidence of this in the literature (Fouyas et al., 2002), although it is generally accepted in clinical practice that neurological function may deteriorate in a stepwise fashion, with periods of stability followed by intervals of rapid deterioration (Nurick, 1972).

Secondly, it is thought that patients with spinal canal stenosis are at a higher risk of SCI after relatively minor trauma (Baron and Young, 2007). A recent study found no evidence of this in patients with asymptomatic cord compression (Bednarik et al., 2011). However, the patients in that study had been advised to avoid activities with a risk of trauma. It is unclear whether the pursuit of normal, unrestricted activities would lead to a greater incidence of traumatic events and therefore of neurological deterioration.

A third factor that mitigates against conservative management is the fact that the existing protocols involve multiple hours of continued traction (Yoshimatsu et al., 2001). This could be associated with significant risks relating to immobility, as well as the impact of such time-consuming and restrictive procedures on quality of life. The lack of evidence leaves the choice of surgical or conservative management as a matter of clinical judgement. In many cases, given the fact that the evidence suggests possible improvement with surgical management and unsatisfactory or unclear outcomes from conservative management, early surgical decompression is often recommended.

The lack of certainty around the management of CSM is further compounded by variable quality of evidence on different surgical options. A number of studies have been criticised for methodological flaws, including retrospective study designs, small sample sizes, tabular interpretation of results without statistical analysis, and poor choice of outcome measures (Jankowitz and Gerszten, 2006). There is a risk that surgery may be performed unnecessarily on some patients whose symptoms might have stabilised, while in others, the fear of complications from surgery, coupled with uncertainty about the benefits of decompression, might lead to delays in surgery until irreversible damage has already occurred (Singh and Crockard, 1999). There is therefore a need for greater
methodological rigour in studies of CSM, including longer follow-up periods and the use of robust, sensitive and reliable outcome measures (Jankowitz and Gerszten, 2006).

2.5 Conclusion and implications

The evidence on the pathophysiology of CSM suggests a primary degenerative process, with some genetic and environmental factors that might precede or accelerate the development of CSM. Signs and symptoms of the disease are somewhat variable. The most common symptoms include gait disturbance, weakness or loss of sensation in the lower limbs, and clumsiness or weakness of the upper limbs, particularly the hands. The diagnosis is suspected in the presence of positive long tract signs and confirmed by MRI evidence of cord compression. Surgical decompression of the spondylotic cervical spine is favoured above conservative management as a treatment for CSM, however previous studies have lacked methodological rigour. The next chapter will explore gait impairment in CSM and its evaluation using three-dimensional gait analysis (3DGA).
Chapter 3: Current Understanding of Gait in Cervical Spondylotic Myelopathy and Implications for Rehabilitation

3.1 Introduction

As outlined in Chapter 2, gait impairment is one of the primary features of CSM. Gait is of interest to physiotherapists, doctors and patients alike. It is the focus of many rehabilitation goals (Field-Fote, 2000) and demands significant amounts of therapy time (Lang et al., 2009). Physiotherapists require a detailed appreciation of the biomechanical and neuromuscular mechanisms underlying gait in order to assess and treat it. Assessment of gait is important to physicians and surgeons as it provides a clinical indication of disease severity. Patients are concerned with the impact of their disease on their quality of life, as their mobility will dictate their ability to participate in their chosen professions or activities. A greater understanding of gait in CSM could therefore inform the interpretation of the course of the disease for patients and clinicians, and provide for improved clinical analysis and treatment strategies in rehabilitation.

Gait analysis is the systematic measurement, description, and assessment of quantities that characterise a person's walking pattern (Gage et al., 1995). Methods of gait analysis can be divided into four categories, 1) visual observation, either in real time or from a video recording, 2) timed walk tests, 3) carpeted recording mats with embedded pressure sensors, such as the GaitRite®, for measurement of temporal-spatial parameters (TSPs), and 4) 3DGA incorporating motion analysis cameras, force plates and sophisticated computer algorithms to determine joint biomechanics, which in some cases also incorporates EMG. Visual observation is the most commonly used method in clinical practice, but it demonstrates poor inter-rater and test-retest reliability and poor criterion validity against the more sophisticated methods (Bilney et al., 2003). A more accurate and reliable method is therefore required for research purposes.

3DGA is widely considered to be the “gold standard” technology for gait analysis (Kirtley, 2006). It has been recommended in incomplete SCI, where three-dimensional measurements of dynamic joint ROM and calculation of joint forces, moments and powers can significantly improve the understanding of a gait deficit (Patrick, 2003). It has been used to evaluate many neurological disorders, such as stroke (Lamontagne et al., 2007), traumatic SCI (Heller et al., 1996, Gil-Agudo et al., 2009), Parkinson’s disease (Morris et al., 2005, Nieuwboer et al., 2007), TBI (Williams et al., 2009b) and CP (Lebiedowska et al., 2004, Gough and Shortland, 2008). In CP, 3DGA is used extensively in the evaluation of complex gait abnormalities and in assessing the effect of surgical interventions to alleviate deformities in the growing child (DeLuca et al., 1997). In seeking
to build a comprehensive picture of gait impairment in CSM, 3DGA is therefore the method of choice.

The aims of this chapter are 1) to review current understanding of gait in CSM, with particular reference to studies using 3DGA, 2) to consider the possible neuromuscular and biomechanical causes of gait impairment, 3) to examine the implications of this knowledge for rehabilitation, and 4) to identify areas in need of further research.

### 3.2 Current understanding of gait in CSM

#### 3.2.1 Literature search

A literature search was conducted using the databases Medline, Cumulative Index of Nursing and Allied Health Literature (CINAHL), and the Physiotherapy Evidence Database (PEDro). The following Medical Subject Heading (MeSH) terms were used in the search strategy: cervical myelopathy, cervical spondylotic myelopathy, compressive myelopathy, spinal cord compression, cervical spondylosis, spondylosis, gait, gait analysis. Studies were included in the literature review if quantitative evaluation of gait in people with CSM had been conducted using either 3DGA, carpeted mats such as the GaitRite®, or timed walk tests.

#### 3.2.2 Identification of studies

Six studies that used 3DGA in the evaluation of gait in CSM were found (Kuhtz-Buschbeck et al., 1999, Maezawa et al., 2001, Suzuki et al., 2002, Moorthy et al., 2005, Kim et al., 2010, Lee et al., 2011). Two studies evaluated gait speed using a validated timed walking test (Singh and Crockard, 1999, Singh et al., 2009). A case report that evaluated changes in gait speed, strength and spasticity in a patient with CSM was also identified (Engsberg et al., 2003). Table 3.1 shows the main methodological features of the studies using 3DGA.
### Table 3.1: Methodological features of studies using 3DGA to evaluate gait in CSM

<table>
<thead>
<tr>
<th>Authors</th>
<th>Sample Size</th>
<th>3DGA System</th>
<th>Healthy Controls</th>
<th>Follow-Up</th>
<th>Parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kuhtz-Buschbeck et al. (1999)</td>
<td>12</td>
<td>Qualisys (Savedalen, Sweden), over treadmill</td>
<td>N = 14, age matched</td>
<td>12 days and 2 months post surgery</td>
<td>TSPs, kinematics (key points)</td>
</tr>
<tr>
<td>Maezawa et al. (2001)</td>
<td>24</td>
<td>Anima (Tokyo, Japan), over ground</td>
<td>N = 72, matched for age and body weight</td>
<td>Variable: mean 32.4 months post surgery, range not specified</td>
<td>TSPs, kinematics (key points), ground reaction forces (peaks)</td>
</tr>
<tr>
<td>Suzuki et al. (2002)</td>
<td>15</td>
<td>Vicon 370 (Oxford, UK)</td>
<td>N = 12, not matched</td>
<td>None</td>
<td>TSPs, kinematics (visual interpretation of curves)</td>
</tr>
<tr>
<td>Moorthy et al. (2005)</td>
<td>6</td>
<td>Not specified</td>
<td>Unpublished normative data, no age, gender or sample size specified</td>
<td>12 months post surgery for 5 patients, 19 months for the 6th patient</td>
<td>TSPs, kinematics (total ROM), ground reaction forces (peaks), EMG (visual interpretation)</td>
</tr>
<tr>
<td>Kim et al. (2010)</td>
<td>36</td>
<td>Motion Analysis (Santa Rosa, California, USA)</td>
<td>None</td>
<td>None</td>
<td>TSPs, kinematics (peak ROM)</td>
</tr>
<tr>
<td>Lee et al. (2011)</td>
<td>38</td>
<td>Motion Analysis (Santa Rosa, California, USA)</td>
<td>N = 36, similar age and gender</td>
<td>None</td>
<td>TSPs, kinematics (ROM at key points)</td>
</tr>
</tbody>
</table>

3DGA = three dimensional gait analysis, N = number of participants, TSPs = temporal-spatial parameters, ROM = range of motion, EMG = electromyography
3.2.3 Changes in temporal-spatial parameters

TSPs, the time and distance measurements of gait, have been described as the “vital signs” of gait analysis (Kirtley, 2006). Studies in CSM have consistently shown reduced gait speed compared to healthy controls (HCs) (Kuhtz-Buschbeck et al., 1999, Maezawa et al., 2001, Suzuki et al., 2002, Lee et al., 2011). One study did not find a significant difference in gait speed between people with CSM and HCs (Moorthy et al., 2005), however the sample size of six participants with CSM may have had insufficient statistical power to detect change.

Gait speed is a product of stride length and cadence (Kirtley, 2006). Three studies found evidence of both reduced cadence and reduced stride length in CSM (Maezawa et al., 2001, Suzuki et al., 2002, Lee et al., 2011). However, cadence did not correlate with scores on the Nurick scale or Myelopathy Disability Index, whereas gait speed showed a moderate correlation (Singh and Crockard, 1999). Achievement of adequate stride and step length depends on the duration of the single support and double support phases in stance, which are in turn influenced by a number of factors such as stability, strength and proprioception (Whittle, 2002). One study reported a significant reduction in the duration of single support in moderate CSM (27.2% gait cycle (GC) duration) and severe CSM (22.5% GC duration) compared to HCs (33% GC duration) (Maezawa et al., 2001). Another study found an increase in double support duration in CSM patients (12.7–13.1% GC duration) compared to HCs (10.3–10.4% GC duration) (Lee et al., 2011). These findings could indicate either 1) impaired stability of the ipsilateral limb in single-leg stance or 2) ineffective momentum generation into swing by the contralateral limb, or both. To date, kinetic and EMG analyses have not evaluated these potential contributing factors.

Dynamic base of support, or step width, has also been measured in gait in CSM. One study found significantly greater step width compared to HCs under three conditions, 1) normal walking on a 13 metre (m) walkway (step width 7.4 centimetres (cm) in HCs compared to 9.9 cm in CSM), 2) blindfolded walking on the walkway (HC 8.9 cm, CSM 13.7 cm), and 3) treadmill walking (HC 10.9 cm, CSM 15.2 cm) (Kuhtz-Buschbeck et al., 1999). The finding of increased step width in CSM was supported by a later study, who found that more severely-affected patients with CSM employed significantly greater step width during overground walking compared to HCs (Maezawa et al., 2001). A further study found no difference in step width between CSM and HC groups (Lee et al., 2011). Step width may be an indicator of dynamic balance during gait (Whittle, 2002), however its significance as a stand-alone measure is unclear. Further investigation of this parameter is warranted.
The characteristics of the comparison HC group are poorly stated in most studies, and this poses a limitation on the interpretation of the results. One study demonstrated adequate gender and age matching, and used similar sample sizes in both groups with 38 in the CSM group and 36 HCs (Lee et al., 2011). Two studies specified that HCs were matched to age and anthropometric characteristics, but did not specify gender matching (Kuhtz-Buschbeck et al., 1999, Maezawa et al., 2001). In one study, different sample sizes were used (72 HCs compared to 24 CSM participants) (Maezawa et al., 2001). This may have reduced the variance of the HC group. Another study did not state the size, gender, age or other characteristics of their HCs, but stated that it comprised “previously unpublished normative data” (Moorthy et al., 2005). In a further study, the HC group was significantly younger (mean age 39 years) than the CSM group (mean age of 55 years) (Suzuki et al., 2002). Age and gender have been found to influence gait parameters, and therefore matching of these factors would be necessary to avoid any confounding effects in the interpretation of findings (Roislien et al., 2009).

3.2.4 Kinematic analysis

A number of studies have examined kinematics in CSM and found deviations in ROM compared to HCs. Each study examined different kinematic key points, leading to difficulty in amassing evidence for a particular abnormality. However, certain trends have emerged. Altered kinematics of the knee in stance have been noted, such as reduced flexion during loading (Kuhtz-Buschbeck et al., 1999, Suzuki et al., 2002) and hyperextension in mid stance (Maezawa et al., 2001). A case report described a patient with CSM whose initial presentation was of a hyperextension disorder at the knee (Moorhead, 1993). Knee flexion in stance is one of Saunders’ determinants of gait (Saunders et al., 1953), as it adjusts the effective length of the leg to keep the height of the hip constant and allow for efficient forward progression of the body mass (Whittle 2003). Therefore, any reduction in knee flexion at loading response could have a significant impact on gait efficiency. Knee flexion in stance also allows for acceptance of body weight through the weight-bearing limb and ensures a smooth transition through mid stance (Perry, 1992). A reduction in the eccentric yield on loading could signify an inability to allow the quadriceps to undergo controlled lengthening, and could be a feature of spasticity as well as of weakness (Kerrigan et al., 2001).

Reduced knee flexion in swing has been found in two studies comparing CSM to HCs. The first study found a reduction of approximately 5° (63.5–63.8° in HCs, 58.2–58.7° in CSM) (Lee et al., 2011), and the second, 10° (56.5° in HCs, 45.2–46.5° in CSM) (Maezawa et al., 2001). Other studies did not evaluate this parameter. Knee flexion in swing could be impaired by a reduced yield in the lengthening quadriceps due to spasticity (Sutherland and Davids, 1993) or by lack of momentum generation in pre swing (Gage, 1991). It could also be an erroneous finding due to differences in gait speed.
between the groups, as knee flexion in swing shows a strong correlation with gait speed (Lelas et al., 2003).

Changes in kinematics at the ankle at toe-off have been identified in two studies. One study found a reduction in peak ankle plantarfexion in CSM (5–10.5°) compared to HCs (15°) (Lee et al., 2011). The other identified a reduction in peak ankle plantarfexion in patients with moderate (7.5°) and severe (8°) CSM compared to HCs (12.8°) (Maezawa et al., 2001). This may indicate a lack of power generation at the ankle during the pre swing phase of gait, however analysis of kinetics would be necessary for a more precise interpretation.

At the hip, no differences in peak flexion or extension, or in total ROM, have been found in CSM compared to HCs (Kuhtz-Buschbeck et al., 1999, Maezawa et al., 2001, Lee et al., 2011). Kinematics at the pelvis were examined in one study, which found a significant reduction in pelvic obliquity in less chronic CSM compared to controls, but also a significant increase in the same parameter in those with more chronic disease, namely symptoms of at least one year’s duration (Suzuki et al., 2002). The magnitude of this change was 1°, and was probably of little clinical significance.

The information from these studies helped to identify features of the gait pattern associated with CSM. However, most studies evaluated only a limited number of parameters. A greater insight could be attained through the systematic analysis of a number of discrete parameters at specific points in the GC across three planes of motion (Hanlon and Anderson, 2006). In addition, the timing of joint motion should be examined, as the timing of peak values must be appropriate to the tasks of that phase in the GC (Perry, 1992). Therefore, although significant contributions have been gleaned from the studies discussed above, a complete kinematic profile of CSM patients, across all planes of motion and multiple key points of interest, has not been described.

A further limitation of previous studies is the presence of potential confounding factors that may affect the interpretation of the findings. One study (Kuhtz-Buschbeck et al., 1999) recorded kinematics during treadmill walking at a standard velocity of 2 kilometres per hour (km/h). This speed was significantly slower than the mean self-selected speed of the CSM group, 4.28 km/h. Changes in joint ROM may have been imposed by forcing a shorter stride length to accommodate the slow speed. The slow speed may also have masked the possible influence of spasticity on joint kinematics, which due to its velocity-dependent nature might be more apparent during faster walking. Furthermore, although treadmill walking has been shown to be equivalent to overground walking in healthy people (Riley et al., 2007), it is not known whether this is the case for people with neurological deficits. The kinematic findings in this study must therefore be considered in the context of these potential limitations.
In order to achieve an accurate evaluation of gait, participants should walk with a pattern that is close to their natural preference (Gormley et al., 1999). One study using the Anima system required participants to fold their arms while walking to avoid interference with the reflective markers during motion capture (Maezawa et al., 2001). This may have had an erroneous influence on the movement of the trunk and pelvis during the GC, and may have impacted on lower limb kinematics.

An important feature of the study of gait is the inter-dependence of many gait variables. Gait speed is known to influence lower limb kinematics, with faster speeds associated with larger ROM, though the associations vary depending on the joint itself and the timing within the GC (Hanlon and Anderson, 2006). In particular peak knee flexion in stance and swing show strong positive correlations with gait speed (Lelas et al., 2003). Apart from Kuhtz-Buschbeck et al. (1999), who assessed gait over a constant speed of 2 km/h, all studies compared the CSM and HC groups at different speeds (Maezawa et al., 2001, Suzuki et al., 2002, Moorthy et al., 2005). While it is important that HCs should walk at self-selected speed in order to evaluate natural walking performance, there is also a need to match the speeds of CSM and HC participants to avoid confounding effects on kinematics (Jordan et al., 2007, Roislien et al., 2009, Williams et al., 2009b). Otherwise, it cannot be known whether reduced joint excursion is caused by the slower gait speed, or co-exists with it as a pathological feature in its own right.

### 3.2.5 Kinetic analysis

The study of kinetics is of particular importance in determining the biomechanical factors that produce movement. The forces produced by the body cannot be measured directly, but can be inferred from the magnitude and position of the ground reaction vector with respect to axes of the lower limb joints during gait. The ground reaction force (GRF) can be recorded during stance by a force plate, and quantitative values for joint moments and powers can then be determined using inverse dynamics (Kirtley, 2006).

There is a lack of reported data on the kinetics of gait in CSM. Two studies have evaluated changes in the components of the GRF. One study found significantly lower anterior-posterior (AP) GRF in the CSM group compared to HCs, but not in vertical or medial-lateral GRF (Moorthy et al., 2005). The second study found significant reductions in peak vertical and AP GRF in patients with moderate and severe CSM compared to HCs (Maezawa et al., 2001). Again, this finding may be confounded by the slower gait speed of the CSM group. Kinetic parameters tend to show a linear or quadratic relationship with speed (Lelas et al., 2003). Without controlling for speed, it is difficult to interpret the changes in GRF in these studies.

Analysis of joint moments and powers would further enhance the current understanding of gait in CSM, as it would allow for the interpretation of the forces that produce the
observed temporal-spatial and kinematic output. No studies have examined moments and powers in gait in CSM to date.

3.2.6 Electromyography analysis

Analysis of the EMG signals generated during gait allows for direct interpretation of the biological signals responsible for muscle activation (Frigo and Crenna, 2009). EMG can evaluate the contributions of individual muscles to a movement pattern. It is therefore an important adjunct to kinetic analysis, which calculates only the net effect of a group of muscles acting about a joint and assumes no co-contraction (Kirtley, 2006). EMG signals from eight lower limb muscles during gait were reported in a cohort of six people with CSM (Moorthy et al., 2005). The signals were interpreted visually. All muscles appeared to show prolonged duration of activation and delayed onset in relaxation, suggesting a problem with co-activation or spasticity (Moorthy et al., 2005). A more complete analysis of EMG, including quantitative measurement of timing and amplitude, is lacking in CSM at present. Such parameters are critical in the interpretation of neurological gait disorders (Frigo and Crenna, 2009). There is a need for future studies to employ more sophisticated analysis methods of EMG signals to determine the effect of muscle activation on the observed gait patterns in CSM.

3.2.7 Effect of disease severity on gait parameters

A number of studies have performed sub-group analysis using various indicators of disease severity to determine its effect on gait in CSM. In one study, a reduction in gait speed compared to HCs was found in people with moderate and severe CSM indicated by lower Japanese Orthopaedic Association (JOA) scores, but not in those with mild CSM (Maezawa et al., 2001). There were no significant differences between the groups themselves, though there was a non-significant trend towards progressively slower gait speed with worsening severity.

Differences in kinematics were also found in this study. This data provided some evidence of the natural history of gait impairment in CSM. Reduced knee flexion was common to all groups. Reduced ankle plantarflexion in stance was present in the moderate and severe groups, whereas the severe group exhibited hyperextension of the knee in stance. Loss of knee flexion in swing was significant in the mildly affected group who had an otherwise normal gait, supporting a similar finding in a previous case study (Moorhead, 1993). This suggests that loss of knee flexion may be one of the first signs of a developing myelopathic gait, and that hyperextension of the knee in stance may indicate more advanced disease.
Another factor that may affect the performance of gait in CSM is the chronicity of the disease. One study on gait performance in CSM dichotomised its participants into two sub-groups, one group with a history of symptoms for more than one year and the other, less than one year. The more chronic group had significantly slower gait speed, shorter stride length and shorter single stance phase duration than the less chronic group, suggesting that gait deteriorates as the disease becomes more prolonged (Suzuki et al., 2002). Chronicity of CSM has been found in previous studies to be predictive of a less favourable outcome (Morio et al., 2001, Suri et al., 2003), and is therefore an important factor to consider in gait analysis.

As outlined in Chapter 2, SI changes on MRI are a known predictor of more severe disease and poorer outcome in CSM. Kim et al. (2010) used 3DGA to evaluate gait in 36 pre-operative patients divided into two groups, those with and without high SI on T2-weighted MRI. Patients with high SI had significantly slower gait speed, longer step time, and increased double support time compared to those without high SI. This study also supported the hypothesis that gait performance deteriorates with worsening radiological outcomes.

3.2.8 Changes in gait parameters following surgery

Changes in TSPs following surgery have been evaluated in five case series, two of which used timed walk tests (Singh and Crockard, 1999, Singh et al., 2009), and three used 3DGA (Kuhtz-Buschbeck et al., 1999, Maezawa et al., 2001, Moorthy et al., 2005). The studies showed a general trend of improvement in gait speed. Singh and Crockard (1999) found that gait speed increased from a mean of 0.35 metres per second (m/s) pre-operatively to 0.47 m/s at two months following surgery in 41 patients. This change had a moderate correlation with the Nurick score and Myelopathy Disability Index, and led to the validation of a 30m timed walk test as an outcome measure for CSM (Singh and Crockard, 1999). Kuhtz-Buschbeck et al. (1999) also found a significant improvement in gait speed at two months post surgery, from 1.18 m/s to 1.33 m/s. An improvement of similar magnitude, from 0.63 m/s to 0.78 m/s, was found by Maezawa et al. (2001), though the follow-up time was not standardised across patients and varied from 12 to 44 months. A longer-term follow-up using the timed 30m walk test as the primary outcome measure showed that an increase in gait speed six months after surgery was maintained at one, two and three-year follow up (Singh et al., 2009). None of the studies stated whether the patients were receiving rehabilitation intervention at the time, a factor that may have influenced the recovery of gait. One study found no significant difference in gait speed after surgery, however with a sample size of six, it may have been inadequately powered (Moorthy et al., 2005).
Post-operative changes in kinematics, kinetics and EMG are somewhat less well defined. A non-significant trend of increased knee flexion in stance at two months post surgery was reported in one study (Kuhtz-Buschbeck et al., 1999). Another noted a statistically significant increase of 5.6° in the total sagittal plane range at the knee at one year follow up, but no changes at the hip or ankle (Moorthy et al., 2005). A third study found statistically significant post-operative increases of 8° in peak hip flexion, 4° in peak knee flexion in swing, 4° in knee extension, and 5° in ankle dorsiflexion in stance (Maezawa et al., 2001). However, the follow-up time ranged from 12 to 44 months, and with such a variable time frame, there may have been other confounding factors affecting gait in this cohort. A standardised follow-up interval would be preferable to clarify changes in gait performance over time following surgery. A further point to consider is whether the magnitude of change in the improved parameters was clinically significant and exceeded measurement error. There are no studies on the repeatability of 3DGA in CSM, and therefore the influence of measurement error on these findings is unknown.

Changes in GRF following surgery have been evaluated in two studies. One study found significant increases in peak AP GRF, but not the medial-lateral or vertical components in six patients at 12 to 19 months post surgery (Moorthy et al., 2005). A second study found that peak vertical GRF decreased significantly at mid stance and increased at terminal stance, but that the AP component was unchanged (Maezawa et al., 2001). These changes in vertical GRF could be explained by the post-operative increase in gait speed in Maezawa’s study (Perry, 1992). Without analysis of joint moments and powers, the contribution of these changes in GRF to the biomechanics of the lower limbs during gait remains unclear.

Visual interpretation of EMG signals during gait in a study of six patients noted a general reduction in the overall activation times of eight lower limb muscles following surgery at 12- to 19-month follow up (Moorthy et al., 2005). However, there were no quantitative data to support this finding. There is therefore no conclusive evidence for changes in EMG parameters, either timing or amplitude, following surgery.

3.2.9 Summary of current understanding of gait in CSM

There is strong evidence for a reduction in gait speed in CSM, and this is associated with increased double support duration and decreased cadence. Kinematic data should be interpreted with caution due to inadequate matching of HC comparison groups, particularly in relation to gait speed. Evidence suggests a reduction in knee flexion during swing in the early stages of the disease, followed by reduced ankle plantarflexion at terminal stance and reduced knee flexion during loading response in more severely affected people. Comparison with HCs of similar age and gender, walking at matched speed, would be necessary to confirm that these findings were not erroneous. Kinetic
analysis was confined to GRF in two studies, and was potentially confounded by differences in speed. These findings are therefore inconclusive and require further study. EMG patterns of the lower limb muscles have been reported subjectively in a small sample, and suggest an impairment of the timing and duration of muscle activation. There is evidence of a correlation between the severity of gait impairment and MRI findings of increased SI. Repeated measures studies using pre- and post-operative gait analysis data have shown some improvements in TSPs and kinematics, however the interpretation of this data in the context of neurological recovery is limited by the variable follow-up time and narrow range of parameters used. Finally, there are no data on the association of these changes with quality of life and function, so their impacts on functional limitation and participation restriction remain unknown.

3.3 Interpreting the causes of gait impairment in CSM

3.3.1 Introduction

The aim of gait analysis is to provide a comprehensive understanding of the actions of the locomotor system during gait, and to identify key features that may relate to the underlying pathophysiology of a disorder. The clinical characteristics of an upper motor neurone lesion (UMNL) include spasticity, paresis, and impaired sensory function and proprioception, all of which influence locomotor performance in people with injuries to the CNS (Dietz, 2002). The influences of paresis, tone and sensory deficits on the gait pattern of CSM will now be discussed, with the objective of determining the key features of the UMNL that lead to changes in gait.

3.3.2 Evidence for paresis

To date, kinetic and EMG analyses have not been sufficient to determine the underlying contributions of muscle action to the biomechanical output. There is a suggestion that a reduction in flexion of the knee in swing occurs prior to any changes in gait speed in the milder stages of the disease, and also a tendency towards knee hyperextension in stance as the disease progresses (Maezawa et al., 2001). This could represent a reduction in eccentric control in the quadriceps, as this finding has been reported in other conditions where eccentric strength is impaired, such as anterior cruciate ligament repairs (Lewek et al., 2002). There is insufficient evidence from the studies of kinematics in CSM to evaluate whether eccentric control may be a problem at other phases of the GC, such as at terminal stance, when controlled lengthening of the hamstrings allows for a gradual extension of the knee in preparation for initial contact (Perry, 1992).

A lack of eccentric control during gait would be consistent with muscle weakness. Subjective weakness or objective findings of paresis on examination are part of the
clinical picture of CSM as discussed in Chapter 2, Section 2.3. One study used
dynamometry to evaluate peak torque production capabilities at the knee in 39 patients
with CSM, who were divided into two comparison groups based on those who could walk
without an aid (A group, 22 patients) and those who required an aid (AA group, 17
patients). The AA group showed a significantly greater reduction in isokinetic strength at
faster velocities in the knee flexors, but not in the extensors, compared to the A group at
the same faster velocities. There were no significant differences in isokinetic strength
between the groups at slower velocities. Isometric strength in the flexors was also
significantly lower in the AA group, while extensors were similar (Sairyo et al., 2001). The
authors suggested that the reduced isokinetic strength of the knee flexors could be
explained by spasticity of the stronger knee extensors. Provocation of the stretch reflex in
the extensors at faster velocities may have overpowered the physiologically weaker
flexors (Sairyo et al., 2001). The lack of a HC comparison group is a limitation of this
study, as the reduction in isometric and isokinetic strength at the knee in the two groups
relative to a healthy population remains unknown. A more recent study evaluated
strength at the knee extensors and flexors using dynamometry in 26 people with CSM
and age- and gender-matched HCs, and found significantly lower peak torques in the
CSM group in both muscle groups (Takayama et al., 2005a). There is therefore some
evidence for paresis as a source of impairment to producing movement in CSM.

The relationship between lower limb strength and the three-dimensional characteristics of
gait has yet to be investigated. Lower limb strength has shown a correlation with
functional walking ability in incomplete SCI (Kim et al., 2004, Wirz et al., 2006, Scivoletto
et al., 2008), however a close relationship exists only in moderately impaired patients
(Dietz, 2002). The correlation between strength and gait performance in CSM has not
been studied. A retrospective study of outcome following anterior cervical decompression
in 75 CSM patients found that, although between 79.1% and 88.1% of patients showed
an improvement in lower limb muscle strength of at least one grade on the Oxford scale
on clinical examination, only 46.7% experienced improvement in lower limb function
assessed by the Cooper lower limb subscale (Chiles et al., 1999). The follow-up time in
this study for these particular outcome measures varied from one to 75 months, so there
may be some confounding factors related to variable follow-up. Despite this limitation,
there is some evidence from Chiles’ study that functional mobility may be slower to
improve than strength, and that other factors may be affecting recovery of function.

3.3.3 Evidence for changes in muscle tone

In a previous study, the finding of a greater reduction in isokinetic torque production as
movement velocity increased was interpreted as evidence of a spastic movement
disorder in CSM (Sairyo et al., 2001). Spasticity is part of the clinical picture of CSM, and
is associated with other positive features of the UMNL, such as hyperreflexia and other
long tract signs. It has been defined by one expert group as “a velocity-dependent increase in the hyperexcitability of the stretch reflex as one component of the upper motor neurone syndrome” (Lance, 1980) and by another expert group as “disordered sensorimotor control resulting from an upper motor neuron lesion presenting as intermittent or sustained involuntary activation of muscles” (Pandyan et al., 2005). There is a clear difference in the scope of these definitions, illustrating the difficulty in defining the clinical entity of spasticity. Spasticity is difficult to measure objectively, particularly during voluntary movement. Scales such as the Ashworth and Modified Ashworth Scales and the Tardieu scale have been criticised in recent years for their lack of validity and reliability (Haugh et al., 2006, Fleuren et al., 2010). Research in physiology suggests that spasticity measured during passive movement and that produced during active movement may be different clinical entities, in that the same neurophysiological pathways do not underlie both phenomena (Ada et al., 1998, Dietz, 2003). Further studies are needed to characterise the influences of spasticity on locomotion in people with CSM.

Despite the lack of conclusive evidence from gait analysis studies on the presence of spasticity during gait, there is strong anecdotal evidence of a spastic gait pattern (Montgomery and Brower, 1992, Salvi et al., 2006, Baron and Young, 2007). There is some evidence, albeit inconsistent and confounded by a lack of matching for gait speed, of an overall reduction in motion amplitude, particularly at the knee (Kuhtz-Buschbeck et al., 1999, Maezawa et al., 2001, Suzuki et al., 2002). An abnormal response to stretch in the quadriceps group during lengthening could reduce the amplitude of knee flexion during swing (Kerrigan and Sheffler, 1995). There is also limited EMG evidence of prolonged duration of activation of the lower limb muscles during gait in CSM (Moorthy et al., 2005). This could reflect spasticity, but it could also be due to the effects of weakness resulting in an increased need for joint stability from co-contraction (Brunner and Romkes, 2008). Other muscles prone to the effects of a velocity-dependent increase in the stretch reflex include the hamstrings during terminal swing and the gastrocnemius during mid stance (Crenna, 1998, Crenna, 1999, Lamontagne et al., 2001). Studies in CP and stroke have found evidence of spasticity of these muscles during lengthening, confirming the existence of hyperexcitability in response to stretch during gait (Crenna, 1999, Lamontagne et al., 2001). However, no such evidence exists in CSM or other form of SCI.

3.3.4 Evidence for impaired proprioception and balance deficits

Proprioception is essential for the maintenance of balance during the single support phase of gait. Lack of proprioception can result in prolonged double support, reduced single support, and a wider dynamic base of support (Whittle, 2002). Studies of TSPs in CSM have noted an increase in step width and a decrease in single support duration (Kuhtz-Buschbeck et al., 1999, Maezawa et al., 2001). This may suggest an impairment
of balance during the GC, which could be proprioceptive in origin. A study of postural control in participants with cervical spondylosis, nine with and eight without myelopathy, and a comparison group of 17 HCs, found increased postural sway on a force platform in both spondylotic groups compared to controls. Although all participants with spondylosis showed an increase in sway, the nine participants with myelopathy were more severely affected, and also showed longer latencies to onset in the soleus and tibialis anterior with postural perturbation (Nardone et al., 2008). The authors concluded that abnormal transmission of proprioceptive input through the spinal cord could account for the unsteady gait patterns observed in CSM.

Proprioception has been evaluated by two further studies, in which joint position sense at the knee was measured by the absolute angular error in reproducing a predetermined position with an electrogoniometer (Takayama et al., 2005b, Okuda et al., 2006). Fifty-four participants with CSM in the first study produced consistently higher errors than their age-matched healthy counterparts (Takayama et al., 2005b). In the second study, 21 participants with CSM were classified according to their lower limb JOA score of CSM severity. It was found that those with the most severe limitation of lower limb function, who were unable to mobilise independently, had significantly higher errors in reproducing a predetermined knee position than those patients with normal or mildly unsteady gait (Okuda et al., 2006). In a post-operative follow-up, angular errors were found to have significantly improved at two-week follow-up in 26 patients who underwent posterior surgical decompression (Takayama et al., 2005a). These studies support the clinical finding of reduced proprioception in CSM, and suggest that there is potential for recovery of this sensory modality following decompressive surgery. Proprioception is, however, intrinsically linked to other impairments, particularly spasticity, because the disinhibition of short-latency reflexes and loss of functionally important long-latency reflexes, as occurs in spasticity, causes a profound alteration of proprioception (Dietz, 2002). The impact of improved sensory and proprioceptive function following surgery on locomotor performance has yet to be established.

### 3.3.5 Summary

Studies using dynamometry and isokinetics have confirmed the presence of paresis in the CSM population compared to HCs. The relationship with gait has not been studied. Follow-up studies using manual muscle testing have found that improvements in functional mobility do not necessarily lead to improvements in strength. Evidence for spasticity is limited, as no study has specifically evaluated electromyographic responses to stretch, however the existence of spasticity as a clinical entity in CSM is generally accepted anecdotally. Impairment of proprioception in a condition affecting the long spinal cord tracts would be expected, and there is evidence of impaired joint position sense and increased postural sway. Again, the clinical significance of these findings in relation to
gait and functional mobility is unclear. Detailed evaluation of the kinematics, kinetics and EMG features of gait in CSM could improve the understanding of the relative contributions of spasticity, paresis and proprioception to locomotion in this cohort.

3.4 Rehabilitation of gait impairment in CSM

Patients with gait impairment due to CSM will require a rehabilitation programme to improve their gait, functional mobility, and quality of life. The evidence base for rehabilitation interventions in neurological disorders is growing, however there are no guidelines for rehabilitation in CSM, either immediately post surgery or over the following months. Studies in incomplete SCI suggest significant potential for neuroplasticity and therefore recovery of function, particularly in individuals where the injury is classified by the American Spinal Injury Association (ASIA) scale level D, where some sensory and motor function are preserved with at least a grade 3 in half of the key muscles (Field-Fote, 2000). There are no statistics on the percentage of CSM patients who present with the various ASIA grades. Clinical experience suggests that most patients are ambulant on presentation, and are therefore representative of a motor incomplete population, most likely ASIA C or D. However, in comparison to a population with traumatic incomplete SCI, CSM patients have the added confounding factor of a potentially progressive degenerative process, so it may be the case that much of their recovery would depend on the success of surgery in stabilising the cervical spine and preventing further impingement of the spinal cord by an ongoing spondylotic process. They also tend to be significantly older than patients with traumatic injury (McKinley et al., 1998), and may have a higher incidence of co-morbidities that impact on rehabilitation potential. Therefore, rehabilitation findings from a general population of incomplete SCI cannot be considered representative of CSM, and this group warrants separate investigation.

A second literature search was conducted to identify papers on the rehabilitation in CSM, using the following MeSH search terms: cervical myelopathy, cervical spondylotic myelopathy, compressive myelopathy, spinal cord compression, cervical spondylosis, spondylosis, rehabilitation, physiotherapy, physical therapy, exercise, exercise therapy. No RCTs of rehabilitation interventions were identified. Four relevant studies were found, two case series on outcomes for CSM patients in a rehabilitation centre (Yap et al., 1993, McKinley et al., 1998), one case series evaluating the effects of robotic treadmill therapy on the autonomic system (Magagnin et al., 2010), and a single case report on the use of electrical stimulation to the hip and knee (Pastor, 2010).

The study by McKinley et al. (1998) reported the findings of 46 individuals who were admitted to a rehabilitation unit with neurological injury caused by spinal stenosis. Nineteen of these patients had CSM and the remaining 27 had cauda equina syndrome secondary to lumbar stenosis. The patients were compared to a group with traumatic SCI with the same ASIA classification, but not matched on age and gender. The CSM group
were on average 28 years older than the traumatic SCI group. Both groups made significant progress from admission to discharge in the self-care, sphincter control, mobility / locomotion and communication / psychosocial sub-categories of the Functional Independence Measure (FIM). There were no significant differences in the rate of improvement between groups, though the traumatic group had a trend towards faster and greater improvement. It was concluded that people with CSM have potential to benefit from inpatient rehabilitation, despite their older age (McKinley et al., 1998).

A similar retrospective study by Yap et al. (1993) reported the outcomes of 18 patients with CSM who were admitted to a rehabilitation centre a mean of 38 days following decompressive cervical surgery. The average length of stay in the rehabilitation centre was 60.4 days. Outcomes were assessed using the Profile System of Disability Classification, an ordinal scale adapted specifically for the study with sub-categories in upper and lower limb strength, bladder and bowel function, independence in self-care, and functional mobility. At the time of admission, the functional status of 50% of the patients had improved following surgery, 27.8% were unchanged and 22.2% had deteriorated. On discharge from the rehabilitation unit, there were significant improvements in upper limb and lower limb strength, mobility and activities of daily living (ADL) status compared to scores on admission (Yap et al., 1993). As with the McKinley study, no information was given on the frequency, intensity or nature of the rehabilitation interventions. The study is also somewhat limited by the use of a little-used ordinal scale of functional ability. Furthermore, the lack of a control group in both studies prevents the improvement being attributed to the intervention. However, it is significant that the patients experienced further improvement following inpatient rehabilitation, even though 50% had already improved after surgery prior to entering the rehabilitation unit. This suggests that rehabilitation may be needed to maximise the potential for recovery following surgery. Further studies are necessary to evaluate this hypothesis.

Few studies have evaluated the effect of specific physiotherapy or rehabilitation interventions. One study evaluated the efficacy of body-weight supported treadmill training (BWSTT) with a robot-driven gait orthosis, the Lokomat®, in six patients with CSM (Magagnin et al., 2010). The patients completed daily 30-minute BWSTT sessions on the Lokomat® for six weeks. The patients were at least 12 months post injury (range, 12–180 months), though it is not clear whether this refers to the time following onset of symptoms or time since surgery. The patients improved significantly in the mobility score of the FIM and on the Barthel index and had a non-significant improvement in JOA score of CSM severity. Scores on the Motricity Index did not change (Magagnin et al., 2010). BWSTT is of particular interest in incomplete SCI due to its potential to influence the spinal reflex circuitry (Field-Fote, 2000). This study was limited by the small sample size and the lack of a control group, however it was interesting that improvements occurred despite the chronicity of CSM in this group. Further studies are necessary with larger
sample sizes, using a control group and comparing BWSTT to more conventional therapies, to ascertain the true value of BWSTT in CSM.

Finally, a case study described positive benefits to one patient with CSM following the use of electrical stimulation to the hip and knee extensors in addition to active ROM exercises, functional mobility training and gait training (Pastor, 2010). The patient participated in therapy five days a week for six weeks, and had electrical stimulation on alternate days. Improvement was demonstrated on manual muscle testing, the FIM and the Walking Index for Spinal Cord Injury (WISCI). However, longer-term studies with larger sample sizes and control groups are necessary to determine if these improvements can be attributed to the interventions described in this case study, and to extrapolate the findings to a wider population of people with CSM.

3.5 Summary

This chapter has reviewed the evidence summarised current understanding of the presentation and nature of gait impairment in people with CSM. It is clear that there are a number of gaps in the literature. In particular, there is a need for evaluation of a more comprehensive range of kinematic key points, exploration of kinetic and EMG analysis, and comparison to healthy controls matched to age, gender and gait speed. There are significant benefits to be gleaned from the use of 3DGA technology, incorporating EMG, in this patient population. These potential benefits will inform the development of the methodology for this study, and underpin its aims and objectives.

The neurophysiological factors responsible for the changes in gait in CSM are poorly understood to date. As outlined in Section 3.3, impairments such as paresis, spasticity, and loss of proprioception may influence gait to varying extents. A recent review of the literature on the physiological evaluation of gait impairment following stroke illustrated the extent to which gait analysis can be used to improve the understanding of gait impairment in a population with a neurological injury (Lamontagne et al., 2007). The same principles could be applied to understanding gait in CSM.

3DGA technology provides the potential for measurement of outcome following surgery using sensitive, robust data. The evaluation of the effect of surgery has been limited in previous studies by the use of insensitive ordinal outcome measures, which may be subject to recall bias and may not capture small but significant changes in the patient’s presentation (Singh and Crockard, 1999, Jankowitz and Gerszten, 2006). The validation of a 30-metre timed walk test has improved the range of outcome measures available for CSM (Singh et al., 2009), however the quality of a gait pattern cannot be captured by a timed walk test alone. 3DGA and EMG would allow for the evaluation of subtle but relevant changes in gait, and may facilitate greater accuracy in the evaluation of surgical decompression and its effect on gait.
Finally, the review in Section 3.4 above shows that rehabilitation protocols for CSM are currently poorly defined. The combined results of the studies indicate that there is potential for improvement in the functional status of these patients with rehabilitation. However, in the absence of any studies of specific intervention, it is difficult to know what elements should be included in these rehabilitation protocols, and how they should be carried out. It has been said that the principles of human motor control are best determined by studying freely moving subjects during natural motor tasks (Capaday, 1997). Detailed, comprehensive evaluation of gait in patients with CSM should contribute to the development of rehabilitation interventions and protocols by allowing the underlying factors contributing to the impairment to be identified.

The next chapter will consider the potential for development of the role of 3DGA and EMG analysis in CSM, leading to the aims and objectives of the thesis.
Chapter 4: Expanding the role of three-dimensional gait analysis and surface electromyography in the evaluation of gait in CSM

4.1 Introduction

It is clear from the evidence presented in Chapter 3 that 3DGA has improved the understanding of some features of gait impairment in CSM. However, there are significant gaps in current knowledge. Studies to date have confined their analyses to TSPs and a limited number of kinematic variables. Kinematics describe the movement of joints during gait and the velocities and accelerations of body segments, but they give no information on the underlying causes of that movement. Little is known about the contribution of individual muscles to the resulting gait pattern. Changes following decompressive surgery are poorly understood, and the physiological impairments underlying gait in CSM have not been determined.

This chapter will explore the potential of 3DGA to improve the scientific understanding of gait in CSM by evaluating its impact in other pathologies. This will lead to the development of aims and objectives for the current study. The thesis will then progress to the consideration of methods to achieve these aims and objectives in Chapter 5.

4.1.1 Factors affecting gait impairment in CSM

Abnormal gait patterns are the result of the combined effects of a primary impairment, such as weakness, pain or sensory loss, and the secondary compensation that ensues to preserve ambulation or the efficiency of ambulation (Dietz, 2002). Unlike other conditions such as stroke, where the injury is sudden and the initial deficit followed by varying degrees of neurological recovery and secondary compensation, the insult to the CNS is more gradual in CSM. It is possible that compensation for a developing neurological deficit may take place as the deficit itself is progressing, particularly if deterioration is slow. CSM varies in its rate of progression and severity of neurological impairment (Baron and Young, 2007). The relationships between primary impairment and compensation cannot be quantified from existing knowledge on gait in this condition.

The underlying contributory factors to the slow speed, prolonged double-support duration and reduced ROM observed in the CSM gait are currently unknown. These features could result from changes in muscle strength, tone, co-activation, the need to compensate for unreliable sensory input, fear of falling, or other factors. The cause and effect relationships between various aspects of gait impairment are of relevance to the interpretation of outcome following surgery, as many people with CSM will be treated with
surgical decompression. It would be valuable to know if certain gait parameters might indicate recovery of neurological function, if recovery does occur. Effective rehabilitation of gait in CSM depends to a large extent on the understanding of the underlying deficits and their interactions.

It was considered beyond the scope of this thesis to consider every possible variable that might influence gait in CSM. However, it was clear that there was potential for 3DGA to explore some of the uncertainties regarding the nature, time frame and recovery of gait impairment. Studies of 3DGA in other pathological conditions were examined with a view to developing specific aims and objectives for this study.

4.2 Improving the understanding of gait using 3DGA

4.2.1 Analysis of gait kinetics

As outlined above and in Chapter 3, most of the work to date using 3DGA in CSM has been confined to the interpretation of TSPs and kinematic variables. Two studies have examined GRFs (Maezawa et al., 2001, Moorthy et al., 2005). These studies did not conduct analysis of the interaction between GRFs and the body segments producing these forces. In other words, joint moments and powers were not calculated. A moment is the product of a force applied and its distance from the pivot point or fulcrum (Kirtley, 2006). In gait, a joint moment represents the body's internal response to an external load (Ounpuu et al., 1996). Moments can be used to infer muscle activity at that joint, provided a number of assumptions are satisfied (Kirtley, 2006). Power is then calculated as the scalar product of the joint moment and the angular velocity of the moving segment (Kirtley, 2006). Power quantifies the net energy absorbed or generated by the working muscles (Lin et al., 2000). Moments and powers cannot be measured directly, but instead they are calculated from the relationship between kinematics and the ground reaction vector using inverse dynamics (Kirtley, 2006).

Information on moments and powers significantly enhances the understanding of a gait deficit. In a study of children with myelomeningocele, kinetic analysis identified the strategies that compensated for weakness of the hip abductors, hip extensors, knee flexors, ankle dorsiflexors, and ankle plantarflexors. For example, compensation for weak or absent hip abductors was achieved by increased lateral trunk sway and greater power absorption by the hip flexors and adductors during stance (Gutierrez et al., 2005). Analysis of joint moments and powers in children with CP identified the strategies behind the gait classifications of jump knee, crouch knee, genu recurvatum and mild impairment. Lin et al. (2000), for example, found that crouch gait required increased power absorption at the knee in terminal stance by the quadriceps to maintain equilibrium with an excessively flexed knee. McNee et al. (2004) supported this finding and suggested that intervention to maintain knee extension in CP might reduce the exposure of the knee
extensor mechanism to increased loads. In other studies, increased hip power generation was identified as a compensatory mechanism for weakness of the ankle plantarflexors in terminal stance in people with stroke (Nadeau et al., 1999) and in older people (DeVita and Hortobagyi, 2000).

Patrick (2003) commented that the information about muscle actions provided by joint moments and powers has improved the management of spasticity in walking in CP, and that incomplete SCI could benefit from a similar approach. The current study will include the analysis of moments and powers at the hip, knee and ankle during gait with the aim of detecting the kinetic contributions to the observed kinematic movement patterns.

4.2.2 Contribution of individual muscles to gait performance

Joint moments are the net product of a combination of forces exerted by active and passive structures. They do not indicate the relative contribution of each individual component force (Lin et al., 2000, Dallmeijer et al., 2011). In a neurological population, the production of moments and powers may be altered by abnormal co-activation between agonist and antagonist muscles (Lamontagne et al., 2000b), abnormal firing of muscles due to spasticity (Dallmeijer et al., 2011), or the influence of contracture (Lin et al., 2000, Dallmeijer et al., 2011). This can lead to difficulty in interpreting the causes of a change in a joint moment.

The role of individual muscles in the production of movement can be evaluated using EMG. Kinesiological EMG, the application of EMG to movement analysis, allows direct analysis of the biological signals responsible for muscle activation during gait (Frigo and Crenna, 2009). Previous studies have described the activity of individual muscles in terms of the appropriateness of their timing during the GC (Buurke et al., 2005), the amplitude of the EMG signal at critical points during gait to indicate intensity of activation (Baddar et al., 2002), and their responses to stretch during periods of lengthening (Crenna, 1999). Combining this information can facilitate interpretation of the causes of an abnormal gait pattern.

4.2.2.1 Evaluation of abnormal timing of activation during gait

The timing of muscle activation during gait is critical to ensure that motor output is optimised to achieve the tasks of the GC (Lauer and Prosser, 2009). Locomotor output depends not only on the force a muscle can produce, but also on the appropriateness of the timing of that force (Shiavi et al., 1987).

Changes in temporal activation patterns can provide insights into the motor control patterns used by people with neurological disorders (Solnik et al., 2010). Gait disturbances in neurological populations have been associated with poor selective
activation of muscle groups, which can manifest as activation of a muscle at an inappropriate time during the GC, or abnormal co-activation between agonists and antagonists (Frigo and Crenna, 2009). Visual observation of EMG patterns during gait in six patients with CSM suggested a prolonged duration of activity of each muscle group (Moorthy et al., 2005), however these observations were subjective. Timing of muscle activation can be quantified by expressing the time for which a muscle is active as a percentage of the total duration of the GC, or by measuring the timing of onset and offset of individual bursts of activity within that GC. In people with CP and stroke, prolonged duration of activation and excessive co-activation between agonist and antagonist muscles have been found in EMG studies of muscle activation (Lamontagne et al., 2000b, Prosser et al., 2010).

Studies on the timing of muscle activation have provided insights into recovery of mobility and function following stroke. In an uncontrolled case series, 13 people with stroke showed significant improvements in functional measures of mobility such as walking speed, the Rivermead Mobility Index, the Functional Ambulation Categories and the Motricity Index from stroke onset to 24 weeks post stroke. However, no significant changes were found in the onset or offset times of eight trunk and lower limb muscles on the hemiparetic side. Instead, there were changes in the timing of muscle activation on the unaffected side. This suggested that functional improvement was related to compensatory, adaptive mechanisms in the unaffected leg, and not to the affected side regaining its pre-stroke patterns of muscle activation (Buurke et al., 2008). Timing is just one aspect of the EMG signal, so it is possible that improvements occurred in other aspects that were not examined. However, the findings suggested that recovery of gait following stroke is not dependent on the re-organisation of temporal aspects of muscle activation on the hemiparetic side (Buurke et al., 2008). This supports the earlier findings of Den Otter et al. (2006), who conducted EMG analysis of gait in people with stroke on a treadmill. They found no changes in timing of muscle activation in the stroke group over a 10-week period, despite improvements in functional mobility. The findings of these studies are important in the field of stroke rehabilitation, as they suggest that factors other than temporal organisation may be of importance in regaining the neuromuscular control of gait. These factors could include changes in EMG amplitude within an altered timing pattern (Den Otter et al., 2006) or the development of compensatory mechanisms by the non-paretic leg and trunk (Buurke et al., 2008).

Co-activation between agonist and antagonist muscles is also a feature of the timing of muscle activation during gait. Co-activation is a motor control strategy that is often employed to increase dynamic joint stiffness where greater stability is required, or to improve movement accuracy (Damiano, 1993). A finding of increased co-activation may reflect a lack of selective muscle activation due to impaired motor control within the CNS (Tedroff et al., 2008). Co-activation is also known to be a compensatory strategy for
muscle weakness and pain in people with osteoarthritis (Heiden et al., 2009), and has a positive association with increasing age (Hortobagyi et al., 2009). Co-activation can be quantified by expressing the time for which both agonist and antagonist are active as the percentage of GC, provided that cross talk has been ruled out (Frigo and Crenna, 2009). Abnormal co-activation has been shown to contribute to deficits in force production in CP (Stackhouse et al., 2005) and to reduce moments acting about a joint at key phases of the GC in stroke (Lamontagne et al., 2002). It is therefore an important factor to consider in the evaluation of deviations in key gait parameters.

4.2.2.2 Evaluation of the intensity of muscle activation on EMG

CSM has been associated with varying degrees of muscle weakness or paresis, similar to other neurological disorders. The amplitude of electrical activity on an EMG trace, measured in millivolts (mV), indicates the intensity of a muscle contraction at a given point in time. However, there is no direct relationship between EMG and muscular force (Heintz and Gutierrez-Farewik, 2007), therefore the interpretation of signal amplitude in the context of physiological muscle activity is a complex task. Methods have been developed to quantify each muscle’s individual force contribution from EMG data (Bogey et al., 2010), but have not been tested in neurological populations.

The value of EMG amplitude as a stand-alone measure has a number of limitations for test-retest and group comparison studies, due to variation in electrode placement and inter-subject variation in muscle morphology and soft tissue depth (Campanini et al., 2007). Amplitude is commonly normalised to the peak value within a GC to facilitate comparison between performances on different test days and between individuals (Lehman and McGill, 1999). Normalisation in this way has the undesirable effect of undermining the absolute magnitude of the signal. Muscles that are activated at very low amplitude may be over-represented (Lamontagne et al., 2007). Furthermore, inability to contract a muscle due to neuromuscular dysfunction will not necessarily be detected (Benoit et al., 2003). Normalisation to a maximum voluntary contraction (MVC) provides the advantage of a neurophysiological reference. It would be expected that participants with greater levels of paresis would demonstrate levels of activation during gait closer to their voluntary maximum, however this may be misleading in individuals with neurological impairment who are unable to produce sufficient muscle activation to achieve a normal force (Damiano et al., 2000).

Despite these limitations, the amplitude of the EMG signal can provide information on whether a muscle’s level of activation is appropriately scaled to the demands of the motor task (Frigo and Crenna, 2009). A number of studies have used timing detection algorithms to determine phases of muscle activity. The ratio of a signal’s amplitude within a burst to its amplitude outside that burst can indicate an individual’s motor control capacities and the ability to scale motor output to the demands of the task (Roetenberg et
A study of EMG timing and amplitude during gait in people with stroke found that the use of gait aids significantly reduced the median amplitude of vastus lateralis and tibialis anterior activity, confirming the hypothesis that less muscular effort was required when walking with an aid (Roetenberg et al., 2003). The EMG amplitudes of soleus, gastrocnemius and tibialis anterior changed significantly during gait in 34 children with CP after multilevel surgical treatment. The amplitudes of these muscles’ EMG signals decreased at initial contact and increased in terminal stance, while those of the thigh muscles did not change (Patikas et al., 2007). Findings such as these could identify aspects of motor control that might be amenable to change with the appropriate intervention.

Measurement of EMG amplitude in gait in people with CSM is a novel undertaking. Possible methods will need to be explored to determine the most valid and reliable approach for this population. These methods will be further examined in Chapter 5, Section 5.4.4.4.

4.2.2.3 Evaluation of abnormal stretch reflexes

Spasticity has been defined as “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 neurone syndrome” (Lance, 1980), and as “disordered sensori-motor control, resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles” (Pandyan et al., 2005). The presence of abnormally increased tone is one of the objective findings in a diagnosis of CSM (Salvi et al., 2006). Gait in CSM is often subjectively described as a “spastic” pattern (Suzuki et al., 2002). Spasticity in this condition therefore deserves further consideration.

Studies have found a poor correlation between static measurements of spasticity and its manifestations during active movement (Dietz, 2003). A study of spasticity in chronic stroke patients found that the stretch reflex in the gastrocnemius muscle was enhanced in stroke patients during passive movement, but not during active movement of the antagonist under conditions designed to simulate limb loading (Ada et al., 1998). Passive muscle stretch stimulates the monosynaptic stretch reflex, however this reflex has very little function in the regulation of gait, except in single leg stance (Dietz, 2002). As a result, it has been suggested that there are differences in the pathophysiology of the clinical signs of spasticity, tested by passive stretch, and the spastic movement disorder that hinders the patient under dynamic conditions (Dietz, 2003).

These findings are significant for CSM because the presence of lower limb spasticity, and the spastic gait that may result, is one of a number of UMNL signs in the typical CSM presentation (Baron and Young, 2007). Lower limb spasticity is usually assessed using
either the Modified Ashworth Scale (Bohannon and Smith, 1987), the Tardieu scale (Haugh et al., 2006), or by simple clinical examination (Barnes, 2008). The poor correlation between resting and dynamic spasticity implies that these scales may be of limited value in the assessment of a condition where gait impairment is a primary symptom. In the clinical setting, the presence of a spastic gait in CSM is normally evaluated by simple observation (Baron and Young, 2007), yet studies have shown the accuracy of observation to be poor compared to 3DGA (Williams et al., 2009a). These findings underpin the need for the development of a locomotor-specific measurement of spasticity in CSM as an objective means of evaluating this clinical feature.

A locomotor-specific measure of spasticity (LSMS) was developed for children with CP by Crenna (1998) and further used for people with stroke by Lamontagne et al. (2001). This measurement was based on the relationship between three variables, 1) the lengthening velocity of a muscle, 2) EMG signal amplitude and 3) the timing of onset of EMG activity during lengthening. Children with spastic CP initiated muscle activity in the hamstrings at a lower lengthening velocity during terminal swing than typically developing children. This suggested that the lengthening velocity threshold for triggering EMG activity was reduced in the CP cohort, indicating higher velocity-related sensitivity to lengthening, namely spasticity (Crenna, 1999). A similar approach was used by Lamontagne et al. (2001), who found that the direction of the slope between the EMG amplitude and lengthening velocity of gastrocnemius was the reverse of normal in people with stroke. In HCs, the slope was negative because EMG amplitude did not increase with increasing lengthening velocity, but in people with stroke, EMG amplitude showed a positive relationship with lengthening velocity. This indicated a pathological response to muscle lengthening and an increase in the gain of the stretch reflex (Lamontagne et al., 2001). These methods are advantageous because they are specific to gait analysis, particularly as the correlations between spasticity during passive and dynamic movement are known to be poor (Ada et al., 1998). They also avoid the use of unnatural stimuli, such as the Hoffmann (H) reflex, in examining the excitability of the stretch reflex during gait (Lamontagne et al., 2001). Further details on the methods involved in the calculation of the locomotor-specific measure of spasticity are provided in Chapter 5, Section 5.4.4.5.

The LSMS has not previously been evaluated in CSM. It has the potential to enhance the understanding of the pathophysiology of this condition. If it were found to be valid and reliable, it could serve as an adjunct to diagnosis in the future, particularly where clinical findings of tone-related changes are unclear on physical examination (Salvi et al., 2006, Baron and Young, 2007).
4.3 Analysis of motor control of gait using 3DGA and EMG data

4.3.1 Relationships between groups of gait variables

Individual TSP, kinematic, kinetic, and EMG parameters are of interest in the evaluation of neurological gait impairment. However, a greater understanding of motor control strategies for the tasks of gait can be obtained by investigating the relationships between individual variables. In a study of people with stroke, Lamontagne et al. (2002) considered the interaction between EMG timing, amplitude, kinematic key points and peak moments in the evaluation of motor control at the ankle during the pre swing phase of gait. Participants who had suffered a stroke were assessed using 3DGA and surface EMG at their comfortable walking speed. They were compared to HCs at both comfortable walking speed and a slower speed approximating that of the stroke group. Decreased peak plantarflexor moments at pre swing were noted in both the paretic and non-paretic lower limbs in the stroke group, but the mechanisms of disturbed motor control differed between the limbs. The paretic limb showed a reduction in the EMG amplitude of medial gastrocnemius and a velocity-sensitive response during lengthening, which indicated both reduced muscle recruitment and hyperactive stretch reflexes. The non-paretic limb showed normal EMG amplitude but greater levels of antagonist co-activation. This indicated an adaptation either to secondary disuse or to poor postural stability during gait (Lamontagne et al., 2000a, Lamontagne et al., 2002). Regression found that more than 50% of the variance of gait speed was explained by the lower peak activation of the medial gastrocnemius of the paretic lower limb in stance. This finding emphasised calf muscle paresis as one of the primary impairments affecting gait after stroke (Lamontagne et al., 2002). However, a limitation of the study was that EMG amplitude was not normalised, therefore inter-subject variation in electrode placement and soft tissue impedance may have affected the results.

It is not yet known whether similar patterns could be expected in CSM. Both CSM and stroke represent injuries to the adult CNS and as such there may be similarities in the overall patterns of gait impairment, but they differ in aetiology, speed of onset and site of pathology. The application of methods to analyse the interaction between multiple gait variables at key phases of the GC, such as those described in the previous paragraph, could improve the understanding of the factors contributing to gait impairment in CSM.

4.3.2 Identification of principal components contributing to gait dysfunction

Gait data is usually interpreted by examining changes in GC curves and specific key points, such as peak ROM or power, or the timing of a key point. However, many parameters are correlated and therefore show a strong interdependency. For this reason, it can be difficult to interpret the dimensions of gait that individual variables represent.
Multivariate statistical analysis methods can uncover more complex relationships between interdependent variables (Perez and Nussbaum, 2003). Principal components analysis (PCA) is one such method. It has been used to classify gait patterns in children with CP (Carriero et al., 2009). In Carriero’s study, lower limb kinematic key points from typically developing children and children with CP were evaluated using PCA to identify the dominant sources of variability in the data and to generate new independent variables, the principal components (PCs). The first PC, accounting for 61% of the variability in the data, was a function of gait speed and hip and knee movement in the sagittal plane, indicating the relationship between these variables. The second PC was a function of cadence, step time and frontal plane kinematics. Its scores were effective in differentiating jump knee from crouch knee gait patterns (Carriero et al., 2009). Cluster analysis of PC scores facilitated the identification of abnormalities that characterised a particular gait pattern, while quantitatively accounting for the interdependence between component variables.

In a similar study, Gaudreault et al. (2011) used PCA to analyse kinematic and kinetic gait data from adults with osteoarthritis of the knee. Two distinct sub-groups of gait impairment were extracted. When the effect of a physiotherapy intervention was evaluated, one of the sub-groups showed changes in kinematic and kinetic key points in response to the intervention, whereas the other group did not. Pre- to post-intervention comparisons of the group as a whole, without accounting for the PCs, found no statistically significant differences in gait parameters. The sub-group who responded had higher peak knee moments prior to treatment than the non-responding group, suggesting that their gait was less severely affected by osteoarthritis. Gaudreault’s study illustrates the contribution of multivariate data reduction techniques when evaluating a complex, multi-dimensional outcome such as gait, particularly where heterogeneity of a population might result in the masking of positive or negative effects of an intervention for a particular sub-group.

A further example of the application of multivariate analysis to 3DGA is the Gait Deviation Index (GDI) (Schwartz and Rozumalski, 2008). The GDI provides a single quantitative measure of deviation in the kinematic patterns of children with CP compared to those of typically developing children. The numerical index generated from the analysis allows the subject’s overall gait pattern to be interpreted in the context of deviation from normal. A GDI score of 100 points or greater suggests no gait pathology, while each 10 points below 100 represents one standard deviation from the overall profile for a typically developing child (Schwartz and Rozumalski, 2008). The reduction of data from numerous GC curves and key points into a single index has potential advantages in repeated measures design studies, particularly where the overall effect of an intervention on a gait pattern is of interest, rather than its effect on an individual joint or variable. The GDI has
been extensively validated in the paediatric population (Molloy et al., 2010), but its applicability to adults with neurological dysfunction is not well established.

The application of multivariate analysis to 3DGA data in CSM has a number of potential advantages. The classification of a large number of interdependent variables would aid in the objective determination of gait patterns in CSM compared to HCs. The reduction of multiple correlated variables into a smaller number of PCs has advantages for communicating an overall change in a gait pattern, which may be of interest when evaluating the effects of an intervention, such as surgery. There is potential benefit in the development of strategies for rehabilitation, as shown by Gaudreault et al. (2011), who found that classifying participants based on their PCA scores led to the identification of a sub-group who responded more positively to an exercise intervention. Studies of rehabilitation interventions in CSM would require adequate statistical power for sub-group analysis if such an objective were to be achieved.

4.4 Aims and objectives

The goal at the outset of this project was to analyse gait impairment in CSM, with a view to understanding its mechanisms, impact on function and quality of life, and response to surgery. Gait is a complex task with many underlying dimensions. It reflects the local force-producing capabilities of individual muscles, the ability of the CNS to modulate force production and co-ordinate muscles to achieve stability and progression, and responsiveness to changes in environmental conditions and functional goals (Dietz, 2002).

This chapter has given an overview of the possibilities of 3DGA and EMG data in providing information on the biomechanical and neuromuscular impairments underlying a pathological gait pattern. Based on the literature review of gait in CSM provided in Chapter 3, and the possibilities for improving the understanding of gait outlined in this chapter, the aims and objectives of this thesis were devised as follows.

4.4.1 First aim: Reliability of 3DGA

The first aim of the thesis was to examine the repeatability of 3DGA measures in CSM, incorporating analysis of test-retest reliability of temporal-spatial, kinematic, kinetic and EMG parameters. This was addressed using a reliability study.

4.4.2 Second aim: Comparison of gait patterns in CSM and healthy controls

The second aim was to compare gait patterns of people with CSM to those of healthy, age- and gender-matched controls. This aim was achieved using a cross-sectional study design, and addressed the following objectives.
1. To describe the gait patterns of people with CSM with reference to age- and gender-based norms.
   a) To compare TSPs, kinematic key points and kinetic key points.
   b) To identify the principal components of these key points in people with CSM and HCs.
   c) To investigate EMG patterns of muscle activation during gait in people with CSM compared to HCs.
   d) To evaluate timing of muscle activity of major lower limb muscles during gait.
   e) To measure co-activation of agonist and antagonist muscles.
   f) To examine indicators of the intensity of muscle activation.
   g) To evaluate dynamic responses to muscle lengthening as a locomotor-specific measurement of spasticity.

2. To identify key areas of impairment in gait in CSM.

4.4.3 Third aim: Analysis of change in gait following surgery for CSM

The third aim was to determine changes in temporal-spatial, kinematic, kinetic, and EMG variables in gait in people with CSM who undergo surgery. This aim was addressed using a repeated-measures experimental study design, with follow-up assessments at six and twelve months following surgery. The experimental study also addressed the following secondary objectives:

1. To determine if changes in gait following surgery can be predicted by other variables.
2. To measure change in functional mobility in people with CSM after surgery.
3. To determine the impact of surgery on health-related quality of life.

4.5 Conclusion

The review of the literature on the pathophysiology of CSM, its associated gait impairment and the impact of 3DGA and EMG analysis on the understanding of other neurological conditions, led to the development of three broad aims for this thesis. The achievement of these aims and their associated objectives led to the design of three separate methodologies, a reliability study, a cross sectional study, and an experimental study. Analysis of gait using 3DGA and EMG was a central component of all three studies. In the next chapter, the development of methodology common to all studies will be discussed, leading to the outline of general methods for recruitment, gait analysis and data processing in Chapter 6.
Chapter 5: Development of Methodology

5.1 Introduction

The aims and objectives outlined in Chapter 4, Section 4.4, required the development of protocols for the primary outcome measures of 3DGA and EMG, and the selection of parameters that would address the research questions. Secondary outcome measures to quantify spasticity, functional mobility, quality of life, and severity of myelopathy were also required. These allowed for interpretation of gait data within the context of a clinical assessment of impairment, functional limitation and participation restriction.

The thesis includes three studies: 1) a test-retest reliability study of 3DGA and EMG analysis in CSM, 2) a cross-sectional study to compare gait in people with CSM to age- and gender-matched controls, 3) an experimental study to identify changes in gait following decompressive surgery for myelopathy. All studies required the recruitment of people with CSM. A consistent method of determining this diagnosis was therefore required. 3DGA and EMG assessment of gait was a central component of all studies, requiring an evidence-based approach to data collection, extraction of parameters, and interpretation of data.

This chapter will outline the development of the methods common to all studies, namely, recruitment of participants with CSM, development of 3DGA and EMG assessment procedures, extraction of clinically relevant parameters from this data, and the selection of secondary outcome measures for CSM.

5.2 Recruitment of participants

As discussed in Chapter 2, there are no definitive clinical tests for CSM. The diagnosis is based on clinical findings with radiological confirmation of spinal cord compression (Cook et al., 2009). In clinical practice, some people fall into an uncertain diagnostic category, with signs and symptoms but no radiological evidence (Wong et al., 2008). It was decided that the CSM cohort in the current study should be representative of people with definite, rather than uncertain, CSM. The diagnostic criteria for CSM participants were the presence of at least one symptom consistent with spinal cord compression, at least one objective long-tract sign, and radiological evidence of spondylotic cord compression with the exclusion of other pathologies. This is in keeping with other studies, for example Fehlings et al. (2008). The full list of diagnostic criteria is provided in Chapter 6, Section 6.3.

In relation to inclusion and exclusion criteria, there were slight differences in the requirements of the studies. Previous operative intervention for CSM was considered a
confounding factor in the cross-sectional and experimental studies. Recruitment to these studies was therefore limited to people with CSM who had no history of operative intervention for this condition. Previous surgery for other clinical presentations in the cervical spine, such as discectomy for nerve root compression, was not considered an exclusion criterion provided that myelopathy was not an indication for that surgery and it did not involve the use of instrumentation. In relation to the reliability study, it was considered that previous surgery would not confound the quantification of an error range of gait parameters specific to CSM. Therefore, recruitment to the reliability study was extended to participants who had undergone surgical intervention, provided they met the diagnostic criteria for CSM and were still clinically myelopathic.

5.3 Development of three-dimensional gait analysis methodology

5.3.1 Introduction

3DGA is considered the “gold standard” in assessment of gait (Kirtley, 2006), and therefore was the method of choice in seeking to build a comprehensive picture of the gait impairment associated with CSM. A number of methodological parameters required consideration to ensure the accuracy of the collected data.

5.3.2 Equipment

5.3.2.1 Motion analysis system

Motion analysis was carried out using a five camera Vicon 250 motion analysis system (Vicon Motion Systems Ltd., Oxford, UK). The system depends on the accurate attachment of lightweight retro-reflective markers to specific bony landmarks, shown in Figure 5.1. These were captured in three-dimensional space at a rate of 50 Hertz (Hz). The three-dimensional position of a marker can be calculated once it is visible to two cameras at the same point in time. Calculation of motion at the centres of each underlying joint was carried out via the Plug-in-Gait® (PiG) software model provided in the Vicon Workstation® application. Data were then exported to the visualisation software Polygon®, where movement at each joint was presented in three planes (sagittal, frontal and transverse) over the duration of a GC. Figure 5.2, Figure 5.3 and Figure 5.4 show the location of the retro-reflective markers in the capture volume, the determination of joint centres using PiG, and the visualisation of time-normalised joint motion in Polygon, respectively.
Figure 5.1: Attachment of retro-reflective markers for motion capture

Figure 5.2: Location of retro-reflective markers in capture space
Ground reaction forces (GRFs) across three planes were captured using a ground-mounted Kistler multi-component force plate (Kistler Instruments Ltd., Winterthur, Switzerland). The force plate was integrated with the Vicon software, allowing for calculation of joint moments and powers from the kinematic and GRF data by inverse dynamics. Force plate data were captured at a rate of 1000 Hz. Figure 5.5 shows a
participant striking the force plate during a gait trial. Figure 5.6 shows the real-time capture of a ground reaction force by the Vicon system.

Figure 5.5: Participant striking the force plate during a gait trial

Figure 5.6: Capture of a ground reaction force during a gait trial
Ground reaction force shown by the vertical yellow line
White dots represent location of retro-reflective markers
5.3.3 Methodological considerations in 3DGA

5.3.3.1 Marker placement using the Modified Helen Hayes model

The Modified Helen Hayes model (MHH) (Davis et al., 1991) is the Vicon system’s default anthropometric model for the calculation of joint centres and subsequently of joint kinematics during movement. The model is based on the placement of 13 retro-reflective markers, eight bilaterally on the anterior superior iliac spine (ASIS), lateral aspect of the knee joint, lateral malleolus, and head of the second metatarsal, and five wand markers, one on the sacrum and four on the lateral aspect of the thigh and lower leg bilaterally, as shown in Figure 5.1 above. Markers are also placed on the calcanei to aid in the identification of heel strike during gait.

Identification of joint centres depends heavily on accurate marker placement. Some of the bony landmarks are not readily identifiable, particularly those for the thigh marker (Baker et al., 1999). A previous study demonstrated the consequences of inaccurate marker placement. Deliberate misplacement of the knee and thigh markers caused offsets from the sagittal to the frontal plane at the knee joint, leading to inaccurate measurement of hip joint rotation and knee moments. Misplacement of the ASIS markers resulted in relocation of the hip joint centres and inaccurate calculation of frontal plane motion. The study concluded that MHH was particularly sensitive marker placement errors (Kirtley, 2002). A further concern is the hierarchical nature of MHH, such that any proximal marker misalignment will result in the propagation of errors to distal joints (Schache et al., 2006).

It is clear from these results that, in any study using 3DGA to assess patient outcomes, particular attention must be given to minimising the errors associated with marker placement. A standard protocol was used in the Movement Laboratory to maximise consistency and repeatability of the data collection process, provided in Appendix 5.1. Test-retest reliability of anthropometric measurements in the laboratory was demonstrated in an earlier study (Meldrum et al., 2005). Marker placement was and anthropometric measurements were standardised as far as possible by providing guidelines within the protocol.

5.3.3.2 Use of the knee alignment device

An aspect of marker placement that is particularly error-prone is the definition of the femoral frontal plane. This plane is based on an estimate of the functional knee joint flexion-extension axis from the thigh co-ordinate system (Baker et al., 1999, Schache et al., 2006). The Knee Alignment Device (KAD) (Motion Lab Systems Inc., Baton Rouge, LA, USA), shown in Figure 5.7, is a spring-loaded clamp that fits over a subject’s knee during static calibration of the marker set. A virtual knee marker can be established at the central joint of the KAD during calibration, enabling more accurate measurement of the
knee flexion-extension axis (Motion Lab Systems Inc., 1998). Errors in the knee joint axis can be quantified by the amount of variability in knee varus–valgus motion and hip rotation (Schache et al., 2006).

Repeatability of knee joint axis determination using the KAD has been compared to two alternative methods, one based on the trans-epicondylar axis and an alternative dynamic optimisation method (Schache et al., 2006). Repeatability of knee varus–valgus motion and hip rotation were measured using the coefficient of multiple determination (CMD), a reliability statistic where a value of 1 indicates perfect agreement and 0 indicates no agreement (Kadaba et al., 1989). Test-retest repeatability of hip rotation using KAD was moderate, with CMD values of 0.69. Absolute variability of knee varus–valgus motion was 2.66–3.45°. An alternative method, the dynamic optimisation method of Baker et al. (1999), showed higher repeatability, with a CMD for hip rotation of 0.78 and knee varus–valgus variability of 1.96°. Schache’s study did not evaluate the repeatability of these parameters using a simple knee marker without the aid of an adjunct such as KAD, and therefore the effect of KAD compared to “blind” knee marker placement cannot be deduced. The study was also limited by the lack of a gold standard method for identification of the knee joint flexion-extension axis during motion, meaning that the true validity of the values determined by KAD or other methods are unknown. The KAD method was the standard protocol in the RCSI Movement Laboratory. Its repeatability values, identified by Schache et al. (2006), were considered acceptable for use in the current study.

![Figure 5.7: Knee alignment device](image)

5.3.3.3 Filtering marker trajectories

The motion of the skin during gait can result in trajectory noise when markers are captured in three-dimensional space. This artefact is normally minimised by the use of a digital off-line low-pass filter, such as the Woltring cross-validatory quintic spline routine
(Woltring, 1986). The Woltring filter is incorporated into the Vicon system. The user is required to specify the mean standard error (MSE) of the filter. Higher MSE values result in a greater level of filtering, potentially leading to a distortion of gait parameters in phase and magnitude (Molloy et al., 2008).

A brief experiment was conducted to determine the optimal MSE for our data. Kinematic and kinetic data from a single participant were captured and saved to three files. Data were then processed with Woltring filters of MSE 27, 20 and 15 mm². The resulting output was compared visually by inspection and quantitatively by measuring the peak values obtained in each parameter. Figure 5.8 compares the effect of the three filters on the knee power curve over one GC.

![Effect of Woltring Filter MSE Values](image)

*Figure 5.8: Effect of filtering data for knee power using a Woltring filter with mean standard error (MSE) values of 27, 20 and 15 mm²*

The higher the MSE value, the greater the diminution of the peak values, illustrated by the second power generation peak in Figure 5.8 at about 15% GC duration. The lower MSE value of 15 mm² appeared on visual inspection to diminish motion artefact sufficiently, without dampening the peak values. A Woltring filter with MSE 15 mm² was
therefore selected. The same MSE was used to filter trajectories in the Vicon system by a number of other studies, for example Beaulieu et al. (2010).

5.4 Development of electromyography methodology

5.4.1 Introduction

EMG is the recording of the electrical signals associated with muscle contraction by needle, fine-wire, or surface electrodes (Winter, 1990, Reaz et al., 2006). The EMG signal itself is a series of individual spikes of varying amplitude and duration, reflecting the underlying action potentials. This characteristic waveform results from the depolarisation of the muscle’s transverse tubular system and the sarcoplasmic reticulum, and its subsequent repolarisation (Winter, 1990). EMG is not a direct measurement of muscular activity, but rather an indirect indicator of muscle function (Perry, 1992).

The use of EMG in motion analysis dates back to the 1940s (Sutherland, 2001). As part of a gait analysis protocol, it is the only available assessment tool that allows direct analysis of the biological signals responsible for muscle activation (Frigo and Crenna, 2009). It can therefore provide information on the relationship between agonist and antagonist muscle groups during a motor task, which is not possible using kinetic analysis alone as only the net torque can be measured. EMG was considered essential in this study, as the aims and objectives required the analysis of individual muscle activity.

5.4.2 Considerations for the recording of EMG via surface electrodes

Surface electromyography (SEMG), the acquisition of the EMG signal via surface electrodes, has been recommended by the American Academy of Neurology as a suitable tool for the kinesiological analysis of movement disorders (Pullman et al., 2000). SEMG has a number of advantages over fine-wire EMG for this purpose, including ease of application and higher reliability (Winter and Scott, 1991). Surface electrodes are non-invasive and painless, and therefore more acceptable to participants. By contrast, indwelling electrodes can interfere with the natural gait pattern. A study found that indwelling electrodes caused a 23.5% reduction in gait speed and a 7.9% decrease in cadence in children with CP (Young et al., 1989).

The main limitation of surface electrodes is their potential to record signals generated by neighbouring muscles as well as the muscle under examination, a phenomenon known as cross talk (Koh and Grabiner, 1993). A contracting muscle generates an electrical signal that is dispersed throughout adjacent tissues by volume conduction, potentially making it difficult to distinguish individual muscle activation patterns (DeLuca and Merletti, 1988). Cross talk has been demonstrated during SEMG examinations of rectus femoris (Nene et al., 2004), gastrocnemius (Perry et al., 1981), peroneus longus (Johanson and
Radtka, 2006), and hamstrings (Koh and Grabiner, 1992). Levels of cross talk of 11–18% MVC from adjacent muscles have been reported in studies of lower extremity muscles (Johanson and Radtka, 2006).

The following factors were considered in the acquisition of SEMG data to ensure that the recorded signal paralleled the underlying signal of the contracting muscle as closely as possible.

5.4.2.1 Electrode placement

The location of the electrode on the examined muscle has a fundamental influence on the output of the EMG signal. A general recommendation is that the electrode should be placed mid-way between the motor end point and the muscle–tendon junction, and be aligned in the direction of muscle fibres insofar as this can be estimated on visual inspection (Basmajian and DeLuca, 1985). Recommendations for optimum electrode placement were published by the Surface Electromyography for Non-Invasive Assessment of Muscles (SENIAM) taskforce (Hermens and Freriks, 1997). These guidelines were followed in the placement of electrodes for this study. Further details are provided in Appendix 5.1.

5.4.2.2 Choice of muscles for examination

In general, large, superficial muscles are less prone to cross talk than smaller, deeper muscles, and can be recorded by SEMG with greater accuracy (Sutherland, 2001). The muscles chosen for analysis in this study were rectus femoris (RF), biceps femoris (BF), tibialis anterior (TA), and medial head of gastrocnemius (MG). They form antagonistic pairs in the thigh and lower leg and thus the influence of neurological changes in timing and co-activation during the GC can be measured. All contribute significantly to the performance of gait, particularly in relation to support and progression in the sagittal plane (Anderson and Pandy, 2003).

5.4.2.3 Maximising fidelity of the SEMG signal

A SEMG electrode will record any signal that reaches its detection area. It is essential to obtain an EMG signal that contains the maximum amount of information from the underlying muscle and the minimum amount of contamination from motion artefact at the electrode-skin interface, noise from the electricity mains, and cross talk from surrounding muscles (DeLuca, 2002). There are two parameters that influence the fidelity of the SEMG signal to the underlying muscle activity: 1) the signal-to-noise ratio (SNR), the ratio of the signal from the muscle of interest to unwanted artefact or noise (Rash, 2004), and 2) the distortion of the relative contribution of the frequency components in the signal (DeLuca, 2002). The factors discussed in the following sections, namely electrodes,
amplification, bandwidth, and sampling frequency, have been recommended for consideration when recording SEMG to ensure a high quality signal.

5.4.2.3.1 Electrodes

The electrodes used in SEMG examination can be either active or passive. Active electrodes were used in this study. These have a built-in pre-amplifier to reduce impedance, which reduces movement artefact and improves SNR compared to passive electrodes (Rash, 2004).

Either monopolar or bipolar electrodes can be used. Bipolar electrodes are considered advantageous due to their ability to reduce cross talk by employing a differentially amplified system, where signals common to both electrodes are rejected as noise (Rash, 2004). The differential amplifier’s accuracy in subtracting these unwanted signals is measured by the Common Mode Rejection Ratio (CMRR). The use of double-differentiated electrodes was found to be effective in reducing cross talk (van Vugt and van Dijk, 2001). The MA-300 system in the RCSI Movement Laboratory used double-differentiated electrodes with a reported CMRR of at least 100 decibels (dB). A CMRR of 90dB is generally sufficient to suppress extraneous electrical noises (DeLuca, 2002).

The input impedance of the differential amplifier should be as large as possible to prevent attenuation and distortion of the signal caused by impedance at the junction of the skin and the electrode. A value of $10^{12}$ Ohms has been recommended (DeLuca, 2002). This was the input impedance of the electrodes used for the current study. The skin was prepared by shaving and cleaning with an alcohol swab prior to application of the electrode, a procedure thought to reduce the resistance of the skin by 200% (Rash, 2004).

5.4.2.3.2 Amplification

Correct amplification ensures that the recorded signal is of sufficient magnitude for analysis. The gain of an amplifier is the amount of amplification applied to the signal and should be sufficient to have an output signal of 1 Volt (V) (Rash, 2004). In this study, a gain range of 2,000 to 13,200 was used to achieve a signal of sufficient amplitude. Signals were inspected in real time using Windaq® software (Dataq Instruments Inc, Akron, Ohio, USA), both at rest and during a maximum voluntary contraction, to ensure optimal detection.

5.4.2.3.3 Bandwidth

The bandwidth is the range of collectable frequencies of the amplifier. The bandwidth should be high enough to reject the low frequency movement artefacts and low enough to attenuate the signal as little as possible. According to DeLuca (2002), the dominant energy of the EMG signal is in the range of 50–150 Hz. There is some variation in the
optimal bandwidth recommended by different authors. A high-pass filter of 10–20 Hz and a low-pass filter of 500 Hz has been recommended to optimise the recording of the entire EMG signal while reducing SNR (Basmajian and DeLuca, 1985, DeLuca, 1997, Hermens et al., 1999). The International Society for Electromyography and Kinesiology (ISEK) recommended a bandwidth within the range of a 5–10 Hz high-pass filter and a 400–450 Hz low-pass filter (Merletti, 1997). The minimum high-pass filter of the MA-300 unit is 20 Hz. The bandwidth was set at 20–500 Hz for this study. This is in agreement with the recommendations of DeLuca (1997) when using a differentially amplified system.

5.4.2.3.4 Sampling

Sampling is the process of acquiring the values of a signal at equally spaced time intervals, designated by the sampling frequency, and recording these values into a computer (Pozzo et al., 2004). The optimal sampling frequency for any signal is dictated by the Nyquist theorem. Nyquist states that the original signal can be fully reconstructed from its samples if the sampling frequency is at least twice that of the highest frequency present in the signal itself (Winter, 1990, Pozzo et al., 2004). Failure to sample at a sufficiently high frequency results in aliasing errors, namely the generation of false frequencies in the sampled data that were not present in the original signal. Over-sampling, or sampling at a rate higher than twice the signal's maximum frequency, has been employed in previous studies, but is unnecessary in determining standard timing and amplitude variables of the signal (Ives and Wigglesworth, 2003). In this study, EMG data were sampled at a rate of 1,000 Hz in accordance with the Nyquist specification. Over-sampling was not employed. Sampling was conducted using a DATAQ® Instruments 32-channel DI-720 Data Acquisition System analogue to digital convertor with 12-bit resolution (DATAQ Instruments Inc., Akron, Ohio, USA).

5.4.3 Processing of EMG

To enhance its validity as a measurement tool for research purposes, the EMG signal should be subjected to further processing, as its variability between individuals precludes interpretation of the raw signal across groups of people (Bogey et al., 1992).

5.4.3.1 Filtering

After the EMG signal has been recorded and sampled into the computer, a filter can then be applied to further clean the data (Rash, 2004). A number of filter types are available for digital signal processing, and of these, the Butterworth filter has been recommended and is most widely used (Winter, 1990).

The choice of filtering frequency can affect the integrity of the EMG signal, and a balance must be sought between removing unwanted frequencies and losing valuable information from the signal. High pass filtering is needed to remove unwanted direct current offset.
created by the half-cell potential of the electrodes, and to remove motion artefact due to movements of the lead wires generated during non-isometric activity in EMG recordings (Pozzo et al., 2004). Frequency components below 20 Hz are usually corrupted by movement artefact and instability in the signal (Hermens and Freriks, 1997, DeLuca, 2002). Low pass filtering is used to remove unwanted noise beyond the bandwidth of interest that could lead to a degradation of the SNR (Pozzo et al., 2004). Most of the power in the muscle signal exists below a frequency of 500 Hz (Soderberg and Knutson, 2000), and the dominant power exists between 50–150 Hz (DeLuca, 2002).

Winter (1990) recommended that a residual analysis be conducted of the difference between filtered and unfiltered signals to ensure the optimal selection of low- and high-pass filtering frequencies. A low-pass filter of 400 Hz and high-pass filter of 25 Hz have been recommended for use in gait analysis (Merletti et al., 2009). In the current study, this filter combination was applied to a number of test signals in a forward and backward direction using a routine in MATLAB® (The Mathworks, Natick, MA, USA), detailed in Appendix 5.2. The effect of the filter was examined by visually inspecting the trace patterns of the original signal and the filtered signal. This verified that the filter appeared to smooth out artefact from the raw EMG, while maintaining a signal that was close in amplitude to the original signal. The effect of the filter is shown Figure 5.9.
The blue trace shows the raw data and the red trace shows the filtered data.
A reduction in the amplitude of some individual spikes of activity is seen, while the overall appearance of the trace is preserved.

### 5.4.3.2 Amplitude processing

The amplitude of the EMG signal can be described in two ways, 1) rectification and linear envelope detection or 2) calculation of root-mean-square (RMS). Rectification involves transposing the negative signals to the positive side of the line, so that positive and negative values do not cancel each other out in subsequent processing (Perry, 1992). The rectified signals can then be smoothed over a given time constant, usually 10–25 ms, to create a function known as the average rectified value (ARV) or linear envelope (Merletti, 1997). RMS involves 1) squaring the individual values of the signal to remove the positive and negative fluctuations, 2) obtaining the average of the squared values over a series of values, and 3) calculating the square root of this average (Rash, 2004).

RMS value is preferred to ARV as it is a direct measure of the signal’s power and therefore has a clear physical meaning (Soderberg and Knutson, 2000, DeLuca, 2002).
The RMS method was therefore chosen for amplitude processing of the SEMG data in the current study.

The time interval over which RMS is calculated is a critical factor in the determination of EMG amplitude (Merletti, 1997). A 30-millisecond (ms) time window with 20 ms overlap was chosen based on observations of differing window lengths (20 ms, 30 ms, 50 ms). The 30 ms window has been reported in previous studies using SEMG data (Mercer et al., 2009). It was tested in a set of signals from the current study and appeared to provide appropriate smoothing while still retaining the quality of the original signal, as shown in Figure 5.10. Over-smoothing the data was undesirable due to the potential to eliminate important information on the quality of the contraction (Frigo and Crenna, 2009).
RMS was calculated over 30 ms windows with 20 ms overlap and interpolated to 101 data points representing gait cycle percentage points from 0 to 100.

The absolute value of the EMG signal amplitude can be affected by intrinsic participant characteristics, such as subcutaneous fat thickness (Lehman and McGill, 1999), as well as extrinsic factors such as heat, humidity, and the location of the electrode over the muscle (Hogrel, 2005). This leads to difficulty in comparing EMG amplitude between different individuals and between test days (Hogrel, 2005). The use of standard protocols
for electrode placement, such as the SENIAM guidelines, can minimise variability due to procedural factors (Gruet et al., 2010). However, despite standard protocols, test-retest reliability studies have shown high absolute variability in EMG amplitude (Campanini et al., 2007, Gruet et al., 2010). SEM values for non-normalised RMS of the quadriceps muscles in people with knee pathology ranged from 0.08 mV for RF to 0.28 mV for vastus lateralis, and indicated high variability relative to the mean in all cases (Callaghan et al., 2009).

The problem can be addressed by normalising a muscle’s amplitude at each point in the signal to a reference level obtained from the muscle in the same assessment (Winter and Scott, 1991). This facilitates physiological interpretation of signal amplitude, and allows for comparison between performance on different test days and between individuals (Lehman and McGill, 1999). EMG signals can be normalised to either a maximal or sub-maximal reference. The first method expresses the amplitude of each point in a signal as a percentage of the mean or peak amplitude of a maximal test of muscle strength. It is referred to as the MVC method (Yang and Winter, 1984). Sub-maximal normalisation references include 1) activities that isolate a muscle’s function, such as an anti-gravity hold (Dankaerts et al., 2004, Mercer et al., 2009), 2) the peak RMS or AMV amplitude in the same activity, known as the Peak Dynamic Method (PDM) (Burden et al., 2003), or 3) the mean RMS or ARV amplitude during the same activity, namely the Mean Dynamic Method (MDM) (Burden et al., 2003). The most valid method of normalisation has not yet been determined (Soderberg and Knutson, 2000).

The advantage of the MVC method is its provision of a meaningful physiological reference (Allison et al., 1998). However, its validity depends largely on the extent to which the intensity of the MVC is a true maximum. There are concerns that the output of MVC can be up to 20–30% less than the muscle’s actual force generation capability (Merletti, 1997). The methods used to obtain MVC vary between studies, with some studies using the EMG signal as biofeedback to maximise the effort given by participants during the test (Benoit et al., 2003). Meldrum et al. (2003) found higher reliability of MVC when a more experienced assessor carried out the tests, which suggested a training effect.

Test-retest reliability of EMG signals from MVC testing has been shown to be high in healthy people, with standard error of measurement (SEM) values in the range of 1.1–6.4% of the mean (Rainoldi et al., 2001). Its application in neurological populations poses some difficulty. People with neurological impairment may not reliably produce MVC due to their motor impairment (Perry, 1992). A recent study found that peak moments at the ankle joint during gait exceeded the maximal isometric strength of the ankle plantarflexors in adolescents with CP (Dallmeijer et al., 2011). The authors suggested that the passive component of the muscle-tendon unit of the triceps surae contributed to torque
generation during gait (Dallmeijer et al., 2011). The finding could also be explained by impaired ability to selectively activate specific muscles in a voluntary isometric test, as shown in CP by Tedroff et al. (2006). A further potential disadvantage of the MVC method is that the contributions of weaker, less effective muscles during gait could be exaggerated because these muscles will appear to contract at a higher percentage of a low maximum (Damiano et al., 2006). There are also concerns about the validity of an isometric test as a normalisation reference, given that the majority of muscle activity in gait is concentric or eccentric (Burden et al., 2003).

The validity of another commonly used alternative, PDM, has not been tested in a neurological population. Its main disadvantage is that the amount of activation cannot be related to any physiological measure, therefore inability to contract a muscle due to pain inhibition or neuromuscular dysfunction may not be detected (Benoit et al., 2003). Variation between individuals may also be diluted using this method (Hsu et al., 2006). However, differences between injured and non-injured limbs were found in EMG amplitude and timing during gait in participants with anterior cruciate ligament deficiency when the PDM normalisation method was used (Benoit et al., 2003). This suggested that the PDM method did not entirely dilute variation in muscular output.

In the current study, the purpose of measuring EMG amplitude was to examine each participant's ability to scale a muscle's output to the demands of the tasks of gait. Normalisation was considered critical to ensure comparison of the same individuals in test-retest situations and to compare the participants in their groups as a whole. In the absence of any definitive evidence on the validity and reliability of normalisation in a neurological population in the study of gait, it was decided to compare both PDM and MVC normalisation methods for test-retest reliability. The method with better reliability would then be used for further analysis. The results of this reliability study are presented in Chapter 7.

5.4.4 Extraction of clinically relevant parameters from EMG data

The EMG signal itself is a series of spikes of varying amplitude and frequency. The processing methods described above, namely filtering, amplitude processing and amplitude normalisation, ensure that the signals can be compared between repeated tests of the same participant and groups of participants. While it is of interest to interpret the signals as a whole over the course of the GC, there are key parameters contained within the signal that represent the nature of the underlying impairment from a physiological point of view (Lamontagne, 2006). This section will address the extraction of clinically relevant parameters that were required to fulfil the aims and objectives outlined in Chapter 4.
5.4.4.1 Determination of the timing of muscle activity during gait

Measurement of the timing and duration of muscle activity bursts is of significant clinical interest in the evaluation of gait impairment. The EMG signal obtained during gait is a continuous sequence of electrical activity, with baseline periods alternating with bursts of activity. The presence of a burst of activity implies that the muscle is contracting with sufficient intensity to contribute to the kinematic and kinetic features of the GC. Temporal analysis of the EMG signal therefore dichotomises the EMG signal into either “activity”, characterised by a sustained series of spikes in the signal, or “baseline”, characterised by a steady state of relatively unchanging background activity, as illustrated in Figure 5.11.
Changes in temporal activation have been associated with a number of clinical conditions and can provide insight into the motor control strategies used by patients (Solnik et al., 2010). As a consequence, accurate distinction of periods of muscle activity from the baseline signal is of particular importance in EMG analysis. EMG signals will vary due to environmental and technical factors, and as such there is no standard reference threshold by which to determine muscle activity from the EMG signal. In general, four methods can be considered: 1) visual determination, 2) defined thresholds, such as a number of standard deviations (SD) above baseline (Hodges and Bui, 1996) or a percentage of maximum contraction (Johanson and Radtka, 2006), 3) computerised statistical methods, such as the approximated generalised likelihood ratio (AGLR) (Staude and Wolf, 1999, Roetenberg et al., 2003), and 4) amplification of the SNR using an off-line equation, such as the Teager-Kaiser Energy Operator (TKEO) (Li et al., 2007), to increase detection capability.

Visual determination by a trained observer is regularly used in day-to-day clinical practice, however, for research purposes, limitations arise with the possibility of observer bias and the excessive time required to process multiple trials (Solnik et al., 2010). Previous studies have used threshold criteria to determine onset and offset of muscle activity. Hodges and Bui (1996) defined the onset of activity using thresholds of one, two and three SDs above the mean of a 50 ms baseline signal lasting for a minimum of 20, 50 or 100 ms. A threshold of 15–18% MVC was recommended in another study (Johanson and Radtka, 2006). These threshold criteria are arbitrary and no consensus has been
reached in the literature regarding the optimal threshold parameters (Roetenberg et al., 2003).

In recent years, mathematical and statistical models have been employed to improve the accuracy of activity detection and reduce the time required to visually process the signals. Two main methods have emerged, the AGLR (Staude and Wolf, 1999, Roetenberg et al., 2003) and TKEO-based methods (Li et al., 2007, Solnik et al., 2010). AGLR uses a log-likelihood ratio calculation to determine the statistical probability of muscle contraction based on probability density functions of the null hypothesis $H_0$ (no change in the EMG signal) and the alternate hypothesis $H_1$ (a change in the signal). A timed determinant window of fixed length 30 ms is moved along the data set, and the hypothesis is tested at each window. An alarm time is signalled when $H_1$ is satisfied (Roetenberg et al., 2003). The use of AGLR to define onset and offset times was more accurate than threshold criteria when applied to the EMG data from multiple GCs (Roetenberg et al., 2003, Solnik et al., 2010). However, the detection of activity requires a distinct change in the log-likelihood ratio from one 30 ms sample to the next, meaning that detection may be more difficult where the change in amplitude is less obvious. Both AGLR and the threshold (SD) method showed an increase in detection latencies as background noise within the EMG signal increased, though the AGLR method was less affected (Lee et al., 2007). Individuals with gait impairment may show high baseline activity and have lower amplitude bursts of activity (Lauer and Prosser, 2009). This can mimic the background noise examined by Lee et al. (2007), and could result in greater inaccuracies in burst activity detection for such populations, even with the more robust AGLR method.

The TKEO was originally developed to compute the energy of sound. It was subsequently proposed as a method to treat EMG signals and aid in the detection of muscle activity by ensuring that both frequency and amplitude information from the EMG signal were incorporated into the detection process (Li et al., 2007). The discrete TKEO $\Psi$ is defined in the time domain for signal amplitude $\chi$ at time point $n$ as follows (Li et al., 2007):

Equation 1: $\Psi(\chi_n) = \chi_n^2 - (\chi_{n+1})(\chi_{n-1})$

The TKEO is directly proportional to both the amplitude and the frequency of the input signal, and therefore it creates a greater distinction between muscle activity and baseline activity than methods based on signal amplitude alone (Li et al., 2007, Lauer and Prosser, 2009). Both AGLR and TKEO out-performed an arbitrary threshold when used with simulated EMG data (Li et al., 2007). The addition of TKEO to the previous three methods of burst detection (visual, threshold, and AGLR) improved the accuracy of all three methods (Solnik et al., 2010). Application of the TKEO to EMG signals from children with CP improved the detection of activity compared to a SD threshold method, despite the high levels of tonic baseline activity in the signals (Lauer and Prosser, 2009). This
improvement in activity detection is thought to be due to the incorporation of frequency information into the detection process. Recent studies have shown that the frequency component of EMG is a particularly sensitive measure in detecting differences between groups or following intervention (Lauer et al., 2007a, Lauer et al., 2007b, Wakeling et al., 2007).

Based on this evidence, it was decided to apply the TKEO equation to the filtered EMG signals to enhance the detection of muscle activity during gait in the current study. The TKEO-treated signals were then smoothed with a second order, 50 Hz Butterworth low-pass filter to avoid errors in activity designation due to the rapid variations in the unsmoothed signal (Solnik et al., 2010). The periods of muscle activation were visually clearer in the TKEO-modified signal than in the original signal as expected. Figure 5.12 shows an example.
The next step in the process was to determine a TKEO-based threshold that would signal a period of muscle activation and then signal its cessation. Previous authors using the TKEO had described the use of a threshold based on a resting baseline. With this method, EMG signals from a muscle at rest were treated with the TKEO equation, and a threshold $T$ was determined using the following equation based on the mean, $\mu$, a constant, $\eta$, and the standard deviation, $\delta$, of the resting trial. This method will be referred to as the Resting Threshold Method (RTM).

\[ T = \mu + \eta \delta \]

**Figure 5.12: Effect of the Teager-Kaiser Energy Operator on an EMG signal of tibialis anterior**

$TKEO = \text{Teager Kaiser Energy Operator, } ms = \text{milliseconds, } VHz^2 = \text{Volts times Hertz squared (units of TKEO)}$
Equation 2: \( T = \mu + \eta \cdot \delta \)

The value of \( \eta \) varied among the studies, from 1 (Lauer and Prosser, 2009), 6–8 (Li et al., 2007), and up to 15 (Solnik et al., 2010). No clear guidelines were available on the optimum value of \( \eta \), as the studies had not reported the validity of a range of values.

In relation to the CSM population, there were concerns regarding the validity of a threshold based on resting EMG activity. Variable resting tone could lead to difficulty in determining an optimum threshold for the participant group as a whole. Given the wide range of impairment among people with CSM, it was expected that participants would vary in the relationship between the amplitude of their EMG signals at rest and during activity. In the current study, preliminary tests were carried out to assess the applicability of the RTM algorithm to EMG signals from the CSM participants. It was found that the algorithm designated activation times that were not always consistent with visual interpretation. A second activation detection method was therefore required for comparison, to determine if it was possible to achieve more consistent parameters for the designation of activation while avoiding the need for visual analysis of all signals.

To develop a new activation detection method, the characteristics of a TKEO-treated EMG signal from a GC were observed and analysed during bursts of activity and outside of these bursts to determine the parameters that might signify a burst of activity. During a visually determined activity burst, the TKEO amplitude was higher, and more rapidly changing, than during periods outside of these bursts. It was hypothesised that measures of TKEO amplitude and rate of change, measured by the slope of the relationship between TKEO amplitude and time, could be combined to create an alternative threshold for muscle activation. Amplitude could be determined by expressing a given point in the TKEO as a percentage of its maximum activity during the same GC. The change in TKEO amplitude between two successive data points could be described by measuring the slope of the line between the amplitudes of those two points.

To determine more exact parameters, signals from each of the four muscles, taken from five participants with CSM during gait of varying mobility impairment, were examined for slope between successive time points and TKEO amplitude at each time point. During periods when visual interpretation of the signal indicated that the muscle was not active, the amplitude of the smoothed TKEO varied from 0.1–1.5% of its maximum amplitude during that GC, and the slope between the amplitude at successive time points varied from 3x10\(^{-5}\) to 2x10\(^{-4}\) VHz\(^2\)/ms. During activation, amplitude exceeded at least 6% of the maximum amplitude during that GC, while the slope of a line between the amplitude of successive time points ranged from 5x10\(^{-5}\) to 0.7 VHz\(^2\)/ms. A novel “double threshold method” (DTM) was developed in the current study to signal activation when the
smoothed TKEO-treated signal exceeded 3% of its maximum amplitude during that GC and when the slope between two successive time points exceeded $10^{-6}$ VHz$^2$/ms.

### 5.4.4.2 Validation of a method to detect the timing of muscle activation

Validation of any new method requires comparison to a “gold standard”, which should be a widely accepted and proven method of performing the same task. There is no gold standard method to determine the timing of muscle activation from EMG signals. In clinical practice, visual interpretation is the most widely used method. It was previously used as a comparison in other validation studies of TKEO-based methods (Solnik et al., 2010). A study was conducted to find the method that showed greatest agreement with visual interpretation. Two methods were compared, the RTM and the newly developed DTM.

A subset of data, comprising the right TA signals from one GC of 12 participants with CSM, was used in the validation process. TA was chosen because it was the most superficial of the four muscles tested, and therefore the least likely to be affected by cross talk. To implement RTM, a sample of EMG data of one second’s duration, taken from the right TA at rest, was extracted using MATLAB. In some signals, the amplification gain had been altered from the resting trial to the gait trials. If this had occurred, the raw EMG data from the resting trial were scaled to the gain used in the gait trials, using a scaling system designed for the MA-300 shown in Appendix 5.3. The TKEO was then applied to the signals using a custom-built routine in MATLAB, detailed in Appendix 5.4. The mean and SD of the TKEO in the resting trial were obtained and three thresholds were determined, 1) one SD above the mean (Lauer and Prosser, 2009), 2) seven SDs above the mean (Li et al., 2007), and 3) 15 SDs above the mean (Solnik et al., 2010).

The filtered EMG signals from Section 5.4.3.1 were then treated with TKEO. The resulting output was smoothed using a second order, 50 Hz Butterworth filter as per Solnik et al. (2010). Using a MATLAB routine, shown in Appendix 5.5, the TKEO amplitude over the GC was compared to the activation threshold at each point in the signal. A window 25 ms in length was moved along each successive point in the signal. Activation was signalled at the central point of this window when at least 24 of 25 data points in the window exceeded the threshold. Cessation of activity was signalled when this condition was no longer satisfied. The resulting output of each threshold was then stored in MATLAB for later comparison with visual interpretation.

DTM was then implemented for each gait trial using a MATLAB routine (Appendix 5.6). The activation threshold consisted of two components, a TKEO amplitude of 3% of the maximum smoothed TKEO amplitude within that trial, and a slope between successive data points of at least $10^{-6}$ VHz$^2$/ms. The threshold was tested using a moving window of
25 ms, with activation signalled when 24 out of the 25 data points exceeded the double threshold.

An independent observer (DM) examined the TKEO-treated signals visually and selected two portions of each signal, one during a clear period of activation and the other, during a clear period of inactivity. The MATLAB routines for both RTM (Appendix 5.5) and DTM (Appendix 5.6) then tested the outputs of the four chosen thresholds over these visually designated “on” and “off” periods. The validation test was set as an all-or-none result, in that the computerised methods were required to signal an “on” phase for the duration of the “on” portion designated by visual observation, and likewise for the “off” portion. Figure 5.13 shows the steps in the validation process.
Results of the validation test are shown in Table 5.1. There was considerable variation in the designation of “on” and “off” phases by RTM compared to visual interpretation, depending on the value of $\eta$ used. The DTM method was more accurate, with 87.5% agreement with visual interpretation, compared to 58.33–79.17% with RTM. Figure 5.14
illustrates the difference in “on” and “off” designation between two participants’ TA signals using these methods. Part A and B show an over-estimation of activity duration in both participants. Part C shows that RTM with a $\gamma_1$ value of 15 achieved a higher level of accuracy in Participant 1, but over-estimated activity in Participant 18. As seen in Part D, DTM designated activity periods that were more consistent with visual interpretation.

Table 5.1: Agreement between visual designation of “on” and “off” phases and four computerised timing methods

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| Total agreement (%) | 58.33 | 75.00 | 79.17 | 87.50 |

RTM, resting threshold method, DTM, double threshold method
RTM thresholds for activation were 1, 7 and 15 standard deviations above mean resting TKEO, termed RTM-1, RTM-7 and RTM-15 respectively.
A value of 1 indicates that the computerised method agreed with visual designation for the duration of that “on” or “off” portion, and a value of 0 indicates a discrepancy.
Figure 5.14: Timing profiles of right tibialis anterior determined by four algorithms for two participants

VHz² = Volts times Hertz squared, TKEO = Teager Kaiser Energy Operator

The raw TKEO (grey trace) was smoothed with a second order 50 Hz low pass Butterworth filter (black trace) to aid detection of activation.

Red line illustrates the designation of timing by the tested algorithm, with a value of 1 indicating activity and 0 indicating baseline.
The validation study therefore found that, when a combined slope and amplitude activation threshold was applied to the TKEO-modified signal, it identified bursts of muscle activation that broadly corresponded with those designated by visual inspection. The activation threshold was set as a percentage of the maximum amplitude of the smoothed TKEO during that same GC, rather than as a number of SDs above a mean resting level as in RTM. This may have minimised the effect of variation in this resting baseline in the designation of activity. The addition of the slope threshold was designed to make the algorithm more specific to neurological problems, where there may be relatively high baseline signal and low peak amplitude during meaningful bursts of muscle activity. The requirement for the slope of the smoothed signal to exceed a threshold ensures that an unchanging baseline, even if abnormally high in amplitude, would not be designated as activation.

The lack of a gold standard in distinguishing muscle activation from baseline signal during gait is a significant limitation in the development of computerised methods for this task. Clinically, visual observation is considered the optimal method of interpreting all aspects of EMG and is the standard against which computerised methods are assessed (Solnik et al., 2010). However, it is inherently subjective, and variation in the EMG signal that is not visible to the naked eye may be missed. Visual interpretation in research is limited by the excessive time needed to process large numbers of signals. A computerised method is therefore desirable for speed and objectivity, but has the disadvantage that it dichotomises the fine detail within the EMG signal into a simple “on” or “off”. The DTM method was the most consistent of the four methods in the designation of activity, but there were still discrepancies between its designation and that of visual observation in three cases. It was interesting that the “on” phase designated by the independent observer in two signals, cases 3 and 6, agreed with none of the computerised methods. On inspection of these signals, it was found that beginning of the visually designated “on” phase occurred just after a short (<20 ms) period of inactivity. All four algorithms required that the threshold be exceeded for at least 24 ms of a 25 ms window. This may have introduced latency in detecting a rapid change in the signal that would have been visible on visual inspection. However, removing the requirement for a 25 ms window might reduce the specificity of the detection algorithm, in that short insignificant spikes might be incorrectly designated as meaningful activity.

It was concluded that the DTM showed best agreement with visual interpretation of muscle activation timing. This method was therefore selected to calculate timing parameters for EMG signals in the study.

5.4.4.3 Co-activation

Gait disturbances can be associated with impaired selective activation of functionally antagonist muscles due to pain inhibition, weakness, or changes in muscle tone, as
discussed in Chapter 4. Determination of co-activation from EMG data depends on the accuracy of the timing data extracted in Section 5.4.4.2. Co-activation between two muscles is reported as the percentage of the GC for which both are simultaneously active. For the current study, two antagonistic pairs were examined, BF and RF as one pair, and TA and MG as the second pair.

5.4.4.4 Interpretation of the intensity of muscle activation

In people with neurological impairment, the representation of an EMG signal into its temporal components of “on” and “off” is insufficient to fully represent a person’s ability to scale motor output to the demands of a task (Roetenberg et al., 2003). The intensity of a muscle’s activation during a burst is also of importance, as it gives an indication of the force produced (Bogey et al., 2005). Three parameters, peak ARV amplitude, timing of this peak, and area under the ARV curve during an activity burst, have been examined for repeatability in HCs with deliberate slight variations in electrode placement (Campanini et al., 2007). Burst durations in Campanini’s study were not extracted for individual GCs, but instead they were pre-determined based on data from a healthy population (Perry, 1992). These activation phases could not be assumed to be accurate in people with pathological gait.

Another study used three amplitude parameters, maximum and median ARV during bursts of activation, and median ARV during baseline phases, to measure the intensity of activation in healthy people and people with CP and stroke (Roetenberg et al., 2003). Bursts of activation were determined using a computerised method, AGLR (see Section 5.4.4.1). Roetenberg’s method was considered to have face validity in the evaluation of gait in CSM, as it was important to obtain a measure of activation intensity during the “off” (baseline) and “on” (activity) phases to fully characterise a muscle’s activation pattern.

A literature search found no studies on the test-retest reliability of these parameters. Regarding validity, one study found significant differences in mean amplitudes during activity bursts of the erector spinae, vastus lateralis, tibialis anterior and gluteal muscles in people with stroke while walking with different gait aids (Buurke et al., 2005). This suggested that the mean amplitude within a burst was a valid measure of the intensity of muscle activation and was responsive to change under varying gait conditions. In the absence of a reliability study on these amplitude parameters, it was decided to examine the test-retest reliability of 1) maximum amplitude during a burst of activity, 2) mean amplitude during a burst of activity, and 3) mean amplitude of the baseline or “off” signal during gait. As the data would be used to compare between individuals and different test days, normalisation of the amplitude parameters was required. The parameters were normalised using both MVC and PDM methods, and the method with higher reliability was then for further use in the thesis.
5.4.4.5 Evaluation of muscle response to lengthening

The potential value of a locomotor-specific measure of spasticity (LSMS) was discussed in Chapter 4, Section 4.2.2.3. This measure was developed to evaluate muscle lengthening during gait in a cohort of children with CP (Crenna, 1998, Crenna, 1999), and then applied to an adult stroke population (Lamontagne et al., 2001). The LSMS in these studies extracted the phases in the GC when a muscle was lengthening, and examined the relationship between lengthening velocity and EMG amplitude.

The method used by Crenna to determine muscle length differed somewhat from the method of Lamontagne. Crenna (1999) measured muscle length in millimetres by means of an anthropometric model for muscle kinematics. Lamontagne et al. (2001) used an equation based on the underlying joint kinematics to assess relative muscle length over the GC, validated in a previous study (Winter and Scott, 1991). Muscle length was compared relative to its length in the anatomical position where hip, knee and ankle joints were in neutral in all planes. Its lengthening velocity was then computed as the rate of change of relative muscle length over time. Lamontagne’s method was adopted for use in this study as it avoided the need for a separate anthropometric model and could be carried out using Vicon PiG®. The lack of actual units of muscle length measurement was not considered a disadvantage, as the hypothesis was concerned with the relationships between relative length, lengthening velocity and EMG output.

The parameters used to evaluate the relationship between muscle length and EMG output also needed consideration. The development studies had described two approaches, 1) determination of the lengthening velocity threshold (LVT) for EMG recruitment (Crenna, 1999), and 2) measurement of the slope between lengthening velocity and EMG amplitude, such that a positive slope would indicate spasticity and its gradient would estimate the reflex gain (Lamontagne et al., 2001). Both approaches have been shown by their respective authors to be valid in discriminating pathological responses to lengthening from normal responses, though they have not been compared in the same population. It was considered that all three measures, 1) the LVT at which onset of EMG activity occurred, 2) the presence or absence of a positive slope, and 3) the gradient of the slope, would be of clinical benefit in distinguishing spasticity during locomotion in the CSM population. The three methods were therefore adopted for use in the current study. It was also considered that the timing of onset of EMG activity during a lengthening contraction would be of clinical importance, as it would indicate whether an abnormal response to lengthening contributed to a change in the timing of activation. The implementation of these measures in the cross-sectional and experimental studies would depend on their test-retest reliability. These results will be reported in Chapter 7.

Some muscles undergo two or more periods of lengthening during the GC. To focus the analysis, it was decided to examine one key lengthening phase for each muscle. These
phases were 1) BF in terminal swing, 2) MG in mid stance, 3) TA in pre swing and 4) RF in pre swing. These phases were chosen because abnormal EMG activity by the lengthening muscles would be disruptive to the GC during these periods of lengthening. Furthermore, pathological responses to lengthening had been shown in other neurological populations to affect MG in mid stance (Lamontagne et al., 2001) and the hamstrings at terminal swing (Crenna, 1999), whereas other lengthening phases, such as that of MG in initial swing, did not show such responses (Lamontagne et al., 2001). It was considered that the analysis of one key phase of lengthening for each muscle would be sufficient to address the research question of whether spasticity was a contributory factor to gait impairment in CSM.

5.5 Secondary outcome measures

In addition to analysis of gait, quantification of disease severity, functional mobility, health-related quality of life (HRQOL) and spasticity were required as secondary outcome measures. The following sections describe the selection of the most suitable scales.

5.5.1 Quantification of disease severity

A number of different scales to quantify the severity of CSM have been developed. A review of the literature identified eight scales, namely the Nurick scale (Nurick, 1972), Japanese Orthopaedic Association (JOA) scale (Japanese Orthopaedic Association, 1994), Cooper myelopathy scale (Cooper and Epstein, 1985), European Myelopathy score (EMS) (Herdmann et al., 1994), Prolo score (Prolo et al., 1986), Myelopathy Disability Index (Casey et al., 1996), Ranawat score (Ranawat et al., 1979), and the Harsh scale (Harsh et al., 1987). Different scales emerged because the clinical symptoms of CSM have resulted in a variety of treatment methods, leading to the development of outcome measures specifically designed to evaluate these methods (Vitzthum and Dalitz, 2007). The most appropriate measure of severity for the current study was selected from a literature review on the reliability and validity of the available scales.

5.5.1.1 Selection of the optimal scale of CSM severity

Vitzthum and Dalitz (2007) conducted a retrospective comparison of five scoring systems used for CSM, the Nurick score, the JOA score, the Cooper myelopathy score, the Prolo score, and the EMS. All five scores were found to be suitable for quantitatively assessing the clinical characteristics and progression of cervical myelopathy. Furthermore, all scales showed a significant correlation with one another using Spearman’s rho, with the significance of the correlations ranging from less than 0.05 (correlation between Cooper and Prolo scales) to less than 0.0001 (correlations between Nurick, JOA, lower limb Cooper scale and EMS). Actual values of Spearman’s rho were not given in the original
The authors concluded that, because all the scales were found to be valid and responsive to change, it was not necessary to develop a new or universal score.

It was concluded from the comparison study of Vitzthum and Dalitz (2007) and the original development studies that, as far as had been examined in the literature, no one particular scale showed superior validity or reliability. The Nurick and JOA scales were observed in the literature to be most commonly used in published reports, and were used in a recent multi-centre study of outcomes following surgery for CSM (Furlan et al., 2011). Comparison of participants of the current study to a wider cohort of people with CSM was desirable. For this reason, the Nurick and JOA were selected as measures of severity.

### 5.5.1.2 Nurick scale

The Nurick scale, detailed in Table 5.2, is the oldest outcome measure for CSM. It focuses on gait function and is therefore unable to detect changes in the upper extremity. A better-functioning score on Nurick was strongly associated with a higher score on the RAND Medical Outcomes Study 36-item Short Form Health Survey (SF36) (King and Roberts, 2002), indicating its validity. No studies of reliability were found. A limitation of Nurick is the influence of a person’s occupation on the score. Progression between a score of 2 and 3 on the scale may depend on the nature of a person’s employment rather than on the severity of neurological deficit. Its focus on gait impairment was however desirable because gait was the subject of the current study’s research and the scale was therefore immediately applicable to this cohort.

**Table 5.2: Nurick scale**

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Signs and symptoms of root involvement, but without evidence of spinal cord disease</td>
</tr>
<tr>
<td>1</td>
<td>Signs of spinal cord disease but no difficulty in walking</td>
</tr>
<tr>
<td>2</td>
<td>Slight difficulty in walking which did not prevent full time employment</td>
</tr>
<tr>
<td>3</td>
<td>Difficulty in walking which prevented full time employment or the ability to do all housework, but which was not so severe as to require someone’s help to walk</td>
</tr>
<tr>
<td>4</td>
<td>Able to walk only with someone else’s help or with the aid of a frame</td>
</tr>
<tr>
<td>5</td>
<td>Chairbound or bedridden</td>
</tr>
</tbody>
</table>

### 5.5.1.3 Japanese Orthopaedic Association scale

The JOA score evaluates severity of myelopathy across four categories, motor dysfunction in the upper extremity, motor dysfunction in the lower extremity, sensory deficit, and sphincter dysfunction (Japanese Orthopaedic Association, 1975). Problems
were identified with the original scoring system, including the ranking of each category, the ratio of the score for categories, and the absence of a category for motor function of the shoulder and elbow (Yonenobu et al., 2001). The scoring system was revised in 1994 to address these limitations (Japanese Orthopaedic Association, 1994). The inter- and intra-rater reliability of the new scoring system were found to be high, with intraclass correlation coefficients (ICCs) of 0.813 and 0.826, respectively (Yonenobu et al., 2001). King et al. (2003) found that the JOA moderately correlated with the SF36 in people with CSM. There were higher correlations between the SF36 and Nurick, Harsh, and Cooper scales, however this was attributed to the broader number of categories of dysfunction that the JOA assesses. When the lower limb sub-section of the JOA was evaluated as a separate entity, it showed strong correlation with the SF36 (King and Roberts, 2002). The JOA provides a wider assessment of CSM severity than most of the other scales as it includes both sensory and motor function of the upper and lower limbs, as well as sphincter dysfunction. The EMS assesses severity of myelopathy across similar domains (Herdmann et al., 1994), however a literature search using Medline found that it had fewer citations than the JOA.

The JOA score was modified for use in Western populations, and there are two modifications. One version by Keller et al. (1993) removed the reference to the ability to eat with chopsticks from the original text and replaced it with a more generic description of upper limb function. A second modification to the original JOA changed the reference to the ability to use chopsticks to the use of Western feeding utensils (Chiles et al., 1999). In the absence of any pre-existing comparison of the validity and reliability of the two Western modifications, the JOA as modified by Chiles et al. (1999) was favoured above the earlier modification of Keller et al. (1993), because its content was considered to show greater similarity to the original JOA score that had been validated in previous studies (Yonenobu et al., 2001). Table 5.3 shows the modified JOA (mJOA) (Chiles et al., 1999).
Table 5.3: Modified Japanese Orthopaedic Association (mJOA) myelopathy score (Chiles et al., 1999)

<table>
<thead>
<tr>
<th>I. Motor Dysfunction of the Upper Extremity</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Unable to feed oneself</td>
</tr>
<tr>
<td>1</td>
<td>Unable to use knife and fork, able to eat with a spoon</td>
</tr>
<tr>
<td>2</td>
<td>Able to use knife and fork with much difficulty</td>
</tr>
<tr>
<td>3</td>
<td>Able to use knife and fork with slight difficulty</td>
</tr>
<tr>
<td>4</td>
<td>No dysfunction</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>II. Motor dysfunction of the Lower Extremity</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Unable to walk</td>
</tr>
<tr>
<td>1</td>
<td>Can walk on flat floor with walking aid</td>
</tr>
<tr>
<td>2</td>
<td>Can walk up and / or down stairs with hand rail</td>
</tr>
<tr>
<td>3</td>
<td>Lack of stability or smooth gait</td>
</tr>
<tr>
<td>4</td>
<td>No dysfunction</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>III (a). Sensory deficit, upper extremity</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Severe sensory loss or pain</td>
</tr>
<tr>
<td>1</td>
<td>Mild sensory loss or pain</td>
</tr>
<tr>
<td>2</td>
<td>No dysfunction</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>III (b). Sensory deficit, lower extremity</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Severe sensory loss or pain</td>
</tr>
<tr>
<td>1</td>
<td>Mild sensory loss or pain</td>
</tr>
<tr>
<td>2</td>
<td>No dysfunction</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>III (c). Sensory deficit, trunk</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Severe sensory loss or pain</td>
</tr>
<tr>
<td>1</td>
<td>Mild sensory loss or pain</td>
</tr>
<tr>
<td>2</td>
<td>No dysfunction</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>IV. Bladder function</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Unable to void</td>
</tr>
<tr>
<td>1</td>
<td>Marked difficulty in micturition (retention)</td>
</tr>
<tr>
<td>2</td>
<td>Difficulty in micturition (frequency, hesitation)</td>
</tr>
<tr>
<td>3</td>
<td>No dysfunction</td>
</tr>
</tbody>
</table>
5.5.2 Measurement of health-related quality of life

The World Health Organisation’s International Classification of Functioning, Disability and Health (ICF) recommended the measurement of health-related quality of life (HRQOL), or participation restriction, to ensure that a complete profile of an individual’s health state is measured (World Health Organisation, 2001). To fulfil this requirement in this study, an outcome measure of HRQOL that had been validated for CSM was needed.

The SF36 been validated for use in CSM by King and Roberts (2002), who confirmed its construct validity and internal consistency in this population. People with CSM exhibited significantly lower scores across all domains of the SF36 compared to a gender and age matched population, indicating poorer HRQOL (King et al., 2003). The results showed that people with CSM experienced problems in HRQOL that extended beyond the realm of physical deficits and included diminished emotional functioning and mental health. The authors recommended that the SF36 be used in conjunction with a disease-specific outcome measure for CSM such as the Nurick or mJOA to provide a complete picture, given that the SF36 was not designed specifically for CSM and may not be sensitive to its particular set of deficits. The applicability and reliability of the SF36, and its validity in this patient group, makes it the measure of choice for our study. Normal values for the SF36 in the Irish population have been collated (Blake et al., 2000), allowing retrospective comparison with the CSM population in the current study relative to normative data. In the current study, the SF36 was scored using the guidelines of (Hays et al., 1993).

5.5.3 Quantification of functional mobility

Assessment of gait impairment using kinematics, kinetics and EMG provides valuable information on the biomechanics and neuromuscular control of the locomotor system, but it does not measure the impact of a gait deficit on an individual’s functional mobility. The Modified Rivermead Mobility Index (MRMI), shown in Table 5.4, is an ordinal scale incorporating eight items related to functional mobility, with each item scored from 0 to 5 depending on ability to perform the task with varying levels of assistance. Validity and reliability of the MRMI has been established in people with stroke (Lennon and Johnson, 2000) and in a general neurological population in a study that reported an ICC for inter-rater reliability of 0.93 and a Spearman’s rho correlation coefficient with the 10m timed walk test of 0.86 (Walsh et al., 2010). It has not been validated specifically for use in CSM, however it has been used to measure of functional mobility in adults with cervical or thoracic SCI (Opara et al., 2007). Its validity and reliability in a mixed neurological population (Walsh et al., 2010) suggested that it should transfer to CSM. Its face and content validity were considered appropriate for measurement of functional mobility in this population.
Table 5.4: The Modified Rivermead Mobility Index

<table>
<thead>
<tr>
<th>Item</th>
<th>Task</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Turning Over.</td>
<td>R L</td>
</tr>
<tr>
<td></td>
<td>Please turn over from your back to your side.</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Lying to Sitting.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Please sit up on the edge of the bed.</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Sitting Balance.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Please sit on the edge of the bed (10 seconds).</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Sitting to Standing.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Please stand up from your chair (&lt;15 seconds).</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Standing.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Please remain standing (10 seconds).</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Transfers.</td>
<td>R L</td>
</tr>
<tr>
<td></td>
<td>Please go from the plinth to the chair and back again.</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Walking Indoors.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Please walk 10 metres in your usual way.</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Stairs.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Please climb up and down this flight of stairs in your usual way.</td>
<td></td>
</tr>
<tr>
<td>TOTAL</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

SCORING SYSTEM

0  Unable to perform
1  Assistance of two people required
2  Assistance of one person required
3  Requires supervision or verbal instruction
4  Requires an aid or an appliance
5  Independent

5.5.4 Quantification of spasticity

CSM is associated with increased muscle tone as one of the features of upper motor neurone pathology. There is considerable debate in the literature regarding the precise nature of spasticity (Pandyan et al., 2005). It has been defined as “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 neurone syndrome” (Lance, 1980), and as “disordered sensori-motor control, resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles” (Pandyan et al., 2005). These two definitions present
somewhat different views of the concept, and illustrate the difficulty in capturing the nature of such a complex phenomenon. As discussed in Chapter 4, the use of a locomotor-based measure of muscle activity during lengthening was a key objective of the study of gait in this project. However, the inclusion of a clinical measure of spasticity was considered advantageous, as this forms part of the regular clinical objective assessment of CSM.

The Ashworth scale was developed in 1964 to grade the resistance encountered in a muscle during passive stretch (Ashworth, 1964). It was modified in 1987, and inter-rater reliability of this modified scale, now known as the Modified Ashworth Scale (MAS), was demonstrated (Bohannon and Smith, 1987). It is the most widely used clinical scale of spasticity (Ansari et al., 2008).

Table 5.5: Modified Ashworth Scale.

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

Several studies have since been carried out on its reliability and validity, with conflicting results (Ansari et al., 2008). One study showed it to be reliable for all muscle groups except the ankle plantarflexors (Gregson et al., 2000), while another found poor inter-rater reliability for the elbow flexor and knee extensor muscles, with kappa values of 0.20 (Fleuren et al., 2010). The validity of the MAS has also been questioned. The lower grades, 1, 1+, and 2 did not correspond with surface EMG readings of the elbow flexors in a trial of botulinum toxin efficacy (Pandyan et al., 2002). A further study found poor correlations between MAS scores and muscle activity in response to stretch, measured using root-mean-square EMG amplitudes, indicating that the MAS demonstrated insufficient construct validity for use as a measurement of spasticity (Fleuren et al., 2010). MAS scores were confounded by the presence of contracture in people more than three years after a stroke, such that the MAS could not distinguish between passive (contracture) and active (spasticity) resistance to stretch (Patrick and Ada, 2006). These findings raised considerable questions on what the MAS actually measures, and some
authors have proposed that it should not be used as a measurement of spasticity due to its limitations (Fleuren et al., 2010). However, a scale that demonstrates satisfactory correlation with EMG measurements in SCI has yet to be developed.

Hsieh et al. (2008) carried out a review of a number of scales measuring spasticity in SCI. It was found that the scales measured different aspects of spasticity, and this could account for the poor correlation between individual tools. It was recommended that a battery of outcome measures be used when assessing spasticity in SCI. To date, no single measure of spasticity has been validated for use in the CSM population. The MAS has been used to grade spasticity in a study on gait impairment in CSM (Kuhtz-Buschbeck et al., 1999), however its validity or reliability in this patient group was not examined.

The studies quoted above indicate a lack of consensus in the literature regarding the reliability and validity of the MAS, and also regarding the optimal measurement of spasticity. However, for the current study, it was considered meaningful to include a measure that is familiar to therapists can be carried out easily in the clinical setting. Furthermore, the literature review in Chapter 2 found that an increase in resting muscle tone is generally accepted as an objective feature of CSM. It was of interest to quantify this change in muscle tone so that the features of gait analysis could be related to the clinical presentation. The inclusion of the MAS to measure spasticity, in combination with the surface EMG data, was therefore considered to provide a more comprehensive measure of this phenomenon than the use of either one of these measures in isolation.

5.6 Conclusion

The objectives of the study required further investigation of a number of methodological issues. The evidence relating to the methodology of 3DGA, SEMG, and the secondary outcome variables of CSM severity, quality of life, functional mobility and resting tone, was reviewed, and methods were developed based on a synthesis of the best available evidence. Chapter 6 will describe the application of the methodology to this study.
Chapter 6: General Methods

6.1 Introduction

The processes described in Chapter 5 led to the development of methods to achieve the aims and objectives of the thesis. The project was divided into three studies as outlined in Chapter 4, Section 4.4. There were a number of general methods that were common to the three studies. All required the recruitment of people with CSM, assessment of the severity of CSM, and gait analysis using 3DGA and EMG technology. This chapter will describe these methods. Additional methods unique to the reliability, cross-sectional and experimental studies will be described in Chapters 7, 8 and 9, respectively.

6.2 Ethical considerations

Ethical approval for the thesis and its protocol was obtained from Beaumont Hospital Ethics (Medical Research) Committee in August 2008. Four amendments to the protocol were submitted to the Committee. In November 2008, the Committee granted approval for a second pre-operative 3DGA assessment to fulfil the aims of the reliability study. A second amendment was sought in December 2008 when it came to attention that one of the possible participants to the study was a staff member of Beaumont Hospital, who would normally have been exempt from taking part in research studies. The Committee approved the recruitment of the staff member in January 2009. In March 2009, approval was granted to include patients from the consultant’s rooms at Beaumont Private Clinic. In February 2010, a final amendment was approved to allow the recruitment of healthy controls for the cross-sectional study, the addition of a one-year follow-up assessment, and the extension of the study’s expected completion date to October 2011. The final version of the approval documentation is provided in Appendix 6.1.

6.3 Participants

6.3.1 Identification of people with CSM

Potential participants with CSM were identified at the outpatient neurosurgical Spinal Assessment Clinic and Beaumont Private Clinic at the national department of neurosurgery, Beaumont Hospital. CSM was diagnosed by a registrar or consultant neurosurgeon based on clinical examination and MRI, using the diagnostic criteria listed in Table 6.1. These criteria were in keeping with those used in other studies, for example Fehlings et al. (2008).
Table 6.1: Criteria for diagnosis of CSM by consultant or registrar in neurosurgery

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Description</th>
</tr>
</thead>
</table>
| One of more of the following symptoms | Clumsy hands  
Numb hands and / or feet  
Weakness of lower limbs  
Unsteady gait  
Bilateral upper and / or lower limb paraesthesiae |
| One or more of the following signs | Lower limb spasticity  
Unsteady gait  
Hyperreflexia, including inverted supinator sign or crossed adductor sign  
Upgoing plantar responses  
Clonus  
Weakness in corticospinal distribution |
| MRI evidence of one or more of the following | Cervical cord compression due to spondylosis, e.g. cord impingement or effacement of cerebrospinal fluid  
Signal change within cord |

6.3.2 Inclusion and exclusion criteria

Participants with a diagnosis of symptomatic CSM using the criteria above were included in the study if they were aged over 18 years, able to give informed consent, and could stand independently and mobilise 10 metres without the need for physical assistance of another person, but with aids if required.

Participants who met the inclusion criteria were excluded from the study if they suffered from 1) a co-existing neurological illness with physical deficits that could confound the assessment of CSM, including a history of stroke with physical deficits, 2) symptomatic lower limb osteoarthritis or rheumatoid arthritis affecting gait, or 3) any condition that would have hindered safe testing according to the study protocol, such as unstable cardiac conditions, severe respiratory difficulties, or severe pain. Skin sensitivity was also an exclusion criterion, if it would preclude the application of surface electrodes and reflective joint markers. Participants with a history of symptomatic tandem lumbar spine stenosis were excluded, as the coexistence of neurogenic claudication would confound the assessment of myelopathy (Lee et al., 2008). Finally, participants who had undergone previous surgical intervention for CSM were included in the reliability study only, and not in the cross-sectional comparison study with HCs or in the repeated-measures design post-operative follow-up study.
Inclusion and exclusion criteria were evaluated by reviewing the participant’s medical record, and by telephone interview prior to the scheduling of the first assessment.

### 6.3.3 Recruitment

When a consultant or registrar in neurosurgery confirmed a diagnosis of CSM, the principal investigator (PI) screened the patient’s medical record for inclusion and exclusion criteria. The PI then sent a letter of recruitment to the potential participant in the post. The letter highlighted the broad aims of the study, and is provided in Appendix 6.2. It included a response form with a stamped addressed envelope to facilitate a reply. The PI’s contact details were listed. Participants who indicated their interest in the study were contacted by telephone to discuss their participation and further check for exclusion criteria, and were then recruited if they wished to do so. If no contact was received from a potential participant, a follow-up phone call was conducted a month following the issue of the recruitment letter to determine whether the participant was willing to take part or preferred to decline.

Participants were provided with a Participant Information Leaflet provided in Appendix 6.3. Following the opportunity to ask questions about the study as they wished, they gave their informed consent using the Consent Form provided in Appendix 6.4. General Practitioner (GP) details were recorded, and each GP was sent a letter informing them of their patient’s participation (Appendix 6.5).

### 6.4 Gait cycle classifications and terminology

A number of classifications have been used in the literature to describe the events of a GC (Sutherland et al., 1988, Perry, 1992, Neptune et al., 2001). In the interpretation of a gait disorder, it is important that the terminology be used consistently to avoid confusion between similar, but not necessarily, equivalent terms such as heel strike and initial contact. This thesis adopted the traditional GC divisions of five stance and three swing phase periods, commonly known as the Ranchos classification (Perry, 1992). In this classification, the GC begins with the contact of one foot with the ground and ends when the same foot contacts the ground again. The event of toe off divides the GC into its two main phases, stance and swing.

The stance phase is further divided into initial double support, single limb support, and second double support. These are defined by the events of opposite foot off, referring to toe-off of the contralateral limb, and opposite foot contact, referring to initial contact of the contralateral limb. To describe the tasks of weight acceptance, single limb support and limb advancement, the GC is further subdivided in the Ranchos classification into the following periods of stance, 1) initial contact, 2) loading response, 3) mid stance, 4) terminal stance, and 5) pre swing, and the following periods of swing, 6) initial swing, 7)
mid swing, and 8) terminal swing. These subdivisions are illustrated in Figure 6.1 and described in Table 6.2.

![Figure 6.1: Gait cycle subdivisions](image)

Fig. 6.1 illustrates the lower limb of interest.

IC = initial contact, LR = loading response, GC = gait cycle.

**Table 6.2: Description of gait cycle subdivisions**

<table>
<thead>
<tr>
<th>Phase</th>
<th>Subdivision</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stance</strong></td>
<td>Initial contact</td>
<td>Contact of foot with ground.</td>
</tr>
<tr>
<td></td>
<td>Loading response</td>
<td>Initial contact to opposite foot off. First double support phase.</td>
</tr>
<tr>
<td></td>
<td>Mid stance</td>
<td>Opposite foot off to heel rise of ipsilateral foot. First half of single limb support.</td>
</tr>
<tr>
<td></td>
<td>Terminal stance</td>
<td>Ipsilateral heel rise to opposite foot contact. Second half of single limb support.</td>
</tr>
<tr>
<td></td>
<td>Pre swing</td>
<td>Contralateral foot contact to ipsilateral toe off. Second double support phase.</td>
</tr>
<tr>
<td><strong>Swing</strong></td>
<td>Initial swing</td>
<td>Foot off to point where swinging leg is adjacent to the contralateral stance limb</td>
</tr>
<tr>
<td></td>
<td>Mid swing</td>
<td>Swing leg moves from a position adjacent to contralateral stance limb to a forward position with tibia aligned vertically</td>
</tr>
<tr>
<td></td>
<td>Terminal swing</td>
<td>Tibia vertical position to initial contact</td>
</tr>
</tbody>
</table>
6.5 Clinical assessment of CSM participants

Assessments were conducted for all CSM participants at baseline in the reliability and cross-sectional studies, and at six- and 12-month post-operative follow-up intervals in the experimental study. All assessments were carried out in the Movement Laboratory, RCSI.

6.5.1 Subjective assessment

A subjective assessment was carried out for each participant. The purpose of the subjective assessment was to ascertain 1) duration, severity and nature of myelopathic symptoms, 2) mobility status, including history of falls, 3) medical history and current medications, 4) social history and occupational activities. The information was recorded in a data collection form (Appendix 6.6), for subsequent analysis and coding.

The severity of the participant’s CSM was graded using the Nurick classification (Nurick, 1972) and the mJOA scale (Chiles et al., 1999). Scores were obtained from a structured interview. Scoring systems for these outcome measures are shown in Chapter 5, Table 5.2 and Table 5.3.

6.5.2 Assessment of resting muscle tone

Lower limb tone was measured in CSM participants using the Modified Ashworth Scale (MAS). Participants lay on a standard plinth. The muscle groups tested were hip adductors, hip flexors, quadriceps, hamstrings, and gastrocnemius. Each muscle group was tested by moving the joint through its full available range in one second, which was standardised by counting “one, one thousand” during the time taken to complete a repetition. Five repetitions were performed and the average resistance encountered was taken as the MAS score (Bohannon and Smith, 1987). The scoring system for the MAS is given in Chapter 5, Table 5.5.

6.5.3 Functional mobility

The Modified Rivermead Mobility Index (MRMI) was assessed using a standard plinth and a flight of stairs with one handrail. Participants were given standard instructions according to the MRMI protocol, detailed in Chapter 5, Table 5.4.

6.5.4 Measurement of health-related quality of life

Finally, participants completed the SF36 questionnaire to assess HRQOL. The SF36 also formed part of the standard assessment protocol of the Spinal Assessment Clinic at Beaumont Hospital. If participants had completed the SF36 at clinic within the previous
month, they were not required to complete it again, and the scores were taken from the most recent clinic records.

6.6 Procedure for gait analysis

6.6.1 Overview of equipment

As described in Chapter 5, Section 5.3, 3DGA was conducted using a five-camera VICON ® 250 motion analysis system (VICON, Oxford Metrics Ltd., Oxford, UK) a Kistler multi-component force plate (Kistler Instruments Ltd., Winterthur, Switzerland), and a Motion Lab Systems MA-300 multi-channel surface EMG system (Motion Lab Systems Inc, Baton Rouge, Louisiana, USA). The capture volume was calibrated prior to each session using the manufacturer’s specified static and dynamic calibration procedures. Calibration residuals of less than 2 mm were accepted.

6.6.2 Anthropometric measurements

The measurement of anthropometric parameters followed the standard protocol of the RCSI Movement Laboratory.

Height was measured with a stadiometer as shown in Figure 6.2. The head was positioned in the Frankfurt plane, a standard plane established by an imaginary line passing through the right tragion and the lowest point of the right eye. The vertical distance from the floor to the vertex of the head, its highest point in the mid-sagittal plane, was measured with the participant standing barefoot.
Weight was recorded in kilograms in unsupported barefoot standing using a Seca digital measuring scales.

Leg length was measured with the participant in supine, lower limbs extended and pelvis in neutral. The inferior aspect of each ASIS was located by palpating caudally to cranially on the anterior hip to find its most inferior aspect. Leg length was measured from this point to the inferior aspect of the medial malleolus to the nearest millimetre.

The distance between right and left ASIS was measured from the inferior aspect of each ASIS, located during measurement of leg length, using a tape measure, to the nearest millimetre.

Knee joint width was measured with the participant lying supine with the knees flexed to 75–80°. The lateral epicondyle was located at the lateral aspect of the knee joint line and the popliteal groove just beneath it was marked with a skin surface marker. The midpoint of the medial collateral ligament at its intersection with the knee joint line was marked medially. The distance between the medial and lateral marks was measured with callipers to the nearest millimetre. The average of three measurements was taken as the knee

Figure 6.2: Measurement of height
joint width. Figure 6.3 demonstrates the measurement of knee joint width using the callipers.

Ankle width was measured in supine with knees extended, using callipers, from the most medial point of the medial malleolus to the most lateral point of the lateral malleolus. The average of three measurements was recorded to the nearest millimetre.

Tibial torsion was measured in supine, with knees extended and the hip in neutral rotation. The arms of a gravity inclinometer known as a tibial torsion device were placed on the most medial point of the medial malleolus and on the most lateral point of the lateral malleolus. The tibial torsion device was held upright to allow the indicator arm to hang feely under gravity, and the number of degrees of tibial torsion was read from the dial with the device in this position, as shown in Figure 6.4.
SEMG electrodes were attached to four muscles on each leg, rectus femoris (RF), biceps femoris (BF), tibialis anterior (TA), and the medial head of gastrocnemius (MG). The area of skin in contact with the electrode was prepared by shaving and cleaning with an alcohol swab to reduce skin impedance, and allowed to dry prior to the attachment of the electrode. In participants with particularly dry skin, the area for electrode contact was further treated with gel to eliminate potential static interference with the signal from the skin surface. Electrodes were aligned along the direction of the muscle fibres insofar as this could be determined from their known anatomical orientation.

Placement of the electrodes followed the SENIAM recommendations (Hermens and Freriks, 1997). To confirm the location for each electrode, the area identified by following the SENIAM guidelines was palpated while resisting the muscle’s action. This ensured that each electrode was placed over an area of muscle bulk. Electrodes were secured to the skin using surgical tape.

The specifications used in the detection, recording, amplification and analogue-to-digital conversion of the EMG signal are listed in Table 6.3.
<table>
<thead>
<tr>
<th>Electrodes</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Specification</td>
<td>Double-differentiated pre-amplified surface electrodes</td>
<td></td>
</tr>
<tr>
<td>Material</td>
<td>Medical-grade stainless steel</td>
<td></td>
</tr>
<tr>
<td>Shape</td>
<td>Circular disks</td>
<td></td>
</tr>
<tr>
<td>Size</td>
<td>12 mm</td>
<td></td>
</tr>
<tr>
<td>Weight</td>
<td>10 g</td>
<td></td>
</tr>
<tr>
<td>Reference contacts</td>
<td>12 mm x 3 mm bar</td>
<td></td>
</tr>
<tr>
<td>Inter-electrode distance</td>
<td>18 mm</td>
<td></td>
</tr>
<tr>
<td>Pre-amplifier body size</td>
<td>38 mm x 19 mm x 8 mm</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>EMG Detection</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Detection mode</td>
<td>Double differential</td>
<td></td>
</tr>
<tr>
<td>Input impedance (pre-amplifiers)</td>
<td>&gt; 100 MΩ</td>
<td></td>
</tr>
<tr>
<td>Common mode rejection ratio (pre-amplifiers)</td>
<td>&gt; 100 dB at 65 Hz</td>
<td></td>
</tr>
<tr>
<td>Signal-to-noise ratio</td>
<td>&gt; 50 dB</td>
<td></td>
</tr>
<tr>
<td>Pre-amplifier gain</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Backpack gain range</td>
<td>10 to 500</td>
<td></td>
</tr>
<tr>
<td>Low pass filter</td>
<td>500 Hz at -3 dB</td>
<td></td>
</tr>
<tr>
<td>High pass filter</td>
<td>20 Hz at -3 dB</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sampling</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sampling rate</td>
<td>1000 Hz</td>
<td></td>
</tr>
<tr>
<td>A/D Card and Resolution</td>
<td>DATAQ® Instruments 32-channel DI-720 Data Acquisition System with 12 bit resolution (DATAQ Instruments Inc., Akron, Ohio, USA)</td>
<td></td>
</tr>
<tr>
<td>Storage</td>
<td>“.c3d” files on PC</td>
<td></td>
</tr>
</tbody>
</table>

* g = grams, mm = millimetres, MΩ = mega Ohms, dB = decibels, Hz = Hertz, PC = personal computer
6.6.4 Recording of MVIC

A maximum voluntary isometric contraction (MVIC) was then recorded for each muscle using SEMG. A practice trial was carried out for each muscle to familiarise the participant with the procedure. At least one second of data from the muscle at rest was recorded prior to the initiation of MVIC, to ensure optimal SNR and to inspect the signal for interference. This data acted as a reference for the Resting Threshold Method timing algorithm described in Chapter 5, Section 5.4.4.2. Amplification gains were adjusted to achieve a signal amplitude of around 1 Volt (V) during MVIC.

MVIC was carried out against manual resistance provided by the principal investigator (AM), using standardised test procedures for BF, RF, TA and MG muscles (Kendall et al., 2005). The test positions and actions are shown in Table 6.4 and Figures 6.5–6.8. The contraction was held for 3 seconds with verbal encouragement to ensure that MVIC was reached.

Table 6.4: Recording of maximum voluntary isometric contraction

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Position</th>
<th>Command</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biceps femoris</td>
<td>Prone, knee in 70° flexion, hip in slight external rotation</td>
<td>&quot;Bend your knee as hard as you can&quot;</td>
<td>Tester applies strong extension force about 5 cm proximal to Achilles tendon, participant resists by attempting to flex knee maximally</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>Prone, knee in 70° flexion, ankle in plantargrade position</td>
<td>&quot;Push up your foot as hard as you can&quot;</td>
<td>Tester applies a dorsiflexion force to the metatarsal heads, participant resists by pushing foot and toes up into tester’s hand</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>Sitting with thighs supported, hands placed at edge of plinth for support, knees in 90° flexion, feet clear of floor</td>
<td>&quot;Hold your knee up and straighten your leg as hard as you can&quot;</td>
<td>Participant flexes hip so that thigh clears the bed, tester places one hand on lower tibia proximal to ankle joint, participant attempts to strongly extend knee while maintaining flexed hip</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>Sitting with thighs supported, feet clear of floor, ankle in 5° dorsiflexion and slight inversion</td>
<td>&quot;Hold your foot up&quot;</td>
<td>Tester attempts to push foot into plantarflexion and eversion, participant resists this action maximally</td>
</tr>
</tbody>
</table>
Figure 6.5: Assessment of maximum voluntary isometric contraction of rectus femoris

Figure 6.6: Assessment of maximum voluntary isometric contraction of biceps femoris
Figure 6.7: Assessment of maximum voluntary isometric contraction of tibialis anterior

Figure 6.8: Assessment of maximum voluntary isometric contraction of medial gastrocnemius
6.6.5 Placement of Vicon retro-reflective markers

Fifteen retro-reflective markers of 25 mm diameter were placed on the participant's lower limbs in standing in accordance with the MHH model (Davis et al., 1991). Marker placement is shown in Figure 6.9 and Figure 6.10. The PI was responsible for marker placement in all assessments. Table 6.5 describes the location and anatomical landmarks used in the placement of markers. Figure 6.11 shows the identification of the location for the heel marker.

Figure 6.9: Frontal plane view of the Modified Helen Hayes marker set and EMG electrodes
Table 6.5: Description of marker placement

<table>
<thead>
<tr>
<th>Marker label</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sacrum</td>
<td>A wand marker was placed over the sacrum at the midpoint between the skin dimples of the posterior superior iliac spine.</td>
</tr>
<tr>
<td>ASIS</td>
<td>Markers were placed over each ASIS.</td>
</tr>
<tr>
<td>Knee</td>
<td>A KAD was attached to the lateral and medial femoral condyles. This was done initially in sitting, with the foot clear of the floor, to determine the location of the KAD at which minimal translation of the horizontal arm occurred during open kinetic chain knee flexion and extension. The KAD was then reapplied in standing at a point slightly posterior to its location in sitting, to compensate for movement of the skin over the knee joint from flexion in sitting to standing.</td>
</tr>
<tr>
<td>Thigh</td>
<td>A short wand was placed over the lateral aspect of the thigh in alignment with the most lateral aspect of the greater trochanter and the lateral knee joint axis marker on the KAD.</td>
</tr>
<tr>
<td>Ankle</td>
<td>Ankle markers were placed over the most lateral aspect of each lateral malleolus.</td>
</tr>
<tr>
<td>Shank</td>
<td>Marker wands were aligned with the ankle joint markers and the knee joint markers at the midpoint of an imaginary line between these markers.</td>
</tr>
<tr>
<td>Forefoot</td>
<td>Markers were attached to the heads of the second metatarsal bones, identified by asking the participant to flex their toes.</td>
</tr>
<tr>
<td>Heel</td>
<td>Markers were attached to the posterior aspect of the calcaneus, at the same height as the forefoot marker, determined by a calliper.</td>
</tr>
</tbody>
</table>

*KAD = knee alignment device, ASIS = anterior superior iliac spine*
6.6.6 Static calibration

A static data capture was then performed with all markers in situ and the KAD placed at the knee joint line to calibrate the participant’s measurements and marker placement in the static position. The position for static calibration is shown in Figure 6.12. The participant was instructed to stand on the force plate for the static capture, with arms folded and knees slightly flexed. If the participant could not achieve stability in standing with slightly flexed knees, the knees were maintained in extension. Usual gait aids were used for participants who had difficulty with unsupported standing. Calibration was completed using PiG®.
The KADs were then removed and single markers were placed over the lateral aspect of the knee joint defined by KAD. A second static calibration was performed to facilitate marker labelling, retaining the rotation parameters defined in the previous calibration. This calibration is shown in Figure 6.13.
Participants were instructed to walk barefoot along the laboratory walkway at their usual, comfortable walking speed, using any habitual gait aids. Stand-by assistance was provided for safety by a co-investigator during the gait trials. Physical assistance was avoided unless the participant became unsteady, in which case that trial was not then used for further analysis. Although the force plate was visible in the centre of the walkway, participants were not informed of its purpose to ensure that their gait patterns were not altered by any attempt to target its location. Data were captured at 50 Hz. Raw data were monitored continuously on the Workstation screen to ensure optimal quality and to detect marker occlusion or displacement that may have occurred during walking. Gait trials were repeated until the participant had achieved ten trials with complete strikes to the force plate, five from each leg. The starting position for each trial was modified if

Figure 6.13: Static calibration without KAD

6.6.7 Gait trials

Participants were instructed to walk barefoot along the laboratory walkway at their usual, comfortable walking speed, using any habitual gait aids. Stand-by assistance was provided for safety by a co-investigator during the gait trials. Physical assistance was avoided unless the participant became unsteady, in which case that trial was not then used for further analysis. Although the force plate was visible in the centre of the walkway, participants were not informed of its purpose to ensure that their gait patterns were not altered by any attempt to target its location. Data were captured at 50 Hz. Raw data were monitored continuously on the Workstation screen to ensure optimal quality and to detect marker occlusion or displacement that may have occurred during walking. Gait trials were repeated until the participant had achieved ten trials with complete strikes to the force plate, five from each leg. The starting position for each trial was modified if
necessary to facilitate the collection of complete, uncontaminated force plate data. For some participants with more severe gait impairment, it was not possible to achieve the required number of force plate strikes, due to short stride length or the onset of fatigue. In these cases, ten trials were collected as normal for TSPs, kinematics, and EMG, but the number of trials containing valid kinetic data may have been less than five. Participants were monitored for fatigue during the assessment by subjective questioning, and given rest breaks as required between trials to minimise fatigue.

6.7 Data processing

6.7.1 Computerised processing of gait trials

The raw video and analogue data, captured by the Vicon cameras and Kistler force plate in Section 6.5, required further processing in Vicon Workstation to generate temporal-spatial, kinematic and kinetic data. Anthropometric data, obtained in Section 6.6.2, were inputted into Workstation. The markers in the static calibration trial were then labelled and Vicon’s “static gait model” procedure was applied to the calibration trial to determine the lower limb joint centres from the anthropometric data and marker locations. This calibration formed the basis of the PiG routine that calculated kinematics and kinetics for the remaining trials in that session.

Individual gait trials were then processed in Workstation. Three-dimensional trajectories were reconstructed from the raw video files, shown in Figure 6.14 and labelled, as shown in Figure 6.15.

![Figure 6.14: Visualisation of markers in Workstation prior to labelling](image)
Gaps in the trajectories were inspected and filled using inbuilt commands in Workstation, either by interpolation for small gaps, or by copying the pattern of an adjacent trajectory if the gaps were large. The trajectories were then filtered using a Woltring filter with a MSE of 15 mm$^2$. PiG then calculated the joint centres and computed the underlying joint motion. GRFs, captured in three planes by the Kistler force plate, were integrated with kinematic data to calculate joint moments and powers using inverse dynamics. Kinetic parameters were normalised to body weight to enable comparison between participants.

GC events of heel strike and toe off were then defined in Workstation to facilitate the calculation of TSPs. These events were derived from force plate data where the participant had achieved a complete strike of the foot to the force plate, without contacting the force plate with the opposite foot. A threshold of 20 Newtons was used to determine the points of heel strike and toe off. Trials without force plate data, or where force plate data were contaminated, were defined by observation of the patterns of the heel and forefoot trajectories, for one GC of each lower limb. Figure 6.16 shows the inspection of heel and forefoot trajectories to determine GC events. The remaining GCs were then computed by Vicon’s auto-correlation system, using the ankle trajectory as a reference, and verified by visual inspection. Errors in the auto-correlation of GC events were manually corrected. TSPs were then calculated for that trial.
The next step in data processing involved the importing of gait trials from Workstation into Vicon Polygon®, a software package for visualisation of temporal-spatial, kinematic and kinetic data. Ten gait trials from each participant were imported into Polygon, comprising five trials each with force plate data from the left and right lower limbs. If a participant could not achieve the specified number of force plate strikes, 10 gait trials were still imported to Polygon, but some of these contained kinematic data only. The Polygon software normalised each GC in time to 51 data points from heel strike (0%) to ipsilateral heel strike (100%) at intervals of 2%. If a gait trial contained data from more than one complete GC for one or both lower limbs, the GC from each lower limb with the best quality data was selected for further analysis, and data from other GCs in that trial were discarded using Polygon’s graphical user interface. The temporal-spatial, kinematic and kinetic data for each trial were then exported to Microsoft Excel using a macro routine in Polygon. An average was obtained for that assessment of each TSP, and of time-normalised kinematic and kinetic curves. Figure 6.17 shows the Polygon interface and the visualisation of kinematic and kinetic graphs. Table 6.6 lists the TSPs and Table 6.7 details the kinematic and kinetic curves that were exported from Polygon.
Figure 6.17: Visualisation of trials in Polygon
<table>
<thead>
<tr>
<th>Variable</th>
<th>Description</th>
<th>Units of measurement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cadence</td>
<td>Number of steps taken in one minute</td>
<td>Steps / minute</td>
</tr>
<tr>
<td>Double support</td>
<td>Time for which both lower limbs are in stance</td>
<td>Seconds</td>
</tr>
<tr>
<td>Double support duration</td>
<td>Duration of double support as a percentage of GC</td>
<td>% GC duration</td>
</tr>
<tr>
<td>Foot off</td>
<td>Percentage of GC at which transition from stance to swing occurs</td>
<td>% GC duration</td>
</tr>
<tr>
<td>Gait speed</td>
<td>Distance covered over time</td>
<td>Metres per second</td>
</tr>
<tr>
<td>Opposite foot contact</td>
<td>Time during stance phase of one limb that the opposite limb begins stance, i.e. beginning of second double support phase</td>
<td>% GC duration</td>
</tr>
<tr>
<td>Opposite foot off</td>
<td>Time during stance phase of one limb that the opposite limb begins swing, i.e. completion of first double support phase</td>
<td>% GC duration</td>
</tr>
<tr>
<td>Single support</td>
<td>Time for which only ipsilateral limb is in contact with the ground</td>
<td>Seconds</td>
</tr>
<tr>
<td>Single support duration</td>
<td>Duration of single support as a percentage of GC</td>
<td>% GC duration</td>
</tr>
<tr>
<td>Step length</td>
<td>Distance covered from heel strike of ipsilateral limb to heel strike of the contralateral limb</td>
<td>Metres</td>
</tr>
<tr>
<td>Step time</td>
<td>Time from heel strike of ipsilateral limb to heel strike of the contralateral limb</td>
<td>Seconds</td>
</tr>
<tr>
<td>Step width</td>
<td>Lateral distance between right and left forefoot markers during double support</td>
<td>Metres</td>
</tr>
<tr>
<td>Stride length</td>
<td>Distance covered from heel strike of one limb to heel strike of the same limb</td>
<td>Metres</td>
</tr>
<tr>
<td>Stride time</td>
<td>Time from heel strike of one limb to heel strike of the same limb</td>
<td>Seconds</td>
</tr>
</tbody>
</table>

GC = gait cycle
<table>
<thead>
<tr>
<th>Name</th>
<th>Description</th>
<th>Positive orientation</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pelvic tilt</td>
<td>Movement of the pelvis in the sagittal plane</td>
<td>Anterior tilt</td>
<td>Degrees</td>
</tr>
<tr>
<td>Pelvic obliquity</td>
<td>Movement of the pelvis in the frontal plane</td>
<td>Upward obliquity</td>
<td></td>
</tr>
<tr>
<td>Pelvic rotation</td>
<td>Movement of the pelvis in the transverse plane</td>
<td>Internal rotation</td>
<td></td>
</tr>
<tr>
<td>Hip flexion / extension</td>
<td>Movement of the hip in the sagittal plane</td>
<td>Flexion</td>
<td></td>
</tr>
<tr>
<td>Hip abduction / adduction</td>
<td>Movement of the hip in the frontal plane</td>
<td>Abduction</td>
<td></td>
</tr>
<tr>
<td>Hip rotation</td>
<td>Movement of the hip in the transverse plane</td>
<td>Internal rotation</td>
<td></td>
</tr>
<tr>
<td>Knee flexion / extension</td>
<td>Movement of the knee in the sagittal plane</td>
<td>Flexion</td>
<td></td>
</tr>
<tr>
<td>Ankle dorsiflexion / plantarflexion</td>
<td>Movement of the ankle in the sagittal plane</td>
<td>Dorsiflexion</td>
<td></td>
</tr>
<tr>
<td>Hip flexor / extensor moment</td>
<td>Net moment of force about the hip in the sagittal plane</td>
<td>External extensor moment</td>
<td>Newton metres</td>
</tr>
<tr>
<td>Hip abduction / adduction moment</td>
<td>Net moment of force about the hip in the transverse plane</td>
<td>External abductor moment</td>
<td></td>
</tr>
<tr>
<td>Knee flexor / extensor moment</td>
<td>Net moment of force about the knee in the sagittal plane</td>
<td>External extensor moment</td>
<td></td>
</tr>
<tr>
<td>Ankle dorsiflexor / plantarflexor moment</td>
<td>Net moment of force about the ankle in the sagittal plane</td>
<td>External plantarflexor moment</td>
<td></td>
</tr>
<tr>
<td>Hip power</td>
<td>Net power at the hip across three planes</td>
<td>Power generation</td>
<td>Watts per kilogram</td>
</tr>
<tr>
<td>Knee power</td>
<td>Net power at the knee across three planes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ankle power</td>
<td>Net power at the ankle across three planes</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
6.7.2 Extraction of kinematic and kinetic key points

The kinematic and kinetic variables, extracted from Polygon for each participant’s assessment and listed in Table 6.7 above, consisted of time-varying curves normalised to 51 data points, each representing the GC at 2% intervals. Key points from each kinematic and kinetic curve were selected based on data from previous studies in CSM (Kuhtz-Buschbeck et al., 1999, Maezawa et al., 2001, Suzuki et al., 2002) and on their clinical relevance (Kirtley, 2006). A large number of variables were included to facilitate exploration of the data in the reliability and cross-sectional studies. This approach was used by a number of studies using 3DGA, particularly in populations whose gait characteristics had not previously been assessed in detail with this technology (Mahaudens et al., 2009, Williams et al., 2009b, Gil-Agudo et al., 2011). Tables 6.8 and 6.9 list and define these kinematic and kinetic key points, respectively. Extraction of the key points from the time-normalised average of each assessment was performed using a customised routine in MATLAB, shown in Appendix 6.7.
<table>
<thead>
<tr>
<th>Joint</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pelvis</strong></td>
<td>Peak pelvic tilt</td>
</tr>
<tr>
<td></td>
<td>Range of pelvic tilt</td>
</tr>
<tr>
<td></td>
<td>Average pelvic tilt</td>
</tr>
<tr>
<td></td>
<td>Peak pelvic obliquity</td>
</tr>
<tr>
<td></td>
<td>Range of pelvic obliquity</td>
</tr>
<tr>
<td></td>
<td>Range of pelvic rotation</td>
</tr>
<tr>
<td><strong>Hip</strong></td>
<td>Hip position in the sagittal plane at initial contact</td>
</tr>
<tr>
<td></td>
<td>Peak hip flexion</td>
</tr>
<tr>
<td></td>
<td>Peak hip extension</td>
</tr>
<tr>
<td></td>
<td>Total range of hip excursion in the sagittal plane</td>
</tr>
<tr>
<td></td>
<td>Peak hip abduction in swing</td>
</tr>
<tr>
<td></td>
<td>Total range of hip excursion in the frontal plane</td>
</tr>
<tr>
<td></td>
<td>Peak hip internal rotation</td>
</tr>
<tr>
<td></td>
<td>Total range of hip rotation</td>
</tr>
<tr>
<td><strong>Knee</strong></td>
<td>Knee position at initial contact</td>
</tr>
<tr>
<td></td>
<td>Peak knee flexion in stance, loading response</td>
</tr>
<tr>
<td></td>
<td>Peak knee flexion in swing</td>
</tr>
<tr>
<td></td>
<td>Peak knee extension in midstance</td>
</tr>
<tr>
<td></td>
<td>Total range of knee excursion in the sagittal plane</td>
</tr>
<tr>
<td><strong>Ankle</strong></td>
<td>Ankle position at initial contact</td>
</tr>
<tr>
<td></td>
<td>Peak ankle dorsiflexion in stance</td>
</tr>
<tr>
<td></td>
<td>Peak ankle dorsiflexion in swing</td>
</tr>
<tr>
<td></td>
<td>Peak ankle plantarflexion</td>
</tr>
</tbody>
</table>
**Table 6.9: Key points extracted from kinetic curves over the gait cycle**

<table>
<thead>
<tr>
<th>Type of Data</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ground reaction force (GRF) (N/kg)</strong></td>
<td>Peak mediolateral GRF</td>
</tr>
<tr>
<td></td>
<td>Peak negative antero-posterior GRF (braking)</td>
</tr>
<tr>
<td></td>
<td>Peak positive antero-posterior GRF (propulsion)</td>
</tr>
<tr>
<td></td>
<td>First vertical GRF peak, loading</td>
</tr>
<tr>
<td></td>
<td>Minimum value of GRF during stance</td>
</tr>
<tr>
<td></td>
<td>Second vertical GRF peak, propulsion</td>
</tr>
<tr>
<td><strong>Moments (Nm/kg)</strong></td>
<td>Peak hip flexor moment</td>
</tr>
<tr>
<td></td>
<td>Peak hip extensor moment</td>
</tr>
<tr>
<td></td>
<td>Peak hip abductor moment</td>
</tr>
<tr>
<td></td>
<td>Peak knee flexor moment</td>
</tr>
<tr>
<td></td>
<td>Peak knee extensor moment</td>
</tr>
<tr>
<td></td>
<td>Peak ankle plantarflexor moment</td>
</tr>
<tr>
<td><strong>Powers (W/kg)</strong></td>
<td>Peak concentric hip extensor power during loading response (H1)</td>
</tr>
<tr>
<td></td>
<td>Peak eccentric hip flexor power during mid-stance (H2)</td>
</tr>
<tr>
<td></td>
<td>Peak concentric hip flexor power during terminal stance (H3)</td>
</tr>
<tr>
<td></td>
<td>Peak eccentric knee extensor power during loading response (K1)</td>
</tr>
<tr>
<td></td>
<td>Peak concentric knee extensor power during mid-stance (K2)</td>
</tr>
<tr>
<td></td>
<td>Peak eccentric knee extensor power in terminal stance (K3)</td>
</tr>
<tr>
<td></td>
<td>Peak eccentric knee flexor power, terminal swing (K4)</td>
</tr>
<tr>
<td></td>
<td>Peak eccentric ankle power in loading response through to mid stance (A1)</td>
</tr>
<tr>
<td></td>
<td>Peak concentric ankle plantarflexor power at terminal stance (A2)</td>
</tr>
</tbody>
</table>

*Abbreviations: W = Watts, kg = kilograms, N = Newtons, Nm = Newton metres*
6.7.3 Processing of EMG data

6.7.3.1 Extraction of EMG

EMG analogue data from each trial were exported to a comma separated values (CSV) text file in ASCII (American Standard Code for Information Interchange) format. A custom built routine in MATLAB, detailed in Appendix 6.8, extracted EMG signals from each channel for the duration of the GCs defined and used in Polygon in section 6.7.1 above. The signals were then filtered with a second-order Butterworth high-pass filter with a cut-off of 25 Hz and a fourth-order Butterworth low-pass filter with a cut-off of 400 Hz, applied in forward and reverse directions using a MATLAB routine, previously shown in Appendix 5.2. The filtered signals were stored in MATLAB ‘mat’ format for further analysis.

6.7.3.2 Detection of the timing of muscle activation

As outlined in Chapter 5, Section 5.4.4.2, the TKEO-based Double Threshold Method (DTM) for detection of muscle activation showed the highest agreement with visual interpretation. It was used for detection of the timing of muscle activation for all EMG signals in the current study. Using a custom built routine in MATLAB, shown in Appendix 6.9, the filtered signals of Section 6.7.3.1 were treated with the TKEO equation. Activation was signalled when the TKEO amplitude at a given time point exceeded 3% of the maximum TKEO amplitude of that trial, in Volts times Hertz squared, and when the slope, or rate of change of the TKEO-treated signal over time, exceeded $10^{-6}$, for at least 24 time points in a 25 point moving window. Cessation of activation was signalled when this condition was no longer satisfied. The application of this algorithm generated two outputs, 1) a vector, termed “ON”, of a series of logical 1’s and 0’s denoting periods of muscle activity and no activity, respectively, and 2) the time points in the GC at which the signal changed from an “on” to an “off” state, and vice versa.

6.7.3.3 Amplitude processing

The amplitude of each EMG signal was extracted using the RMS method. RMS was generated over a time interval of 30 ms with 20 ms overlap using a previously written routine in MATLAB (Ajiboye, 2006), provided in Appendix 6.10. The resulting output was interpolated to 101 data points using one-dimensional linear interpolation, thereby normalising the data in time to the GC at successive percentage points from 0 to 100. This facilitated the interpretation of RMS amplitude in relation to timing data, kinematics and kinetics.

6.7.3.4 Normalisation of amplitude

Normalisation of a signal’s RMS amplitude to a given reference amplitude is necessary to facilitate comparison between participants on different test days, and between groups of participants. As outlined in Chapter 5, Section 5.4.4.4, both the maximum amplitude of a
muscle’s EMG signal in one session of gait analysis and a muscle’s signal during MVC, have been used as normalisation references, but their reliability has not been tested. It was therefore necessary to test both methods for reliability and to choose the most reliable method for further analysis.

To obtain the PDM reference, the maximum value of each muscle’s time-normalised RMS, over the ten extracted trials in that session, was identified using MATLAB. For MVC normalisation, MVC signals obtained in Section 6.6.4 were exported to MATLAB and their RMS were calculated. In some cases, the MVC trial was recorded using different amplification gains to those used in gait trials. If this had occurred, raw data from the MVC trial was scaled to the gains used in the gait trials for that session, using a scaling system designed for the MA-300 (Appendix 5.3). The maximum RMS value over the 0.5 s interval with the highest mean amplitude was then extracted and used as the MVC normalisation reference (Hsu et al., 2006).

6.7.4 Extraction of clinically relevant EMG parameters

6.7.4.1 Timing and co-activation

The total duration of activation for each muscle in a GC was summed and expressed as a percentage of GC duration to allow for comparison between GCs of varying length and between individuals. The mean activation time of ten gait trials in one session was used for further analysis.

To determine co-activation, the activation of antagonistic muscle pairs, RF–BF and TA–MG, were compared at each time point in the GC. Co-activation was defined as simultaneous activation of both muscles, when the “ON” vector of Section 6.6.3.2 denoted “1” for both muscles at a particular data point. The duration of co-activation for each pair was then summed and expressed as a percentage of GC duration. The mean co-activation of 10 trials in one session was used for further analysis.

6.7.4.2 Amplitude parameters to determine intensity of muscle activation

The RMS of each signal over the GC was further processed to extract the peak and mean RMS amplitude of each burst of muscle activity, as well as the mean RMS during the periods of the GC when the muscle in a baseline state (“off”). Phases of activity for each muscle were determined based on their relevance to the GC from the work of (Perry, 1992). The phases used for analysis of each muscle are listed in Table 6.10. Stance and swing were determined from the point at which toe-off occurred in each individual GC, previously obtained as a TSP in Section 6.7.1.
**Table 6.10: Bursts of muscle activity examined for amplitude**

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Activity burst</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medial gastrocnemius</td>
<td>Stance</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>Loading response</td>
</tr>
<tr>
<td></td>
<td>Swing</td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>Stance</td>
</tr>
<tr>
<td></td>
<td>Swing</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>Loading response</td>
</tr>
<tr>
<td></td>
<td>Pre swing to initial swing</td>
</tr>
<tr>
<td></td>
<td>Swing</td>
</tr>
</tbody>
</table>

Figure 6.18 illustrates the process of extracting amplitude data for each activity burst. The timing of muscle activation within each phase was extracted based on the data of Section 6.6.4.1. The timing was then applied to the RMS of the signal obtained in Section 6.6.3.2. For each phase listed in Table 6.10, the RMS values at the time points in which the muscle was active in that phase were extracted, and the peak and mean RMS were calculated from this data. The RMS of the “off” phases was calculated by extracting the RMS values at the time points in the GC when the muscle was not active, and calculating the mean of these values.
Figure 6.18: Processing of the EMG signal to obtain timing and amplitude parameters, showing the right rectus femoris muscle from one participant’s baseline (pre-operative) assessment

A) Raw signal, extracted over the gait cycle from heel strike to heel strike
B) Signal after filtering off-line with a Butterworth filter to remove motion artefact
C) TKEO-treated signal with the straight grey line (Y axis) showing the periods in which the muscle was “on” (active) or “off” in the gait cycle
D) the root-mean-square (RMS) of the signal, normalised to the gait cycle from 0 to 100%
E) extraction of RMS from the bursts of each clinically-relevant phase

V, Volts, ms, milliseconds, VHz², Volts times Hertz squared, RMS, root-mean-square amplitude, TO, toe off, RF, rectus femoris
6.7.4.3 Locomotor-specific measure of spasticity

As discussed in Chapter 5, Section 5.4.4.5, a locomotor specific measure of spasticity (LSMS) was developed according to the methods of Lamontagne et al. (2001) and Crenna (1999). The LSMS evaluated the relationship between a muscle’s EMG signal and its lengthening velocity over the GC. The relative length and lengthening velocities of TA, MG, BF and RF during the GC were calculated using formulae derived by Winter and Scott (1991). The resting length of each muscle was defined as its length in the anatomical position, with hips, knees and ankles at 0° flexion and extension. The muscle’s length at any given point during the GC was a function of the angle of the joint or joints it crossed, its angle of pennation, and three (for single joint muscles) or six (for multi joint muscles) constants. Length was expressed in relation to the resting length in the anatomical position. Lengthening velocity was then calculated as a function of the GC duration. Equations are listed in Appendix 6.11. Calculations of muscle length and lengthening velocity were performed using a custom-built routine in MATLAB (Appendix 6.12). Lengthening velocity was interpolated to 101 data points to facilitate interpretation with the EMG signal.

One key lengthening phase was examined for each muscle, namely, BF lengthening contraction during terminal swing, MG during mid stance, TA during terminal stance through initial swing, and RF during the same period. Three parameters were extracted as part of the LSMS, 1) the lengthening velocity at the point of onset of EMG activity during the lengthening contraction, namely LVT, 2) the time point at which EMG activity onset occurred, termed the “critical time” and expressed as a percentage of GC duration, and 3) the slope of the line of EMG RMS, expressed as a percentage of the maximum RMS in that session, plotted against lengthening velocity, with positive slopes indicating an abnormal response (Lamontagne et al., 2001). The onset of EMG activity during lengthening was extracted from the timing data of Section 6.7.4.1.

6.8 Conclusion

This chapter has described the general methods of recruitment, data collection, and processing that were common to all studies in the project. The next three chapters will detail the methods and results of the individual reliability, cross-sectional and experimental studies.
Chapter 7: Reliability Study

7.1 Introduction

Before a measurement instrument can be applied to a clinical population, it is good scientific practice to estimate its reliability in that population. This ensures that the results can be interpreted within the context of the variation in scores that occurs from test to retest (Bruton et al., 2000). In the absence of previous studies of reliability on 3DGA and EMG in the CSM population, it was necessary for the current study to undertake this task. About 70% of reliability studies conducted to date have evaluated healthy participants (McGinley et al., 2009). Each clinical population will have its own characteristics regarding variability (Bruton et al., 2000), and therefore estimates of reliability from healthy participants cannot be extrapolated to those with a specific impairment such as CSM. Reliability is a key concern where the data will be used to compare participants and to evaluate the outcome of an intervention (Dankaerts et al., 2004), as was the case with the current study.

7.2 Previous research on reliability of 3DGA and EMG

7.2.1 Reliability of temporal-spatial, kinematic and kinetic parameters

A number of studies have evaluated the reliability of temporal-spatial, kinematic and kinetic parameters in populations including healthy participants, CP, and stroke. Approaches to the analysis of reliability in 3DGA have differed among studies. Some studies examined the reliability of kinematic and kinetic curves over the complete GC (Kadaba et al., 1989, Steinwender et al., 2000, Schwartz et al., 2004, Delval et al., 2008), while others extracted key points from those curves, such as a peak value or a range (Maynard et al., 2003, Monaghan et al., 2007, Yavuzer et al., 2008). Key points have been considered more meaningful, as they are easier to compare and interpret than complete curves, and tend to include the most clinically relevant features of the curves (Redekop et al., 2008).

A systematic review, based on the results of fifteen studies of 3DGA, found highest reliability for kinematic parameters in the sagittal plane (ICC 0.8), with the exception of pelvic tilt (ICC 0.6), and lowest reliability in the transverse plane (ICC <0.7). Standard errors of measurement (SEM) of around 4º in the sagittal plane and 2º in the frontal plane were calculated. The review concluded that most kinematic parameters showed moderate to good reliability, however the measurement errors were not small enough to be ignored in clinical interpretation (McGinley et al., 2009). In neurological populations, intra-session reliability was found to be higher in children with better-functioning scores.
on the Gross Motor Function Classification Scale, and lower in those with more severe CP (Redekop et al., 2008), suggesting that the severity of a mobility disorder impacts negatively on reliability. Studies have found kinetic parameters to be more reliable than kinematics in children with CP (Steinwender et al., 2000), adults with stroke (Campanini and Merlo, 2009), and healthy adults (Kadaba et al., 1989, Monaghan et al., 2007). In healthy adults, GRFs were more repeatable than joint moments, possibly due to variation in marker placement contributing to moments (Kadaba et al., 1989).

No studies on the reliability of 3DGA in SCI have been reported to date. It would be expected that some of the trends shown in previous studies would apply to the current study in CSM. Variation in marker placement is considered the most important source of extrinsic or measurement error in test-retest reliability (McGinley et al., 2009). Efforts to minimise this error in the current study were discussed in Chapter 5, Section 5.3.3.1. Intrinsic variability, or true inherent variation in a participant’s gait, may also be a factor in people who have limited mobility (Schwartz et al., 2004), such as those with CSM, and may increase the minimal detectable change of the 3DGA in this population (Haley and Fragala-Pinkham, 2006).

7.2.3 Reliability of SEMG parameters

7.2.3.1 Reliability of timing data in gait

To date, no studies have examined the reliability of SEMG timing data in gait in a neurological population. In healthy participants, the timing of the peak value of the linear envelope, normalised to GC duration, was found to vary by less than 10% GC duration for peroneus longus, TA, MG and soleus, despite deliberate changes in electrode location (Campanini et al., 2007). This would suggest that timing is relatively robust to slight differences in electrode placement from test to retest. However, Campanini’s study did not consider variability in estimates of the total duration of bursts of muscle activation over the GC, which may be affected by the relative location of innervation zones and tendons with respect to the overlying electrodes, and may therefore vary from test to retest (Farina et al., 2001).

In neurological populations, the timing and amplitude of bursts of activity may be prone to greater levels of intrinsic variability (Schwartz et al., 2004). This may be due to the presence of spasticity with its variable manifestations depending on pain, temperature, or fatigue, or to inconsistencies in achieving the optimum motor strategy for a task in gait. Therefore, it cannot be assumed that the duration of muscle activation over the GC will be robust in the current study’s CSM population, and an estimate of test-retest variability is required.
Test-retest reliability of SEMG amplitude data was also a key concern for the current study. Conflicting results have been found by studies in addressing this issue, and as yet, there are no findings relating to the reliability of amplitude data from EMG signals in a neurological population. Three factors need to be considered in the reliability of amplitude data: 1) the effect of test-retest variability when electrodes are removed and re-applied, as opposed to intra-test reliability, where electrodes are not usually removed and environmental changes will not affect the final signal, 2) the units reported, that is, whether the RMS or average rectified value (ARV) amplitude in Volts is examined for reliability or whether normalisation is applied, 3) if normalisation is applied, which method is used.

The most reliable normalisation reference has not yet been determined. Norcross et al. (2010) compared the reliability of ARV amplitude of EMG of the gluteal muscles, RF, vastus lateralis, BF and adductors at the hip from two normalisation tasks, conventional MVC and muscle activity during single leg stance. Both methods demonstrated good-to-excellent reliability (ICC, 0.85–0.95, SEM, 8.5–201 mV). However, the effect of removing and reapplying the electrodes was not examined, a factor which must be considered in clinical interpretation of EMG data where results will usually be compared on different test days. A study of test-retest reliability of non-normalised ARV amplitudes in a maximal and sub-maximal test of trunk muscle function found higher test-retest reliability in the sub-maximal test (ICC, 0.88, SEM, 8%) compared to the maximal test (ICC 0.7, SEM 17%) (Dankaerts et al., 2004). A study of reliability in healthy people and those with knee pathology found high variability in non-normalised RMS parameters, with ICCs ranging from 0.23 to 0.84, and SEM of up to 0.28 mV (Callaghan et al., 2009). Although these studies examined the reliability of the normalisation reference itself, they did not examine the reliability of EMG signals from other tasks that were scaled to that reference value. This was the key question for the current study, which was not so much concerned with the reliability of the amplitude of SEMG signals in the reference test, but rather, the amplitude of signals during gait when normalised to that reference.

In relation to normalised SEMG amplitude data, Knutson et al. (1994) found that normalising gastrocnemius activity to MVC provided more repeatable results in people with anterior cruciate ligament injuries, than normalisation to mean and peak amplitudes from gait. Reliability of the MVC method was also found to be superior to PDM and MDM methods for EMG assessment of the hip musculature in HCs during open and closed kinetic chain exercises (Bolgla and Uhl, 2007).

These studies show that reliability of amplitude parameters varies considerably, depending on the population studied, the normalisation method used, and the time interval between tests. In the absence of any definitive data on reliability, it was
necessary that the current study evaluate reliability of both PDM-normalised and MVC-normalised bursts amplitudes during gait in CSM. The most reliable method could then be used for analysis in the cross-sectional and experimental studies.

### 7.2.3.3 Reliability of locomotor-specific measure of spasticity

As discussed in Chapter 5, Section 5.4.4.5, a measure of the lower limb muscles’ responses to lengthening during the GC was required to fulfill the objective of determining if spasticity was a factor in gait impairment. Although aspects of the LSMS have been validated in children with CP (Crenna, 1999) and in adults following stroke (Lamontagne et al., 2001), its repeatability has not been assessed. This has limited its use in monitoring locomotor recovery (Lamontagne, 2006). The current study therefore included analysis of the test-retest reliability of three aspects of the LSMS, namely 1) the lengthening velocity threshold (LVT) for muscle activation, 2) the critical time during lengthening at which activation occurred, and 3) the slope of the relationship between EMG amplitude and lengthening velocity, in the CSM group. The methods involved in calculation of the LSMS have been described in Chapter 6, Section 6.7.4.3.

### 7.2.4 Methodological considerations in the statistical analysis of reliability

A number of statistical methods have been used in the evaluation of reliability. Of these, the coefficient of variation, the coefficient of multiple correlation (CMC), and its derivate for curves, the coefficient of multiple determination (CMD), have been criticised because they are influenced by the size of the parameter itself. Parameters with a larger range, for example knee flexion in swing, will invariably record higher reliability than parameters with a smaller range, such as pelvic obliquity, using these methods (McGinley et al., 2009). The ICC is a more commonly used measure of relative reliability, and has been recommended for use in reliability studies (Fleiss, 1986, Rankin and Stokes, 1998).

Recent literature has recognised that correlation indices alone do not determine whether a measure is sufficiently reliable for clinical use. It is therefore necessary to obtain a measure of absolute reliability, reported in the same units as the parameter itself, to ensure that the results can be interpreted in a clinical context (Bruton et al., 2000, McGinley et al., 2009). Absolute measures of reliability include the SEM (Streiner and Norman, 2008), which is based on the ICC (Shrout and Fleiss, 1979), and Bland–Altman 95% limits of agreement (LOA) (Bland and Altman, 1986).

### 7.3 Aims and objectives

The aims of the reliability study were 1) to evaluate the reliability of temporal-spatial, kinematic, kinetic, and EMG parameters in a cohort of people with clinical and
radiological evidence of CSM, and 2) to estimate the change required to exceed measurement error for parameters used in the evaluation of gait impairment in CSM.

7.4. Methods

7.4.1. Study design

The reliability study was a prospective cohort design with consecutive recruitment of participants from a neurosurgical clinic. This design has been recommended for reliability studies of gait analysis (McGinley et al., 2009).

7.4.2 Participants

Participants were recruited from a neurosurgical clinic between December 2008 and February 2010. The inclusion and exclusion criteria described in Chapter 6, Section 6.3 were applied. As discussed in Chapter 5, section 5.32, previous surgery for CSM was not an exclusion criterion for participation in the reliability study.

7.4.3 Timing of test-retest assessments

Participants attended the RCSI Movement Laboratory on two separate test days, between two and seven days apart. This time interval was considered short enough to avoid a true change in a participant’s gait pattern, an important factor in a potentially progressive condition such as CSM. It was also long enough to minimise recall bias on the part of the PI, for example, with respect to marker placement or anthropometric measurements. It aimed to avoid a potential learning effect on the part of the participant who might recall a self-generated cue in relation to gait. The time interval was in keeping with recommendations from other reliability studies (McGinley et al., 2009).

7.4.4 Capture of 3DGA and EMG data

Motion analysis and EMG data were captured according to the methods described in Chapter 6, Section 6.6. The PI performed anthropometric measurements, marker application and data processing for all assessments, and was blinded to the results of the participants’ first assessment on the second test day. Data were processed according to the methods described in Chapter 6, Section 6.7.

Reliability was calculated on the average of ten gait trials from each session. The use of multiple gait trials within one 3DGA assessment has been shown to improve reliability by stabilising the average of a number of slightly variable trials, and a figure of ten trials for kinematics has been reported (Monaghan et al., 2007). Kinetic data are generally more reliable (Kadaba et al., 1989, Campanini and Merlo, 2009), but also more difficult to
obtain because a complete and uncontaminated strike to the force plate is required. For this study, it was decided to capture ten trials for temporal-spatial, kinematic, and EMG data, and five trials from each lower limb for kinetic data, to achieve a stable average for reliability tests while avoiding excessive data collection time and possible participant fatigue.

The parameters examined for reliability were listed in Chapter 6, Tables 6.6, 6.8 and 6.9. Data for the participants' left lower limbs were analysed for reliability of TSPs, kinematics and kinetics, while data from the right lower limbs were analysed for reliability of EMG parameters. These sides were chosen by coin toss to condense the analysis, as it was not expected that there would be systematic differences in reliability between lower limbs. Furthermore, each individual contributes their own intrinsic variability to the data set, regardless of whether left or right is analysed, and including both in the data set may have artificially increased reliability by reducing the variability of the overall scores.

### 7.4.5. Statistical analysis

#### 7.4.5.1 Sample size calculation

A sample size calculation was based on a formula provided by Streiner and Norman (2008), shown in Appendix 7.1. The lower acceptable limit for reliability was set at an ICC of 0.70, the minimum recommended level when using a measurement in research (Nunnally, 1978). The desired level of reliability was set at 0.85, leaving a predicted confidence interval width of 0.7–1.0. The levels for type I and type II statistical errors were set at 0.05 and 0.8, respectively. Each patient performed ten trials to enhance reliability as previously recommended (Monaghan et al., 2007), thereby generating five trials with kinetic data for each lower limb. A sample size of 13 was calculated.

#### 7.4.5.2. Statistical measurement of reliability

The distribution of each variable was tested for normality using quantile-quantile (QQ) plots, stem-and-leaf plots and the Shapiro-Wilk test in Stata 11 (StataCorp, Texas, USA). Test-retest reliability was examined for each parameter using three statistical methods, 1) the ICC one-way random effects model for single measures, recommended for use in test-retest reliability studies (Fleiss, 1986), 2) the SEM calculated from the ICC and the parameter’s SD, using the formula described by (Streiner and Norman, 2008): \( SEM = SD \times \sqrt{1-ICC} \), and 3) Bland–Altman plots and 95% LOA (Bland and Altman, 1986). ICC values were calculated in Predictive Analytical Software (PASW) 18 (IBM Corporation, Armonk, NY, USA), while SEM and LOA were calculated in Excel for Macintosh 2008 (Microsoft Corporation, Redmond, WA, USA).
7.5 Results

7.5.1 Participants

Twenty-six people met the inclusion criteria between December 2008 and February 2010, and 13 of these, seven female and six male, agreed to participate. Of the 13 who did not take part, five participated in the cross-sectional and experimental studies, but declined to attend a second baseline assessment for reliability. Five declined to participate in any aspect of the study, and the remaining three participants were excluded, two due to pre-existing neurological conditions, and one due to inability to mobilise. In accordance with the inclusion criteria, all 13 had signs and symptoms of CSM, MRI evidence of cord compression, and no history of co-existing neurological problems or other medical conditions affecting gait. Three participants had a history of previous surgery for CSM. Their participation was limited to the reliability study only, and they did not go on to participate in the cross-sectional study or the experimental study. One participant complained of a painful knee on the second test day, resulting in an alteration in his gait pattern. His data were therefore excluded, as an appreciable change in his gait had occurred that was not related to CSM.

The characteristics of the remaining 12 participants are shown in Table 7.1. The mean age of the cohort was 54.3 years (range, 34–73 years). Participants had a median Nurick score of 2 (range, 1–4), a median mJOA score of 10.5 (range, 8–14), and a median MRMI score of 39 (range, 34–40). One participant (case 14) required the use of a crutch to mobilise independently. The remaining 11 could walk unaided. Kinetic data were not collected for two participants, as one failed to strike the force plate cleanly due to a short stride length, while the other used a crutch that contacted the force plate, contaminating the data.
Table 7.1: Characteristics of participants of the reliability study

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (years)</th>
<th>Gender</th>
<th>Duration of symptoms (months)</th>
<th>Previous surgery (Y/N)</th>
<th>Nurick</th>
<th>mJOA</th>
<th>MRMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>01</td>
<td>50</td>
<td>F</td>
<td>12</td>
<td>Y</td>
<td>3</td>
<td>12</td>
<td>38</td>
</tr>
<tr>
<td>02</td>
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<td>47</td>
<td>M</td>
<td>12</td>
<td>N</td>
<td>3</td>
<td>11</td>
<td>39</td>
</tr>
</tbody>
</table>

mJOA = modified Japanese Orthopaedic Association score, MRMI = Modified Rivermead Mobility Index

Case numbers reflect sequential recruitment to the overall study

Missing values from the sequence are those participants who did not attend for a second pre-operative or baseline assessment for reliability

7.5.2 Reliability of 3DGA parameters

7.5.2.1 Reliability of temporal-spatial parameters

TSPs showed excellent test-retest reliability. As shown in Table 7.2, ICCs were above 0.9 for all parameters with the exception of opposite foot contact (ICC 0.61). SEM values and Bland–Altman 95% LOA were low for all parameters. Figure 7.1 shows Bland–Altman plots for each TSP.
<table>
<thead>
<tr>
<th>Parameter</th>
<th>ICC</th>
<th>95% CI</th>
<th>SEM</th>
<th>Bland–Altman 95% LOA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICC</td>
<td>95% CI</td>
<td>D</td>
<td>SD(D)</td>
</tr>
<tr>
<td>Cadence (steps/min)</td>
<td>0.99</td>
<td>0.95</td>
<td>0.99</td>
<td>1.70</td>
</tr>
<tr>
<td>Double Support (s)</td>
<td>0.99</td>
<td>0.96</td>
<td>0.99</td>
<td>0.01</td>
</tr>
<tr>
<td>Foot Off (%)</td>
<td>0.97</td>
<td>0.90</td>
<td>0.99</td>
<td>0.81</td>
</tr>
<tr>
<td>Opposite Foot Contact (%)</td>
<td>0.61</td>
<td>0.11</td>
<td>0.87</td>
<td>1.01</td>
</tr>
<tr>
<td>Opposite Foot Off (%)</td>
<td>0.91</td>
<td>0.72</td>
<td>0.97</td>
<td>0.77</td>
</tr>
<tr>
<td>Single Support (s)</td>
<td>0.96</td>
<td>0.86</td>
<td>0.99</td>
<td>0.01</td>
</tr>
<tr>
<td>Step Length (m)</td>
<td>0.99</td>
<td>0.98</td>
<td>0.99</td>
<td>0.01</td>
</tr>
<tr>
<td>Step Width (m)</td>
<td>0.91</td>
<td>0.72</td>
<td>0.97</td>
<td>0.01</td>
</tr>
<tr>
<td>Stride Length (m)</td>
<td>0.99</td>
<td>0.98</td>
<td>0.99</td>
<td>0.02</td>
</tr>
<tr>
<td>Stride Time (s)</td>
<td>0.99</td>
<td>0.96</td>
<td>0.99</td>
<td>0.02</td>
</tr>
<tr>
<td>Step Time (s)</td>
<td>0.97</td>
<td>0.89</td>
<td>0.99</td>
<td>0.02</td>
</tr>
<tr>
<td>Gait Speed (m/s)</td>
<td>0.99</td>
<td>0.98</td>
<td>0.99</td>
<td>0.02</td>
</tr>
</tbody>
</table>

ICC = Intraclass correlation coefficient, CI = 95% confidence intervals for ICCs, SEM = standard error of measurement, LOA = Bland–Altman 95% limits of agreement, D = mean difference, SD(D) = standard deviation of the difference, s = seconds, m = metres, m/s = metres per second
Bland–Altman LOA were calculated using the formula 95% LOA = D ± 2 x SD(D) (Bland and Altman, 1986)
All values are rounded up to two decimal places for presentation in the table
Figure 7.1: Bland–Altman plots for temporal-spatial parameters
The x-axis shows the mean score and the y-axis shows the difference in scores
Dashed lines indicate 95% limits of agreement
7.5.2.2 Reliability of kinematic parameters

Reliability indices of kinematic parameters showed greater variation than those of TSPs. In general, total joint ROM in a plane was more reliable than a peak or specific point within that range, for example, the ICC for total hip sagittal plane motion was 0.95, whereas peak hip flexion and extension had ICC values of 0.89 and 0.74, respectively. Two parameters had ICC values below 0.6, and these were peak hip internal rotation (0.54) and ankle position at initial contact (0.33). SEMs were below 4° for all parameters except peak hip internal rotation (5.81°). Bland–Altman plots with 95% LOA are shown in Figures 7.2, 7.3 and 7.4. LOA values ranged from -1.63° to 1.73° for pelvic tilt range, to -20.44° to 18.47° for peak hip internal rotation. Reliability statistics for each parameter are listed in Table 7.3.
### Table 7.3: Reliability of kinematic parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>ICC</th>
<th>95% CI</th>
<th>SEM</th>
<th>Bland–Altman 95% LOA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak pelvic tilt</td>
<td>0.84</td>
<td>0.56</td>
<td>0.95</td>
<td>2.38</td>
</tr>
<tr>
<td>Total range pelvic tilt</td>
<td>0.87</td>
<td>0.63</td>
<td>0.96</td>
<td>0.55</td>
</tr>
<tr>
<td>Average pelvic tilt</td>
<td>0.78</td>
<td>0.43</td>
<td>0.93</td>
<td>2.57</td>
</tr>
<tr>
<td>Peak pelvic obliquity</td>
<td>0.64</td>
<td>0.16</td>
<td>0.88</td>
<td>1.62</td>
</tr>
<tr>
<td>Total range pelvic obliquity</td>
<td>0.92</td>
<td>0.74</td>
<td>0.98</td>
<td>0.92</td>
</tr>
<tr>
<td>Total range pelvic rotation</td>
<td>0.87</td>
<td>0.62</td>
<td>0.96</td>
<td>1.14</td>
</tr>
<tr>
<td>Peak hip flexion</td>
<td>0.89</td>
<td>0.68</td>
<td>0.97</td>
<td>2.99</td>
</tr>
<tr>
<td>Peak hip extension</td>
<td>0.74</td>
<td>0.33</td>
<td>0.92</td>
<td>3.19</td>
</tr>
<tr>
<td>Total hip range in sagittal plane</td>
<td>0.95</td>
<td>0.84</td>
<td>0.99</td>
<td>1.28</td>
</tr>
<tr>
<td>Peak hip abduction</td>
<td>0.69</td>
<td>0.24</td>
<td>0.90</td>
<td>2.25</td>
</tr>
<tr>
<td>Total hip range in frontal plane</td>
<td>0.85</td>
<td>0.58</td>
<td>0.95</td>
<td>1.05</td>
</tr>
<tr>
<td>Peak hip internal rotation</td>
<td>0.54</td>
<td>0.00</td>
<td>0.84</td>
<td>5.81</td>
</tr>
<tr>
<td>Total range hip rotation</td>
<td>0.87</td>
<td>0.62</td>
<td>0.96</td>
<td>3.66</td>
</tr>
<tr>
<td>Knee position at initial contact</td>
<td>0.66</td>
<td>0.19</td>
<td>0.89</td>
<td>3.11</td>
</tr>
<tr>
<td>Peak knee flexion in stance</td>
<td>0.62</td>
<td>0.12</td>
<td>0.87</td>
<td>3.67</td>
</tr>
<tr>
<td>Peak knee flexion in swing</td>
<td>0.65</td>
<td>0.17</td>
<td>0.88</td>
<td>3.32</td>
</tr>
<tr>
<td>Peak knee extension</td>
<td>0.62</td>
<td>0.12</td>
<td>0.87</td>
<td>3.12</td>
</tr>
<tr>
<td>Total knee range in sagittal plane</td>
<td>0.87</td>
<td>0.62</td>
<td>0.96</td>
<td>2.62</td>
</tr>
<tr>
<td>Ankle position at initial contact</td>
<td>0.33</td>
<td>-0.25</td>
<td>0.74</td>
<td>2.35</td>
</tr>
<tr>
<td>Peak ankle dorsiflexion in stance</td>
<td>0.62</td>
<td>0.12</td>
<td>0.87</td>
<td>1.37</td>
</tr>
<tr>
<td>Peak ankle dorsiflexion in swing</td>
<td>0.79</td>
<td>0.43</td>
<td>0.93</td>
<td>2.00</td>
</tr>
<tr>
<td>Peak ankle plantarflexion in swing</td>
<td>0.76</td>
<td>0.37</td>
<td>0.92</td>
<td>2.90</td>
</tr>
</tbody>
</table>

**Note:**
- ICC = Intraclass correlation coefficient, CI = 95% confidence intervals for ICCs, SEM = standard error of measurement, LOA = Bland–Altman 95% limits of agreement, D = mean difference, SD(D) = standard deviation of the difference.
- Bland–Altman LOA calculated using the formula 95% LOA = D ± 2 x SD(D) (Bland and Altman, 1986).
- All values are rounded up to two decimal places for presentation in the table.
- Abbreviations: °, degrees.
The x-axis shows the mean score and the y-axis shows the difference in scores.
Dashed lines indicate 95% limits of agreement.

Figure 7.2: Bland–Altman plots for kinematic parameters of the pelvis
Figure 7.3: Bland–Altman plots for kinematic parameters of the hip

The x-axis shows the mean score and the y-axis shows the difference in scores.
Dashed lines indicate 95% limits of agreement.
Figure 7.4: Bland–Altman plots for kinematic parameters of the knee and ankle
The x-axis shows the mean score and the y-axis, the difference in scores
Dashed lines indicate 95% limits of agreement
7.3.3.3 Reliability of kinetic parameters

Kinetic parameters generally showed excellent reliability, with most ICCs at least 0.85, as shown in Table 7.4. However, low ICCs were found for medio-lateral GRF (0.12), peak hip abductor moment (0.54), and peak eccentric power at the knee during loading response, K1 (0.56). SEM values for kinetic parameters are listed in Table 7.4, and Bland–Altman plots with 95% LOA are provided in Figures 7.5, 7.6 and 7.7. SEMs were generally small when referenced to the mean values for each parameter shown in the Bland–Altman plots. Bland–Altman 95% LOA are significantly larger than SEM values for the same parameters. Outliers are visible in some plots, including knee power at loading response and peak hip abductor moments.
Table 7.4: Reliability of kinetic parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>ICC</th>
<th>95% CI</th>
<th>SEM</th>
<th>Bland–Altman 95% LOA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICC</td>
<td>95% CI</td>
<td>D</td>
<td>SD (D)</td>
</tr>
<tr>
<td>Medio-lateral GRF</td>
<td>0.12</td>
<td>-0.50</td>
<td>0.67</td>
<td>0.05</td>
</tr>
<tr>
<td>First peak, antero-posterior GRF</td>
<td>0.87</td>
<td>0.60</td>
<td>0.97</td>
<td>0.16</td>
</tr>
<tr>
<td>Second peak, antero-posterior GRF</td>
<td>0.95</td>
<td>0.81</td>
<td>0.99</td>
<td>0.10</td>
</tr>
<tr>
<td>First peak, vertical GRF</td>
<td>0.84</td>
<td>0.51</td>
<td>0.96</td>
<td>0.40</td>
</tr>
<tr>
<td>Minimum vertical GRF in midstance</td>
<td>0.92</td>
<td>0.72</td>
<td>0.98</td>
<td>0.36</td>
</tr>
<tr>
<td>Second peak, vertical GRF</td>
<td>0.86</td>
<td>0.56</td>
<td>0.96</td>
<td>0.23</td>
</tr>
<tr>
<td>Hip extensor moment</td>
<td>0.66</td>
<td>0.13</td>
<td>0.90</td>
<td>122</td>
</tr>
<tr>
<td>Hip flexor moment</td>
<td>0.91</td>
<td>0.70</td>
<td>0.98</td>
<td>88</td>
</tr>
<tr>
<td>Hip abductor moment</td>
<td>0.54</td>
<td>-0.06</td>
<td>0.86</td>
<td>69</td>
</tr>
<tr>
<td>Knee extensor moment</td>
<td>0.89</td>
<td>0.63</td>
<td>0.97</td>
<td>56</td>
</tr>
<tr>
<td>Knee flexor moment</td>
<td>0.91</td>
<td>0.70</td>
<td>0.98</td>
<td>58</td>
</tr>
<tr>
<td>Ankle plantarflexor moment</td>
<td>0.94</td>
<td>0.79</td>
<td>0.98</td>
<td>53</td>
</tr>
<tr>
<td>Hip concentric power, loading (H1)</td>
<td>0.76</td>
<td>0.31</td>
<td>0.93</td>
<td>0.19</td>
</tr>
<tr>
<td>Hip eccentric power, midstance (H2)</td>
<td>0.86</td>
<td>0.56</td>
<td>0.96</td>
<td>0.10</td>
</tr>
<tr>
<td>Hip concentric power, terminal stance (H3)</td>
<td>0.95</td>
<td>0.82</td>
<td>0.99</td>
<td>0.12</td>
</tr>
<tr>
<td>Knee eccentric power, loading response (K1)</td>
<td>0.56</td>
<td>-0.03</td>
<td>0.87</td>
<td>0.42</td>
</tr>
<tr>
<td>Knee concentric power, midstance (K2)</td>
<td>0.80</td>
<td>0.40</td>
<td>0.94</td>
<td>0.17</td>
</tr>
<tr>
<td>Knee eccentric power, terminal stance (K3)</td>
<td>0.92</td>
<td>0.73</td>
<td>0.98</td>
<td>0.11</td>
</tr>
<tr>
<td>Knee eccentric power, terminal swing (K4)</td>
<td>0.81</td>
<td>0.43</td>
<td>0.95</td>
<td>0.12</td>
</tr>
<tr>
<td>Ankle eccentric power, loading (A1)</td>
<td>0.91</td>
<td>0.69</td>
<td>0.98</td>
<td>0.12</td>
</tr>
<tr>
<td>Ankle concentric power, terminal stance (A2)</td>
<td>0.91</td>
<td>0.71</td>
<td>0.98</td>
<td>0.36</td>
</tr>
</tbody>
</table>

ICC = Intraclass correlation coefficient, CI = 95% confidence intervals for ICCs, SEM = standard errors of measurement, LOA = Bland–Altman 95% limits of agreement, D = mean difference, SD(D) = standard deviation of the difference, GRF = ground reaction force
Bland–Altman LOA calculated using the formula 95% LOA = D ± 2 x SD(D) (Bland and Altman, 1986)
Units of measurement: GRF, Newtons per kilogram (N/kg), moments, Newton millimetres per kilogram (Nmm/kg), powers, Watts per kilogram (W/kg)
All values are rounded up to two decimal places for presentation in the table.
Figure 7.5: Bland–Altman plots for peak ground reaction forces
The x-axis shows the mean score and the y-axis, the difference in scores
Dashed lines indicate 95% limits of agreement
GRF = ground reaction force, N = Newtons, kg = kilograms
Figure 7.6: Bland–Altman plots for peak joint moments
The x-axis shows the mean score and the y-axis, the difference in scores
95% limits of agreement (LOA) are shown in the dashed lines
Nmm = Newton millimetres, kg = kilograms
The x-axis shows the mean score and the y-axis, the difference in scores. 95% limits of agreement (LOA) are shown in the dashed lines.

\[ W \text{ = Watts, } \text{kg = kilograms} \]
7.5.3 Reliability of EMG parameters

7.5.3.1 Reliability of EMG timing parameters

The total duration of each muscle’s activation time was calculated for individual GCs using the DTM based on the TKEO equation, as previously described in Chapter 6, Section 6.7.3.2. Reliability indices of the mean activation times of 10 GCs in each of the two test days were then calculated. Results are shown in Table 7.5. Timing of TA demonstrated the highest test-retest reliability overall, with an ICC of 0.81 and a SEM of 5.5% GC duration. RF, BF and MG had ICCs of 0.59, 0.56 and 0.55 respectively. SEMs were 5.5% GC duration or below for three of the four muscles with the exception of BF, which had a SEM of 9.3% GC duration. Bland–Altman 95% LOA were larger than the corresponding SEMs. Bland–Altman plots are shown in Figure 7.8.

Table 7.5: Reliability of the total timing of muscle activity in the gait cycle

<table>
<thead>
<tr>
<th>Muscle</th>
<th>ICC</th>
<th>SEM (% GC)</th>
<th>Bland–Altman LOA (% GC)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICC</td>
<td>95% CI</td>
<td>D</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>0.59</td>
<td>.080</td>
<td>.861</td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>0.56</td>
<td>.030</td>
<td>.847</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>0.81</td>
<td>.474</td>
<td>.939</td>
</tr>
<tr>
<td>Medial gastrocnemius</td>
<td>0.55</td>
<td>.015</td>
<td>.843</td>
</tr>
</tbody>
</table>

*ICC = intraclass correlation coefficient, SEM = standard error of measurement, CI = confidence interval, GC = gait cycle, LOA = limits of agreement, D = difference, SD (D) = standard deviation of the difference*
Figure 7.8: Bland–Altman plots for timing profiles of the rectus femoris, biceps femoris, tibialis anterior and medial gastrocnemius

The x-axis shows the mean activation time of the muscle across the two assessments, and the y-axis shows the difference in activation time between the two assessments. Dashed lines indicate 95% limits of agreement. Mean and difference are expressed as a percentage of gait cycle duration (% GC).

7.5.3.2 Reliability of EMG amplitude parameters

Reliability of mean and peak RMS amplitudes of key bursts of muscle activity, and the mean RMS amplitude during a muscle’s baseline phases of inactivity over the GC, were analysed. Two sets of amplitude data were generated, one normalised to MVC, and the other normalised using PDM. Both methods were described in Chapter 6, Section 6.7.3.3. The reliability of amplitude parameters normalised to the two methods will be presented separately.

7.5.3.2.1 MVC normalisation method

The MVC normalisation method yielded poor results for reliability. Cronbach’s alpha was negative for some variables, including the peak amplitude of the stance and swing bursts of activity of TA, MG and RF, and the mean amplitude of the stance bursts of TA and RF. This indicated a lack of internal consistency between the values obtained in the two test days, and therefore ICC and SEM were not calculated for these variables. ICCs were poor for the remaining parameters. Three of these, peak amplitude of BF stance burst, peak amplitude of BF swing burst, and mean amplitude of BF swing burst, achieved ICC values above 0.6. SEM values and 95% LOA were of the order of 30% MVC for mean and peak burst amplitudes for all muscles. The SEM values for amplitude during baseline...
phases were less than 5% MVC for BF and RF, but close to 19% for TA, and could not be calculated for MG. Results of the reliability of amplitude variables using MVC normalisation are shown in Table 7.6.
### Table 7.6: Reliability of the amplitude of key muscle activity bursts, normalised to maximum voluntary contraction (MVC)

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Amplitude</th>
<th>ICC</th>
<th>95% CI</th>
<th>SEM</th>
<th>Bland–Altman 95% limits of agreement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>D</td>
</tr>
<tr>
<td><strong>Rectus femoris</strong></td>
<td>Mean burst amplitude, stance</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>35.14</td>
</tr>
<tr>
<td></td>
<td>Mean burst amplitude, swing</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>80.42</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, stance</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>12.59</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, swing</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>-21.66</td>
</tr>
<tr>
<td></td>
<td>Mean amplitude, baseline</td>
<td>0.07</td>
<td>-0.49</td>
<td>0.59</td>
<td>4.82</td>
</tr>
<tr>
<td></td>
<td>Mean burst amplitude, pre swing</td>
<td>0.00</td>
<td>-0.58</td>
<td>0.60</td>
<td>15.43</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, pre swing</td>
<td>-0.07</td>
<td>-0.62</td>
<td>0.55</td>
<td>22.30</td>
</tr>
<tr>
<td><strong>Biceps femoris</strong></td>
<td>Mean burst amplitude, stance</td>
<td>0.54</td>
<td>0.00</td>
<td>0.84</td>
<td>13.65</td>
</tr>
<tr>
<td></td>
<td>Mean burst amplitude, swing</td>
<td>0.62</td>
<td>0.13</td>
<td>0.87</td>
<td>15.00</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, stance</td>
<td>0.64</td>
<td>0.16</td>
<td>0.88</td>
<td>23.37</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, swing</td>
<td>0.60</td>
<td>0.09</td>
<td>0.86</td>
<td>26.66</td>
</tr>
<tr>
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<td>Mean amplitude, baseline</td>
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<td>0.05</td>
<td>0.85</td>
<td>4.74</td>
</tr>
<tr>
<td><strong>Tibialis anterior</strong></td>
<td>Mean burst amplitude, stance</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>-0.02</td>
</tr>
<tr>
<td></td>
<td>Mean burst amplitude, swing</td>
<td>0.20</td>
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<td>50.28</td>
</tr>
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<td></td>
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<td>0.66</td>
<td>87.28</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, swing</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>33.54</td>
</tr>
<tr>
<td></td>
<td>Mean amplitude, baseline</td>
<td>0.12</td>
<td>-0.45</td>
<td>0.63</td>
<td>18.95</td>
</tr>
<tr>
<td><strong>Medial gastrocnemius</strong></td>
<td>Mean burst amplitude, stance</td>
<td>0.08</td>
<td>-0.48</td>
<td>0.60</td>
<td>49.18</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, stance</td>
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<td>*</td>
<td>*</td>
<td>11.73</td>
</tr>
<tr>
<td></td>
<td>Mean activity, baseline</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>18.37</td>
</tr>
</tbody>
</table>

*Results for SEM and Bland–Altman 95% LOA are expressed as a percentage of MVC.
* indicates that Cronbach’s alpha was negative for internal consistency, therefore ICC and SEM were not calculated.
ICC = intraclass correlation coefficient, SEM = standard error of measurement, CI = confidence interval, D = mean difference between scores of first and second assessment, SD(D) = standard deviation of the difference between scores of the first and second assessment.
PDM normalisation method

PDM normalisation yielded higher reliability results than the MVC method. Four parameters, mean amplitude of RF stance burst, peak amplitude of BF stance burst, and mean amplitude during MG and TA baseline phases, had ICCs of 0.7 or higher. However, one of these, peak amplitude of BF stance burst, had a high SEM of over 30% RMS_{MAX}, indicating high absolute variability of test-retest scores. SEM values for other variables were of the order of 1% RMS_{MAX} for mean activity at baseline during gait, 4–10% RMS_{MAX} for mean amplitude during bursts, and 7–15% RMS_{MAX} for peak amplitude during bursts. The mean and peak amplitudes during RF swing were not internally consistent according to Cronbach’s alpha, and therefore their ICC and SEM were not calculated. The peak amplitude of MG during stance had a constant value of 100% RMS_{MAX} for all participants, and therefore reliability statistics could not be calculated. Table 7.7 shows the reliability results for amplitude parameters normalised using PDM. Bland–Altman plots with 95% LOA are shown in Figures 7.9 and 7.10.
<table>
<thead>
<tr>
<th>Muscle</th>
<th>Amplitude</th>
<th>ICC</th>
<th>SEM</th>
<th>Bland–Altman 95% limits of agreement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>ICC</td>
<td>95% CI</td>
<td>SEM</td>
</tr>
<tr>
<td><strong>Rectus femoris</strong></td>
<td>Mean burst amplitude, stance</td>
<td>0.70</td>
<td>0.25</td>
<td>0.90</td>
</tr>
<tr>
<td></td>
<td>Mean burst amplitude, swing</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, stance</td>
<td>0.63</td>
<td>0.15</td>
<td>0.88</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, swing</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>Mean amplitude, baseline</td>
<td>0.25</td>
<td>-0.33</td>
<td>0.70</td>
</tr>
<tr>
<td></td>
<td>Mean burst amplitude, pre swing</td>
<td>0.32</td>
<td>-0.33</td>
<td>0.77</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, pre swing</td>
<td>0.18</td>
<td>-0.45</td>
<td>0.70</td>
</tr>
<tr>
<td><strong>Biceps femoris</strong></td>
<td>Mean burst amplitude, stance</td>
<td>0.63</td>
<td>0.14</td>
<td>0.88</td>
</tr>
<tr>
<td></td>
<td>Mean burst amplitude, swing</td>
<td>0.46</td>
<td>-0.11</td>
<td>0.80</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, stance</td>
<td>0.79</td>
<td>0.43</td>
<td>0.93</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, swing</td>
<td>0.36</td>
<td>-0.22</td>
<td>0.76</td>
</tr>
<tr>
<td></td>
<td>Mean amplitude, baseline</td>
<td>0.54</td>
<td>0.00</td>
<td>0.84</td>
</tr>
<tr>
<td><strong>Tibialis anterior</strong></td>
<td>Mean burst amplitude, stance</td>
<td>0.55</td>
<td>0.02</td>
<td>0.84</td>
</tr>
<tr>
<td></td>
<td>Mean burst amplitude, swing</td>
<td>0.50</td>
<td>-0.05</td>
<td>0.82</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, stance</td>
<td>0.47</td>
<td>-0.09</td>
<td>0.81</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, swing</td>
<td>0.41</td>
<td>-0.16</td>
<td>0.78</td>
</tr>
<tr>
<td></td>
<td>Mean amplitude, baseline</td>
<td>0.89</td>
<td>0.67</td>
<td>0.97</td>
</tr>
<tr>
<td><strong>Medial gastrocnemius</strong></td>
<td>Mean burst amplitude, stance</td>
<td>0.39</td>
<td>-0.18</td>
<td>0.77</td>
</tr>
<tr>
<td></td>
<td>Peak burst amplitude, stance</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>Mean activity, baseline</td>
<td>0.79</td>
<td>0.44</td>
<td>0.93</td>
</tr>
</tbody>
</table>

**Table 7.7: Reliability of the amplitude of key muscle activity bursts, normalised using the peak dynamic method (PDM)**

ICC = intraclass correlation coefficient, SEM = standard error of measurement, CI = confidence interval, D = mean difference between scores, SD(D) = standard deviation of the difference between scores, LOA = limits of agreement

SEM and Bland–Altman 95% LOA are expressed as a percentage of RMSMAX

* indicates that Cronbach’s alpha was negative for internal consistency, therefore ICC and SEM were not calculated

** The peak activity during MG stance was 100% RMSMAX for all participants and had zero variability, therefore reliability could not be calculated
The x-axis shows the mean activation time of the muscle across the two assessments, and the y-axis shows the difference in activation time between the two assessments.

Dashed lines indicate 95% limits of agreement.

Mean and difference are expressed as a percentage of gait cycle duration (% GC).

Figure 7.9: Bland–Altman plots for PDM-normalised amplitude parameters of medial gastrocnemius (a), rectus femoris (b)
The x-axis shows the mean activation time of the muscle across the two assessments, and the y-axis shows the difference in activation time between the two assessments. Dashed lines indicate 95% limits of agreement. Mean and difference are expressed as a percentage of gait cycle duration (% GC).
7.5.3.3 Reliability of locomotor-specific measure of spasticity

The reliability of three indices to describe a muscle’s response to lengthening were examined, 1) critical time of EMG onset during lengthening, 2) LVT, the lengthening velocity at onset of EMG activity during muscle lengthening, and 3) the slope of the muscle’s time- and amplitude-normalised EMG signal versus its lengthening velocity, indicating the rate of change of EMG amplitude with respect to lengthening velocity. A full description of these indices was provided in Chapter 6, Section 6.7.4.3. LVT was the most reliable of the three indices, with ICCs ranging from 0.77 (MG) to 0.89 (BF). SEM values ranged from 0.18 normalised lengths per second (l0/s) for BF to 0.42 l0/s for RF. The critical time of EMG onset was very reliable for TA (ICC 0.91, SEM 2.36% GC duration), but less reliable (ICC 0.35) for BF and MG. Reliability of the slope index was the least favourable of the three indices, with ICC values below 0.6 for all muscles and large SEM and LOA values, particularly for TA (SEM 11.18 % RMSMAX/ms, LOA -29.59 → 42.61 % RMSMAX/ms). Table 7.8 shows the ICC, SEM and LOA values for each muscle’s critical time, LVT and slope. Bland–Altman plots are shown in Figure 7.11.
Table 7.8: Reliability of locomotor-specific measure of spasticity parameters

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Index</th>
<th>ICC</th>
<th>95% CI</th>
<th>SEM</th>
<th>ICC</th>
<th>95% CI</th>
<th>D</th>
<th>SD (D)</th>
<th>95% LOA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Critical time</td>
<td>0.63</td>
<td>0.13</td>
<td>0.87</td>
<td>3.70</td>
<td>0.63</td>
<td>6.02</td>
<td>-11.40</td>
<td>12.67</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>LVT</td>
<td>0.81</td>
<td>0.49</td>
<td>0.94</td>
<td>0.41</td>
<td>0.05</td>
<td>0.63</td>
<td>-1.21</td>
<td>1.31</td>
</tr>
<tr>
<td></td>
<td>Slope</td>
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<td>-0.14</td>
<td>0.79</td>
<td>1.60</td>
<td>0.61</td>
<td>2.71</td>
<td>-4.81</td>
<td>6.03</td>
</tr>
<tr>
<td></td>
<td>Critical time</td>
<td>0.35</td>
<td>-0.23</td>
<td>0.75</td>
<td>3.04</td>
<td>1.42</td>
<td>5.27</td>
<td>-9.11</td>
<td>11.95</td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>LVT</td>
<td>0.89</td>
<td>0.68</td>
<td>0.97</td>
<td>0.17</td>
<td>0.06</td>
<td>0.25</td>
<td>-0.44</td>
<td>0.56</td>
</tr>
<tr>
<td></td>
<td>Slope</td>
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<td>0.04</td>
<td>0.85</td>
<td>6.90</td>
<td>-8.18</td>
<td>7.76</td>
<td>-23.69</td>
<td>7.33</td>
</tr>
<tr>
<td></td>
<td>Critical time</td>
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<td>0.72</td>
<td>0.97</td>
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<td>-0.91</td>
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<td>-7.42</td>
<td>5.61</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>LVT</td>
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<td>0.57</td>
<td>0.95</td>
<td>0.29</td>
<td>-0.18</td>
<td>0.41</td>
<td>-1.00</td>
<td>0.64</td>
</tr>
<tr>
<td></td>
<td>Slope</td>
<td>0.37</td>
<td>-0.21</td>
<td>0.76</td>
<td>9.55</td>
<td>5.87</td>
<td>15.90</td>
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<td>37.68</td>
</tr>
<tr>
<td></td>
<td>Critical time</td>
<td>0.35</td>
<td>-0.23</td>
<td>0.75</td>
<td>3.54</td>
<td>1.53</td>
<td>6.16</td>
<td>-10.79</td>
<td>13.86</td>
</tr>
<tr>
<td>Medial gastrocnemius</td>
<td>LVT</td>
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<td>0.40</td>
<td>0.93</td>
<td>0.22</td>
<td>-0.12</td>
<td>0.31</td>
<td>-0.75</td>
<td>0.51</td>
</tr>
<tr>
<td></td>
<td>Slope</td>
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<td>0.08</td>
<td>0.86</td>
<td>1.19</td>
<td>-0.04</td>
<td>1.96</td>
<td>-3.97</td>
<td>3.88</td>
</tr>
</tbody>
</table>

LVT = lengthening velocity threshold, LOA = limits of agreement

Critical time is the time, as a percentage of gait cycle duration, at which EMG onset occurs during lengthening.

LVT is the lengthening velocity (in normalised lengths per second) at which EMG onset occurs.

Slope is the change in EMG amplitude per unit change in lengthening velocity during the phase of lengthening (see Section 6.7.4.3 for a detailed description).
Figure 7.11: Bland–Altman plots for locomotor-specific measurement of spasticity

The x-axis shows the mean activation time of the muscle across the two assessments, and the y-axis shows the difference in activation time between the two assessments. Dashed lines indicate 95% limits of agreement (LOA). Mean and difference are expressed as a percentage of gait cycle duration. GC = gait cycle, l/s = normalised muscle lengths per second, % RMS\textsubscript{MAX} = normalised amplitude.
The aims of this study were to evaluate the reliability of 3DGA and SEMG in measuring gait in CSM, and to estimate the change required to exceed measurement error when applying these variables to clinical practice and research. It was found that TSPs demonstrated high reliability, with SEM values below what would be considered meaningful change in clinical practice. For example, the SEM for gait speed, an outcome measure commonly used to evaluate severity of CSM (Singh and Crockard, 1999), was 0.02 m/s, whereas a change of 0.1 m/s, five times the magnitude of the SEM, has been suggested as a clinically-meaningful change (Judge et al., 1996b).

When measuring kinematic parameters, it has been suggested that errors larger than 5° could mislead clinical interpretation (McGinley et al., 2009). With the exception of peak hip internal rotation, the SEMs for kinematic key points in this CSM cohort were all below 4°, and most were below 3°. This indicates a sufficiently high level of reliability for use in clinical practice. Similarly, kinetic parameters showed a general trend of high ICCs and low SEMs. Medio-lateral GRF had a low ICC, consistent with other studies (Rabuffetti and Frigo, 2001), but its SEM of 0.05 Newtons per kilogram (N/kg) was also low, indicating a high degree of absolute agreement between test-retest scores.

Bland–Altman 95% LOA were reported for all parameters, however these values are significantly greater than the corresponding SEM. This may be explained by the small sample size in this study. Sample sizes of at least 50 have been recommended when calculating LOA (Rankin and Stokes, 1998). In the current study, Bland–Altman plots for some parameters revealed a number of outliers that may have contributed to a large standard deviation of the difference. The effect of these outliers might be reduced in a larger sample size. There are no general guidelines on the interpretation of the 95% LOA, and each must be considered with reference to the range of the raw data (Monaghan et al., 2007).

The ICC has been recommended for use in reliability studies because of its flexibility in evaluating different study designs, and its ability to isolate factors affecting reliability, such as intra-tester compared to inter-tester variation (Streiner and Norman, 2008). A criticism of the ICC is the extent to which it is influenced by between-subjects variance. It measures the ratio of true score variance to true variance plus error, and as such it will invariably be low in situations where there is little variation among subjects. This limitation is illustrated by ankle position at initial contact, which had a low ICC of 0.33 but a SEM of 2.35°, still within an acceptable error range. Similarly, the low ICC of peak medio-lateral GRF was also associated with an acceptable SEM (0.05 N/kg).
not the value of the reliability coefficient, but rather, whether the measurement error renders the instrument practical for clinical use (Bruton et al., 2000).

7.6.2 Reliability of EMG parameters of timing, amplitude and response to lengthening

The reproducibility of many EMG indices of locomotor function was previously unknown and limited their applicability in repeated measures design studies (Lamontagne, 2006). The current study provides some evidence for the reproducibility of timing and amplitude parameters, and the LSMS. With regard to timing, the SEM values for RF, MG and TA indicated that a change in activation time of around 5.5% GC duration could be considered a real change. This represents a change of about 55 ms, based on an average GC duration of one second. Activity bursts of 30 ms or less are have little effect on the resulting kinetic output (Bogey et al., 1992). It is therefore likely that a SEM of 5% would not cause clinically relevant differences to be masked by error.

The large SEM for BF timing was not in keeping with the results from the other muscles. Bland–Altman plots of Figure 7.8 show some outlying data points that may have adversely affected the reliability analysis. It is possible that the effect of outliers on the timing data of BF, and indeed the other muscles, would be reduced if a larger sample size were employed. BF is less superficial than the other muscles, and may be more prone to greater test-retest variation in electrode placement. It is also possible that its timing may be more intrinsically variable in this neurological population. Further investigation in larger sample sizes and in other populations could identify the contributory factors to this finding.

Reliability analysis of the amplitude of EMG activity, an indirect measure of the intensity of muscle activation, yielded mixed results. Inter-session and inter-participant variations in electrode placement, the variable number and size of motor units sampled, and environmental conditions, preclude the analysis of the absolute measurement of signal amplitude in Volts (Lehman and McGill, 1999). Normalisation to a reference level of EMG amplitude is therefore required for reproducibility, however the reliability of the resulting normalised parameters has not previously been examined in relation to gait in a neurological population. This study found that, with the possible exception of BF, normalisation to MVC yielded unacceptably poor test-retest reliability in determining EMG amplitude parameters in gait in CSM. No parameter had an ICC above 0.7, and only three parameters, pertaining to BF, had ICCs above 0.6. SEM values were below 5% MVC for two parameters, the mean amplitude of the “off” phases of BF and RF, but generally of the order of at least 15–20%, and in some cases up to 50%, MVC.

It is likely that the high variability in the results between test days is due to variation in the performance of MVC itself. The ability of people with neurological impairment to reliably
produce MVC has been questioned (Perry, 1992, Damiano et al., 2000), but its relevance to gait has not previously been tested. The current study was limited by the lack of a dynamometer to ensure that a consistent force was produced during the performance of MVC on both days. This might have established whether the source of error was the production of MVC, or the recording of its associated EMG activity. However, from a clinical point of view, the methodology of this study was carefully applied using standardised test procedures and instructions to participants. Aside from the variation in electrode placement, which in theory should not be a factor for the normalised signal as the gait signals are referenced to a maximum value obtained with the same electrode location, the most plausible reason for variation in test-retest scores was variability in the number of motor units activated, and their intensity, in performing MVC on the two test days.

PDM normalisation uses the peak amplitude obtained during gait itself as the normalisation reference. This method has been used in studies of gait in neurological populations, such as Lamontagne et al. (2001). The current study took the PDM normalisation reference for each test day to be the maximum RMS value achieved over the ten gait trials from that session, designated RMS\text{MAX}. Each data point in the time-normalised RMS of the signal was then expressed as a percentage of RMS\text{MAX}. Test-retest reliability analysis yielded somewhat more favourable results for PDM compared to MVC normalisation. ICCs of 0.7 or higher were achieved for the mean amplitude of the RF loading response burst, peak amplitude of the BF stance burst, and mean amplitude of TA and MG during baseline phases. However, a number of parameters achieved ICC values of 0.6 or lower. SEM values were of the order of 1% RMS\text{MAX} for mean activity during the baseline phases, 4–10% RMS\text{MAX} for mean activity during bursts, and 7–15% RMS\text{MAX} for peak activity during bursts. The results suggest that mean normalised RMS amplitude during a burst of activity may be a more reliable indicator of the intensity of muscle activation, than the peak RMS amplitude. Although the signal’s RMS was smoothed by the use of a 30 ms time window, it is not possible, or indeed desirable, to remove all signal fluctuations. It is possible that the peak amplitude in a burst is more prone to variation due to these inherent fluctuations in the signal, and that mean amplitude is a more consistent indicator of the intensity of a burst of activity, as it reflects the overall work done by a muscle during that burst of activity.

Finally, test-retest reliability of indices of a muscle’s response to lengthening, the LSMS, showed that LVT was the most repeatable measure. Its ICCs ranged from 0.77 to 0.89, indicating good to excellent reliability. SEM values for LVT were 0.17–0.41 l0/s. Clinically meaningful change for this measure in CSM is as yet unknown. A study of medial hamstrings activity in gait in children with spastic CP and typically-developing children found a statistically significant difference in LVT of 0.6 l0/s (reported in percentages as 60% total length per second) between the two groups (Crenna, 1999). This value of 0.6
lo/s is more than three times the SEM of BF LVT in this study, 0.17 lo/s, indicating that clinically meaningful change would probably not be masked by measurement error.

The critical time of EMG activity onset during lengthening showed mixed relative reliability, with ICCs of 0.35–0.91, but better absolute reliability, with SEMs of 2.24–3.7% GC duration for all muscles. This parameter is less useful than LVT as an indicator of spasticity, as it measures only the time at which muscle activation occurs during muscle lengthening, and not the length of the muscle or its lengthening velocity. However, it may serve as a useful indicator of premature activity in relation to timing of the GC, and allow correlations to be generated with the timing of other parameters. Its SEM of 2–3% GC duration equates to around 20–30 ms based on an average GC duration of one second. This is considered below the time of a clinically meaningful change in muscle activation (Bogey et al., 1992), suggesting that critical time is sufficiently reliable for use in clinical studies and research.

The slope parameter, adapted for use from Lamontagne et al. (2001), showed the poorest reliability of the three LSMS indices. As a measure of rate of change of one normalised parameter (RMS amplitude) with respect to another normalised parameter (muscle lengthening velocity), it is more difficult to interpret intuitively than LVT. It showed greater absolute variability, as indicated by the variation in the mean scores of the Bland–Altman plots shown in Figure 7.11. Its poorer reliability may be due to the fact that it depends on RMS amplitude, which in itself shows poor test-retest reliability as discussed in the previous paragraphs, whereas LVT is based on the time at which muscle activation occurs and not the amplitude of this activation.

Lamontagne et al. (2001) suggested that the frequency of positive slopes, rather than the magnitude of the slope itself, could be used as an indicator of spasticity, as a positive slope is considered to represent a pathological response to lengthening. As shown in the Bland–Altman plots, positive slopes were evident in one participant for BF, six participants for TA, and all participants for MG, while no participant had a positive slope for RF. It is possible that the direction of the slope, rather than its magnitude, could distinguish between people with pathological responses to lengthening and those with normal responses. In people with stroke, the frequency of positive slopes was significantly higher in MG during gait than in matched HCs at the same speed (Lamontagne et al., 2001). Further research is necessary to determine whether this is true of all muscles, and whether it applies equally to other neurological populations, including CSM.

### 7.6.3 Sources of variability in gait data

Variability in gait data can be attributed to two sources, intrinsic variability or true variation in the patient’s gait pattern, and extrinsic variability due to methodological errors in
marker application, anthropometric measurements, or calibration of the motion capture system (Schwartz et al., 2004). It is generally accepted that the major source of extrinsic error in 3DGA data is in marker application (McGinley et al., 2009). We found that total ROM in a plane was generally more reliable than peaks within that range, for example, total sagittal plane range at the knee joint had an ICC of 0.87 and a SEM of 2.62°, whereas the values for peak flexion in swing were 0.65 and 3.32°, and for peak extension, 0.62 and 3.12°. This might be due to variations in marker placement, resulting in an offset from flexion to extension where the total range is in fact unchanged.

Intrinsic variability can be estimated by measuring the reliability of gait trials within the same session, when methodological issues such as marker placement and calibration errors would not influence gait variability (Schwartz et al., 2004). There may also be some intrinsic variability in gait performance from day to day, particularly in patients with neurological conditions, where factors such as spasticity or fatigue may influence the strategies used in gait (Redekop et al., 2008). This intrinsic variability can provide valuable information about the condition of the patient, however it may be difficult to distinguish from measurement error. The current study found that two parameters pertaining to the action of the plantarflexors, peak ankle plantarflexion angle and concentric ankle power at terminal stance, were less reliable than other peak values in the same curves. This may suggest that certain phases of the GC could be inherently more variable in the CSM population. Further studies will be needed to investigate these findings, and to establish the characteristics of CSM gait in people with different levels of disease severity.

It is known that reliability will vary for the same instrument across populations and across different levels of severity (Streiner and Norman, 2008). Our study included a wide range of CSM severity, from those with little or no difficulty in walking (Nurick grade 1), to one patient who required an aid (Nurick grade 4). A previous study found that reliability of gait data was lower in children with more severe CP (Redekop et al., 2008). The current study was not sufficiently powered to examine reliability within sub-groups. However, such a wide range of impairment represents the spectrum of disease severity in the CSM population. It has been recommended that studies of reliability should recruit samples that represent the populations of interest (McGinley et al., 2009).

The lower reliability, and thus greater variability, of EMG indices compared to TSP, kinematic and kinetic data, is of interest in the interpretation of gait. The inherent limitations of EMG in relation to electrode placement, including variable sampling of motor units, and the need for filtering and extraction of key parameters from an inherently variable signal, have been discussed in Chapter 5, Section 5.4. These extrinsic factors may have affected the test-retest reliability, and have previously been acknowledged as the most important source of variability in EMG studies (Lehman and McGill, 1999).
However, it is possible that other factors contribute to the changes in EMG measurements from test to retest. TSPs, kinematics and kinetics represent the end products of an interaction between the biological signals responsible for muscle activation and the musculoskeletal system. Reliability analysis demonstrates that this end product is relatively consistent from test to retest, so the question must then be posed as to why there is so much variability in the underlying EMG signals. There are a number of possible reasons for this. The first possibility is that the level of processing needed to extract clinically relevant parameters from the EMG signal’s random series of spikes reduces the accuracy of the relationship between the extracted parameter and the clinical entity it is designed to measure. This may be the case particularly with measures of EMG amplitude, when condensing a rapidly varying signal into a mean or peak value over a large burst of activity may not reflect the complexities of the task achieved by the smaller peaks and troughs within that burst. In other words, the problem may be one of validity, in that the calculation of EMG amplitude over a burst does not measure the intensity of muscle activation, as it is designed to do.

The second possibility is that there is greater intrinsic variability in muscle activation as measured by EMG, perhaps relating to fatigue or pain, than the outputted kinematic and TSPs would indicate. In other words, the locomotor system may be very adept at producing a consistent output using motor strategies that are not necessarily constructed in a consistent manner. However, it is difficult to understand the extent of this without similar studies of reliability in healthy populations. In addition to these factors, which are intrinsic to the muscles themselves, there may also be slight fluctuations in the orientation of the limbs and the centre of mass, and in the activation of other untested agonist muscles. These would require varying degrees of compensation by the tested muscles, but are not reflected in the calculation of lower limb kinematics and kinetics. Muscles need to respond to changes in posture, the environment, and sensory input from the supporting surface, and in people with neurological injuries, these responses may not be consistent. These inconsistencies could be reflected in the variable amplitude parameters measured by EMG.

Variation in test-retest scores cannot therefore be dismissed entirely as measurement error, particularly in this neurological population. However, intrinsic variability has the effect of increasing the minimal detectable change of a measure, the minimal amount of change that is not likely to be due to chance or measurement error (Haley and Fragala-Pinkham, 2006). Therefore, a true improvement cannot be reported with certainty until the post-intervention test result exceeds the variability of the pre-intervention test by at least one SEM, regardless of whether the source of that variation is intrinsic or extrinsic.
7.6.4 Choice of parameters for evaluation of gait in CSM and healthy controls

The relative (ICC) and absolute (SEM) reliability of an array of TSP, kinematic, kinetic and EMG parameters showed acceptable reliability for use in clinical practice and in repeated measures design studies, with the exception of some parameters such as MVC normalisation and peak hip internal rotation range. The choice of parameters from the list of those tested for reliability depends on the aspects of gait that are of greatest clinical interest in the CSM population. On review of the kinematic parameters, it was decided to exclude peak and range of pelvic tilt from the remainder of the thesis, as the average position of the pelvis in the sagittal plane, average pelvic tilt, has been quoted in previous studies of gait in neurological populations (Williams et al., 2009b), and is of greater clinical interest. Peak hip internal rotation and peak hip abduction were also excluded, as the total ranges in these planes were more reliable than the peaks. The remaining TSP, kinematic and kinetic parameters were retained for further analysis in the cross-sectional and repeated measures design studies.

In relation to EMG, the reliability of timing parameters, extracted using the TKEO-based DTM routine, was considered acceptable. The high SEM of BF timing parameters meant that changes in test-retest scores would need to exceed almost 10% GC duration to be deemed clinically meaningful. However, as no superior method to determine timing in this muscle had been identified, and changes of this magnitude could occur in people with more severe CSM compared to HCs, it was considered acceptable to include BF timing as a parameter.

Amplitude parameters showed high test-retest variability and comparatively low reliability, compared to other parameters. Of the two methods of normalisation, amplitude parameters normalised using PDM showed higher reliability than when normalised to MVC. From a validity point of view, PDM lacks a clear physiological reference, which is a potential advantage of MVC. However, the poor reliability results of MVC normalisation suggest that the ability of people with CSM to produce a true maximum voluntary activation should be questioned, and that MVC may not be a valid normalisation reference in this population. Therefore, despite its limitations, PDM was chosen as the normalisation method for further use in the current study. The bursts of activation that were tested in the reliability study were included in the cross-sectional and repeated measures design studies, but limited to the analysis of mean amplitude, rather than peaks, of these bursts due to the superior reliability of the mean values. The results, however, will need to be interpreted in the context of limited reliability and the potential contributing factors to test-retest variability discussed in Section 7.4.4.

Finally, of the possible parameters included in the LSMS, LVT showed highest reliability, and SEM values suggested that the measurement error was small enough to avoid
masking clinically relevant change. Critical time of activation onset was more variable, but it was decided to include it as a descriptive indicator of muscle response to lengthening, as it is of clinical interest to know the point during lengthening at which muscle activation occurs. The slope criterion demonstrated poor reliability, and therefore it was decided not to test this parameter for statistically significant differences in the cross-sectional or repeated measures studies. However it was decided to retain the presence or absence of positive slopes as a descriptive indicator, as recommended in the original paper (Lamontagne et al., 2001). Observation of trends in the CSM cohort suggested that there may be a tendency towards positive slopes in some muscles during gait, so it may be useful to compare this trend to healthy controls and to evaluate pre- to post-operative changes.

7.7 Conclusion

This chapter determined the relative and absolute test-retest reliability of TSPs, kinematic, kinetic and EMG parameters in CSM. Most parameters demonstrated acceptable reliability for use in the cross-sectional and experimental studies. Some parameters, such as EMG amplitude normalised to MVC, were discarded from further use in the thesis due to unacceptable reliability. An estimate of the change required to exceed measurement error is now known. This information will inform the interpretation of differences in gait between the CSM participants and HCs in the cross-sectional study, and the change scores following surgery in the experimental study.
Chapter 8: Cross-sectional study of gait impairment in people with CSM compared to matched healthy controls

8.1 Introduction

This chapter reports the results of a cross-sectional study to address the second aim of the thesis, the comparison of gait patterns of people with CSM to those of age- and gender-matched HCs. The objectives of this study were 1) to describe the gait patterns of people with CSM with reference to age- and gender-based norms, and 2) to identify key areas of impairment in gait in CSM.

8.2 Development of protocol for healthy controls

8.2.1 Matching of healthy controls to CSM participants

A number of factors were considered in the selection of a cohort of HCs, aiming 1) to ensure that the HC group was an accurate match for the CSM participants, and 2) to avoid confounding of the kinematic, kinetic and SEMG data by extraneous factors. Functional data analysis of gait in 48 healthy individuals found that age, gender and gait speed had statistically significant influences on kinematic data (Roislien et al., 2009). Gait speed was unaffected by the presence of other variables, including height and weight in the regression models. It was therefore decided to match HCs based on age and gender. Previous studies using age-matched HCs to evaluate neurological gait impairment had an age criterion of within five years (Williams et al., 2009b), and this was considered achievable.

The literature indicated conflicting opinions on the need to match gait speed when comparing gait data between groups. Gait speed is a known confounding variable for kinematics and kinetics, particularly in the sagittal plane (Lelas et al., 2003, Roislien et al., 2009, Chung and Wang, 2010). Some authors have cautioned that, unless gait analysis is carried out at matched speed, conclusions cannot be drawn on the cause and effect relationship between speed and speed sensitive parameters (Chen et al., 2005). A reduction in gait speed, especially in older people, could be due to biomechanical changes (DeVita and Hortobagyi, 2000) or to the conscious choice of a more cautious gait pattern (Winter et al., 1990), rather than to an inability to generate the moments and powers required for a faster gait. Many studies of gait in neurological populations have matched the speed of the HC group to the group with pathological gait for these reasons (Lamontagne et al., 2002, Chen et al., 2005, Williams et al., 2009b).
On the other hand, it is known that individual preference for a particular gait speed occurs at the point where energy consumption per unit distance is minimised. This allows the locomotor system to take advantage of the passive mechanical properties of the lower limb in the absorption and generation of power (Jordan et al., 2007). Therefore, the imposition of a slower gait speed on healthy people may be counter-productive. Although the end product, gait speed, is the same, the neuromuscular and biomechanical strategies required to achieve this speed are not. This poses difficulties in the interpretation of the results of a matched speed comparison. A study of 30 healthy people walking at different percentages of preferred gait speed found that energy cost of walking and sagittal plane kinematics did not show any statistically significant differences between 80% and 120% of preferred walking speed (PWS), indicating that 80% PWS is not slow enough to change the demands of the locomotion task (Chung and Wang, 2010). This study did not compare the effect of actual gait speed, but instead focused on the use of a percentage of PWS as the speed criterion.

Based on the above studies, it appeared that there were advantages and disadvantages to the comparison of gait data at comfortable and matched speeds. Both methods appeared to have validity for different aspects of gait performance. As a result, it was decided to assess gait in HCs at both self-selected comfortable walking speed and at the speed of the participant with CSM to whom they were matched.

8.2.2 Principal components analysis

The interdependence of many gait variables can lead to difficulties in the interpretation of data. The relationships between variables may not be clear from assessing them individually. Furthermore, the large number of variables increases the likelihood of a type two statistical error, or finding a significant result by chance.

Principal components analysis (PCA) is a multivariate statistical technique designed to reduce the number of variables in a data set into a smaller number of dimensions or principal components (PCs). Each PC is a linear weighted combination of the original variables (Vyas and Kumaranayake, 2006). PCA has been used to classify gait patterns in children with CP (Schutte et al., 2000, Carriero et al., 2009) and in adults with osteoarthritis of the hip (Gaudreault et al., 2011). In the current study, its advantage was that it could be implemented on the pooled data of the CSM and HC participants at comfortable gait speed, as it would account for the interdependence of gait speed and other parameters. Stability tests of PCA on gait data from two groups of children found that the PCs were robust to changes in the data sets (Carriero et al., 2009), therefore it could reasonably be assumed that the exclusion of matched speed data would not affect the output of PCA.
The aim of PCA was to identify the dominant variability of the data and reduce its dimensionality into smaller numbers of PC variables. This would improve the understanding of the differences in gait between HC and CSM participants, and contribute to the identification of key areas of impairment that might otherwise be masked by the inter-dependency of variables in the original data set.

8.3 Methods

8.3.1 Participants

Participants with CSM were recruited from a neurosurgical clinic between December 2008 and December 2010. The diagnosis of CSM was confirmed by a neurosurgeon, either consultant or registrar, using the criteria outlined in Chapter 6, Table 6.1. Inclusion and exclusion criteria were described in Chapter 6, Section 6.4.1.1.

HCs were recruited from a local population. The need for participants was advertised via email at the PI’s workplace. Recipients were encouraged to forward the email to others. The inclusion criteria for HCs were 1) age within 5 years of a recruited CSM participant, 2) same gender as that participant, 3) ability to attend for assessment at the Movement Laboratory. HCs were excluded if they had a history of lower limb joint replacement surgery, complained of acute or chronic musculoskeletal injuries affecting gait, suffered a cardiovascular or respiratory impairment hindering mobilisation, or had a history of neurological disorders with physical deficits.

Upon recruitment, HCs were provided with a Controls Information Leaflet (Appendix 8.1). All HCs gave informed consent using the Healthy Controls Consent Form (Appendix 8.2). Beaumont Hospital Ethics (Medical Research) Committee granted ethical approval (Appendix 6.1).

8.3.2 Gait analysis

CSM and HC participants attended the Movement Laboratory for 3DGA and EMG assessment, following the procedures described in Chapter 6, Section 6.6. Gait analysis was firstly conducted at self-selected comfortable walking speed for both groups. Ten trials, comprising five left and five right force plate strikes, were captured.

Each HC then completed a second assessment at the walking speed of the CSM participant to whom he or she was matched, termed the matched speed assessment. Each trial was timed with a stopwatch. The goal speed was indicated by verbal feedback at the end of each trial, and not by external cues during the trial itself. Trials were included in the representative average if they were within 0.1 m/s of the goal speed. The assessment concluded when ten trials at goal speed were achieved.
8.3.3 Processing of gait trials and extraction of parameters

Gait trials were processed and TSP, kinematic, kinetic and EMG parameters extracted according to the methods described in Chapter 6, Section 6.7. The average of ten captured trials of each condition, comfortable and matched speeds, was used to represent the gait pattern of each participant at that speed. Data from each CSM participant’s more affected lower limb were analysed, and compared to data from the same leg of his or her HC match, as previously reported in a neurological population (Williams et al., 2009b). The more affected lower limb was determined by subjective questioning.

8.3.4 Statistical analysis

A sample size calculation was performed in Stata. At a significance level of 0.05, 13 pairs of participants were required for 90% power to detect a difference in gait speed of 0.1 m/s between CSM and HC participants, based on a SD of 0.11 m/s.

Two sets of statistical analysis were conducted, the first comparing CSM with HC participants at comfortable speed, and the second comparing the pairs at matched speed. The distribution of the difference between paired CSM and HC scores of each variable was checked visually using QQ plots and stem and leaf plots, and theoretically using the Shapiro-Wilk test and tests of skewness and kurtosis in Stata. Data from CSM and HC participants were compared using two-tailed paired t-tests. The non-parametric Wilcoxon signed-rank test was used to compare variables that were not normally distributed. In the case of the LSMS (see Chapter 5, Section 5.4.4.5 and Chapter 6, Section 6.7.4.3), the incidence of positive slopes between CSM and HC participants was compared for each muscle using Fisher’s exact test of proportions. Significance for all tests was established a priori at a probability ($p$) level of 0.05.

PCA of the temporal-spatial, kinematic and kinetic variables was performed in Stata on the pooled data of the CSM and HC participants at comfortable gait speed. The groups were not defined a priori when pooling the data (Carriero et al., 2009). EMG data were excluded from the first PCA in order to determine the PCs that emerged from a smaller data set of more correlated variables. A second PCA was then performed with EMG data included.

The contribution of each independent variable to the PCs was examined in both analyses. A variable was considered to contribute to a PC if its prediction score for that PC exceeded 0.15. Previous studies incorporating kinematic data had used a threshold prediction score of 0.2 (Carriero et al., 2009). Prediction scores in the current study were expected to be lower due to the larger number of parameters in the data set. The aspect of gait that was represented by each PC was then interpreted. Scatter plots of the scores
of the PCs were generated to visualise differences between the HC and CSM participants.

8.4 Results

8.4.1 Participants

Forty-four people with CSM met the inclusion criteria for the study over the 24-month recruitment period. A total of 15 were excluded for reasons of pre- or co-existing neurological conditions (six), immobility (four), previous surgery for CSM (four), and rheumatoid arthritis affecting gait (one). Ten people declined to participate in the study, leaving a total of 19 participants in the CSM group. One participant was later diagnosed with a co-existing neurological disorder. His data were excluded from analysis in accordance with the exclusion criteria. Age- and gender-matched HCs were recruited for 16 of the remaining 18 participants. One participant was not matched to a HC because he habitually mobilised with an aid. The effects of this would have confounded comparison with an independently mobile HC. A match was not found within the required time frame for the oldest participant in the CSM group, who was 77 years old. However, as the power calculation indicated the need for 13 pairs of participants to detect a difference in gait speed of 0.1 m/s, it was considered that 16 participants in each group would be sufficient for statistical analysis. The two participants who were not matched to HCs participated in the experimental study, presented in Chapter 9.

Characteristics of the participants in the CSM and HC groups are shown in Table 8.1. Paired t-tests confirmed that there were no statistically significant differences between the CSM and HC pairs in height ($p = 0.08$) or weight ($p = 0.75$).
### Table 8.1: Characteristics of participants

<table>
<thead>
<tr>
<th>Case</th>
<th>Gender</th>
<th>Age (years)</th>
<th>Height (metres)</th>
<th>Weight (kg)</th>
<th>Duration of symptoms (months)</th>
<th>Nurick</th>
<th>mJOA</th>
<th>MRMI</th>
<th>Age (years)</th>
<th>Height (metres)</th>
<th>Weight (kg)</th>
</tr>
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<td>8</td>
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<td>75.5</td>
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<td>11</td>
<td>39</td>
<td>54.8</td>
<td>1.71</td>
<td>73.1</td>
<td></td>
</tr>
</tbody>
</table>

mJOA = modified Japanese Orthopaedic Association score, MRMI = Modified Rivermead Mobility Index, kg = kilograms, M = male, F = female
8.4.2 Temporal-spatial parameters

8.4.2.1 Comfortable speed

The comfortable gait speed of HC participants was 1.49 m/s, significantly faster than that of their CSM counterparts, 1.12 m/s ($p < 0.0001$). This faster speed resulted from both a longer mean stride length of 1.45 m compared to 1.19 m ($p = 0.0001$), and a higher mean cadence of 122 steps per minute compared to 113 steps per minute ($p = 0.005$). CSM participants spent a longer proportion of the GC in double support, 26%, compared to 20% GC duration for HCs ($p = 0.0001$). This was associated with shorter single support duration of 36.7% in CSM compared to 39.9% in HCs ($p < 0.0001$). The decrease in single support duration was caused by both a delay in opposite foot off, which occurred at 12.9% GC in CSM compared to 10.4% GC in HC ($p = 0.0006$), and in foot off, at 62.7% GC in CSM compared to 60.2% GC in HC ($p = 0.003$), as illustrated in Figure 8.1. There were no significant differences in step width between pairs (CSM 16.9 cm, HC 15.9, $p = 0.4$). Full data are presented in Table 8.2.

![Figure 8.1: Relative durations of double support, single support and swing phases of gait for CSM and HC participants at comfortable speed](image)

DS1 = first phase of double support, OFO = opposite foot off, SS = single support phase, OFC = opposite foot contact, DS2 = second phase of double support, FO = foot off
Table 8.2: Temporal-spatial parameters of CSM and HC participants at comfortable speed

<table>
<thead>
<tr>
<th>Variable</th>
<th>CSM Mean (SD)</th>
<th>HC Mean (SD)</th>
<th>Difference Mean (SD)</th>
<th>95% Confidence Intervals</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cadence (steps / min)</td>
<td>113.22 (10.40)</td>
<td>122.97 (8.62)</td>
<td>-9.75 (0.08)</td>
<td>-16.17 (0.05) -3.30</td>
<td>0.006</td>
</tr>
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<td>Double support (s)</td>
<td>0.28 (0.06)</td>
<td>0.20 (0.03)</td>
<td>0.08 (0.06)</td>
<td>0.05 (0.11)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Single support (s)</td>
<td>0.39 (0.04)</td>
<td>0.39 (0.02)</td>
<td>0.00 (0.04)</td>
<td>-0.02 (0.02)</td>
<td>0.9</td>
</tr>
<tr>
<td>Double support duration (% GC)</td>
<td>26.06 (3.82)</td>
<td>20.32 (2.03)</td>
<td>5.74 (4.14)</td>
<td>3.54 (7.95)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Single support duration (% GC)</td>
<td>36.66 (2.06)</td>
<td>39.86 (0.85)</td>
<td>-3.20 (1.99)</td>
<td>-4.26 (2.10)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Foot off (% GC)</td>
<td>62.71 (2.19)</td>
<td>60.18 (1.33)</td>
<td>2.53 (2.81)</td>
<td>1.03 (4.03)</td>
<td>0.003</td>
</tr>
<tr>
<td>Gait speed (m/s)</td>
<td>1.12 (0.24)</td>
<td>1.49 (0.18)</td>
<td>-0.36 (0.24)</td>
<td>-0.49 (0.2)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Opposite foot contact (% GC)</td>
<td>49.56 (1.25)</td>
<td>50.26 (0.59)</td>
<td>-0.69 (1.57)</td>
<td>-1.53 (0.14)</td>
<td>0.09</td>
</tr>
<tr>
<td>Opposite foot off (% GC)</td>
<td>12.91 (2.14)</td>
<td>10.40 (1.13)</td>
<td>2.51 (2.33)</td>
<td>1.27 (3.75)</td>
<td>0.0006</td>
</tr>
<tr>
<td>Step length (m)</td>
<td>0.59 (0.10)</td>
<td>0.73 (0.06)</td>
<td>-0.14 (0.11)</td>
<td>-0.19 (0.1)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Step time (s)</td>
<td>0.54 (0.05)</td>
<td>0.49 (0.03)</td>
<td>0.05 (0.06)</td>
<td>0.02 (0.08)</td>
<td>0.002</td>
</tr>
<tr>
<td>Step width (m)</td>
<td>0.17 (0.04)</td>
<td>0.16 (0.03)</td>
<td>0.01 (0.01)</td>
<td>-0.01 (0.03)</td>
<td>0.4</td>
</tr>
<tr>
<td>Stride length (m)</td>
<td>1.19 (0.19)</td>
<td>1.45 (0.11)</td>
<td>-0.26 (0.20)</td>
<td>-0.37 (0.2)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Stride time (s)</td>
<td>1.07 (0.11)</td>
<td>0.98 (0.06)</td>
<td>0.09 (0.11)</td>
<td>0.03 (0.15)</td>
<td>0.006</td>
</tr>
</tbody>
</table>

SD = standard deviation, GC = gait cycle, m = metres, s = seconds

8.4.2.2 Matched speed

In the matched speed trials, HCs walked at a mean gait speed of 1.11 m/s to match their CSM counterparts at 1.12 m/s. There were differences in how this speed was achieved. HCs had a lower cadence (104 steps per minute) compared to CSM participants (113
steps per minute, \( p = 0.0009 \), and a longer stride length (1.27 m compared to 1.19 m, \( p = 0.03 \)). In keeping with the lower cadence, HC had a significantly longer stride time (CSM 1.07 s, HC 1.17 s, \( p = 0.0008 \)), and achieved this by spending longer portion of the GC in single support (CSM 36.7% GC, HC 37.6% GC, \( p = 0.049 \)) rather than in double support (CSM 26.1% GC, HC 24.9% GC, \( p = 0.14 \)). Full results are shown in Table 8.3.

**Table 8.3: Temporal-spatial parameters of CSM and HC participants at matched speed**

<table>
<thead>
<tr>
<th>Variable</th>
<th>CSM Mean</th>
<th>CSM SD</th>
<th>HC Mean</th>
<th>HC SD</th>
<th>Difference Mean</th>
<th>Difference SD</th>
<th>95% Confidence Intervals</th>
<th>( p ) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cadence (steps / min)</td>
<td>113.22</td>
<td>10.40</td>
<td>103.91</td>
<td>12.02</td>
<td>9.31</td>
<td>9.00</td>
<td>4.52 14.11</td>
<td>0.0009</td>
</tr>
<tr>
<td>Double support (s)</td>
<td>0.28</td>
<td>0.06</td>
<td>0.30</td>
<td>0.07</td>
<td>-0.02</td>
<td>0.03</td>
<td>-0.03 0.00</td>
<td>0.07</td>
</tr>
<tr>
<td>Single support (s)</td>
<td>0.39</td>
<td>0.04</td>
<td>0.44</td>
<td>0.04</td>
<td>-0.05</td>
<td>0.04</td>
<td>-0.07 -0.02</td>
<td>0.0005</td>
</tr>
<tr>
<td>Double support duration (GC)</td>
<td>26.06</td>
<td>3.82</td>
<td>24.94</td>
<td>3.32</td>
<td>1.12</td>
<td>2.84</td>
<td>-0.40 2.64</td>
<td>0.14</td>
</tr>
<tr>
<td>Single support duration (GC)</td>
<td>36.66</td>
<td>2.06</td>
<td>37.56</td>
<td>1.66</td>
<td>-0.90</td>
<td>1.67</td>
<td>-1.79 0.00</td>
<td>0.049</td>
</tr>
<tr>
<td>Foot off (GC)</td>
<td>62.71</td>
<td>2.19</td>
<td>62.49</td>
<td>1.76</td>
<td>0.21</td>
<td>2.01</td>
<td>-0.85 1.28</td>
<td>0.7</td>
</tr>
<tr>
<td>Gait speed (m/s)</td>
<td>1.12</td>
<td>0.24</td>
<td>1.11</td>
<td>0.21</td>
<td>0.02</td>
<td>0.07</td>
<td>-0.02 0.06</td>
<td>0.3</td>
</tr>
<tr>
<td>Opposite foot contact (GC)</td>
<td>49.56</td>
<td>1.25</td>
<td>50.27</td>
<td>0.65</td>
<td>-0.71</td>
<td>1.41</td>
<td>-1.46 0.04</td>
<td>0.06</td>
</tr>
<tr>
<td>Opposite foot off (GC)</td>
<td>12.91</td>
<td>2.14</td>
<td>12.69</td>
<td>1.73</td>
<td>0.22</td>
<td>1.54</td>
<td>-0.61 1.04</td>
<td>0.6</td>
</tr>
<tr>
<td>Step length (m)</td>
<td>0.59</td>
<td>0.10</td>
<td>0.64</td>
<td>0.06</td>
<td>-0.05</td>
<td>0.07</td>
<td>-0.09 -0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>Step time (s)</td>
<td>0.54</td>
<td>0.05</td>
<td>0.58</td>
<td>0.06</td>
<td>-0.04</td>
<td>0.05</td>
<td>-0.07 -0.01</td>
<td>0.007</td>
</tr>
<tr>
<td>Step width (m)</td>
<td>0.17</td>
<td>0.04</td>
<td>0.16</td>
<td>0.04</td>
<td>0.01</td>
<td>0.05</td>
<td>-0.02 0.03</td>
<td>0.5</td>
</tr>
<tr>
<td>Stride length (m)</td>
<td>1.19</td>
<td>0.19</td>
<td>1.27</td>
<td>0.13</td>
<td>-0.08</td>
<td>0.14</td>
<td>-0.16 -0.01</td>
<td>0.03</td>
</tr>
<tr>
<td>Stride time (s)</td>
<td>1.07</td>
<td>0.11</td>
<td>1.17</td>
<td>0.13</td>
<td>-0.10</td>
<td>0.10</td>
<td>-0.15 -0.05</td>
<td>0.0008</td>
</tr>
</tbody>
</table>

\( SD = \) standard deviation, \( GC = \) gait cycle, \( m = \) metres, \( s = \) seconds
8.4.3 Kinematic parameters

8.4.3.1 Comfortable speed

Paired t-tests were used to compare all kinematic variables, with the exception of peak ankle dorsiflexion in swing. This variable was tested using a Wilcoxon signed-rank test because the differences between HC and CSM pairs did not satisfy a normal distribution.

At the pelvis, there were no differences in average pelvic tilt (CSM 9.37°, HC 8.76°, \( p = 0.75 \)) or in range of pelvic rotation (CSM 8.2°, HC 10.76°, \( p = 0.48 \)). However, HCs had a significantly greater range of pelvic obliquity (8.78°) compared to CSM (6.34°, \( p = 0.003 \)).

At the hip, HCs had significantly greater total sagittal plane excursion than the CSM participants (HC 49.1°, CSM 44.3°, \( p = 0.004 \)). There were no significant differences in peak hip extension, hip position at initial contact, or in hip ROM in the frontal or transverse planes.

At the knee, HCs showed significantly greater peak flexion in stance (HC 22.1°, CSM 13.7°, \( p = 0.0005 \)), peak flexion in swing (HC 57.5°, CSM 48.6°, \( p = 0.0005 \)), and total sagittal plane motion of the knee (HC 59.9°, CSM 51.9°, \( p = 0.004 \)), but no significant difference in peak knee extension.

There was no significant difference in ankle position in the sagittal plane at initial contact, though the CSM group tended to strike with slight plantarflexion (CSM –0.61, HC 0.52°, \( p = 0.3 \)). Peak ankle dorsiflexion was not different between the groups in either stance or swing, however the graph of ankle movement over the GC, shown in Figure 8.2, “Ankle Dorsi / Plantar ROM”, indicated that HCs achieved dorsiflexion earlier in the mid stance phase than CSM participants. HCs had significantly greater peak plantarflexion of -18.4° compared to CSM, -11.3° (\( p = 0.013 \)). Table 8.4 shows the full results. Figure 8.2 illustrates kinematic curves over the GC.
Table 8.4: Kinematic key points of CSM and HC participants at comfortable speed

<table>
<thead>
<tr>
<th>Variable</th>
<th>CSM</th>
<th>HC</th>
<th>Difference</th>
<th>95% Confidence Intervals</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Pelvic obliquity range</td>
<td>6.3</td>
<td>3.2</td>
<td>8.8</td>
<td>2.8</td>
<td>-2.4</td>
</tr>
<tr>
<td>Pelvic rotation range</td>
<td>8.2</td>
<td>3.6</td>
<td>10.8</td>
<td>4.1</td>
<td>-0.6</td>
</tr>
<tr>
<td>Average pelvic tilt</td>
<td>9.4</td>
<td>5.9</td>
<td>8.8</td>
<td>4.2</td>
<td>0.6</td>
</tr>
<tr>
<td>Hip position at initial contact</td>
<td>30.3</td>
<td>6.9</td>
<td>31.3</td>
<td>5.6</td>
<td>-1.0</td>
</tr>
<tr>
<td>Peak hip extension</td>
<td>-13.4</td>
<td>6.3</td>
<td>-16.5</td>
<td>5.3</td>
<td>3.1</td>
</tr>
<tr>
<td>Total sagittal plane excursion of hip</td>
<td>44.3</td>
<td>5.3</td>
<td>49.1</td>
<td>4.9</td>
<td>-4.8</td>
</tr>
<tr>
<td>Range of hip abduction / adduction</td>
<td>12.0</td>
<td>3.2</td>
<td>13.0</td>
<td>3.0</td>
<td>-1.1</td>
</tr>
<tr>
<td>Range of hip rotation</td>
<td>19.1</td>
<td>10.1</td>
<td>16.2</td>
<td>4.8</td>
<td>2.9</td>
</tr>
<tr>
<td>Knee position at initial contact</td>
<td>4.2</td>
<td>6.0</td>
<td>3.3</td>
<td>3.7</td>
<td>0.9</td>
</tr>
<tr>
<td>Peak knee flexion in stance</td>
<td>13.7</td>
<td>6.4</td>
<td>22.1</td>
<td>6.9</td>
<td>-8.3</td>
</tr>
<tr>
<td>Peak knee extension</td>
<td>-3.3</td>
<td>4.4</td>
<td>-2.4</td>
<td>4.4</td>
<td>-0.9</td>
</tr>
<tr>
<td>Peak knee flexion in swing</td>
<td>48.6</td>
<td>5.6</td>
<td>57.5</td>
<td>4.2</td>
<td>-8.9</td>
</tr>
<tr>
<td>Total sagittal plane excursion of knee</td>
<td>51.9</td>
<td>6.9</td>
<td>59.9</td>
<td>5.4</td>
<td>-8.0</td>
</tr>
<tr>
<td>Ankle position at initial contact</td>
<td>-0.6</td>
<td>3.7</td>
<td>0.5</td>
<td>3.1</td>
<td>-1.1</td>
</tr>
<tr>
<td>Peak ankle dorsiflexion in stance</td>
<td>14.3</td>
<td>2.6</td>
<td>14.4</td>
<td>2.7</td>
<td>-0.1</td>
</tr>
<tr>
<td>Peak ankle plantarflexion</td>
<td>-11.3</td>
<td>6.9</td>
<td>-18.4</td>
<td>7.6</td>
<td>7.1</td>
</tr>
<tr>
<td>Peak ankle dorsiflexion in swing</td>
<td>6.2</td>
<td>4.2</td>
<td>4.7</td>
<td>2.5</td>
<td>1.5</td>
</tr>
</tbody>
</table>

* denotes a non-normally distributed variable with p value calculated using Wilcoxon signed rank test
Confidence intervals were not calculated for non-normally distributed variables
SD = standard deviation
All variables are measured in degrees
Figure 8.2: Kinematic curves of HC and CSM participants at comfortable speed

Significant differences between HC (green) and CSM (red) are indicated using boxes and arrows. Ant = anterior, Post = posterior, Flex = flexion, Ext = extension, Plantar = plantarflexion, Dorsi = dorsiflexion, Abd = abduction, Add = adduction, Int = internal, Ex = external.

The vertical dashed lines indicate toe-off for CSM (red) and HC (green).
There were no significant differences in kinematics at the hip or pelvis at matched speed. At the knee, HCs showed greater peak flexion during swing (HC 54.6°, CSM 48.6°, \( p = 0.006 \)), and this resulted in greater total sagittal plane excursion (HC 56.8°, CSM 51.9°, \( p = 0.03 \)). At the ankle, HCs achieved greater peak ankle dorsiflexion during stance (HC 16.2°, CSM 14.3°, \( p = 0.02 \)) and greater peak plantarflexion at pre swing (HC –16.6°, CSM –11.3°, \( p = 0.02 \)). Full results are listed in Table 8.5. Graphs of GC curves are shown in Figure 8.3.
<table>
<thead>
<tr>
<th>Variable</th>
<th>CSM Mean</th>
<th>CSM SD</th>
<th>HC Mean</th>
<th>HC SD</th>
<th>Difference Mean</th>
<th>Difference SD</th>
<th>95% Confidence Intervals</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pelvic obliquity range</td>
<td>6.3</td>
<td>3.2</td>
<td>7.0</td>
<td>2.3</td>
<td>-0.7</td>
<td>2.3</td>
<td>-1.9 - 0.5</td>
<td>0.2</td>
</tr>
<tr>
<td>Pelvic rotation range</td>
<td>8.2</td>
<td>3.6</td>
<td>8.4</td>
<td>2.1</td>
<td>1.2</td>
<td>3.5</td>
<td>-0.7 - 3.0</td>
<td>0.2</td>
</tr>
<tr>
<td>Average pelvic tilt</td>
<td>9.4</td>
<td>5.8</td>
<td>8.8</td>
<td>4.0</td>
<td>0.6</td>
<td>7.2</td>
<td>-3.2 - 4.5</td>
<td>0.74</td>
</tr>
<tr>
<td>Hip position at initial contact</td>
<td>30.3</td>
<td>6.9</td>
<td>29.3</td>
<td>5.3</td>
<td>1.0</td>
<td>9.4</td>
<td>-4.0 - 6.0</td>
<td>0.67</td>
</tr>
<tr>
<td>Peak hip extension</td>
<td>-13.4</td>
<td>6.3</td>
<td>-13.5</td>
<td>5.9</td>
<td>0.1</td>
<td>8.6</td>
<td>-4.5 - 4.7</td>
<td>0.97</td>
</tr>
<tr>
<td>Total sagittal plane excursion of hip</td>
<td>44.3</td>
<td>5.3</td>
<td>43.7</td>
<td>3.2</td>
<td>0.6</td>
<td>4.2</td>
<td>-1.6 - 2.9</td>
<td>0.55</td>
</tr>
<tr>
<td>Range of hip abduction / adduction</td>
<td>12.0</td>
<td>3.2</td>
<td>11.4</td>
<td>2.6</td>
<td>0.5</td>
<td>3.0</td>
<td>-1.1 - 2.1</td>
<td>0.49</td>
</tr>
<tr>
<td>Range of hip rotation</td>
<td>19.1</td>
<td>10.1</td>
<td>17.0</td>
<td>4.6</td>
<td>2.1</td>
<td>10.8</td>
<td>-3.6 - 7.9</td>
<td>0.45</td>
</tr>
<tr>
<td>Knee position at initial contact</td>
<td>4.2</td>
<td>6.0</td>
<td>1.7</td>
<td>4.5</td>
<td>2.5</td>
<td>6.9</td>
<td>-1.2 - 6.1</td>
<td>0.17</td>
</tr>
<tr>
<td>Peak knee flexion in stance</td>
<td>13.7</td>
<td>6.4</td>
<td>16.2</td>
<td>7.9</td>
<td>-2.5</td>
<td>7.3</td>
<td>-6.4 - 1.4</td>
<td>0.19</td>
</tr>
<tr>
<td>Peak knee extension</td>
<td>-3.3</td>
<td>4.4</td>
<td>-2.2</td>
<td>4.0</td>
<td>-1.1</td>
<td>5.3</td>
<td>-3.9 - 1.7</td>
<td>0.4</td>
</tr>
<tr>
<td>Peak knee flexion in swing</td>
<td>48.6</td>
<td>5.6</td>
<td>54.6</td>
<td>5.0</td>
<td>-6.0</td>
<td>7.5</td>
<td>-10.0 - -2.0</td>
<td>0.006</td>
</tr>
<tr>
<td>Total sagittal plane excursion of knee</td>
<td>51.9</td>
<td>6.9</td>
<td>56.8</td>
<td>5.2</td>
<td>-4.9</td>
<td>8.0</td>
<td>-9.2 - -0.6</td>
<td>0.03</td>
</tr>
<tr>
<td>Ankle position at initial contact</td>
<td>-0.6</td>
<td>3.7</td>
<td>-0.7</td>
<td>3.5</td>
<td>0.1</td>
<td>4.0</td>
<td>-2.1 - 2.2</td>
<td>0.95</td>
</tr>
<tr>
<td>Peak ankle dorsiflexion in stance</td>
<td>14.3</td>
<td>2.6</td>
<td>16.2</td>
<td>2.5</td>
<td>-2.0</td>
<td>3.0</td>
<td>-3.6 - -0.4</td>
<td>0.02</td>
</tr>
<tr>
<td>Peak ankle plantarflexion</td>
<td>-11.3</td>
<td>6.9</td>
<td>-16.6</td>
<td>6.4</td>
<td>5.3</td>
<td>8.0</td>
<td>1.1 - 9.6</td>
<td>0.02</td>
</tr>
<tr>
<td>Peak ankle dorsiflexion in swing</td>
<td>6.2</td>
<td>4.2</td>
<td>6.2</td>
<td>4.0</td>
<td>0.1</td>
<td>4.5</td>
<td>-2.3 - 2.5</td>
<td>0.96</td>
</tr>
</tbody>
</table>

SD = standard deviation
All variables are measured in degrees
Figure 8.3. Kinematic curves of CSM and HC participants at matched speed
Significant differences are indicated using boxes and arrows
Ant = anterior, Post = posterior, Flex = flexion, Ext = extension, Plantar = plantarflexion, Dorsi = dorsiflexion, Abd = abduction, Add = adduction, Int = internal, Ex = external
Vertical dashed lines indicate toe-off for both groups
8.4.4 Kinetic parameters

8.4.4.1 Comfortable speed

Full results for kinetic parameters are listed in Table 8.6. Analysis of GRF showed no difference in peak medio-lateral shear force between HC and CSM pairs ($p = 0.3$). Differences in peak vertical GRF were not normally distributed, however when analysed with a Wilcoxon signed-rank test, there were significantly higher forces in HCs ($p = 0.0004$). Analysis of the AP GRF showed significant differences in both the deceleration (CSM 1.39 N/kg, HC 2.1 N/kg, $p = 0.0003$) and acceleration components (CSM –1.6 N/kg, HC –2.53 N/kg, $p < 0.0001$). Figure 8.4 illustrates the GRF curves.

Peak hip abductor and extensor moments showed a trend towards lower scores in CSM. There were significant differences in peak hip flexor moments (HC –0.81 Newton metres per kilogram (Nm/kg), CSM –0.62 Nm/kg, $p = 0.02$). At the knee, peak extensor moments were significantly higher in HCs (0.56 Nm/kg) than in CSM (0.27 Nm/kg, $p = 0.0005$), however there were no differences in peak flexor moments. Peak ankle plantarflexor moments were significantly higher in HC (1.6 Nm/kg) than in CSM (1.41 Nm/kg, $p = 0.0007$). Figure 8.4 illustrates the hip, knee and ankle moments.

Figure 8.5 shows net power at the hip, knee and ankle, summed across the three axes of rotation, over the GC. Analysis revealed significant differences in hip power absorption in mid stance, H2 (HC –0.96 Watts per kilogram (W/kg), CSM –0.61 W/kg, $p = 0.004$) and hip power generation in pre swing, H3 (HC1.71 W/kg, CSM 1.0 W/kg, $p = 0.0001$). There were significant differences between pairs in all of four power peaks at the knee, with greater power generation and absorption by the HC participants. Ankle power generation at pre swing, A2, was higher in HC (4.85 N/kg) than in CSM (2.82 N/kg, $p = 0.0001$), whereas there was no difference in power absorption at the ankle in mid stance, A1.
Table 8.6: Peak kinetic parameters of CSM and HC participants at comfortable speed

<table>
<thead>
<tr>
<th>Variable</th>
<th>CSM</th>
<th></th>
<th>HC</th>
<th></th>
<th>Difference</th>
<th></th>
<th></th>
<th>95% Confidence Intervals</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td></td>
</tr>
<tr>
<td>Medio-lateral GRF</td>
<td>0.66</td>
<td>0.19</td>
<td>0.59</td>
<td>0.19</td>
<td>0.07</td>
<td>0.27</td>
<td>-0.07</td>
<td>0.21</td>
<td>0.3</td>
</tr>
<tr>
<td>Antero-posterior GRF, deceleration</td>
<td>1.39</td>
<td>0.46</td>
<td>2.10</td>
<td>0.53</td>
<td>-0.71</td>
<td>0.61</td>
<td>-1.03</td>
<td>-0.38</td>
<td>0.0003</td>
</tr>
<tr>
<td>Antero-posterior GRF, acceleration</td>
<td>-1.60</td>
<td>0.52</td>
<td>-2.53</td>
<td>0.41</td>
<td>0.93</td>
<td>0.59</td>
<td>0.61</td>
<td>1.24</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Vertical GRF</td>
<td>10.58</td>
<td>0.74</td>
<td>12.23</td>
<td>1.42</td>
<td>-1.65</td>
<td>1.57</td>
<td>*</td>
<td>*</td>
<td>0.004*</td>
</tr>
<tr>
<td>Hip extensor moment</td>
<td>0.78</td>
<td>0.20</td>
<td>0.88</td>
<td>0.22</td>
<td>-0.10</td>
<td>0.21</td>
<td>-0.21</td>
<td>0.01</td>
<td>0.075</td>
</tr>
<tr>
<td>Hip flexor moment</td>
<td>-0.62</td>
<td>0.27</td>
<td>-0.81</td>
<td>0.23</td>
<td>0.19</td>
<td>0.28</td>
<td>0.04</td>
<td>0.34</td>
<td>0.02</td>
</tr>
<tr>
<td>Hip abductor moment</td>
<td>0.84</td>
<td>0.18</td>
<td>0.98</td>
<td>0.22</td>
<td>-0.14</td>
<td>0.30</td>
<td>-0.30</td>
<td>0.02</td>
<td>0.08</td>
</tr>
<tr>
<td>Knee extensor moment</td>
<td>0.27</td>
<td>0.18</td>
<td>0.56</td>
<td>0.26</td>
<td>-0.30</td>
<td>0.27</td>
<td>-0.44</td>
<td>-0.15</td>
<td>0.0005</td>
</tr>
<tr>
<td>Knee flexor moment</td>
<td>-0.52</td>
<td>0.11</td>
<td>-0.57</td>
<td>0.14</td>
<td>0.06</td>
<td>0.17</td>
<td>-0.03</td>
<td>0.14</td>
<td>0.2</td>
</tr>
<tr>
<td>Ankle plantarflexor moment</td>
<td>1.41</td>
<td>0.21</td>
<td>1.60</td>
<td>0.12</td>
<td>-0.18</td>
<td>0.18</td>
<td>-0.28</td>
<td>-0.09</td>
<td>0.0007</td>
</tr>
<tr>
<td>Hip power generation, H1</td>
<td>0.51</td>
<td>0.29</td>
<td>0.58</td>
<td>0.41</td>
<td>-0.07</td>
<td>0.45</td>
<td>-0.31</td>
<td>0.17</td>
<td>0.5</td>
</tr>
<tr>
<td>Hip power absorption, H2</td>
<td>-0.61</td>
<td>0.27</td>
<td>-0.96</td>
<td>0.39</td>
<td>0.35</td>
<td>0.41</td>
<td>0.13</td>
<td>0.56</td>
<td>0.004</td>
</tr>
<tr>
<td>Hip power generation, H3</td>
<td>1.00</td>
<td>0.45</td>
<td>1.71</td>
<td>0.47</td>
<td>-0.71</td>
<td>0.54</td>
<td>-1.00</td>
<td>-0.42</td>
<td>0.0001</td>
</tr>
<tr>
<td>Knee power absorption, K1</td>
<td>-0.70</td>
<td>0.87</td>
<td>-2.10</td>
<td>1.36</td>
<td>1.40</td>
<td>1.47</td>
<td>0.62</td>
<td>2.18</td>
<td>0.002</td>
</tr>
<tr>
<td>Knee power generation, K2</td>
<td>0.59</td>
<td>0.28</td>
<td>0.97</td>
<td>0.33</td>
<td>-0.38</td>
<td>0.39</td>
<td>-0.58</td>
<td>-0.17</td>
<td>0.0015</td>
</tr>
<tr>
<td>Knee power absorption, K3</td>
<td>-0.68</td>
<td>0.42</td>
<td>-1.21</td>
<td>0.50</td>
<td>0.53</td>
<td>0.63</td>
<td>0.20</td>
<td>0.87</td>
<td>0.004</td>
</tr>
<tr>
<td>Knee power absorption, K4</td>
<td>-0.70</td>
<td>0.27</td>
<td>-1.56</td>
<td>0.63</td>
<td>0.86</td>
<td>0.69</td>
<td>0.49</td>
<td>1.22</td>
<td>0.0002</td>
</tr>
<tr>
<td>Ankle power absorption, A1</td>
<td>-0.99</td>
<td>0.34</td>
<td>-0.92</td>
<td>0.24</td>
<td>-0.07</td>
<td>0.29</td>
<td>-0.22</td>
<td>0.09</td>
<td>0.37</td>
</tr>
<tr>
<td>Ankle power generation, A2</td>
<td>2.82</td>
<td>1.34</td>
<td>4.85</td>
<td>1.19</td>
<td>-2.03</td>
<td>1.56</td>
<td>-2.86</td>
<td>-1.20</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

* denotes a non-normally distributed variable analysed using Wilcoxon signed rank test

Ground reaction forces (GRF) are reported in Newtons per kilogram, moments in Newton metres per kilogram, and powers in Watts per kilogram

SD = standard deviation, GRF = ground reaction force
Figure 8.4: Ground reaction forces and joint moments of HC and CSM participants at comfortable speed
Med = medial, Lat = lateral, N = Newtons, kg = kilograms, Nm = Newton metres, Ant = anterior, Post = posterior, Ext = extensor, Flex = flexor
Table 8.7 shows the kinetic parameters of CSM and HC participants at matched speed. There were fewer differences between pairs at this speed. The acceleration component of AP GRF was higher in HC, with a peak of –1.9 N/kg, compared to –1.6 N/kg in CSM (p = 0.02). The CSM group generated higher peak hip extensor moments (CSM 0.78 Nm/kg, HC 0.61 Nm/kg, p = 0.013), a parameter that was not different at comfortable speed. In keeping with this finding, peak hip power generation during loading response (H1) was higher in the CSM group (0.51 W/kg) compared to HCs (0.25 W/kg, p = 0.05). There were no differences in power generation and absorption at the knee, although there was a non-significant tendency for greater absorption peaks at K1 and K4 in HCs. At the ankle, the CSM participants absorbed significantly more power at the A1 peak in mid stance (CSM –0.91 W/kg, HC –0.8 W/kg, p = 0.03). There was a trend towards higher power generation at the ankle at toe-off in HC (3.32 W/kg) compared to CSM (2.82 W/kg), however this did not reach statistical significance (p = 0.075). Figures 8.6 and 8.7 show kinetic curves over the GC for CSM and HC participants.

8.4.4.2 Matched speed

Figure 8.5: Joint powers of CSM and HC participants at comfortable speed

W = Watts, kg = kilograms, Gen = generation, Abs = absorption, H = Hip power peak, K = knee power peak, A = ankle power peak
Table 8.7: Peak kinetic parameters of CSM and HC participants at matched speed

<table>
<thead>
<tr>
<th>Variable</th>
<th>CSM Mean</th>
<th>SD</th>
<th>HC Mean</th>
<th>SD</th>
<th>Difference Mean</th>
<th>SD</th>
<th>95% Confidence Intervals</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medio-lateral GRF</td>
<td>0.66</td>
<td>0.19</td>
<td>0.59</td>
<td>0.19</td>
<td>0.07</td>
<td>0.27</td>
<td>-0.07 - 0.21</td>
<td>0.3</td>
</tr>
<tr>
<td>Antero-posterior GRF, deceleration</td>
<td>1.39</td>
<td>0.46</td>
<td>1.53</td>
<td>0.45</td>
<td>-0.13</td>
<td>0.44</td>
<td>-0.37 - 0.10</td>
<td>0.24</td>
</tr>
<tr>
<td>Antero-posterior GRF, acceleration</td>
<td>-1.60</td>
<td>0.52</td>
<td>-1.90</td>
<td>0.45</td>
<td>0.30</td>
<td>0.45</td>
<td>0.05 - 0.54</td>
<td>0.02</td>
</tr>
<tr>
<td>Vertical GRF</td>
<td>10.58</td>
<td>0.74</td>
<td>10.82</td>
<td>0.57</td>
<td>-0.24</td>
<td>0.60</td>
<td>-0.56 - 0.08</td>
<td>0.12</td>
</tr>
<tr>
<td>Hip extensor moment</td>
<td>0.78</td>
<td>0.20</td>
<td>0.61</td>
<td>0.23</td>
<td>0.16</td>
<td>0.23</td>
<td>0.04 - 0.29</td>
<td>0.01</td>
</tr>
<tr>
<td>Hip flexor moment</td>
<td>-0.62</td>
<td>0.27</td>
<td>-0.58</td>
<td>0.27</td>
<td>-0.04</td>
<td>0.26</td>
<td>-0.18 - 0.10</td>
<td>0.5</td>
</tr>
<tr>
<td>Hip abductor moment</td>
<td>0.84</td>
<td>0.18</td>
<td>0.90</td>
<td>0.21</td>
<td>-0.06</td>
<td>0.25</td>
<td>-0.19 - 0.07</td>
<td>0.3</td>
</tr>
<tr>
<td>Knee extensor moment</td>
<td>0.27</td>
<td>0.18</td>
<td>0.33</td>
<td>0.22</td>
<td>-0.06</td>
<td>0.21</td>
<td>-0.18 - 0.05</td>
<td>0.25</td>
</tr>
<tr>
<td>Knee flexor moment</td>
<td>-0.52</td>
<td>0.11</td>
<td>-0.51</td>
<td>0.16</td>
<td>0.00</td>
<td>0.18</td>
<td>-0.10 - 0.09</td>
<td>0.95</td>
</tr>
<tr>
<td>Ankle plantarflexor moment</td>
<td>1.41</td>
<td>0.21</td>
<td>1.48</td>
<td>0.13</td>
<td>-0.07</td>
<td>0.17</td>
<td>-0.16 - 0.02</td>
<td>0.12</td>
</tr>
<tr>
<td>Hip power generation, H1</td>
<td>0.51</td>
<td>0.29</td>
<td>0.32</td>
<td>0.25</td>
<td>0.19</td>
<td>0.36</td>
<td>0.00 - 0.38</td>
<td>0.05</td>
</tr>
<tr>
<td>Hip power absorption, H2</td>
<td>-0.61</td>
<td>0.27</td>
<td>-0.57</td>
<td>0.31</td>
<td>-0.05</td>
<td>0.28</td>
<td>-0.20 - 0.10</td>
<td>0.5</td>
</tr>
<tr>
<td>Hip power generation, H3</td>
<td>1.00</td>
<td>0.45</td>
<td>0.94</td>
<td>0.45</td>
<td>0.06</td>
<td>0.40</td>
<td>-0.15 - 0.28</td>
<td>0.54</td>
</tr>
<tr>
<td>Knee power absorption, K1</td>
<td>-0.70</td>
<td>0.87</td>
<td>-0.84</td>
<td>0.56</td>
<td>0.15</td>
<td>0.73</td>
<td>-0.24 - 0.53</td>
<td>0.43</td>
</tr>
<tr>
<td>Knee power generation, K2</td>
<td>0.59</td>
<td>0.28</td>
<td>0.56</td>
<td>0.25</td>
<td>0.03</td>
<td>0.31</td>
<td>-0.13 - 0.19</td>
<td>0.7</td>
</tr>
<tr>
<td>Knee power absorption, K3</td>
<td>-0.68</td>
<td>0.42</td>
<td>-0.66</td>
<td>0.44</td>
<td>-0.02</td>
<td>0.50</td>
<td>-0.28 - 0.25</td>
<td>0.9</td>
</tr>
<tr>
<td>Knee power absorption, K4</td>
<td>-0.70</td>
<td>0.27</td>
<td>-0.84</td>
<td>0.44</td>
<td>0.14</td>
<td>0.41</td>
<td>-0.08 - 0.36</td>
<td>0.19</td>
</tr>
<tr>
<td>Ankle power absorption, A1</td>
<td>-0.99</td>
<td>0.34</td>
<td>-0.80</td>
<td>0.24</td>
<td>-0.19</td>
<td>0.31</td>
<td>-0.36 - 0.02</td>
<td>0.03</td>
</tr>
<tr>
<td>Ankle power generation, A2</td>
<td>2.82</td>
<td>1.34</td>
<td>3.32</td>
<td>1.09</td>
<td>-0.50</td>
<td>1.04</td>
<td>-1.06 - 0.06</td>
<td>0.075</td>
</tr>
</tbody>
</table>

SD = standard deviation, GRF = ground reaction force

GRFs are reported in Newtons per kilogram, moments in Newton metres per kilogram, and powers in Watts per kilogram.
Figure 8.6: Ground reaction forces and joint moments of HC and CSM participants at matched speed
Med = medial, Lat = lateral, N = Newtons, kg = kilograms, Nm = Newton metres, Ant = anterior, Post = posterior, Ext = extensor, Flex = flexor
Significant differences at key points are highlighted with text and arrows
Vertical dashed lines indicate toe-off for both groups
Figure 8.7: Joint powers of HC and CSM participants at matched speed

Gen = power generation, Abs = power absorption, W = Watts, kg = kilograms
Significant differences at key points are highlighted with text and arrows
Vertical dashed lines indicate toe-off for both groups

8.4.5 Analysis of EMG timing parameters

8.4.5.1 Comfortable speed

The total duration of activation of RF, BF, TA and MG muscles over the GC was determined using the TKEO-based DTM algorithm described in Chapter 5, Section 5.4.4.2. Paired t-tests found that the duration of RF, BF and TA activation was significantly longer in CSM participants. MG showed no differences between pairs. The actual differences of 10.9% for RF, 9.8% for BF and 11.2% for TA exceeded the SEM for timing of those muscles, previously discussed in Chapter 7, Section 7.5.3.1. Timing parameters are listed in Table 8.8.

The CSM group showed significantly greater co-activation between RF and BF, 14.5% GC duration compared to the HCs’ 9.3% GC duration ($p = 0.04$). There was a non-significant trend for greater co-activation between TA and MG in CSM (7.5% GC) than in HC (4% GC, $p = 0.17$). Table 8.8 shows the results of the analysis of co-activation.
Table 8.8: Duration of muscle activation and co-activation of CSM and HC participants at comfortable speed

<table>
<thead>
<tr>
<th>Muscle</th>
<th>CSM</th>
<th>HC</th>
<th>Difference</th>
<th>Confidence Interval</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>32.97</td>
<td>18.22</td>
<td>22.06</td>
<td>6.00</td>
<td>10.92</td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>31.81</td>
<td>10.96</td>
<td>21.93</td>
<td>5.09</td>
<td>9.88</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>42.16</td>
<td>9.86</td>
<td>30.93</td>
<td>8.84</td>
<td>11.23</td>
</tr>
<tr>
<td>Medial gastrocnemius</td>
<td>31.75</td>
<td>9.68</td>
<td>28.88</td>
<td>8.09</td>
<td>2.88</td>
</tr>
<tr>
<td>Rectus–biceps co-activation</td>
<td>14.44</td>
<td>8.73</td>
<td>9.32</td>
<td>4.38</td>
<td>5.12</td>
</tr>
<tr>
<td>Tibialis–gastrocnemius co-activation</td>
<td>7.47</td>
<td>7.91</td>
<td>4.02</td>
<td>4.03</td>
<td>3.45</td>
</tr>
</tbody>
</table>

Activation times are expressed as a percentage of gait cycle duration
SD = standard deviation

8.4.5.2 Matched speed

Analysis of timing parameters at matched speed mirrored the results of the comfortable speed analysis. The CSM group had significantly greater total activation time of RF, BF and TA, though RF did not reach statistical significance ($p = 0.07$). The timing of MG showed no significant differences between groups, though there was a non-significant trend of longer activation time in CSM (31.75% GC) than in HC (26.81% GC, $p = 0.14$).

Co-activation between RF and BF was of significantly greater duration in CSM compared to HC. There was no difference between pairs in relation to MG and TA co-activation. Table 8.9 shows the results of analysis of timing and co-activation at matched speed.
Table 8.9: Duration of muscle activation and co-activation of CSM and HC participants at matched speed

<table>
<thead>
<tr>
<th>Muscle</th>
<th>CSM</th>
<th>HC</th>
<th>Difference</th>
<th>Confidence Interval</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>32.97</td>
<td>18.22</td>
<td>24.45</td>
<td>6.50</td>
<td>8.53</td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>31.81</td>
<td>10.96</td>
<td>19.30</td>
<td>6.52</td>
<td>12.51</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>42.16</td>
<td>9.86</td>
<td>29.76</td>
<td>10.88</td>
<td>12.40</td>
</tr>
<tr>
<td>Medial gastrocnemius</td>
<td>31.75</td>
<td>9.68</td>
<td>26.81</td>
<td>8.03</td>
<td>4.95</td>
</tr>
<tr>
<td>Rectus–biceps co-activation</td>
<td>14.44</td>
<td>8.73</td>
<td>9.30</td>
<td>3.79</td>
<td>5.14</td>
</tr>
<tr>
<td>Tibialis–gastrocnemius co-activation</td>
<td>7.47</td>
<td>7.91</td>
<td>3.93</td>
<td>3.60</td>
<td>3.54</td>
</tr>
</tbody>
</table>

Activation times are expressed as a percentage of GC duration
SD = standard deviation

8.4.6 EMG amplitude parameters

8.4.6.1 Comfortable speed

The mean amplitude of a muscle’s EMG signal during bursts of activity and during “off” phases were extracted according to the methods described in Chapter 6, Section 6.7.3.3. Mean amplitudes were normalised using the PDM method described in Section 6.7.3.4. The CSM group had significantly higher mean amplitude in the “off” or baseline phases of RF (CSM 17.9% RMSMAX, HC 13.7% RMSMAX, p = 0.02) and BF (CSM 15.6% RMSMAX, HC 11% RMSMAX, p = 0.006). The CSM group also showed significantly lower BF activity during stance (CSM 47.6% RMSMAX, HC 58.5% RMSMAX, p = 0.04). There were no differences in mean amplitude of MG or TA. Results are shown in Table 8.10 and Figure 8.8.
Table 8.10: Amplitude of muscle activity bursts and baseline phases during gait of CSM and HC participants at comfortable speed

<table>
<thead>
<tr>
<th>Muscle Burst</th>
<th>CSM Mean</th>
<th>CSM SD</th>
<th>HC Mean</th>
<th>HC SD</th>
<th>Difference Mean</th>
<th>Difference SD</th>
<th>Confidence Interval</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RF loading response</td>
<td>56.15</td>
<td>9.82</td>
<td>60.88</td>
<td>7.89</td>
<td>-4.72</td>
<td>11.06</td>
<td>-10.62</td>
<td>1.17</td>
</tr>
<tr>
<td>RF pre swing</td>
<td>45.70</td>
<td>15.86</td>
<td>43.36</td>
<td>12.93</td>
<td>-5.78</td>
<td>13.84</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>RF swing</td>
<td>19.34</td>
<td>12.21</td>
<td>25.11</td>
<td>9.63</td>
<td>5.44</td>
<td>18.96</td>
<td>-13.15</td>
<td>1.60</td>
</tr>
<tr>
<td>RF baseline</td>
<td>17.86</td>
<td>6.08</td>
<td>13.71</td>
<td>3.73</td>
<td>4.15</td>
<td>6.65</td>
<td>0.61</td>
<td>7.70</td>
</tr>
<tr>
<td>BF stance</td>
<td>47.63</td>
<td>11.10</td>
<td>58.50</td>
<td>15.10</td>
<td>-9.56</td>
<td>16.54</td>
<td>-18.72</td>
<td>-0.40</td>
</tr>
<tr>
<td>BF swing</td>
<td>43.87</td>
<td>12.88</td>
<td>39.67</td>
<td>12.03</td>
<td>4.20</td>
<td>14.56</td>
<td>-3.56</td>
<td>11.96</td>
</tr>
<tr>
<td>BF baseline</td>
<td>15.55</td>
<td>4.03</td>
<td>10.97</td>
<td>3.93</td>
<td>4.58</td>
<td>5.52</td>
<td>1.64</td>
<td>7.52</td>
</tr>
<tr>
<td>TA stance</td>
<td>55.78</td>
<td>8.68</td>
<td>55.61</td>
<td>14.02</td>
<td>0.18</td>
<td>18.41</td>
<td>-9.63</td>
<td>9.98</td>
</tr>
<tr>
<td>TA swing</td>
<td>47.73</td>
<td>6.72</td>
<td>45.25</td>
<td>9.49</td>
<td>2.48</td>
<td>12.91</td>
<td>-4.40</td>
<td>9.36</td>
</tr>
<tr>
<td>TA baseline</td>
<td>15.91</td>
<td>4.23</td>
<td>14.16</td>
<td>2.70</td>
<td>1.75</td>
<td>4.92</td>
<td>-0.87</td>
<td>4.38</td>
</tr>
<tr>
<td>MG stance</td>
<td>57.35</td>
<td>6.97</td>
<td>62.03</td>
<td>4.66</td>
<td>-4.68</td>
<td>9.13</td>
<td>-9.54</td>
<td>0.19</td>
</tr>
<tr>
<td>MG baseline</td>
<td>13.29</td>
<td>3.42</td>
<td>11.84</td>
<td>6.60</td>
<td>1.46</td>
<td>8.11</td>
<td>-2.86</td>
<td>5.78</td>
</tr>
</tbody>
</table>

Amplitude of each burst is expressed as a percentage of the muscle’s peak root-mean-square (RMS) amplitude during gait.

RF = rectus femoris, BF = biceps femoris, TA = tibialis anterior, MG = medial gastrocnemius, SD = standard deviation.

The symbol * denotes results of a Wilcoxon signed-rank test.
Amplitude is expressed as a percentage of maximum root-mean-square (RMS) amplitude obtained during 10 gait trials of each session (%RMS_max)

* denotes statistically significant difference between CSM and HC

Figure 8.8: Box and whisker plots of mean EMG amplitude of CSM and HC participants at comfortable speed

Amplitude is expressed as a percentage of maximum root-mean-square (RMS) amplitude obtained during 10 gait trials of each session (%RMS_MAX)

* denotes statistically significant difference between CSM and HC
8.4.6.2 Matched speed

At matched speed, CSM participants showed statistically higher normalised amplitudes during the “off” phases of RF ($p = 0.02$) and BF ($p = 0.01$), in keeping with the findings at comfortable speed. No other differences in amplitude were found at matched speed. Differences in RF activity during swing and pre swing did not follow a normal distribution. These activity bursts were examined with the non-parametric Wilcoxon signed-rank test. The results of EMG amplitude analysis at matched speed are shown in Table 8.11.

Table 8.11: Amplitude of muscle activity bursts and baseline phases during gait of CSM and HC participants at matched speed

<table>
<thead>
<tr>
<th>Muscle Burst</th>
<th>CSM Mean</th>
<th>CSM SD</th>
<th>HC Mean</th>
<th>HC SD</th>
<th>Difference Mean</th>
<th>Difference SD</th>
<th>Confidence Interval</th>
<th>$p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RF loading response</td>
<td>56.15</td>
<td>9.82</td>
<td>57.25</td>
<td>6.39</td>
<td>-1.10</td>
<td>7.80</td>
<td>-5.26</td>
<td>3.06</td>
</tr>
<tr>
<td>RF pre swing</td>
<td>45.70</td>
<td>15.86</td>
<td>38.10</td>
<td>14.57</td>
<td>-10.72</td>
<td>17.99</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>RF swing</td>
<td>19.34</td>
<td>12.21</td>
<td>30.06</td>
<td>10.56</td>
<td>8.89</td>
<td>25.93</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>RF baseline</td>
<td>17.86</td>
<td>6.08</td>
<td>13.28</td>
<td>3.18</td>
<td>4.59</td>
<td>6.69</td>
<td>1.02</td>
<td>8.16</td>
</tr>
<tr>
<td>BF stance</td>
<td>47.63</td>
<td>11.10</td>
<td>53.99</td>
<td>8.32</td>
<td>-5.05</td>
<td>11.12</td>
<td>-11.20</td>
<td>1.11</td>
</tr>
<tr>
<td>BF swing</td>
<td>43.87</td>
<td>12.88</td>
<td>38.49</td>
<td>13.35</td>
<td>5.38</td>
<td>15.38</td>
<td>-2.82</td>
<td>13.57</td>
</tr>
<tr>
<td>BF baseline</td>
<td>15.55</td>
<td>4.03</td>
<td>11.70</td>
<td>3.29</td>
<td>3.85</td>
<td>5.54</td>
<td>0.90</td>
<td>6.80</td>
</tr>
<tr>
<td>TA stance</td>
<td>55.78</td>
<td>8.68</td>
<td>58.66</td>
<td>11.81</td>
<td>-2.88</td>
<td>18.28</td>
<td>-12.62</td>
<td>6.86</td>
</tr>
<tr>
<td>TA swing</td>
<td>47.73</td>
<td>6.72</td>
<td>42.44</td>
<td>7.84</td>
<td>5.29</td>
<td>11.34</td>
<td>-0.75</td>
<td>11.33</td>
</tr>
<tr>
<td>TA baseline</td>
<td>15.91</td>
<td>4.23</td>
<td>13.35</td>
<td>3.11</td>
<td>2.56</td>
<td>5.19</td>
<td>-0.21</td>
<td>5.33</td>
</tr>
<tr>
<td>MG stance</td>
<td>57.35</td>
<td>6.97</td>
<td>58.93</td>
<td>5.10</td>
<td>-1.58</td>
<td>9.31</td>
<td>-6.54</td>
<td>3.38</td>
</tr>
<tr>
<td>MG baseline</td>
<td>13.29</td>
<td>3.42</td>
<td>10.93</td>
<td>5.33</td>
<td>2.37</td>
<td>6.68</td>
<td>-1.20</td>
<td>5.93</td>
</tr>
</tbody>
</table>

$RF = rectus$ femoris, $BF = biceps$ femoris, $TA = tibialis$ anterior, $MG = medial$ gastrocnemius, $SD = standard$ deviation
Amplitude is expressed as a percentage of peak root-mean-square (RMS) amplitude during gait
* denotes that the groups were compared using the Wilcoxon signed rank test
8.4.7 Locomotor-specific measure of spasticity

8.4.7.1 Comfortable speed

Methods to derive a locomotor-specific measure of spasticity (LSMS) were implemented as described in Chapter 6, Section 6.7.4.3. Two quantitative parameters were extracted, 1) the lengthening velocity threshold (LVT) that triggered muscle activation during a lengthening phase for that muscle, and 2) the time (percentage GC duration) at which this activation was initiated, termed the critical time, tc. The frequency of a positive slope between the EMG RMS and lengthening velocity was also presented for descriptive analysis. The CSM group showed a trend of lower LVT during lengthening, indicating greater velocity-related sensitivity to stretch, in all muscles except MG. This trend was significant for TA (p = 0.05). Similarly, the time of EMG activity onset during the GC was earlier in RF and BF in CSM, but not in MG or TA, however this trend was not significant. The results of the LSMS are presented in Table 8.12.

<table>
<thead>
<tr>
<th>Measure</th>
<th>CSM Mean</th>
<th>CSM SD</th>
<th>HC Mean</th>
<th>HC SD</th>
<th>Difference Mean</th>
<th>Difference SD</th>
<th>Confidence Interval</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RF LVT (l0/s)</td>
<td>2.78</td>
<td>1.12</td>
<td>3.52</td>
<td>1.56</td>
<td>-0.74</td>
<td>1.82</td>
<td>-1.71</td>
<td>0.23</td>
</tr>
<tr>
<td>RT tc (% GC)</td>
<td>84.08</td>
<td>6.70</td>
<td>87.82</td>
<td>6.06</td>
<td>-3.74</td>
<td>8.72</td>
<td>-8.39</td>
<td>0.90</td>
</tr>
<tr>
<td>BF LVT (l0/s)</td>
<td>1.47</td>
<td>0.43</td>
<td>1.59</td>
<td>0.47</td>
<td>-0.13</td>
<td>0.63</td>
<td>-0.46</td>
<td>0.21</td>
</tr>
<tr>
<td>BF tc (% GC)</td>
<td>79.49</td>
<td>3.86</td>
<td>81.41</td>
<td>4.81</td>
<td>-1.91</td>
<td>6.55</td>
<td>-5.40</td>
<td>1.58</td>
</tr>
<tr>
<td>TA LVT (l0/s)</td>
<td>1.02</td>
<td>0.71</td>
<td>1.67</td>
<td>1.09</td>
<td>-0.65</td>
<td>1.20</td>
<td>-1.29</td>
<td>0.01</td>
</tr>
<tr>
<td>TA tc (% GC)</td>
<td>56.83</td>
<td>5.24</td>
<td>55.92</td>
<td>4.37</td>
<td>0.91</td>
<td>8.18</td>
<td>-3.45</td>
<td>5.27</td>
</tr>
<tr>
<td>MG LVT (l0/s)</td>
<td>0.50</td>
<td>0.71</td>
<td>0.47</td>
<td>1.46</td>
<td>0.03</td>
<td>1.54</td>
<td>-0.79</td>
<td>0.85</td>
</tr>
<tr>
<td>MG tc (% GC)</td>
<td>20.39</td>
<td>9.13</td>
<td>20.97</td>
<td>8.90</td>
<td>-0.58</td>
<td>10.86</td>
<td>-6.36</td>
<td>5.21</td>
</tr>
</tbody>
</table>

RF = rectus femoris, BF = biceps femoris, TA = tibialis anterior, MG = medial gastrocnemius, l0/s = normalised muscle lengths per second, GC = gait cycle

Data on the direction of the slope between lengthening velocity and EMG amplitude are shown in Figure 8.9. The dominant direction of the slope out of ten trials for each participant was analysed, such that if five or more trials showed a positive slope during lengthening, then the dominant direction for that participant was deemed to be positive. A noteworthy finding was the predominance of positive slopes in MG in both groups,
indicating a velocity-sensitive response to lengthening. This was in keeping with this muscle’s lower LVT (Table 8.10). For each muscle, the proportions of dominantly positive slopes in CSM and HC participants were compared using Fisher’s exact test. Participants with CSM showed a higher incidence of dominant positive slopes in TA, however this did not reach statistical significance ($p = 0.07$). No differences in dominant slope direction were found for MG, RF or BF, suggesting that the gain of the stretch reflex was not different between CSM and HC participants in these muscles during gait.

Positive slopes suggest a velocity sensitive response to lengthening

**Figure 8.9: Dominant direction of the slope of muscle lengthening velocity with respect to EMG amplitude ($\% \text{RMS}_{\text{Max}}$) for CSM and HC participants at comfortable speed**

Positive slopes suggest a velocity sensitive response to lengthening

### 8.4.7.2 Matched speed

The critical time of onset of RF and TA activity during lengthening occurred significantly earlier in CSM than in HC (RF: CSM 84% GC, HC 89.4% GC, $p = 0.02$; TA: CSM 56.8% GC, HC 61.3%, $p = 0.03$). There were no differences in the LVT of these muscles, in contrast to the results at comfortable speed. The findings for MG were consistent with comfortable speed analysis, with no differences in either LVT or $t_c$. However, the LVT of BF was significantly lower in HC, 1.11 l/s, compared to 1.47 l/s in CSM. Results are shown in Table 8.13.
Table 8.13: Locomotor-specific measure of spasticity of CSM and HC participants at matched speed

<table>
<thead>
<tr>
<th>Measure</th>
<th>CSM</th>
<th>HC</th>
<th>Difference</th>
<th>Confidence Interval</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td></td>
</tr>
<tr>
<td>RF LVT (l0/s)</td>
<td>2.78</td>
<td>1.12</td>
<td>3.26</td>
<td>1.20</td>
<td>-0.49</td>
</tr>
<tr>
<td>RT tc (% GC)</td>
<td>84.08</td>
<td>6.70</td>
<td>89.40</td>
<td>5.07</td>
<td>-5.32</td>
</tr>
<tr>
<td>BF LVT (l0/s)</td>
<td>1.47</td>
<td>0.43</td>
<td>1.11</td>
<td>0.40</td>
<td>0.36</td>
</tr>
<tr>
<td>BF tc (% GC)</td>
<td>79.49</td>
<td>3.86</td>
<td>82.76</td>
<td>5.53</td>
<td>-3.26</td>
</tr>
<tr>
<td>TA LVT (l0/s)</td>
<td>1.02</td>
<td>0.71</td>
<td>1.44</td>
<td>0.94</td>
<td>-0.42</td>
</tr>
<tr>
<td>TA tc (% GC)</td>
<td>56.83</td>
<td>5.24</td>
<td>61.27</td>
<td>4.27</td>
<td>-4.45</td>
</tr>
<tr>
<td>MG LVT (l0/s)</td>
<td>0.50</td>
<td>0.71</td>
<td>0.71</td>
<td>0.49</td>
<td>-0.21</td>
</tr>
<tr>
<td>MG tc (% GC)</td>
<td>20.39</td>
<td>9.13</td>
<td>18.33</td>
<td>4.43</td>
<td>2.06</td>
</tr>
</tbody>
</table>

RF = rectus femoris, BF = biceps femoris, TA = tibialis anterior, MG = medial gastrocnemius, lengthening velocity threshold = LVT, normalised lengths per second = l0/s, GC = gait cycle

The slope of the relationship between EMG amplitude and lengthening velocity was also calculated for each trial, and the dominant slope direction was determined for each participant. Results on slope direction were identical to those at comfortable speed, presented in Section 8.4.7.1. In other words, the dominant slope direction did not change in HCs when speed was reduced.

8.4.8 Principal components analysis of gait data

8.4.8.1 Principal components analysis of temporal-spatial, kinematic and kinetic data

PCA was conducted on the pooled data of temporal-spatial, kinematic and kinetic variables listed in Tables 8.2, 8.4 and 8.6. In total, 50 variables were included in the PCA algorithm. The first PC, labelled PC1, accounted for 44.4% of the total variability of the data, the second, PC2, accounted for a further 9.8% of the variability, and the third PC, PC3, accounted for a further 8.2%. In all, 62.3% of the total variability of the data set was accounted for by the first three PCs. The remaining PCs each accounted for no more than 6.9% of the total variability of the data set, and these were not considered further. Table 8.14 shows the first 3 PCs and their contributing variables.
Table 8.14: Interpretation of the first three principal components for pooled temporal-spatial, kinematic and kinetic data of CSM and HC participants

<table>
<thead>
<tr>
<th>PC</th>
<th>Proportion</th>
<th>Positive Variables</th>
<th>Score</th>
<th>Negative Variables</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>PC1</td>
<td>42.9%</td>
<td>Gait speed</td>
<td>0.207</td>
<td>Anteroposterior acceleration GRF</td>
<td>-0.199</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Step length</td>
<td>0.197</td>
<td>Double support time, seconds</td>
<td>-0.196</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stride length</td>
<td>0.196</td>
<td>Double support duration, % GC</td>
<td>-0.189</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Single support duration, % GC</td>
<td>0.193</td>
<td>Opposite foot off, % GC</td>
<td>-0.184</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anteroposterior deceleration GRF</td>
<td>0.185</td>
<td>Hip absorption power, H2</td>
<td>-0.170</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vertical GRF</td>
<td>0.182</td>
<td>Knee absorption power, K1</td>
<td>-0.168</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hip power, pre swing (H3)</td>
<td>0.181</td>
<td>Foot off, % GC</td>
<td>-0.167</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ankle power generation, stance (A2)</td>
<td>0.178</td>
<td>Step time</td>
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</tr>
<tr>
<td></td>
<td></td>
<td>Knee sagittal plane range</td>
<td>0.172</td>
<td>Knee absorption power, K4</td>
<td>-0.164</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hip sagittal plane range</td>
<td>0.166</td>
<td>Stride time</td>
<td>-0.160</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Knee power generation, K2</td>
<td>0.165</td>
<td>Knee absorption power, K3</td>
<td>-0.158</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cadence</td>
<td>0.163</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Knee flexion in swing</td>
<td>0.160</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pelvic obliquity range</td>
<td>0.158</td>
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<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ankle plantarflexor moment</td>
<td>0.151</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PC2</td>
<td>9.8%</td>
<td>Average pelvic tilt</td>
<td>0.386</td>
<td>Single support time, seconds</td>
<td>-0.323</td>
</tr>
<tr>
<td></td>
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<td>Hip position, initial contact</td>
<td>0.371</td>
<td>Ankle position, initial contact</td>
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<tr>
<td></td>
<td></td>
<td>Hip rotation range</td>
<td>0.247</td>
<td>Ankle plantarflexor moment</td>
<td>-0.207</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hip extension</td>
<td>0.225</td>
<td>Stride time</td>
<td>-0.173</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hip extensor moment</td>
<td>0.165</td>
<td>Step time</td>
<td>-0.166</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cadence</td>
<td>0.165</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pelvic rotation range</td>
<td>0.162</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Foot off, % GC</td>
<td>0.159</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pelvic obliquity range</td>
<td>0.159</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PC3</td>
<td>8.5%</td>
<td>Knee extension</td>
<td>0.380</td>
<td>Knee absorption power, K1</td>
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</tr>
<tr>
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<td></td>
<td>Knee flexion in stance</td>
<td>0.360</td>
<td>Knee sagittal plane range</td>
<td>-0.166</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hip power generation, H1</td>
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</tr>
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<td>Knee position, initial contact</td>
<td>0.273</td>
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</tr>
<tr>
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<td>Knee extensor moment</td>
<td>0.257</td>
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<td></td>
<td>Medio-lateral GRF</td>
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<tr>
<td></td>
<td></td>
<td>Hip extension</td>
<td>0.224</td>
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<tr>
<td></td>
<td></td>
<td>Hip flexor moment</td>
<td>0.223</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hip position, initial contact</td>
<td>0.222</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Knee flexor moment</td>
<td>0.182</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Positive variables are those that increase as the PC increases
Negative variables are those that have an inverse relationship with the PC score
Kinematic and kinetic variables refer to peak values unless range is stated
GRF = ground reaction force, PC = principal component, GC = gait cycle
The symbol * denotes variable with a negative sign by convention
A review of the component scores indicated that the variables contributing to PC1 were functions of propulsion and the generation of momentum. PC1 was influenced by the relationship between walking speed and sagittal plane range at the hip and knee, as well as the power bursts associated with gait speed. The variables contributing to PC2 and PC3 occurred during the stance phase of gait, and this suggested that PC2 and PC3 reflected different aspects of stability in stance. Many of PC2’s contributing variables centred on the mid and terminal stance phases of gait, while PC3 variables tended to centre on the loading response to mid stance phases. Individual PC scores are shown in Figure 8.10. The differences between the groups were not tested for significance because the aim of PCA was to explore the relationships between the variables, and not to test a hypothesis. Figure 8.11 illustrates the dispersion of scores of CSM and HC participants in the PC1 and PC2 components, and shows a pattern of clustering with CSM participants generally showing lower scores on PC1 and greater variability on PC2.

Figure 8.10: Box-and-whisker plots of the three principal component variables from 3DGA data in CSM and HC participants
Mean scores are shown by the central line, standard deviations by the boxes, and minimum and maximum values by the whiskers
PC = principal component
The arrows and boxed variables show the relationship between these individual variables and the overall PC score.

A-P GRF = antero-posterior ground reaction force, GC = gait cycle, PC = principal component.

**Figure 8.11: Scatterplot of individual participant scores on principal components 1 & 2**

The arrows and boxed variables show the relationship between these individual variables and the overall PC score.

A-P GRF = antero-posterior ground reaction force, GC = gait cycle, PC = principal component.

### 8.4.8.2 Principal components analysis including EMG data

PCA was then repeated with the inclusion of EMG timing, co-activation, amplitude, and lengthening data. A higher total number of PCs were extracted compared to PCA without EMG data. The first three PCs remained dominant, representing 30.9%, 8.9% and 7.4% of the total variability in the data, respectively. The 3DGA variables contributing to the PCs were consistent with those of the first PCA, albeit with some differences in the order of contribution. This suggested that the PCs were consistent in their representation of the components of gait, and were relatively stable to the inclusion of the EMG data. The overall scores on PC3, however, were slightly different when EMG data were included (see Figure 8.12), suggesting that the inclusion of EMG data had a greater effect on this PC than on PC1 and PC2. PC1 was negatively influenced by RF–BF co-activation duration and by BF duration, although the weightings for these variables, −0.124 and −0.142, did not reach the threshold of 0.15 required for significant contributions. PC2 was positively influenced by duration of RF activity, amplitude of RF during stance, and BF duration, suggesting that RF and BF had a role in increasing the components of stability in stance represented by PC2. PC3 was positively affected by TA burst amplitude in stance and by the critical time of MG activity onset during lengthening, suggesting that...
the EMG variables contributing to PC3 reflected stability during stance at the lower leg and foot. The variables contributing to the PCs of the combined TSP, kinematic, kinetic and EMG data set are listed in Table 8.15. Figure 8.12 shows the dispersion of scores on PC1 and PC2 for CSM and HC participants. Figure 8.13 illustrates box-and-whisker plots of the HC and CSM scores on the first three PCs. As illustrated by these graphs, the overall clustering of HC and CSM scores is well preserved compared to the first PCA without EMG.
<table>
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<tr>
<th>PC</th>
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<td>Hip flexor moment*</td>
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<td></td>
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<td>TA stance burst amplitude</td>
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<td>MG critical time</td>
<td>0.166</td>
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</tr>
<tr>
<td></td>
<td></td>
<td>Foot off, % GC</td>
<td>0.151</td>
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Positive variables are those that increase as the PC increases
Negative variables are those that have an inverse relationship with the PC score
Kinematic and kinetic variables refer to peak values unless range or position is stated
GRF = ground reaction force, MG = medial gastrocnemius, RF = rectus femoris, BF = biceps femoris, TA = tibialis anterior, GC = gait cycle, PC = principal component
The symbol * denotes that variable has a negative sign by convention
Figure 8.12: Scatterplot of individual participant scores on principal components 1 & 2, with EMG data included in the analysis

RF = rectus femoris, BF = biceps femoris, TA = tibialis anterior, PC = principal component

Figure 8.13: Box and whisker plots of the three principal component variables from 3DGA and EMG data of CSM and HC participants

PC = principal component
8.5 Discussion

8.5.1 Differences in gait between CSM and HC participants

8.5.1.1 Biomechanical changes in gait in CSM compared to HCs at comfortable speed

The aim of this cross-sectional study was to compare the gait patterns of people with untreated CSM to those of healthy age- and gender-matched controls. The first comparison was conducted at the comfortable gait speed of both groups to illustrate the natural walking pattern adopted by each participant. In keeping with other studies (Kuhtz-Buschbeck et al., 1999, Singh and Crockard, 1999, Maezawa et al., 2001, Suzuki et al., 2002, Lee et al., 2011), the current study also found that people with CSM walked at a significantly slower comfortable walking speed, representing a decrease of 0.36 m/s on the speed of the HCs (1.48 m/s). The fact that the comfortable gait speed of the slowest HC, 1.22 m/s, was still 0.1 m/s faster than the mean of the CSM group, 1.12 m/s, illustrates the severity of loss of natural gait speed in CSM.

Analysis of TSPs at comfortable speed indicated that CSM participants had difficulty in generating adequate stride and step length, and also required a longer time to complete each stride. They spent a longer proportion of the GC in double support and less in single support and swing, suggesting either a lack of stability in single leg stance, weak contralateral push off at pre swing, premature cessation of swing due to hyperactivity of the hamstrings, or limited hip extension on the stance leg (Winter, 1985, Kirtley, 2006).

Analysis of kinematics and kinetics provided further insight into the underlying movement patterns. CSM participants differed from their HC counterparts in their range of pelvic obliquity over the GC, with HCs showing an average of 2.4° more obliquity, which appeared to be due to reduced upward rather than downward movement as shown in Figure 8.2. The difference between groups exceeded the SEM of pelvic obliquity range of 0.92°, as per Chapter 7, Section 7.5.2.2. This was partly in keeping with the findings of a previous study, which found that pelvic obliquity was reduced in people who had a history of CSM for less than one year, but normal in those who had CSM for more than a year, compared to HCs (Suzuki et al., 2002). The significance of the reduction in pelvic obliquity in the current study was unclear. Upward pelvic obliquity was considered to increase the effective leg length of the trail limb during stance, and was deemed to be one of the six determinants of gait (Saunders et al., 1953). It was later shown that this movement occurred too late in the GC to have such an effect (Della Croce et al., 2001). It was possible that reduced pelvic obliquity in CSM was a secondary effect of other factors affecting stability in stance.

At the hip, there were no differences in ROM in the frontal or transverse planes. Peak hip extension showed a mean reduction of 3° in CSM compared to HC. This did not reach
statistical significance, nor did it exceed the SEM of peak hip extension of 3.2°. However, it led to a significant overall reduction in total sagittal plane range at the hip in CSM. Stimulation of afferent receptors in the hip joint occurs during hip extension and signals the transition from stance to swing, contributing to appropriate muscle activation. If the hip is prevented from reaching an extended position, the generation of the flexor burst and the onset of swing may be inhibited (Dietz, 2002). There were significant decreases in the H2 and H3 hip powers during terminal stance and pre swing in the CSM cohort. This may have been a feature of the reduced speed of the CSM group (Kirtley, 2006). It may also have resulted from decreased hip extension, which then impaired the generation of propulsion into swing.

Significant differences were found in knee flexion in stance, swing, and total knee sagittal plane range, in keeping with the findings of previous studies on gait in CSM (Kuhtz-Buschbeck et al., 1999, Maezawa et al., 2001, Suzuki et al., 2002, Lee et al., 2011). As discussed in Chapter 3, Section 3.2.4, a previous study found reduced knee flexion during swing in people with symptoms of CSM who did not have a reduction in gait speed, suggesting that it was an early sign of gait impairment in CSM (Maezawa et al., 2001). The current study also found lower power absorption peaks of the knee at loading response (K1) and initial swing (K3) in CSM, confirming that the losses of knee flexion in stance and swing were associated with reduced eccentric activity. In particular, loss of the K1 peak at loading response indicated impairment of the loading response due to weak quadriceps (Kirtley, 2006).

At the ankle, CSM participants reached significantly lower peak plantarflexion than their HC counterparts at the terminal stance and pre swing phases of gait. This was associated with reduction in the ankle plantarflexor moment and the A2 power generation peak, with a decrease of more than 2 W/kg in the latter. The plantarflexors have been shown to provide most of the body’s support during terminal stance (Anderson and Pandy, 2003). The loss of their concentric power burst at this point would have limited the propulsion of the leg into swing (Kirtley, 2006). This loss of propulsion was not compensated by an increase in the H3 hip power generation peak, as noted in other populations such as stroke (Nadeau et al., 1999). An effective swing phase, and therefore stride length, depends on the generation of sufficient momentum at the toe-off phase of gait (Kirtley, 2006). The lack of plantarflexion and A2 power at pre swing in CSM could have contributed to the shorter stride length and reduced single support time noted in the analysis of the TSPs.

8.5.1.2 Biomechanical changes in gait in CSM compared to HCs at matched speed

There is some difficulty in the interpretation of kinematic and kinetics between two sets of participants walking at different speeds. Despite the obvious advantage that it reflects the natural capabilities of each participant, many variables, particularly kinetics, have a
predictive dependency with gait speed (Riley et al., 2001, Lelas et al., 2003). Other variables, such as sagittal plane kinematics, show a positive correlation with speed (Kirtley et al., 1985). The differences between CSM and HC kinematics and kinetics could simply restate the fact that people with CSM walk more slowly, rather than providing insight into the causes of the slower gait, as suggested in stroke (Chen et al., 2005).

To control for the potential confounding effects of gait speed, each HC performed ten trials at the walking speed of the CSM participant to whom he or she was paired. This necessitated a mean reduction in walking speed of 0.36 m/s, with a range of 0–0.68 m/s, depending on the severity of the CSM participant’s mobility limitation. HCs achieved the target speed by reducing cadence, while maintaining a stride length close to that of their comfortable speed. The proportion of the GC spent in single support was significantly shorter in CSM than in HC participants at matched speed, confirming the analysis at comfortable speed that people with CSM lacked either stability in stance or the propulsive power to initiate swing (Kirtley, 2006).

Analysis of kinematics and kinetics further highlighted the differences between groups. Motion at the pelvis and hip were similar at matched speed, however peak power generation at the hip in loading response, H1, and peak hip extensor moment at loading response, were higher in the CSM group. A pathological increase in H1 power can compensate for a disruption of forward progression during stance (Kirtley, 2006).

The HC group demonstrated greater peaks of ankle dorsiflexion during stance, ankle plantarflexion at toe-off and AP GRF at pre swing, as well as higher knee flexion in swing. These findings indicated an impairment of the CSM participants’ ability to 1) control forward progression and generate propulsion through initial lengthening of the Achilles tendon in dorsiflexion, and 2) release that energy through ankle plantarflexion at pre swing (Kirtley, 2006). This was not just a consequence of slower gait speed. The finding of reduced knee flexion in swing was also consistent with impaired swing initiation caused by inadequate propulsion (Gage, 1991). The combination of reduced ankle plantarflexion at pre swing and reduced knee flexion in swing has previously been reported in the paretic limbs of people with stroke (Chen et al., 2005) and with traumatic central cord syndrome (Gil-Agudo et al., 2009). Anecdotally, people with CSM commonly cite toe catching as a cause of falls. This could be related to reduced knee flexion in swing because this variable is a key component in ensuring adequate foot clearance (Gage et al., 1995).

The condition of matched speed aimed to control for the association of many kinematic and kinetic variables with gait speed, however it had the disadvantage of imposing an unfamiliar locomotor pattern on one set of participants. Individual preference for a particular gait speed occurs close to the speed at which energy consumption per unit distance is minimised, allowing the locomotor system to take advantage of the passive
mechanical properties of the lower limb in the absorption and generation of power (Jordan et al., 2007). The strategies used by the HCs in the current study to achieve slower speeds did not reflect the natural preferences of their neuromusculoskeletal systems. The deliberate enforcement of a reduced speed may have had confounding effects on the kinematic and kinetic patterns of the HCs because they may have adopted alternative and unfamiliar strategies to impede the generation of their natural momentum. Some evidence of this was noted in the EMG analysis.

8.5.1.3 EMG analysis of the neuromuscular control of locomotion

Further insight into the muscular activation patterns that produced the observed kinematics and kinetics was gleaned from EMG analysis of four lower limb muscles. There were key differences between HC and CSM participants in the timing of muscle activation. In CSM, the duration of activation of RF, BF and TA was prolonged by around 10% GC duration. The increases in duration of RF and BF activation were associated with an increase in their co-activation of more than 5% GC duration, with HCs showing BF–RF co-activation of 9.3% GC duration and CSM participants, 14.4% GC duration. The timing of muscle activation in HCs was relatively robust to changes in speed. Total activation duration varied for each muscle by around 1–2% GC duration, even though gait speed varied by a significantly greater amount under the comfortable and matched speed conditions. This provided further evidence that the timing of activation in CSM was a feature of abnormal neuromuscular control, and not a consequence of slower gait speed. This was the first study to quantify timing abnormalities in gait in CSM.

Co-activation occurs during normal movement and may improve movement efficiency during performance of lower limb activities by providing increased joint stabilisation (Busse et al., 2006). However, a point is reached when increased co-activation will excessively stiffen a joint and impair its movement (Damiano, 1993). Prolonged duration of muscle activation and excessive co-activation have been attributed to an inability to voluntarily activate intended muscles due to impaired corticospinal function and defective motor commands (Shiavi et al., 1987). This interpretation suggests that timing abnormalities are a direct consequence of the CNS lesion.

In contrast, recent research has provided evidence for prolonged activation and co-activation as adaptive compensatory strategies. In one study of people with orthopaedic problems, prolonged duration of muscle activity during gait was found in 66% of the lower limbs studied (Brunner and Romkes, 2008). This finding was attributed to the need to compensate for weakness. The patterns of prolonged muscle activation were similar to those reported in neurological disorders, such as CP. This led to the hypothesis that people with neurological disorders essentially used the same adaptive strategies for muscle weakness as people without neurological dysfunction, and that weakness was the primary impairment underlying the timing abnormalities (Brunner and Romkes, 2008). A
finding of particular interest to the current study was that co-activation between hamstrings and quadriceps was the second most common timing abnormality noted in people with orthopaedic conditions, present in 17% of the lower limbs studied. This was attributed to the need for increased proximal stabilisation during stance. It is possible that people with CSM used this co-activation strategy for similar reasons. The most common timing abnormality in the orthopaedic population was prolonged duration of MG activation, but this was not observed in the current study.

Further evidence of co-activation as an adaptive strategy was found in a study of gait following stroke. People with stroke had less co-activation during gait on the paretic side, but greater co-activation on the non-paretic side, compared to HCs (Lamontagne et al., 2000b). There was little evidence to suggest that increased co-activation on the non-paretic side resulted from ipsilaterally mediated effects of the CNS lesion. Rather, it was considered more likely that this was an adaptive behaviour by the non-paretic limb to help maintain postural stability, particularly in the double support phases of gait when it was assisting the paretic limb in the support function (Lamontagne et al., 2000b). This provided further evidence for the hypothesis that co-activation and prolonged timing of muscle activation are compensatory mechanisms, rather than primary consequences of the CNS injury.

Comparison between the findings of the current study and other studies was limited by the differences in the methods used to determine the timing of muscle activation. Brunner and Romkes (2008) used semi-quantitative visual analysis. This facilitated assessment of subtle changes in the signal, but may have led to greater variation in the results than if a computerised algorithm had been used. Lamontagne et al. (2001) used an amplitude threshold of 20 microvolts (µV) to designate activation, however this method may have been confounded by the higher baseline activity of people with neurological impairment (Roetenberg et al., 2003). Future studies could address this problem by consistently applying a validated timing detection method, such as the DTM method of the current study.

In addition to the timing of activation of a particular muscle, a measure of its intensity was also required. The intensity of muscle activation over the GC was indicated by the mean amplitudes of bursts of activity and inactivity, normalised to the maximum amplitude obtained during the GC in that session. The BF activity burst in stance was of higher normalised mean amplitude in HC at comfortable speed than in CSM, but this difference did not occur at matched speed. Other muscle activity bursts showed no difference between pairs. In contrast, the mean baseline amplitudes of BF and RF were significantly higher in CSM than in HCs at both comfortable and matched speeds.

Normalisation of a muscle’s EMG amplitude to the peak amplitude from the GC posed some difficulty in the interpretation of the results. Lower peak amplitude would over-
emphasise the amplitude at other phases of the GC. This meant that the higher amplitude of the inactive phases could have been caused by an inability to scale down the intensity of a muscle's output when its activation was not required, or by an inability to increase the intensity during periods when activation was required. The use of a gait-based normalisation reference may have diluted the variability between participants, as there was no way to know whether each individual's peak amplitude was high or low compared to normal.

Some authors have reported the absolute value of the amplitude at specific phases of the GC in V, and have found significant differences in burst amplitudes of people with neurological gait impairment compared to HCs (Lamontagne et al., 2002). However, there are difficulties with this approach. The absolute value of the EMG signal amplitude can be affected by intrinsic participant characteristics, such as subcutaneous fat thickness (Lehman and McGill, 1999), as well as extrinsic factors such as heat, humidity, and the location of the electrode over the muscle (Hogrel, 2005). Although muscle morphology in CSM has not been studied with reference to atrophy or soft tissue infiltration, in the current study it was subjectively more difficult to palpate the lower limb muscles of CSM compared to HC participants during the placement of electrodes described in Chapter 6, Section 6.6.3. The muscles of HCs tended to be more prominent, and identification of the optimum location for electrode placement was therefore easier. If the absolute amplitude of the EMG signal had been studied, it would not be possible to know whether a reduced amplitude in CSM were due to inability to generate a signal of higher intensity, or whether the signal had been attenuated by a higher ratio of non-contractile soft tissue to muscle tissue.

In the absence of clear indication of the intensity of muscle activation, some authors suggested that the ratio of the mean amplitude of bursts of activity to baseline activity provided a relevant indication of the motor control capacities of the patient (Roetenberg et al., 2003). Compared to HCs at both matched and comfortable speed, the higher mean amplitude of BF and RF of CSM participants during the “off” phases suggested that these muscles were less efficient at scaling their intensity appropriately. Unfortunately, the limitations of the amplitude normalisation method prevented any further interpretation of whether this were due to higher baseline signal, reduced peak output, or a combination of both.

Abnormal timing and amplitude parameters appeared to affect the proximal lower limb muscles of RF and BF, with less effect on MG and TA. This was in keeping with the findings of other studies on the manifestation of motor problems following incomplete SCI. In a study of 27 people with incomplete SCI, including six with CSM, timing of activation and accuracy of movement of dorsi- and plantarflexors were relatively unaffected compared to a comparison group of people with stroke, even though the peak
torques of these muscles were similarly reduced in both groups (van Hedel et al., 2010). Similar findings were shown using smaller sample sizes of the same populations by (Wirth et al., 2008). Van Hedel's study evaluated a task involving the application of dorsiflexor and plantarflexor torque to move a visual target. The findings from the current study suggested that the timing of activation of MG and co-activation between TA and MG were relatively preserved during gait in CSM. The scaling of motor output by MG and TA, as measured by the mean amplitude during and outside their key bursts, was comparable to that of HCs.

It was of interest that timing and amplitude abnormalities appeared to affect the muscles of the thigh, while most kinematic and kinetic differences between CSM and HC participants occurred at the knee and ankle. This may be explained by the need for greater proximal co-activation and activation duration to stabilise the lower limb due to a loss of distal power absorption and generation capability. Prolonged activation of BF may, for example, have produced the higher hip extensor moment and H1 power at loading response. As discussed in Section 8.5.1.2, increased H1 is a compensatory strategy to facilitate forward translation of the trunk over the supporting limb where the contralateral pre swing phase has failed to generate adequate propulsion (Kirtley, 2006).

8.5.1.4 Analysis of the hyperexcitability of the stretch reflex during locomotion

EMG analysis then progressed to the interpretation of the LSMS, which described a muscle's response to lengthening during the GC. Hyperexcitability of the stretch reflex is just one component of the abnormal muscle activity observed in spasticity, and is associated with both an increase in the gain of the stretch reflex and a reduction in the stretch receptors' threshold for activation (Pandyan et al., 2005). If stretch reflex hyperexcitability contributed to the abnormal muscle activity observed in CSM, it would be expected that 1) EMG activity would occur at a lower lengthening velocity in CSM than in HC, indicating reduced stretch receptor threshold, and 2) a given lengthening velocity would elicit a greater efferent response in EMG activity, indicating increased reflex gain.

At comfortable speed, CSM participants showed a trend of lower LVTs for all muscles with the exception of MG. Differences were significant only for TA during the terminal stance phase of gait, where TA activity was initiated during at a lengthening velocity of just over 1 l/s compared to the HC threshold of 1.7 l/s. The incidence and frequency of positive slopes of TA were also significantly higher in CSM than in HC participants. This confirmed that the TA showed earlier activation during lengthening, indicating higher sensitivity of the stretch reflex, and greater EMG activity as lengthening velocity increased, indicating an increase in the gain of the stretch reflex compared to HCs. It is possible that this activation of TA impeded the progression of the ankle into plantarflexion and contributed to the limitation of the A2 power burst, two findings that were noted in the analysis of kinematics and kinetics compared to HC at comfortable speed.
It was previously suggested that the slope of the relationship between normalised EMG amplitude and lengthening velocity determined whether the response to lengthening was normal, indicated by a negative slope, or pathological, indicated by a positive slope. This was shown to have discriminative validity in stroke when compared to data from HCs (Lamontagne et al., 2001). However, the current study found that the dominant slope direction was positive in a number of HCs, indicating that slope direction alone did not distinguish between pathological and non-pathological responses to lengthening. In the case of MG, a positive slope was the dominant direction in both HC and CSM participants. The reasons for this may lie in the nature of the eccentric contraction to be studied. In the evaluation of spasticity, the question is whether muscle activation allows normal physiological lengthening to take place in a controlled manner, or whether the activation, through premature timing or excessive amplitude, impedes joint movement and thus interferes with the tasks of the GC. It has been proven that the H reflex, a measure of the excitability of the monosynaptic stretch reflex, increases towards the end of stance, allowing the stretch reflex to assist in the push-off phase of gait (Capaday and Stein, 1987). The dominance of positive slopes in MG in HCs suggested that, rather than being an abnormal response, it was normal for EMG activity to increase during this lengthening phase in the GC. This reinforced the fact that the monosynaptic stretch reflex was more excitable at the end of stance. In contrast, the dominant slope direction for TA, BF and RF was negative in HCs, indicating that a normal gait allowed some yield to occur during lengthening before muscle activity was initiated to control or halt this movement. The current study therefore provided no evidence for hyperexcitability of the stretch reflex of MG as a factor in the abnormal gait of CSM.

Analysis of the LSMS at matched speed yielded largely similar findings for TA, RF and MG responses to lengthening, although the difference in LVT of TA did not reach statistical significance, as it had done at comfortable speed. An unexpected finding was the reversal of the expected relationship in BF LVT at terminal swing, which was significantly lower in HCs than their CSM counterparts. As HCs by definition had no neurological impairment, this lower LVT could not be due to spasticity. This highlighted a limitation of LVT as an indicator of spasticity during locomotion at an imposed speed. LVT measured the velocity at which a muscle activated to control its lengthening by initiating an eccentric contraction, but it did not give an indication of whether this activation was due to a reduction in the threshold of the stretch receptors, or a voluntary activation. In response to the imposition of the unnaturally slow gait required by the matched speed condition, it was likely that HCs were consciously trying to slow the advancement of the tibia to increase their stride time, reduce cadence and ensure that the slower speed was achieved. They may have activated their hamstring muscles deliberately at a lower than normal lengthening velocity to impair the progression of the swing phase, thereby delaying initial contact and lowering cadence.
In addition to the lack of distinction of voluntary versus reflexive activation on lengthening, the LSMS was considered to have a further limitation. Calculations of muscle lengthening by this method did not discriminate between lengthening of the muscle belly and lengthening of its tendon, but instead they were analysed as one unit. Studies using ultrasound technology revealed key differences in the behaviour of muscles and tendons in response to lengthening. In healthy children, lengthening of the gastrocnemius–soleus complex was characterised by near-isometric muscle contraction and passive stretching of the Achilles tendon by up to 7 mm, followed by an elastic recoil of the tendon at push-off to contribute to the A2 power generation peak (Fukunaga et al., 2001). Prior to the publication of Fukunaga’s findings, it was thought that gastrocnemius contracted eccentrically, not isometrically, during the mid stance phase of gait. However, it is not known whether this relationship holds true in children or adults with neurological dysfunction. It has been hypothesised that children with CP may have insufficient ability to control lengthening with an isometric contraction, and this may force their gastrocnemius muscles to act eccentrically (Shortland, 2011).

The objective of the LSMS in the current study was to examine the lower limb muscles for evidence of hyperexcitability of the stretch reflex as a manifestation of spasticity during gait. It was beyond the scope of the study to examine relative lengthening of the various components of the musculotendinous units. The lack of distinction between lengthening of muscle versus tendon is acknowledged as a limitation. Nonetheless, the LSMS provided quantitative data on the responses of a muscle when a lengthening force was imposed on it, even if it did not identify the structures that actually lengthened, or whether the associated contraction was reflexive or voluntary. The evaluation of spasticity during gait is a complex task, and the method used in this study was the best available at the time of study design. Its specificity to gait and avoidance of the need for unnatural stimuli were considered advantages to this method (Lamontagne et al., 2001).

**8.5.1.5 Principal components analysis**

Principal components analysis (PCA) was performed on the pooled TSP, kinematic and kinetic parameters of the CSM and HC participants to explore the data, identify its dominant variability, and reduce its dimensionality into smaller numbers of principal component variables (PCs). This approach has been used successfully in CP, resulting in the classification of gait patterns that were broadly consistent with those identified by observation (Carriero et al., 2009). In the current study, three dominant PCs were extracted from the pooled data. In keeping with the findings of Carriero et al. (2009), the first PC, accounting for 42.9% of the total variability of the data, was a function of gait speed, sagittal plane kinematics and the absorptive and propulsive powers associated with speed. This suggested that PC1 represented the ability of the locomotor system to generate momentum. CSM participants tended to score below zero on this PC, while HC participants scored above zero. This was not surprising, given the slower gait speed of
CSM participants and their lower scores on many of the variables contributing to PC1. PCA did not determine whether the lower peak powers and sagittal plane motion were the cause or the result of the slower gait speed, however it provided support to the findings that the generation of momentum was a deficiency in the CSM gait.

The second PC included variables that were associated with the single support phase of gait. Interpretation of these as a group suggested that PC2 represented stability in single leg stance. Earlier analysis of TSPs had shown that CSM participants spent a shorter proportion of the GC in single support compared to HCs at both matched and comfortable speed. It was not clear from kinematics and kinetics whether this was due to lack of stability in single leg stance, or to reduced generation of momentum at toe-off affecting the duration of swing and consequently the contralateral single support phase. Visual interpretation of PC2 suggested that there were differences between HC and CSM pairs in stability in single leg stance, but that these differences were not as pronounced as those of PC1. The lack of clear differences suggested that, although stability in single leg stance was somewhat impaired in CSM and contributed to the relationship between single and double support duration, it was not the key factor in this relationship.

The inclusion of EMG variables in the PCA data set resulted in higher overall variability in the data. This was reflected by the reduction in the overall percentage of variability accounted for by each PC. The first two PCs retained their overall profile of contributing variables and showed consistency in scores of CSM and HC participants with the first PCA. PC3 showed a greater change in its contributing variables and its overall scores when EMG data were included. This rendered it more difficult to interpret the aspect of gait that it represented. In relation to PC1 and PC2, the scores were much more stable, allowing some insight to be gleaned into the aspects of EMG that influence these PCs. The momentum generation represented by PC1 was negatively influenced by duration of BF activation and RF–BF co-activation. However, these variables did not achieve the weighting threshold to contribute significantly to that PC. In contrast, the stability component PC2 was positively influenced by both of these variables, as well as by the duration of RF activation and its amplitude during its stance phase burst. These findings could reflect the contribution of prolonged duration of muscle activation, particularly of BF and RF, to the task of stability in stance. To date, no studies could be found that included both 3DGA and EMG data in a PCA for gait analysis, so it is not known whether the EMG variables would contribute to the PCs in this way in other populations.

8.5.2 Factors contributing to gait impairment in CSM

In an UMNL, impaired movement is not merely the direct consequence of a central lesion, as evidenced by defective programming and reflex function. It also reflects the secondary compensatory processes induced by the primary lesion (Dietz, 2002). It is known that an
UMNL leads to an increase in the excitability of the short latency monosynaptic reflexes, a decrease in the functionally more important long latency reflexes, and a decrease in supraspinal drive. These neurophysiological changes are expressed during movement by a lack of force generating capability (paresis), abnormal muscle tone, and reduced proprioceptive input to the lower limb muscles (Dietz, 2002, Dietz, 2003). The locomotor system then adopts a movement pattern that best preserves the ability to ambulate under the constraints imposed by these impairments, and that represents the best integration between the biomechanical system and the control properties of the CNS (Taga, 1995). The challenge for the current study was to evaluate the influences of these neuromuscular and biomechanical interactions, and to determine the primary and secondary compensatory mechanisms underlying gait impairment in CSM.

8.5.2.1 Spasticity as a contributing factor

In a physical examination, a finding of spasticity is one of a cluster of signs that can indicate the presence of an upper motor neurone lesion and eventually lead to a diagnosis of myelopathy (Baron and Young, 2007). The characteristic gait of CSM has been described in observational gait analysis as a “spastic pattern” (Suzuki et al., 2002). However, in relation to neurological disorders in general, Pandyan et al. (2005) noted that there was insufficient evidence in the literature to support the hypothesis that the abnormal muscle activity observed in people with spastic movement disorders resulted from stretch reflex hyperexcitability.

The current study did not find strong evidence of stretch reflex hyperexcitability as a contributing factor to gait impairment in CSM. There was some indication of velocity-related sensitivity to lengthening in TA from its lower LVT during terminal stance and pre-swing, and its higher percentage of positive slopes. Abnormal activation of TA at this point could have contributed to the reductions in peak ankle plantarflexion and the A2 power, thereby impairing propulsion and leading to the observed shortened step and stride lengths and shortened swing phase duration. It was noted that TA LVT did not feature as a contributing variable to any of the first three PCs, meaning that it probably had little influence on the tasks of momentum generation and stability in stance. It is also unlikely that it contributed to the prolonged duration of activation of TA that was determined in the analysis of timing, as the lower LVT did not cause the TA to activate earlier in the GC in CSM.

Evidence of spasticity of the plantarflexor muscles has been found in gait in other neurological conditions (Crenna, 1998, Lamontagne et al., 2001). The current study found evidence of neither hyperexcitability of the MG stretch reflex nor premature activation of MG during stance. LVTs for activation were similar between HC and CSM participants. The incidence and frequency of positive slopes were also similar. Previous studies have shown that the presence of excessive EMG activity during passive lengthening of a
muscle in stroke was not associated with excessive activity during lengthening under
dynamic conditions (Ada et al., 1998). The current study did not examine the EMG
responses to passive lengthening in the CSM cohort, however the finding of a lack of MG
spasticity during gait were similar to those of Ada et al. (1998), but in contrast to those of
Lamontagne et al. (2001), both studies in stroke.

Similarly, there was no conclusive evidence of spasticity in RF and BF muscles. Both
muscles showed prolonged duration of activation, excessive co-activation and
excessively high EMG amplitude outside of bursts of muscle activity, compared to HCs.
The phenomenon of “sustained involuntary activation of muscles”, such as might be
indicated by excessive activation duration or co-activation, was included in the revised
definition of spasticity by the SPASM consortium (Pandyan et al., 2005). This finding
could reflect spasticity in the CSM cohort, but this assumption is confounded by the
identification of similar activation patterns in people without neurological disorders
(Brunner and Romkes, 2008).

In summary, the current study identified limited evidence of hyperexcitability of the stretch
reflex of TA only, not of the other muscles, as a possible limiting factor to the generation
of push-off at the pre swing phase of gait. It was not clear whether the abnormal timing of
muscle activation or sustained activation outside of functional bursts of activity were due
to primary manifestations of spasticity as defined by Pandyan et al. (2005), or whether
they were compensatory mechanisms. However again, these findings must be
considered within the limitations of the LSMS outlined in Section 8.5.1.4, and within the
lack of clear consensus on the manifestation of spasticity during gait.

8.5.2.2 Paresis as a contributing factor

The definition of spasticity of Pandyan et al. (2005) included the signs and symptoms of
the positive features of an UMNL, but excluded the negative features. These include
paresis, defined as a lack of force-producing capability, and lack of dexterity, defined as a
reduced ability to accurately activate selected muscle groups within the available strength
range (Pandyan et al., 2005, van Hedel et al., 2010).

The evidence for paresis as a factor in gait impairment in CSM was more compelling than
that of spasticity. There were significant differences in several peak kinetic and kinematic
parameters, particularly at the knee and ankle, suggesting an inability to generate and co-
ordinate sufficient muscular force to walk at a speed typical of people of similar age and
gender. Compared to HCs at matched speed, CSM participants showed reductions in
peak ankle plantarflexion, the acceleration component of AP GRF, and peak knee flexion
in swing, three variables that are associated with the generation of propulsive power.
These findings indicated that CSM participants had difficulty in generating momentum at
this critical phase of the GC, and that the lack of momentum was not simply due to a
slower gait speed. Furthermore, at matched speed, CSM participants showed an increase in the first hip power peak at H1, a known compensatory strategy for loss of forward progression during stance (Kirtley, 2006). The principal component PC1 was a function of progression and generation of momentum, and showed the largest difference between CSM and HC participants of the three PCs.

Evidence from EMG analysis also supported the hypothesis that paresis was a factor. Mean normalised EMG amplitudes outside the key bursts of activity of RF, BF, and, at comfortable speed, TA, were abnormally high. This may have been due to excessive background activity or to lower peak activation intensity, or both. Although the exact nature of this relationship could not be determined, it pointed to an impairment of the motor control capabilities of the CSM participants (Roetenberg et al., 2003).

Differences in the timing of muscle activation were more conclusive than analysis of amplitude, as the TKEO-based DTM routine had shown high validity and reliability. The increases in BF and RF duration of activation and in their co-activation were of sufficient magnitude to be of clinical interest. Their persistence at matched speed suggested that these findings were important features of the control of ambulation in CSM and not just the result of a slower gait speed. Co-activation of proximal musculature during gait is a known compensatory strategy for muscle weakness in people with orthopaedic problems (Brunner and Romkes, 2008) and for contralateral lower limb paresis (Lamontagne et al., 2000b) and may have been used as a similar compensatory strategy here.

In summary, there was strong evidence for paresis as a primary contributory factor to gait impairment in CSM. This was evidenced by reduced ability to generate power, reduced scaling of motor output in the accomplishment of the tasks of the GC, and the need for co-activation to enhance stability and compensate for weakness.

**8.5.2.3 Impaired proprioception as a contributing factor**

Complex movements are programmed by the CNS and adapted by proprioceptive feedback (Dietz, 2002). Impairment of one aspect of proprioception has been shown in previous studies of people with CSM using joint position sense of the knee as an outcome measure (Takayama et al., 2005b, Okuda et al., 2006). The single support phase provides the greatest proprioceptive challenge to the locomotor system during gait. Parameters related to single leg stance have been used as measures of stability in stroke (Lamontagne et al., 2002). However, the single support phase also depends on the ability of the stance leg to generate sufficient muscle power (Anderson and Pandy, 2003), as well as its ability to receive proprioceptive input and adjust the alignment of the body accordingly. It is therefore not possible to separate the various influences of paresis and proprioception in the single support phase of gait.
Whether the reduction in single support duration in CSM was due to a lack of power or a lack of proprioceptive input was unclear. Riley et al. (1990) hypothesised that it is during the double support phase that the CNS integrates the afferent signals of proprioceptive input and relays the information to the neuromuscular system to make the required postural adjustments and produce the intended movement. Lack of proprioceptive input at this point would require more time to co-ordinate the postural adjustments needed to initiate forward progression of the centre of mass and change from bipedal to unilateral stance (Buckley et al., 2005). It is possible that the decrease in single support and associated increase in double support duration reflected a lack of reliable proprioceptive input during stance.

In addition to single leg support, instability during walking is primarily assessed by key parameters in the medio-lateral direction (Judge et al., 1996a). People with CSM tended to have a slightly higher step width and medio-lateral GRF, but these differences did not reach statistical significance. There was therefore no clear evidence of impaired medio-lateral stability in CSM. However, some studies have not found step width to be an indicator of impaired balance (Krebs et al., 2002, Moe-Nilssen and Helbostad, 2005). Further studies are needed to determine indicators of reduced stability in CSM, as the lack of difference in step width cannot rule out an instability component in this population.

The second PC was a function of stability in single leg stance. Its scores were more variable among CSM participants, who tended towards positive scores while HCs had a mean negative score. Other studies using PCA have dichotomised their populations in sub-groups based on whether the scores on a PC variable were above or below zero. The scores of the PCs on the pooled data of all participants are centred on a zero mean, and therefore the sign of the score could be considered to reflect distinct sub-groupings (Gaudreault et al., 2011). It is possible that the difference in sign of the mean PC2 score of CSM and HC participants reflected fundamental differences in the stability in single leg stance of both groups, however the magnitude of the difference was small compared to that of PC1.

It was also possible that the deficiencies in the generation of momentum and propulsion at terminal stance and pre-swing were not just related to paresis, but were also a compensatory strategy for loss of proprioceptive input. The CSM participants may have decreased the time spent in the unstable situation of single leg support by using less muscle force to avoid the generation of momentum and base of support excursions associated with propulsion, in an effort to protect the locomotor system from these proprioceptive challenges.

The current study was limited in its ability to evaluate instability during gait. It examined gait in CSM participants at comfortable speed only. The locomotor systems were not challenged by faster gait speeds, or other tasks such as obstacle avoidance or tandem
walking. These conditions would have allowed further discrimination of the ability of people with CSM to control their centre of mass in more challenging conditions (Schrager et al., 2008). Furthermore, evaluation of stride-to-stride variability, an independent predictor of falls in the elderly (Hausdorff et al., 2001, Maki and McIlroy, 2006), may also have provided further information on instability. However, the aim of this study centred on the evaluation of habitual gait patterns in CSM. Many participants were challenged by normal locomotion at comfortable speed, and did not have the capacity to increase their speed further or achieve tasks such as tandem walking.

8.6 Conclusion

This chapter has presented and discussed the findings of gait impairment in CSM compared to HCs at comfortable and matched gait speeds. Key biomechanical and neuromuscular changes in the CSM gait were identified, and their significance as primary impairments or compensatory strategies was evaluated. This information has provided a profile of gait impairment in CSM and highlighted key deficits that contributed to this impairment.

The next chapter will evaluate the ability of the CNS in CSM to recover its locomotor ability following surgical decompression of the spinal cord.
Chapter 9: Changes in gait at six and twelve months following surgery for cervical spondylotic myelopathy

9.1 Introduction

Surgery is widely used as a treatment for CSM, particularly in people whose symptoms show neurological progression over time. The aim of surgical intervention is to stabilise the disease by minimising spinal cord compression at the spondylotic levels, thereby preventing further neurological deterioration (Rao et al., 2006). However, surgery itself carries numerous risks including vertebral artery injuries, damage to neural structures, and instrumentation failure (Rao et al., 2006). For many people with CSM, the decision to undergo surgery can be a difficult one. The lack of anticipated improvement in symptoms and the risk of adverse events must be weighed against the possibility of further irreversible neurological deterioration if the cord is not decompressed.

Gait impairment is one of the cardinal symptoms of CSM. General measures of gait performance, such as speed and the ability to mobilise with or without an aid, are often used as general indicators of CSM severity. There is some evidence from follow-up studies that temporal-spatial and kinematic variables can improve after surgery (Kuhtz-Buschbeck et al., 1999, Maezawa et al., 2001). The clinical significance of these improvements is not known. A more detailed examination of gait before and after surgical intervention is required to assess the true potential for recovery, as well as the clinical significance of that recovery. This would provide further information to surgeons and patients on the expected outcomes of surgery, and contribute to the expanding base of knowledge on the potential for changes in gait following spinal cord injury.

The aim of this study was to determine changes in temporal-spatial, kinematic, kinetic, and EMG variables in gait in people with CSM at six months and one year following surgery. The study was also concerned with the following secondary objectives: 1) to determine if an association existed between measures of gait impairment and severity of CSM, 2) to compare functional mobility in people with CSM to measures of gait impairment, and 3) to determine the impact of gait impairment on HRQOL.

9.2 Methods

9.2.1 Recruitment

Participants with CSM were recruited from the neurosurgical spinal assessment clinic at Beaumont Hospital over a two-year period from December 2008 to December 2010. The
procedures used for the diagnosis of CSM and the inclusion and exclusion criteria for the study were described in Chapter 6, Section 6.3.

9.2.2 Data collection

Participants attended the Movement Laboratory, RCSI, for assessment on three occasions, once before surgery, and twice after surgery at six and twelve months post-operatively. Gait analysis was conducted using 3DGA and EMG as described in Chapter 6, Section 6.6. Secondary outcome measures of CSM severity, spasticity, functional mobility and HRQOL were examined, using the procedures described in Chapter 6, Section 6.5.

3DGA and EMG data were processed as described in Chapter 6, Section 6.7. Ten gait trials were extracted from each assessment to create a representative average.

9.2.3 Statistical analysis

9.2.3.1 Power calculation

The literature review in Chapter 3 found that gait speed was the most commonly used outcome measure for gait in CSM. Reports have considered the minimal clinically important difference in gait speed to be 0.1 m/s, and although this is not specific to CSM, it shows a good correlation with the ability to carry out functional activities of daily living (Judge et al., 1996b). A power calculation based on a paired t-test found that a sample size of 13 participants would have 90% power at the 5% significant level to detect a change in gait speed of 0.1 m/s from pre- to post-operative assessment, with a SD of the difference of 0.11 m/s. The power calculation was conducted in Stata.

9.2.3.2 Hypothesis testing

Univariate analysis was conducted on individual clinically meaningful gait parameters, comprising the key points, ranges and EMG parameters previously selected and outlined in Chapter 6, Sections 6.7.2 and 6.7.4. A number of considerations were made in relation to the optimal method to test the pre- and post-operative individual parameters for statistically significant changes post surgery. Data from two follow-up assessments at six and twelve months following surgery were available. Analysis of the differences between mean scores at three time points would normally require a repeated measures analysis of variance (ANOVA). There were a number of difficulties with the application of this test to the current study. A repeated measures ANOVA assumes the property of sphericity, namely that the variance in the difference in scores between two time points is the same as the variance between two other time points (Park et al., 2009). It was expected that this assumption would not be satisfied by the small sample size of the current study,
particularly as one set of time points, baseline to six month follow-up, included surgical intervention, whereas the second time points, six and 12-month follow-up, had no intervention. Analysis of the differences in mean scores at multiple time points was further complicated by the possibility that not all participants would attend a one-year follow-up by September 2011.

In relation to the primary research question, the differences at individual time points were of less interest than the overall changes in gait, and the secondary outcome measures, over the entire follow-up period. To address this question, and to avoid the limitations of the repeated-measures ANOVA test, it was decided to conduct hypothesis testing on the differences between baseline scores and last follow-up, using paired t-tests. This would achieve the aim of identifying overall changes in gait following surgery. Descriptive and visual analysis of scores at six and twelve months would allow for interpretation of progress over the follow-up period. Prior to the application of a paired t-test, the assumption of normal distribution was verified for each variable using QQ plots and stem-and-leaf plots in Stata. The Shapiro-Wilk test and tests of skewness and kurtosis were carried out as numerical tests of normal distribution.

Secondary outcome measures of MRMI, MAS, mJOA and Nurick were non-parametric in nature. Changes in these scores from baseline to last follow-up were tested for significance using the Wilcoxon signed rank test.

The null hypothesis of the current study could therefore be stated as follows: 1) the CSM cohort would not experience change in key gait parameters following surgery, and 2) no significant differences would be found from pre-operative assessment to latest post-operative follow-up.

9.2.3.3 Post hoc comparison with healthy control data

If a significant change was found in a variable from pre- to post-operative assessment, post hoc comparison of the changed score with HC data was carried out. Two of the 13 participants in the experimental study did not have a matched HC for reasons outlined in Chapter 8, Section 8.4.1. An independent t-test was therefore required to compare the groups, as they no longer represented matched pairs. To assess the impact of change of any variable post-operatively, its mean value from 16 HCs and 13 CSM participants post-operatively were compared using an independent t-test, once the assumption of normal distribution was satisfied. The null hypothesis, that there would be no difference between the post-operative CSM cohort and HCs, was in this case considered a positive outcome.

9.2.3.4 Principal components analysis of gait data

PCA was conducted on the pooled 3DGA and EMG data of the CSM participants before and after surgery and the cohort of 16 HCs who participated in the cross-sectional study.
HCs were included to provide a normal reference. It was expected that the PCs extracted from this multivariate analysis would reflect those found in the cross-sectional study. Data were plotted and interpreted visually. The purpose of PCA was to assess the interaction between individual gait parameters and their contributions to the overall aspects of gait represented by the PCs. Differences in PCs themselves from pre- to post-surgery were not tested for statistical significance, because although the PCs gave an overall numerical impression of performance in an aspect of gait, it was not known whether changes in the contributing parameters represented improvement, deterioration, or compensation. Furthermore, the PC scores are dimensionless and have no clear interpretation in the clinical setting, and therefore tests of statistically significant change could not be interpreted for their clinical relevance.

9.2.3.5 Predictors of outcome following surgery for CSM

Three variables, age, symptom duration, and severity of symptoms at baseline, were considered possible predictors of outcome following surgery. Previous studies reported that older age and longer symptom duration were associated with poorer outcomes after surgery for CSM (Morio et al., 2001, Suri et al., 2003). The severity of symptoms at baseline did not show significant correlation with post-operative recovery in previous studies (Morio et al., 2001), however many studies analysed the mJOA score as a parametric test using percentage recovery rates as the primary outcome measure (Hirabayashi et al., 1981), rather than using gait data. The effect of these possible predictor variables on gait data was not known prior to the current study. Severity of symptoms at baseline was measured using the pre-operative mJOA score and pre-operative gait speed.

These three predictor variables were examined for correlation with post-operative change in gait speed as the response variable. Pearson’s correlation coefficient was used to examine correlation between age and symptom duration and the independent variable of change in gait speed, while Spearman’s correlation coefficient was used for the non-parametric mJOA score. If a significant correlation was found to exist, the variables, with the exception of the non-parametric variable mJOA, were entered into a linear regression model to determine whether a dependency existed between the two variables. The dependency was quantified using hypothesis tests of the significance of the regression coefficient $R^2$, and by calculating its confidence interval.
9.3 Results

9.3.1 Participants

9.3.1.1 Recruitment

The participants identified in the cross-sectional study were also eligible to participate in the experimental study, as the same inclusion and exclusion criteria applied. As previously described in Chapter 8, Section 8.4.1, there were 44 people who met the inclusion criteria over the 24-month recruitment period. Fifteen were excluded for reasons previously described in Chapter 8, Section 8.4.1, and a further ten declined to participate. The remaining 19 participated in the study. One participant was diagnosed with a co-existing neurological disorder after the first assessment, and his data were therefore excluded as the neurological disorder may have confounded the gait data. Five participants did not undergo surgery within the time frame of the thesis, which required that surgery take place before March 2011 to ensure a six-month follow-up prior to September 2011. Their pre-operative baseline data were included in the cross-sectional study. The experimental study therefore includes the data of the 13 participants who underwent surgery between December 2008 and March 2011. Eleven had also participated in the cross-sectional study and eight had participated in the reliability study. The flow chart in Figure 9.1 illustrates the recruitment process and participation of the CSM cohort in the three studies.
9.3.1.2 Time frames and protocol deviations

One participant of the cohort of 13 was unable to attend for six-month follow-up assessment, but attended the 12-month assessment as scheduled. Three participants
attended follow-up at six months but were unavailable to attend at 12 months. Last post-operative follow-up therefore took place at six months for three participants and at 12 months for 10 participants.

The protocol aimed to schedule the pre-operative assessment as close as possible to the date of surgery, however a number of difficulties arose with this. Many participants received less than 24 hours’ notice of their admission. Several planned admissions were cancelled due to lack of availability of beds, theatre time, or the emergency admission of other patients. It was therefore not possible to know the date of surgery in advance with certainty. The mean time from pre-operative assessment to surgery was 2.2 months (range, 0–4.9 months).

9.3.1.3 Characteristics of participants

The characteristics of the 13 participants are detailed in Table 9.1. The mean age was 56.6 years (range, 34–77 years). Participants reported a history of myelopathic symptoms for a mean of 59 months (range, 9–420 months). The median Nurick score was 3 (range, 1–4), median mJOA score, 10 (range, 8–13), and median MRMI score, 39 (range, 34–40). Two participants had a history of lower limb osteoarthritis, and three had a history of low back pain, however these problems were not acutely symptomatic and therefore did not preclude participation in the study. Two participants had non-insulin dependent diabetes mellitus with no history of associated lower limb neuropathy. One participant had a history of myocardial infarction, treated at the time with angiographic stenting, and had no persistent cardiac symptoms or signs since that event.

9.3.2 Surgical intervention

9.3.2.1 Description of surgical intervention

The most commonly involved cervical levels were C3/4, C4/5 and C5/6. Eleven participants underwent surgery at one or more of these levels (85%). One participant had additional involvement of C6/7, and one participant had compression of the spinal cord at C0/1 and C1/2. Seven participants underwent surgery via an anterior approach, involving an anterior cervical disectomy and fusion (ACDF) in six cases and an anterior cervical corpectomy and fusion (ACCF) in one case. The remaining six participants had a posterior approach, three of which involved laminectomy and lateral mass plating (LLMP), one, a decompression and lateral mass screws, and two, an occipital-cervical fusion.

9.3.2.2 Post-operative complications and adverse events

Three participants of the cohort of 13 (23%) suffered adverse events relating to their CSM. One participant sustained a fractured neck of femur following a fall. This was
reduced and fixed with a hemiarthroplasty. Surgery to the cervical spine followed three weeks later.

Two participants (15%) suffered post-operative complications, one involving wound infection at the surgical site and subsequent sepsis, and the other involving instrumentation failure. In both cases, the instrumentation was removed and the cervical spine stabilised in a halo ring and brace. The second participant then underwent a repeat fusion following the removal of the halo brace. Follow-up for this participant was scheduled from the date of this repeat surgery, rather than from the date of the original surgery. The first participant achieved adequate decompression from the halo brace and did not require further surgery, and therefore no adjustments to follow-up dates were required in this case.

### 9.3.2.3 Physiotherapy intervention following surgery

One participant (8%) had no physiotherapy intervention following surgery. Three participants (23%) were assessed by a physiotherapist during their in-patient stay in the days immediately following surgery and given general post-operative advice, but received no further out-patient follow-up. The remaining nine participants (69%) had multiple sessions of outpatient or domiciliary physiotherapy following surgery.
# Table 9.1: Baseline characteristics of participants and description of surgical intervention

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Duration of symptoms (months)</th>
<th>PMH</th>
<th>Nurick</th>
<th>mJOA</th>
<th>MRMI</th>
<th>Surgical Approach</th>
<th>Level</th>
<th>Procedure</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>02</td>
<td>67</td>
<td>12</td>
<td>OA hip &amp; knee</td>
<td>3</td>
<td>8</td>
<td>34</td>
<td>Anterior</td>
<td>C4/5, C5/6</td>
<td>C5 ACCF</td>
<td>Fracture NOF</td>
</tr>
<tr>
<td>03</td>
<td>57</td>
<td>12</td>
<td>Lumbar discectomy, OA knee</td>
<td>3</td>
<td>11</td>
<td>38</td>
<td>Anterior</td>
<td>C4/5, C5/6</td>
<td>ACDF</td>
<td></td>
</tr>
<tr>
<td>05</td>
<td>34</td>
<td>24</td>
<td>Nil</td>
<td>2</td>
<td>10</td>
<td>40</td>
<td>Anterior</td>
<td>C4/5, C5/6</td>
<td>ACDF</td>
<td></td>
</tr>
<tr>
<td>07</td>
<td>45</td>
<td>60</td>
<td>Nil</td>
<td>3</td>
<td>12</td>
<td>39</td>
<td>Posterior</td>
<td>C0/1, C1/2</td>
<td>OCF</td>
<td>Instrumentation failure ***</td>
</tr>
<tr>
<td>08</td>
<td>62</td>
<td>36</td>
<td>LBP</td>
<td>3</td>
<td>9</td>
<td>39</td>
<td>Anterior</td>
<td>C4/5, C5/6</td>
<td>ACDF</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>50</td>
<td>48</td>
<td>Nil</td>
<td>2</td>
<td>10</td>
<td>40</td>
<td>Anterior</td>
<td>C3/4, C4/5, C5/6</td>
<td>ACDF</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>73</td>
<td>420</td>
<td>Angina</td>
<td>3</td>
<td>12</td>
<td>38</td>
<td>Posterior</td>
<td>C3/4, C4/5, C5/6</td>
<td>LLMP</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>51</td>
<td>60</td>
<td>MI &amp; angioplasty, NIDDM</td>
<td>4</td>
<td>10</td>
<td>35</td>
<td>Posterior</td>
<td>C3/4, C4/5, C5/6, C6/7</td>
<td>LLMP</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>72</td>
<td>10</td>
<td>Nil</td>
<td>2</td>
<td>10</td>
<td>40</td>
<td>Posterior</td>
<td>C3/4</td>
<td>LLMP</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>47</td>
<td>48</td>
<td>LBP</td>
<td>1</td>
<td>13</td>
<td>40</td>
<td>Anterior</td>
<td>C4/5, C5/6</td>
<td>ACDF</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>47</td>
<td>12</td>
<td>GORD</td>
<td>3</td>
<td>11</td>
<td>39</td>
<td>Posterior</td>
<td>C3/4, C4/5, C5/6</td>
<td>Lateral mass screws, decompression</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>54</td>
<td>18</td>
<td>NIDDM</td>
<td>3</td>
<td>10</td>
<td>39</td>
<td>Anterior</td>
<td>C5/6</td>
<td>ACDF</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>77</td>
<td>9</td>
<td>Nil</td>
<td>3</td>
<td>12</td>
<td>39</td>
<td>Posterior</td>
<td>C1/2</td>
<td>OCF</td>
<td></td>
</tr>
</tbody>
</table>

PMH = past medical history, mJOA = Japanese Orthopaedic Association score, MRMI = Modified Rivermead Mobility Index, OA = osteoarthritis, LBP = low back pain, MI = myocardial infarction, NIDDM = non insulin dependent diabetes mellitus, GORD = gastro-oesophageal reflux disorder, ACCF = anterior cervical corpectomy & fusion, ACDF = anterior cervical discectomy & fusion, OCF = occipital-cervical fusion, LLMP = laminectomy & lateral mass plating, NOF = neck of femur

* removal of instrumentation and fusion with halo brace, ** re-do of surgery and replacement of instrumentation following removal of halo brace
9.3.3 Subjective reports of change following surgery

Standard verbal questions were used to assess each participant’s subjective impression of their gait pattern and general symptoms following surgery. At last post-operative follow-up, eight participants (62%) stated that their gait had improved, while three (23%) reported deterioration. Two participants (15%) reported no change. When asked about symptoms in general, excluding gait, nine (69%) reported that they had generally improved, while one (8%) noted an overall deterioration. Two participants (15%) reported no change.

Six participants (46%) reported a history of at least one fall in the six months prior to the pre-operative assessment. All pre-operative falls were reported to be as a result of unsteady gait and mobility impairment, rather than adverse environmental circumstances. Following surgery, two participants (15%) had sustained a fall by their last follow-up, however in both cases, the participants felt that the falls were unrelated to their CSM gait impairment, as one (a previous faller) had slipped on ice and the other (no history of pre-operative falls) had tripped on an unexpected obstacle. Results are depicted in Figure 9.2.
Results of the secondary outcome measures are shown in Table 9.2.

9.3.4 Secondary outcome measures

Results of the secondary outcome measures are shown in Table 9.2.
Table 9.2: Median (range) scores of secondary outcome measures

<table>
<thead>
<tr>
<th>Scale</th>
<th>Pre-op</th>
<th>6 months post-op</th>
<th>12 months post-op</th>
<th>Last follow-up</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>mJOA</td>
<td>10 (8–13)</td>
<td>13.5 (8–17)</td>
<td>14 (10–17)</td>
<td>14 (10–17)</td>
<td>0.005</td>
</tr>
<tr>
<td>Nurick</td>
<td>3 (1–4)</td>
<td>2 (0–4)</td>
<td>1.5 (0–4)</td>
<td>2 (0–4)</td>
<td>0.009</td>
</tr>
<tr>
<td>MRMI</td>
<td>39 (34–40)</td>
<td>40 (35–40)</td>
<td>40 (36–40)</td>
<td>40 (35–40)</td>
<td>0.03</td>
</tr>
</tbody>
</table>

SF36

<table>
<thead>
<tr>
<th>Scale</th>
<th>Pre-op</th>
<th>6 months post-op</th>
<th>12 months post-op</th>
<th>Last follow-up</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Physical functioning</strong></td>
<td>40 (10–80)</td>
<td>60 (15–90)</td>
<td>65 (25–90)</td>
<td>60 (15–90)</td>
<td>0.009</td>
</tr>
<tr>
<td><strong>Physical role functioning</strong></td>
<td>50 (0–75)</td>
<td>50 (0–93.75)</td>
<td>53.13 (13–94)</td>
<td>56.25 (12.5–93.75)</td>
<td>0.06</td>
</tr>
<tr>
<td><strong>Bodily pain</strong></td>
<td>32.5 (0–100)</td>
<td>67.5 (22.5–100)</td>
<td>46.25 (10–100)</td>
<td>65 (10–100)</td>
<td>0.02</td>
</tr>
<tr>
<td><strong>General health</strong></td>
<td>50 (20–95)</td>
<td>55 (15–85)</td>
<td>65 (20–80)</td>
<td>65 (20–85)</td>
<td>0.06</td>
</tr>
<tr>
<td><strong>Vitality</strong></td>
<td>50 (19–94)</td>
<td>56.25 (6.25–81.25)</td>
<td>50 (19–81)</td>
<td>56.25 (6.25–81.25)</td>
<td>0.57</td>
</tr>
<tr>
<td><strong>Social functioning</strong></td>
<td>50 (0–100)</td>
<td>62.5 (0–100)</td>
<td>43.75 (38–88)</td>
<td>50 (0–100)</td>
<td>0.61</td>
</tr>
<tr>
<td><strong>Emotional role functioning</strong></td>
<td>66.7 (0–100)</td>
<td>75 (0–100)</td>
<td>79.2 (25–100)</td>
<td>92 (25–100)</td>
<td>0.07</td>
</tr>
<tr>
<td><strong>Mental health</strong></td>
<td>65 (50–90)</td>
<td>75 (30–100)</td>
<td>67.5 (35–90)</td>
<td>75 (35–90)</td>
<td>0.16</td>
</tr>
<tr>
<td><strong>Health transition</strong></td>
<td>25 (0–50)</td>
<td>75 (25–100)</td>
<td>50 (25–100)</td>
<td>75 (25–100)</td>
<td>0.004</td>
</tr>
</tbody>
</table>

mJOA = Japanese Orthopaedic Association score, MRMI = Modified Rivermead Mobility Index, SF36 = RAND Medical Outcomes Study 36-Item Short Form Health Survey

9.3.4.1 Severity of myelopathy

Figure 9.3 shows mJOA and Nurick scores before, six months and twelve months following surgery. MJOA scores improved from a median of 10 before surgery to a median of 13.5 at six months and 14 at 12 months following surgery. Of the ten participants who attended 12-month follow-up, two were unchanged, one had deteriorated, and the remaining seven had improved. Six-month follow-up of the other three participants found one unchanged and two improved. The median Nurick score was 3 before surgery, 2 at six months and 1.5 at 12 months. Six participants had no change in Nurick score and seven showed an improvement at their latest follow-up.
When last follow-up scores were pooled as post-operative scores, the Wilcoxon signed rank test showed a statistically significant improvement in mJOA scores ($p = 0.005$) and in Nurick scores ($p = 0.009$) following surgery.

### 9.3.4.2 Functional mobility

Functional mobility was assessed using the MRMI. A significant ceiling effect was noted with this outcome measure. At pre-operative assessment, four of 13 participants scored
the maximum score of 40, while at post-operative assessment, eight of 13 participants reached the maximum score. The median MRMI score was 39 (range, 34–40) pre-operatively and 40 (range, 35–40) post-operatively. The Wilcoxon signed rank test found this change to be statistically significant ($p = 0.03$), but it did not exceed the minimal detectable change of the MRMI of four points (Lennon and Johnson, 2000).

### 9.3.4.3 Health-related quality of life

HRQOL was assessed using the SF36. The nine domain scales of physical functioning, physical role functioning, bodily pain, general health, vitality, social functioning, emotional role functioning, mental health and health transition were compared separately from pre- to post-operative assessment using a Wilcoxon signed rank test. There were statistically significant improvements in physical functioning, bodily pain and health transition. No significant changes occurred in the other domains, although physical role functioning, emotional role functioning and general health showed trends of improvement.

### 9.3.4.4 Resting muscle tone

Tone in the lower limb muscles of RF, BF, TA and MG was assessed using the Modified Ashworth Scale (MAS). The numbers of participants scoring at each point on the MAS before and after surgery are depicted in Figure 9.4. Post-operative MAS scores of RF were lower, depicting lower resting tone, in three participants, while one participant’s score increased. Two participants had a lower score of BF after surgery, while one had a higher score. TA scores increased post-operatively in one participant. Finally, in MG, post-operative MAS scores were lower in five participants and higher in two participants. A Wilcoxon signed rank test found no difference in MAS scores of RF ($p = 0.34$), BF ($p = 0.6$), TA ($p = 0.3$) or MG ($p = 0.27$), from pre- to post-operative assessment.
Columns show the number of participants who scored at each point on the Modified Ashworth Scale before and after surgery.
See Chapter 5, Table 5.5 for an explanation of the tone depicted by each score.

9.3.5 Gait analysis

9.3.5.1 Temporal-spatial parameters

Individual participant trends in gait speed, cadence, double support duration, single support duration, stride length and step width are shown in Figure 9.5.
Gait speed increased from a mean of 1.05 m/s pre-operatively to 1.08 m/s at last post-operative assessment. This was not statistically significant ($p = 0.62$). There were no changes in stride length (mean difference, 0.02 m, $p = 0.57$), cadence (mean difference, 1.1 steps per minute, $p = 0.83$) or single support as a percentage of GC duration (mean...
difference, 0.2% GC duration, \( p = 0.82 \)). No differences were found in the individual timing of the events of opposite foot off, opposite foot contact or foot off, or in step width. The results are summarised in Table 9.3.

**Table 9.3: Changes in temporal-spatial parameters following surgery**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre Op</th>
<th>Post Op</th>
<th>Difference</th>
<th>95% Confidence Intervals</th>
<th>( p ) value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td></td>
</tr>
<tr>
<td>Gait speed (m/s)</td>
<td>1.05</td>
<td>0.32</td>
<td>1.08</td>
<td>0.37</td>
<td>-0.03</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.20</td>
</tr>
<tr>
<td>Cadence (steps / min)</td>
<td>110.30</td>
<td>12.44</td>
<td>111.37</td>
<td>14.87</td>
<td>-1.06</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>17.96</td>
</tr>
<tr>
<td>Stride length (m)</td>
<td>1.12</td>
<td>0.26</td>
<td>1.14</td>
<td>0.31</td>
<td>-0.02</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.11</td>
</tr>
<tr>
<td>Double support duration (% GC)</td>
<td>27.99</td>
<td>6.90</td>
<td>28.72</td>
<td>8.93</td>
<td>-0.72</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4.88</td>
</tr>
<tr>
<td>Single support duration (% GC)</td>
<td>35.89</td>
<td>2.82</td>
<td>35.68</td>
<td>4.46</td>
<td>0.22</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3.27</td>
</tr>
<tr>
<td>Opposite foot off (% GC)</td>
<td>13.38</td>
<td>2.78</td>
<td>13.79</td>
<td>3.88</td>
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<td></td>
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<td>2.83</td>
</tr>
<tr>
<td>Opposite foot contact (% GC)</td>
<td>49.27</td>
<td>1.31</td>
<td>49.48</td>
<td>1.38</td>
<td>-0.21</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.25</td>
</tr>
<tr>
<td>Foot off (% GC)</td>
<td>63.87</td>
<td>4.57</td>
<td>64.39</td>
<td>4.66</td>
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<tr>
<td></td>
<td></td>
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<td></td>
<td></td>
<td>2.20</td>
</tr>
<tr>
<td>Step width (m)</td>
<td>0.16</td>
<td>0.04</td>
<td>0.17</td>
<td>0.05</td>
<td>-0.01</td>
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<td></td>
<td></td>
<td>0.02</td>
</tr>
</tbody>
</table>

SD = standard deviation, m = metres, s = seconds, min = minute, GC = gait cycle
* denotes non-normally distributed variable tested with Wilcoxon signed rank test

9.3.5.2 Kinematic parameters

Figure 9.6 shows kinematics of the pelvis, hip, knee and ankle over the GC at pre-operative, six-month and 12-month assessments.
The graphs indicate a small decrease in pelvic tilt, an increase in peak hip extension, and an increase in peak knee flexion in swing. Results of the analysis of pre- to post-operative assessments are provided in Table 9.4. Paired t tests found no significant differences in the kinematic key points tested.
Table 9.4: Changes in kinematic key points following surgery

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre Op</th>
<th>Post Op</th>
<th>Difference</th>
<th>95% Confidence Intervals</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td></td>
</tr>
<tr>
<td>Pelvic obliquity range</td>
<td>6.17</td>
<td>3.49</td>
<td>6.84</td>
<td>3.04</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>-0.67</td>
<td>2.11</td>
<td>-1.95</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.60</td>
</tr>
<tr>
<td>Average pelvic tilt</td>
<td>8.76</td>
<td>5.43</td>
<td>8.10</td>
<td>6.48</td>
<td>-2.23</td>
</tr>
<tr>
<td></td>
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<td>0.67</td>
<td>4.79</td>
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<td></td>
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<td></td>
<td></td>
<td>3.56</td>
</tr>
<tr>
<td>Peak hip extension</td>
<td>-12.24</td>
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<td>-13.10</td>
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</tr>
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<td></td>
<td></td>
<td></td>
<td>0.85</td>
<td>5.68</td>
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<td>4.29</td>
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<td>Hip total sagittal plane excursion</td>
<td>42.46</td>
<td>7.62</td>
<td>42.52</td>
<td>8.03</td>
<td>-3.38</td>
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<td>-0.07</td>
<td>5.48</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3.24</td>
</tr>
<tr>
<td>Hip abduction adduction range</td>
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<td>3.47</td>
<td>11.07</td>
<td>4.07</td>
<td>-0.88</td>
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<td></td>
<td></td>
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<td>0.93</td>
<td>3.00</td>
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<td></td>
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<td>2.74</td>
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<tr>
<td>Peak knee flexion in stance</td>
<td>14.82</td>
<td>5.83</td>
<td>16.20</td>
<td>6.27</td>
<td>-4.17</td>
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<td>-1.39</td>
<td>1.28</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.40</td>
</tr>
<tr>
<td>Peak knee flexion in swing</td>
<td>47.80</td>
<td>1.73</td>
<td>50.07</td>
<td>2.21</td>
<td>-5.32</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>-2.26</td>
<td>1.40</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.79</td>
</tr>
<tr>
<td>Knee total sagittal plane excursion</td>
<td>49.59</td>
<td>8.67</td>
<td>50.37</td>
<td>8.94</td>
<td>-2.89</td>
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<td>-0.79</td>
<td>3.49</td>
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<td>1.32</td>
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<td>Peak ankle dorsiflexion in stance</td>
<td>15.08</td>
<td>2.73</td>
<td>15.79</td>
<td>2.53</td>
<td>-0.71</td>
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<td>2.54</td>
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</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>16.73</td>
</tr>
<tr>
<td>Peak ankle dorsiflexion in swing</td>
<td>8.31</td>
<td>6.14</td>
<td>10.34</td>
<td>4.41</td>
<td>-2.03</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>5.16</td>
<td>3.54</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-5.15</td>
</tr>
<tr>
<td>Peak ankle plantarflexion</td>
<td>-10.50</td>
<td>7.36</td>
<td>-9.86</td>
<td>5.25</td>
<td>-3.28</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>-0.64</td>
<td>4.37</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2.00</td>
</tr>
</tbody>
</table>

Means, standard deviations (SD) and confidence intervals are reported in degrees

9.3.5.3 Kinetic parameters

Figure 9.7 shows GRFs and joint moments at the hip, knee and ankle at the three time points. Figure 9.8 shows the joint powers. The graphs indicated some increases in hip, knee and ankle powers, particularly in the terminal stance and pre swing phases of gait, from pre- to post-operative assessment. Increases were noted in peak knee extensor moment during loading response and in its corresponding power, K1.
Figure 9.7: Ground reaction forces and joint moments at pre-operative and six- and 12-month post-operative assessments

N/kg = Newtons per kilogram, Nm/kg = Newton metres per kilogram, Lat = lateral, Med = medial, Ext = extensor, Flex = Flexor

Dashed grey lines show one standard deviation of HC data

Arrows indicate statistically significant differences between CSM participants at pre-operative and last post-operative assessment
Paired t tests comparing pre-operative with last post-operative assessments found a significant increase in ankle A2 power at pre-swing from 2.63 W/kg before surgery to 3.18 W/kg after surgery ($p = 0.03$). There was also a statistically significant increase in the magnitude of the knee K4 power at terminal swing from 0.66 W/kg at pre-operative assessment to 1.08 W/kg post-operatively ($p = 0.01$). Knee power in initial swing, K3, increased from 0.69 W/kg pre-surgery to 0.93 W/kg after surgery, but this did not reach statistical significance at the two-tailed level ($p = 0.099$). Similarly, hip H3 power at pre-swing increased from 0.89 W/kg to 1.24 W/kg, again without reaching statistical significance ($p = 0.098$).

Peak hip extensor moments increased from 0.72 Nm/kg to 0.81 Nm/kg, but this was not significant ($p = 0.15$). There were no differences in peak hip abductor ($p = 0.82$) or peak knee extensor moments ($p = 0.32$). Peak ankle plantarflexor moment did not satisfy the assumption of a normal distribution, and was tested with a Wilcoxon signed rank test. This showed towards higher peak plantarflexor moments in the post-operative assessment (1.34 Nm/kg pre-operatively, 1.44 Nm/kg post-operatively, $p = 0.05$), and the difference of 0.1 Nm/kg exceeded the SEM of this variable of 53 Nmm/kg (0.053 Nm/kg).
The acceleration component of AP GRF increased from 1.48 N/kg to 1.57 N/kg at post-operative assessment, but this was not significant (\(p = 0.28\)). Results are shown in Table 9.5.

**Table 9.5: Kinetic key points before and after surgery**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre Op Mean</th>
<th>Pre Op SD</th>
<th>Post Op Mean</th>
<th>Post Op SD</th>
<th>Difference Mean</th>
<th>Difference SD</th>
<th>95% Confidence Intervals</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vertical GRF, peak 1</td>
<td>10.50</td>
<td>0.82</td>
<td>10.83</td>
<td>0.78</td>
<td>-0.33</td>
<td>0.87</td>
<td>*</td>
<td>* 0.07</td>
</tr>
<tr>
<td>Anteroposterior GRF, acceleration</td>
<td>-1.48</td>
<td>0.62</td>
<td>-1.57</td>
<td>0.67</td>
<td>0.09</td>
<td>0.28</td>
<td>-0.08</td>
<td>0.26</td>
</tr>
<tr>
<td>Peak hip extensor moment</td>
<td>0.72</td>
<td>0.24</td>
<td>0.81</td>
<td>0.24</td>
<td>-0.09</td>
<td>0.21</td>
<td>-0.21</td>
<td>0.04</td>
</tr>
<tr>
<td>Peak hip abductor moment</td>
<td>0.85</td>
<td>0.17</td>
<td>0.86</td>
<td>0.28</td>
<td>-0.01</td>
<td>0.22</td>
<td>-0.15</td>
<td>0.12</td>
</tr>
<tr>
<td>Peak knee extensor moment</td>
<td>0.27</td>
<td>0.19</td>
<td>0.32</td>
<td>0.22</td>
<td>-0.05</td>
<td>0.17</td>
<td>-0.16</td>
<td>0.06</td>
</tr>
<tr>
<td>Peak ankle plantarflexor moment</td>
<td>1.34</td>
<td>0.28</td>
<td>1.44</td>
<td>0.36</td>
<td>-0.10</td>
<td>0.23</td>
<td>*</td>
<td>* 0.05</td>
</tr>
<tr>
<td>Hip power generation, H1</td>
<td>0.48</td>
<td>0.26</td>
<td>0.42</td>
<td>0.34</td>
<td>0.06</td>
<td>0.20</td>
<td>-0.06</td>
<td>0.18</td>
</tr>
<tr>
<td>Hip power absorption, H2</td>
<td>-0.56</td>
<td>0.32</td>
<td>-0.63</td>
<td>0.32</td>
<td>0.07</td>
<td>0.32</td>
<td>-0.12</td>
<td>0.27</td>
</tr>
<tr>
<td>Hip power generation, H3</td>
<td>0.89</td>
<td>0.48</td>
<td>1.24</td>
<td>0.95</td>
<td>-0.35</td>
<td>0.70</td>
<td>-0.77</td>
<td>0.08</td>
</tr>
<tr>
<td>Knee power absorption, K1</td>
<td>-0.69</td>
<td>0.95</td>
<td>-0.93</td>
<td>1.11</td>
<td>0.24</td>
<td>0.57</td>
<td>-0.10</td>
<td>0.59</td>
</tr>
<tr>
<td>Knee power absorption, K3</td>
<td>-0.61</td>
<td>0.43</td>
<td>-0.91</td>
<td>0.77</td>
<td>0.31</td>
<td>0.62</td>
<td>-0.07</td>
<td>0.68</td>
</tr>
<tr>
<td>Knee power absorption, K4</td>
<td>-0.66</td>
<td>0.28</td>
<td>-1.08</td>
<td>0.55</td>
<td>0.42</td>
<td>0.52</td>
<td>0.10</td>
<td>0.73</td>
</tr>
<tr>
<td>Ankle power generation, A2</td>
<td>2.63</td>
<td>1.58</td>
<td>3.18</td>
<td>1.61</td>
<td>-0.55</td>
<td>0.80</td>
<td>-1.04</td>
<td>0.07</td>
</tr>
</tbody>
</table>

The symbol * denotes a non-normally distributed variable with p value calculated using Wilcoxon signed rank test.

Ground reaction forces (GRF) are reported in Newtons per kilogram, moments in Newton metres per kilogram, and powers in Watts per kilogram.
9.3.6 EMG analysis

9.3.6.1 Duration of muscle activation

Figure 9.9 shows the total duration of muscle activation during the GC at pre-operative, six-month and 12-month assessments. Table 9.6 shows the results of statistical analysis from pre-operative assessment to last follow-up. The duration of activation of TA increased from 37% GC duration pre-operatively to 41.7% post-operatively ($p = 0.02$). There was a non-significant decrease in RF activation duration from 34% to 30% GC duration at post-operative assessment, however this was associated with a large dispersion (SD, 18% GC duration) and did not reach statistical significance ($p = 0.46$). No other changes in the activation duration of individual muscles were noted. There was a 0.9% GC duration decrease in BF–RF co-activation following surgery, and a 1.9% increase in TA–MG co-activation, however these changes were not significant. Figure 9.10 shows representative pre-operative and post-operative EMG signals from an individual participant, with the timing parameters highlighted.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Pre Op Mean</th>
<th>Pre Op SD</th>
<th>Post Op Mean</th>
<th>Post Op SD</th>
<th>Difference Mean</th>
<th>Difference SD</th>
<th>95% confidence intervals</th>
<th>$p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rectus femoris</td>
<td>33.97</td>
<td>19.11</td>
<td>29.99</td>
<td>16.14</td>
<td>3.97</td>
<td>18.63</td>
<td>-7.28</td>
<td>15.23</td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>32.15</td>
<td>11.97</td>
<td>31.81</td>
<td>10.13</td>
<td>0.34</td>
<td>13.77</td>
<td>-7.98</td>
<td>8.66</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>36.99</td>
<td>11.16</td>
<td>41.67</td>
<td>10.83</td>
<td>-4.68</td>
<td>6.46</td>
<td>-8.58</td>
<td>-0.78</td>
</tr>
<tr>
<td>Medial gastrocnemius</td>
<td>26.90</td>
<td>10.83</td>
<td>28.30</td>
<td>6.09</td>
<td>-1.40</td>
<td>7.42</td>
<td>-5.89</td>
<td>3.08</td>
</tr>
<tr>
<td>RF / BF co-activation</td>
<td>13.64</td>
<td>8.69</td>
<td>12.71</td>
<td>10.90</td>
<td>0.93</td>
<td>8.99</td>
<td>-4.50</td>
<td>6.36</td>
</tr>
<tr>
<td>TA / MG co-activation</td>
<td>3.97</td>
<td>3.79</td>
<td>5.83</td>
<td>5.65</td>
<td>-1.85</td>
<td>5.00</td>
<td>*</td>
<td>*</td>
</tr>
</tbody>
</table>

* denotes variable tested with non-parametric Wilcoxon signed rank test
Duration of activation is expressed as a percentage of gait cycle duration
RF = rectus femoris, BF = biceps femoris, TA = tibialis anterior, MG = medial gastrocnemius
Figure 9.9: Box and whisker plots of total duration of muscle activation and co-activation during gait
Signals were treated with a Butterworth filter to remove motion artefact.
RF = rectus femoris, BF = biceps femoris, TA = tibialis anterior, MG = medial gastrocnemius
9.3.6.2 EMG amplitude

The EMG amplitudes of key bursts of muscle activity during the GC before and after surgery are shown in Figures 9.11 and 9.12. Pre- and last post-operative comparisons are detailed in Table 9.7. There were no significant changes after surgery. In the cross-sectional study, RF and BF baseline activity was shown to be higher in CSM than in HCs. The amplitude of RF baseline activity reduced from 18.2% to 17.2% GC duration, and BF reduced from 16.8% to 15.2% GC duration, however these were not statistically significant ($p = 0.5$ and $p = 0.24$, respectively).

Table 9.7: Amplitude of muscle activity bursts during the gait cycle before and after surgery

<table>
<thead>
<tr>
<th>Muscle Burst</th>
<th>Pre Op</th>
<th>Post Op</th>
<th>Difference</th>
<th>Confidence Interval</th>
<th>$p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td></td>
</tr>
<tr>
<td>RF loading response</td>
<td>56.40</td>
<td>8.87</td>
<td>56.31</td>
<td>11.87</td>
<td>-0.09</td>
</tr>
<tr>
<td>RF pre swing</td>
<td>52.10</td>
<td>11.88</td>
<td>44.79</td>
<td>13.67</td>
<td>7.31</td>
</tr>
<tr>
<td>RF swing</td>
<td>17.46</td>
<td>12.59</td>
<td>21.31</td>
<td>9.06</td>
<td>-3.85</td>
</tr>
<tr>
<td>RF baseline</td>
<td>18.18</td>
<td>6.64</td>
<td>17.03</td>
<td>4.83</td>
<td>1.15</td>
</tr>
<tr>
<td>BF stance</td>
<td>43.76</td>
<td>9.22</td>
<td>45.21</td>
<td>8.26</td>
<td>-1.46</td>
</tr>
<tr>
<td>BF swing</td>
<td>46.13</td>
<td>10.12</td>
<td>44.39</td>
<td>16.87</td>
<td>1.74</td>
</tr>
<tr>
<td>BF baseline</td>
<td>16.81</td>
<td>3.95</td>
<td>15.17</td>
<td>3.93</td>
<td>1.64</td>
</tr>
<tr>
<td>TA stance</td>
<td>55.46</td>
<td>8.89</td>
<td>55.01</td>
<td>9.33</td>
<td>0.45</td>
</tr>
<tr>
<td>TA swing</td>
<td>48.73</td>
<td>8.50</td>
<td>48.99</td>
<td>4.69</td>
<td>-0.26</td>
</tr>
<tr>
<td>TA baseline</td>
<td>15.42</td>
<td>4.28</td>
<td>16.84</td>
<td>3.57</td>
<td>-1.41</td>
</tr>
<tr>
<td>MG stance</td>
<td>59.19</td>
<td>4.39</td>
<td>57.27</td>
<td>4.90</td>
<td>1.92</td>
</tr>
<tr>
<td>MG baseline</td>
<td>12.40</td>
<td>3.77</td>
<td>11.93</td>
<td>3.71</td>
<td>0.47</td>
</tr>
</tbody>
</table>

Amplitude of each muscle is expressed as a percentage of its peak root-mean-square (RMS) amplitude during gait.
RF = rectus femoris, BF = biceps femoris, TA = tibialis anterior, MG = medial gastrocnemius, SD = standard deviation.
Figure 9.11: Box and whisker plots of mean EMG amplitude of key RF and BF bursts of activity before and six and 12 months after surgery

Amplitude is expressed as a percentage of maximum root-mean-square (RMS) amplitude obtained during 10 gait trials of each session.
Amplitude is expressed as a percentage of maximum root-mean-square (RMS) amplitude obtained during 10 gait trials of each session.

Table 9.8 shows the lengthening velocity threshold (LVT) and time of onset of EMG activity during lengthening, critical time (t_C), of the four lower limb muscles during gait before and after surgery. There was a significant increase in LVT of RF from 2.74 l/s to 3.2 l/s after surgery (p = 0.02), indicating a reduction in velocity-related sensitivity to lengthening. There were no significant changes in the LVT or t_C of the remaining muscles.

9.3.6.3 Locomotor-specific measure of spasticity

Table 9.8 shows the lengthening velocity threshold (LVT) and time of onset of EMG activity during lengthening, critical time (t_C), of the four lower limb muscles during gait before and after surgery. There was a significant increase in LVT of RF from 2.74 l/s to 3.2 l/s after surgery (p = 0.02), indicating a reduction in velocity-related sensitivity to lengthening. There were no significant changes in the LVT or t_C of the remaining muscles.
### Table 9.8: Locomotor-specific measure of spasticity before and after surgery

<table>
<thead>
<tr>
<th>Measure</th>
<th>Pre Op</th>
<th>Post Op</th>
<th>Difference</th>
<th>Confidence Interval</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>RF LVT (l(_0)/s)</td>
<td>2.74</td>
<td>1.12</td>
<td>3.20</td>
<td>1.21</td>
<td>-0.46</td>
</tr>
<tr>
<td>RT t(_c) (% GC)</td>
<td>83.55</td>
<td>8.03</td>
<td>85.51</td>
<td>6.20</td>
<td>-1.96</td>
</tr>
<tr>
<td>BF LVT (l(_0)/s)</td>
<td>1.43</td>
<td>0.44</td>
<td>1.31</td>
<td>0.48</td>
<td>0.12</td>
</tr>
<tr>
<td>BF t(_c) (% GC)</td>
<td>78.79</td>
<td>5.83</td>
<td>78.68</td>
<td>6.16</td>
<td>0.11</td>
</tr>
<tr>
<td>TA LVT (l(_0)/s)</td>
<td>1.16</td>
<td>0.73</td>
<td>1.13</td>
<td>0.68</td>
<td>0.03</td>
</tr>
<tr>
<td>TA t(_c) (% GC)</td>
<td>57.33</td>
<td>7.50</td>
<td>57.19</td>
<td>5.63</td>
<td>0.14</td>
</tr>
<tr>
<td>MG LVT (l(_0)/s)</td>
<td>0.63</td>
<td>0.62</td>
<td>0.59</td>
<td>0.83</td>
<td>0.03</td>
</tr>
<tr>
<td>MG t(_c) (% GC)</td>
<td>20.11</td>
<td>12.39</td>
<td>18.78</td>
<td>10.43</td>
<td>1.33</td>
</tr>
</tbody>
</table>

\(LVT = \) lengthening velocity threshold, normalised lengths per second \(= l_0/s\), \(GC = \) gait cycle, \(RF = \) rectus femoris, \(BF = \) biceps femoris, \(TA = \) tibialis anterior, \(MG = \) medial gastrocnemius

Data on the direction of the slope of lengthening velocity versus EMG amplitude are shown in Figure 9.13. The dominant direction of the slope over ten trials from each participant was analysed. There was a slight reduction in the number of participants with dominant positive slopes of TA and MG post-operatively, and a slight increase in dominant positive slopes of RF. However, these resulted from changes in dominant slope direction for one (TA) and two (MG and RF) participants, and did not reach statistical significance on Fisher’s exact test.
Positive slopes suggest a velocity sensitive response to lengthening.

Comparison of 3DGA and EMG variables in participants with CSM before and after surgery showed statistically significant differences in five variables, peak ankle plantarflexor moment, A2 and K4 powers, duration of activation of TA, and LVT of RF. The post-operative measurements from these variables were compared to the HC cohort of the cross-sectional study. Results are shown in Table 9.9. There were statistically significant differences between the CSM participants post-operatively and the HCs in K4 and A2 power and TA duration of activation. There were no significant differences between the groups with respect to RF LVT or peak ankle plantarflexor moment. K4 power and peak ankle plantarflexor moment did not follow a normal distribution, and were tested using the non-parametric Mann Whitney U test.

### Figure 9.13: Dominant direction of the slope of muscle lengthening velocity with respect to EMG amplitude (% $\text{RMS}_{\text{MAX}}$) before and after surgery

Positive slopes suggest a velocity sensitive response to lengthening.
Table 9.9: Comparison of variables that changed following surgery in the CSM cohort with data from healthy controls at comfortable speed

<table>
<thead>
<tr>
<th>Variable</th>
<th>CSM post op</th>
<th>HC</th>
<th>Difference</th>
<th>Confidence interval</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle plantarflexor moment (Nm/kg)</td>
<td>1.44</td>
<td>1.6</td>
<td>0.16</td>
<td>*</td>
<td>*0.75</td>
</tr>
<tr>
<td>K4 power (W/kg)</td>
<td>-1.08</td>
<td>-1.56</td>
<td>0.63</td>
<td>-0.48</td>
<td>*</td>
</tr>
<tr>
<td>A2 power (W/kg)</td>
<td>3.18</td>
<td>4.85</td>
<td>1.19</td>
<td>1.67</td>
<td>0.60</td>
</tr>
<tr>
<td>TA activation duration (% GC)</td>
<td>41.67</td>
<td>30.93</td>
<td>8.84</td>
<td>-10.74</td>
<td>-18.23</td>
</tr>
<tr>
<td>RF LVT (lo/s)</td>
<td>3.20</td>
<td>3.52</td>
<td>1.56</td>
<td>0.32</td>
<td>-0.76</td>
</tr>
</tbody>
</table>

Nm/kg = Newton metres per kilogram, W/kg = Watts per kilogram, G = gait cycle, TA = tibialis anterior, RF = rectus femoris, LVT = lengthening velocity threshold, lo/s = relative lengths per second

* denotes non normally distributed variable tested with Mann Whitney U test

9.3.8 Principal components analysis of 3DGA and EMG data

PCA was conducted on the pooled 3DGA and EMG data of the 13 participants pre- and post-surgery (last follow-up) and the 16 HCs from the cross-sectional study. The PCs extracted were similar to those of the cross-sectional study, with the first three PCs accounting for 33.5%, 6.9% and 6.6% of the variability in the data set, respectively. Table 9.10 lists the 3DGA and EMG variables contributing to each PC and their weightings.

Figure 9.14 shows the clustering of scores on PC1 and PC2 before and after surgery. Figure 9.15 shows box and whisker plots of each PC. The plot of PC1 indicated that post-operative scores moved towards HC scores, although mean PC1 scores remained below zero. There was little change in PC2 and PC3 scores from pre- to post-operative assessments.
<table>
<thead>
<tr>
<th>PC</th>
<th>Score</th>
<th>Positive</th>
<th>Weight</th>
<th>Negative</th>
<th>Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>PC1</td>
<td>33.5%</td>
<td>Gait speed</td>
<td>0.194</td>
<td>Antero-posterior GRF, acceleration*</td>
<td>-0.188</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Step length</td>
<td>0.186</td>
<td>Double support, seconds</td>
<td>-0.174</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stride length</td>
<td>0.186</td>
<td>Double support duration, % GC</td>
<td>-0.171</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Antero-posterior GRF, deceleration</td>
<td>0.176</td>
<td>Opposite foot off, % GC</td>
<td>-0.164</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Single support duration, % GC</td>
<td>0.173</td>
<td>Step time</td>
<td>-0.159</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ankle power generation, A2</td>
<td>0.171</td>
<td>Foot off, % GC</td>
<td>-0.158</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Knee sagittal plane range</td>
<td>0.169</td>
<td>Stride time</td>
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</tr>
<tr>
<td></td>
<td></td>
<td>Vertical GRF</td>
<td>0.161</td>
<td>Knee absorption power, K1*</td>
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</tr>
<tr>
<td></td>
<td></td>
<td>Cadence</td>
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</tr>
<tr>
<td></td>
<td></td>
<td>Hip sagittal plane range</td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hip power generation, H3</td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Knee power generation, K2</td>
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<td></td>
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<tr>
<td>PC2</td>
<td>6.9%</td>
<td>Hip position, initial contact</td>
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<td></td>
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<tr>
<td></td>
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<td>TA critical time</td>
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<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>BF critical time</td>
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</tr>
<tr>
<td></td>
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<td>BF LVT</td>
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<td></td>
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<tr>
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<tr>
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<td>Hip sagittal plane range</td>
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<td></td>
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<td>Hip extension*</td>
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<tr>
<td>PC3</td>
<td>6.6%</td>
<td>BF critical time</td>
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<td>RF pre-swing burst amplitude</td>
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<td>TA stance burst amplitude</td>
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<td>TA duration</td>
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<td>MG critical time</td>
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<td></td>
<td></td>
</tr>
<tr>
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<td></td>
<td>Opposite foot contact, % GC</td>
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</table>

GRF = ground reaction force, GC = gait cycle, PC = principal component, RF = rectus femoris, BF = biceps femoris, TA = tibialis anterior, MG = medial gastrocnemius, LVT = lengthening velocity threshold

Kinematic and kinetic variable names refer to peak values unless range or position is stated

* denotes a variable with a negative sign by convention
Scatter plots, shown in Figure 9.16, were generated to examine the relationship between post-operative change in gait speed and four potential predictors, age, symptom duration, baseline mJOA and baseline gait speed. The relationships were examined using tests of correlation. Pearson’s correlation coefficient found no statistically significant associations between age and change in gait speed (correlation coefficient $R = 0.076$, $p = 0.8$) or baseline gait speed and change in gait speed ($R = -0.047$, $p = 0.88$). Symptom duration
was not normally distributed and its relationship with change in gait speed was tested for significance using Spearman’s rho, as was the case for mJOA, an ordinal scale. Spearman’s rho found no correlation between baseline mJOA and change in speed (rho = 0.102, p = 0.74) or between symptom duration and change in gait speed (rho = −0.03, p = 0.92). These pairs of variables were not entered into a linear regression model to assess for dependency because they did not satisfy the assumption of a linear association between predictor and response variables.

Figure 9.16: Scatterplots of post-operative change in gait speed and a) age, b) baseline gait speed, c) symptom duration, and d) Japanese Orthopaedic Association score
Symptom duration is shown with the outlier at 420 months removed to illustrate the relationship between the variables at the lower end of the x-axis
JOA = Japanese Orthopaedic Association score
9.4 Effect of complications and adverse events

As discussed in Section 9.3.1.5, three participants experienced adverse events resulting from either CSM itself or surgical treatment for it. One participant, case 02, sustained a fractured neck of femur as a result of a fall that occurred after her baseline assessment and three weeks before her planned surgery. She underwent a hemiarthroplasty and post-operative rehabilitation, and then had decompressive surgery to the cervical spine as planned. At six-month follow-up, she was mobilising independently. She reported no falls in the time since surgery, however her gait speed had decreased from 0.83 m/s to 0.61 m/s. Her mJOA score increased from 8 to 14, showing an overall improvement in myelopathic symptoms. The same participant did not attend a one-year follow-up because she had sustained an injury to her ankle that significantly hindered mobilisation at that time.

Another participant, case 07, suffered post-operative failure of her cervical instrumentation. She deteriorated medically and required intubation and mechanical ventilation in the intensive care unit. Following removal of instrumentation, her cervical spine was immobilised in a halo ring and vest for four months. She then underwent repeat occipital-cervical fusion five months after the first surgery. She was unavailable for follow-up at six months following that surgery, but returned at 12 months. Her gait speed decreased from 1.04 m/s pre-operatively to 0.61 m/s post-operatively, while mJOA score decreased from 13 to 12.

The third participant, case 14, experienced a post-operative infection at the surgical site with progression to sepsis. He underwent removal of surgical instrumentation and multiple wound debridement procedures, and was subsequently immobilised in a halo vest and jacket. This was removed after 12 weeks and he had no further operative intervention after that event. At six-month follow-up, there was a decrease in gait speed from 0.53 m/s to 0.38 m/s, and a decrease in mJOA from 10 to 8. At 12-month follow-up, gait speed increased to 0.6 m/s, and mJOA returned to the baseline score of 10. He attended a rehabilitation centre for a four-week period of in-patient rehabilitation in the time between his six-month and twelve-month follow-up assessments.

In total, three participants with adverse events experienced a mean decrease in gait speed of 0.19 m/s (SD, 0.25 m/s). The ten participants who did not experience complications showed a mean increase of 0.09 m/s (SD, 0.14 m/s). The differences were not tested for statistical significance due to the small number of participants with complications.
9.5 Discussion

9.5.1 Introduction

This was the first study to comprehensively evaluate gait in CSM before and after surgery using 3DGA and EMG technology. The aim of the study was to determine whether people with CSM experienced changes in gait following surgery. The first part of the discussion will be addressed under three clinical questions: 1) Did surgery stabilise the gait impairment? 2) Did gait improve following surgery? 3) If gait improved, was it comparable to normal healthy gait? The second part of the discussion will examine the implications of the findings for the management of CSM.

9.5.2 Overview of changes in gait following surgery

9.5.2.1 Did surgery stabilise the gait impairment?

The aim of surgical decompression in CSM is to halt the progression of neurological deterioration by decompressing the spinal cord and stabilising the affected spondylotic cervical levels (Rao et al., 2006). Although there are no long-term prospective studies on the natural history of CSM, step-wise deterioration over time has been described in the literature (Clarke and Robinson, 1956). If the course of CSM is one of progressive neurological deterioration, then a lack of improvement in gait following surgery is not necessarily a negative outcome. Rather, a lack of deterioration implies that the goal of stabilisation of the deficit has been achieved.

The results found no evidence of deterioration in gait following surgery over the three time points. Considering the cohort as a whole, there was a mean increase in gait speed of 0.03 m/s. This change was neither statistically nor clinically significant, but this small increase shows a trend contrary to what would be expected in a situation of worsening CSM. Similarly, other kinematic key points that had been different to HCs in the cross-sectional study, such as hip total sagittal plane range, peak knee flexion in swing, peak ankle plantarflexion in stance, and range of pelvic obliquity, showed no significant change following surgery, indicating that the kinematics of the gait pattern were preserved. In relation to kinetics, any statistically significant changes were due to increases rather than decreases in peak values. There were no negative changes in kinetic key points or curves over the GC. EMG data were more difficult to interpret in the context of stabilisation of the gait impairment, as some changes may have been compensatory and there was no obvious direction of improvement or deterioration in timing and amplitude parameters. LVT did not significantly decrease, however, which suggests no increase in spasticity-related effects on gait.
In summary, it can be concluded that in this cohort of 13 participants, gait showed on average no deterioration in the six- to 12-month post-operative period.

9.5.2.2 Did gait improve following surgery?

The primary goal of surgery is to prevent deterioration in neurological function, however in some cases, a secondary goal may be to facilitate improvement (Rao et al., 2006). Statistical analysis of TSPs and kinematic key points revealed no differences in post-operative scores. Visual inspection of the graphs did not reveal trends that may have been undetected due to lack of statistical power. In this respect, the time, distance and movement features that characterised the participants’ gait patterns did not improve.

However, the kinetics and neuromuscular control that contributed to these gait patterns showed some post-operative changes. Power generation at the ankle in pre swing (A2), power absorption by the knee in terminal swing (K4), and peak ankle plantarflexor moment were significantly higher post-operatively. The H3 propulsive power of the hip at pre-swing and the knee absorption powers of K1 in loading response and K3 in terminal swing all showed mean post-operative increases that were of clinical interest and exceeded SEM, although they did not reach statistical significance. On the whole, these changes indicated that the locomotor system had become more adept at absorbing and generating power at the hip, knee and ankle at appropriate times during gait.

EMG data on the neuromuscular control of force production yielded somewhat inconclusive results. The significant increase in the duration of activation of TA over the GC was unlikely to have produced the increased A2 power, as this is normally a function of the gastrocnemius–soleus complex. No changes in gastrocnemius function were noted in the EMG data, although these parameters had not differed from HCs at baseline. There was a statistically significant increase in the LVT of RF, indicating an improvement in the ability of that muscle to yield to stretch. This indicated reduced hyperexcitability of the stretch reflex and therefore reduced RF spasticity. This was not associated with a significant reduction in the duration of activation of RF. Similarly, peak knee flexion angle in swing, a parameter often impaired by spasticity of RF (Sutherland and Davids, 1993), was not different following surgery. In other words, if it was the case that RF was responding more appropriately to lengthening as the post-operative change in its LVT suggested, then this improvement was not reflected in the corresponding kinematics or in the duration of activation of RF over the GC.

In summary, there was evidence of some improvement in kinetic data in the CSM cohort following surgery, however this did not result in measurable improvements in kinematics or TSPs. Participants appeared to be in better control of power absorption and generation, but did not use this control to increase the excursion of the lower limb joints in the sagittal plane and walk faster.
9.5.2.3 Did gait return to normal following surgery?

The next research question considered whether the improved kinetic and EMG parameters had returned to normal levels. Visual interpretation of the hip, knee and ankle powers and ankle plantarflexor moment over the GC indicated that the post-operative curves were closer to those of HCs. Two parameters that were significantly improved in CSM post-operatively, A2 and K4 peak powers, continued to show significant differences from HC data, while there were no differences in peak ankle plantarflexor moment between post-operative CSM data and HCs.

Chapter 8, Section 8.4.5, reported that the pre-operative duration of activation of TA was significantly prolonged compared to HCs. Post-operatively, TA activation duration had further increased from 37% to 41.7% GC duration, compared to HC duration of 31%. This parameter was therefore more “abnormal” than it had been pre-operatively. The LVT of RF increased significantly following surgery, and its post-operative value was not significantly different to that of HCs. Interpretation of this change was hindered by the lack of significant difference between CSM participants and HCs pre-operatively, despite the large mean difference in scores at that time (HC 3.5 l/s, CSM 2.8 l/s pre-operatively, 3.2 l/s post-operatively). Analysis of the implications of these changes in EMG will be considered in Section 9.5.4.

PCA allowed further exploration of the relationships between gait variables. The first PC, representing propulsion and momentum generation, showed clear differences between HC and CSM participants pre-operatively. Post-operatively, the mean score for the CSM cohort moved closer to that of HC participants, but differences remained in the clustering of the groups, as shown in Figures 9.14 and 9.15. In contrast, the CSM participants’ post-operative score on PC2, representing aspects of stability in stance, showed a small decrease on the pre-operative scores. This change was in the opposite direction to HC data. PCA therefore indicated a change in momentum generation following surgery that brought CSM participants closer to their HC counterparts, but also indicated an opposite change in the aspects of stability in stance represented by PC2.

In summary, there was little evidence that gait in CSM participants returned to normal following surgery. There was a trend of improvement towards normal values in kinetic variables, though significant differences between CSM and HCs remained. The first PC, reflecting propulsion and generation of momentum, followed this trend. For the most part, the features of the myelopathic gait that were identified in Chapter 8 were still present following surgery.
9.5.3 Comparison of findings with other studies

The current study found no significant change in gait speed in people with CSM after decompressive surgery. This finding is in contrast to previous reports. Post-operative increases in gait speed of 0.12 m/s (Singh and Crockard, 1999), 0.15 m/s (Kuhtz-Buschbeck et al., 1999, Maezawa et al., 2001) and 0.19 m/s (Singh et al., 2009) have been reported, although one study of six participants found no significant change (Moorthy et al., 2005). Similarly, other studies found significant post-operative changes in kinematics. One reported a 5.6° increase in total sagittal plane excursion of the knee (Moorthy et al., 2005). A further study identified an 8° increase in peak hip flexion, a 4° increase in peak knee flexion in stance, and a 5° increase in peak ankle dorsiflexion in stance (Maezawa et al., 2001). None of these features were apparent from the data of the current study. The increase in speed in the cohort was just 0.03 m/s (95% CI from pre- to post-operative assessment, -0.15–0.09 m/s), and would not be considered to be of clinical importance.

There is a possibility that the 0.03 m/s increase in speed could reflect the beginning of further increases after the twelfth post-operative month. Anecdotally, it has been reported that any neurological improvement following surgery for CSM could take up to 24 months to manifest. However, Singh et al. (2009) assessed performance on a 30m timed walk test at 6, 12, 24 and 36 months post-surgery in CSM, and found that most improvement in gait speed occurred in the first six months. This was supported by a recent prospective study, which found the greatest increase in Berg Balance Scale scores in the first six post-operative months (Furlan et al., 2011). It is worth noting that while Singh et al. (2009) found a significant increase in 30m timed walk scores from pre- to post-operative follow-up, their participants may have represented a more severely affected population. Their mean pre-operative gait speed was 0.56 m/s, in contrast to the current study, in which participants walked at a mean speed of 1.05 m/s before surgery.

In relation to kinematics, non-significant increases were found in the current study in some parameters including peak knee flexion in swing, peak ankle dorsiflexion in swing, and peak hip extension. These were increases of 1–2° and did not exceed the SEM of those variables, suggesting that they were within the realm of measurement error. Even if one could be confident of exceeding measurement error, changes of this magnitude could not be considered of clinical importance. The lack of statistical significance of these findings was therefore considered to reflect the true state of the participants, and was not a type two statistical error.
9.5.4 Interpretation of gait in CSM following surgery

The overall finding was that the temporal-spatial and kinematic features of gait in CSM were unchanged following surgery, but there were some changes in how these features were produced by kinetics and EMG. Significant increases in ankle power generation at pre-swing, knee power absorption in terminal swing, and peak ankle plantarflexor moment were found. A strong predictive relationship between peak powers and gait speed has been shown, with regression coefficients of above 0.8 for A2 and K4 (Lelas et al., 2003). In the current study, the mean increases of 0.55 W/kg in A2 and 0.42 W/kg in K4 did not significantly affect gait speed. This was a surprising finding. Winter (1983) reported that the A2 power at pre swing was responsible for generating around 30% of the total energy of the GC. The K4 power at terminal swing reflects the eccentric contraction of the hamstring muscles to control forward advancement of the swinging tibia and prevent abrupt hyperextension of the knee (Kirtley, 2006). It is clear that, in the post-operative assessment, the stance leg underwent greater propulsion into swing by the ankle plantarflexors and the hamstrings then dampened this additional momentum in terminal swing. These changes were reflected in the increase in PC1 scores. However, it is not clear why the additional forward momentum created by these power peaks did not translate into faster gait speed or increased stride length. It was not the case that the greater propulsion in swing was offset by longer period in stance, as double support duration and cadence were unchanged.

EMG identified a significant increase in the duration of activation of TA post-operatively. TA duration had already been abnormally prolonged at baseline compared to HCs. This further increase was a trend away from HC data. It is possible that this reflected neurological deterioration and further impaired selective control of muscle activation, however there was no other evidence of neurological deterioration in this cohort. That TA duration was even more prolonged following surgery, despite a trend to improvement of other gait parameters, suggested that it was a compensatory feature of gait in CSM. Visual analysis of individual TA signals showed that, across a number of signals, the additional duration of activation occurred mainly in stance. It may have reflected a strategy to improve control around the ankle during weight bearing, creating a more stable ankle joint and allowing the A2 power to be transmitted more effectively in propulsion. TA did not show increased co-activation with MG following surgery. Its additional activation therefore must have occurred during times when MG was not active. This supports the theory that its purpose was to create stability in loading response and mid stance. However, without EMG signals from other muscles acting about the ankle joint for stability, including peroneus longus and tibialis posterior, the exact function of this additional TA activation can only be speculated upon. Increases in the duration of muscle activity have been noted as compensatory strategies in the non-paretic lower limb after
stroke (Lamontagne et al., 2000b), and this finding suggests that similar strategies are possible in CSM.

Changes in the effects of spasticity during the GC were assessed using the LSMS. RF did not show a significantly lower LVT compared to HCs pre-operatively, suggesting that it wasn’t impaired by spasticity, although the SD of the difference of 1.82 l/s between HC and CSM pairs indicated a large dispersion in the scores. Following surgery, there was a significant increase in RF LVT, indicating less spasticity of RF post-operatively compared to the pre-operative assessment. It could be the case that this spasticity of RF had little effect on the kinematics of the gait pattern at baseline, and therefore a higher LVT was not reflected in the motion of the hip or knee. RF spasticity is usually associated with prolonged activation time (Sutherland and Davids, 1993). This was the case compared to HCs at baseline, and post-operatively, there was a mean decrease in RF activation duration of close to 4% GC duration. This was not statistically significant and did not exceed SEM, but could reflect a slight change in neuromuscular function that did not affect kinematics.

The lack of quantitative change in TSPs and kinematics despite changes in other aspects of biomechanical and neuromuscular function, merits further discussion. The consistency of the kinematics of gait from pre- to post-operative assessment was not in keeping with the subjective finding that 62% of participants felt an improvement in their gait, the reduction in falls incidence from 46% in the pre-operative year to 15% in the post-operative year, and the improvements in MRMI and SF36 physical functioning scores. An abnormal gait pattern in a person with neurological impairment reflects the direct consequences of the primary CNS lesion and the secondary compensatory processes that determine the optimal gait pattern for a given CNS lesion (Dietz, 2002). It could be the case that this gait pattern becomes embedded into the CNS, even if the biomechanical and neuromuscular factors contributing to this pattern have the potential to change their output. Similar findings have been reported in people with hip osteoarthritis, who have reported relief of pain and improved HRQOL following total hip arthroplasty, but without changes in gait kinematics and kinetics of either the affected or contralateral lower limb (Beaulieu et al., 2010). This lack of change in gait has been attributed to the preservation of the pain-avoidance strategies that characterised gait prior to joint replacement (Beaulieu et al., 2010). In the current study, it is possible that fear of falling or perceived lack of stability may have caused the CSM participants to retain a gait pattern characterised by slower gait speed with shorter stride lengths, less time in single support duration and smaller kinematic joint excursions. Their subjective improvement in gait could reflect that the kinetics and neuromuscular activity were more efficient, even if not more effective, at producing movement. Most people with CSM experience a neurological deficit for a number of months or, in some cases years, prior to operative
intervention, and this would allow time for the abnormal gait pattern to embed itself in the CNS.

Another possibility is that the increases in powers in the current study were of insufficient magnitude to induce changes in kinematics. There may be a threshold beyond which A2 and K4 must improve before they affect the kinematic and temporal-spatial output. This hypothesis would not be supported by previous studies of the dependency between powers and gait speed, which suggested a quadratic predictive relationship (Lelas et al., 2003). However, the relationship between these variables has not been studied in gait in people with neurological disorders. Interestingly, even though these powers improved significantly in the CSM cohort following surgery, they still differed from HCs, albeit at a faster speed. This may indicate that they were a significant but insufficient improvement on the pre-operative assessment. The differences in gait in the cross-sectional study of people with CSM and HCs at matched speed, reported in Chapter 8, suggested that equal gait speeds do not result from equal kinematic and kinetic parameters when one cohort has a neurological impairment.

9.5.5 Implications for the management of CSM

Although the current study evaluated gait before and after surgical intervention, it was observational in nature and did not set out to evaluate the effect of that intervention. Without an RCT and sufficient statistical power for two groups, it cannot be known whether the changes in gait resulted directly from surgery. However, from an observational viewpoint, the study shows with relative certainty that gait did not deteriorate over the 12-month (in three cases, six month) follow-up period, in contrast to the generally accepted trend of progressive deterioration in untreated CSM. In addition, there was a mean post-operative improvement in mJOA scores, improved subjective perception of gait, and a reduced incidence of falls in the cohort, suggesting that, if a change does occur, it tends to be in the positive direction. This is the first study to provide quantitative data on the biomechanical and neuromuscular changes in gait following surgery. This new information may assist surgeons in effectively counselling people with CSM who seek greater clarity on the balance of risks and benefits in advance of a decision to undergo surgical intervention.

The issue of adverse events deserves further consideration. Three of the 13 participants experienced adverse events, representing 23% of the cohort. One of these adverse events, a fall leading to a fractured neck of femur and subsequent hemiarthroplasty, was not a direct result of surgery. Surgical complications occurred in two participants, 15% of the cohort. As might be expected, there was a general trend of poorer outcomes in those who experienced complications, but the number was too small for sub-group analysis. Furthermore, additional factors were likely to have impacted on these outcomes.
including the prolonged intensive care unit stays in two participants, and lengthy periods of time of immobility for all three participants that would have adversely affected gait, regardless of the neurological state. However, not all participants who experienced complications had an overall negative outcome. One improved his gait speed by 0.07 m/s at one-year follow-up compared to his pre-operative speed, despite initial deteriorations in his gait at six months. However, prospective surgical candidates should probably be advised that the likelihood of preservation of gait performance and possible improvement in some aspects of gait appears to be linked to an uncomplicated pre- and post-operative course.

The results of the current study hold important implications for the rehabilitation of gait in people with CSM following surgery. The improvements in kinetics and changes in EMG parameters suggested that some adaptation had taken place over the post-operative time period, although these did not translate into temporal-spatial or kinematic changes. Even if surgery does not directly alter the CNS lesion or lead to an improvement in neurological impairment, as found by Sampath et al. (2000), the capacity for improvement in gait by neuroplasticity and compensation may be preserved (Dietz and Harkema, 2004). Studies using functional MRI in CSM have provided evidence of reorganisation in the sensorimotor cortices following surgical decompression of cervical spine stenosis (Holly et al., 2007, Duggal et al., 2010). The current study’s findings of improvement in aspects of gait are in keeping with these studies’ evidence of adaptation within the CNS following surgery. In a systematic review, Kokotilo et al. (2009) described the reorganisation of brain function in people with CNS damage as one of the fundamental mechanisms involved in recovery of sensorimotor function, and commented that the brain networks involved in different aspects of motor control remain responsive, even in chronic paralysis. Therapeutic strategies aimed at restoring spinal cord function, such as body-weight supported treadmill training, could build on this cortical reorganisation, even in people with chronic SCI (Lucareli et al., 2011).

The participants in the current study received variable levels of rehabilitation input, depending on the severity of their gait deficit. All participants were otherwise independently mobile, with deficits that were probably not as severe as those of people with other neurological disorders. This, along with the expectation that surgery stabilises rather than improves gait, may cause patients and clinicians to accept a gait pattern that has changed little following surgery. An RCT of more intensive rehabilitation intervention, targeted at locomotor recovery following surgery for CSM, is necessary to determine whether people with CSM can build on the changes in kinetics and EMG to maximise their potential to improve. The changes in gait outlined in the current study, coupled with developing knowledge on the ability of the spinal cord to recover its locomotor capacity after injury, suggest that there is potential for further improvement in gait in CSM.
9.5.6 Limitations of the study

9.5.6.1 Interpretation of cause and effect

This experimental study aimed to assess gait in people with CSM before and after surgical intervention. Its goal was not to determine whether surgery was superior to conservative management, and as such, the changes in gait cannot be causally attributed to surgical intervention without comparison to a control (non-surgical) group. Instead, the study can be considered within its remit as an observational study of change following an intervention. An RCT of the effects of surgery compared with conservative management would not be ethical, as surgery has over time become accepted as the gold standard for treatment in CSM, particularly in people with a deteriorating neurological status (Jankowitz and Gerszten, 2006, Rao et al., 2006). All participants in the current study had experienced deterioration in their gait since the onset of their symptoms, and all chose surgical intervention after the option was presented to them with the known risks and benefits. The current study adds an additional dimension to knowledge of these risk and benefits by confirming that, for the most part, gait does not deteriorate, and that positive changes are possible, particularly in an uncomplicated post-operative course.

9.5.6.2 Evaluation of impairment associated with gait deficits

The study examined gait in freely moving participants with CSM at their comfortable gait speed to gain an insight into the preferred locomotor strategy of the CNS in response to the neurological lesion. The features of an UMNL, namely spasticity, paresis, impaired sensation and proprioception, would have influenced these locomotor patterns to varying degrees. The study did not include measurements of impairment of proprioception or muscle strength. The influences of these impairments therefore cannot be quantified. However, only weak associations between voluntary muscle strength and gait have been shown in other neurological conditions, such as CP (Dallmeijer et al., 2011).

An outcome measure of one aspect of the UMNL, spasticity, was used, but again this was known to have poor association with changes in tone observed during gait and during active movement in general (Ada et al., 1998, Dietz, 2003). In the current study, the MAS provided information on resting tone as a separate entity. It was not used to inform the analysis of gait.

With regard to measurement of proprioception, the complex interaction between the various sensory pathways involved in the regulation of gait could not have been inferred by simple clinical tests of sensation and joint position sense. More intensive tests, such as static posturography, would be informative and should be considered for future studies. However, for the current study, the addition of such measures would have significantly increased the research burden on participants who were already required to
contribute significant time to multiple gait analysis assessments with several repeated trials.

The assessment of various aspects of neurological function in locomotion is complex and time-consuming, with a long list of potential research questions. The study needed to focus its methods to its primary question, the performance of gait. Information on signs of impairment, such as power and sensation, can lead to a diagnosis and an overall impression of the severity of CNS involvement, but give little information about the cause and effect of a locomotor disorder (Dietz, 2003). Now that the key aspects of gait impairment in CSM have been identified, and their response to surgery documented, future studies could further assess the contributions of impaired proprioception, paresis and spasticity to the observed gait patterns.

9.5.6.3 Effect of heterogeneity among participants

Finally, although the study was statistically powered to detect change following surgery with gait speed as the primary outcome measure, the sample size of 13 was vulnerable to the effects of heterogeneity within the cohort. The standard deviation of the difference for gait speed was 0.2 m/s, almost twice the predicted variance of 0.11 m/s. Much of this increased variance was contributed by two of the participants with complications, who experienced deterioration in gait speed that was not in keeping with the trend of the cohort in general. However, post-operative complications are a feature of surgery, and were reported in 18.5% of cases in a recent prospective study of 81 people with CSM (Furlan et al., 2011). The incidence of complications in the current study was in keeping with general trends.

9.6 Conclusion

The study concludes that, at one-year follow-up after surgical decompression, people with CSM experienced subjective improvements in their ability to mobilise and the severity of their CSM. This was associated with improved functional mobility and health-related quality of life. TSPs and kinematic patterns showed no change following surgery, however there were increases in the absorption and generation of power, particularly by the knee and ankle, at key phases of the GC. EMG data indicated preservation of the potential for neuromuscular adaptation by altering the duration of activation of key muscle groups. Future studies of gait in CSM should focus on analysis of gait in more challenging environments, and the implementation of rehabilitation strategies to maximise the changes in kinetic and EMG parameters in improving locomotor performance following surgery.
Chapter 10: Conclusion and Implications

10.1 Introduction

The aims of this thesis were 1) to examine the repeatability of 3DGA and EMG measures of gait in CSM, 2) to evaluate gait impairment in people with CSM compared to healthy age- and gender-matched controls, and 3) to investigate changes in gait following decompressive surgery. There were a number of novel aspects to this thesis. The repeatability of 3DGA had not previously been examined in CSM or other forms of incomplete SCI. Furthermore, this was the first known investigation of the repeatability of EMG indices of timing, amplitude and response to lengthening in gait in a neurological population. This was the first study to compare gait in CSM to age- and gender-matched HCs, and to account for the speed dependency of gait parameters. It was also the first study of gait in CSM to systematically evaluate a range of kinematic key points, to investigate joint moments and powers, and to quantify muscle activity by EMG in a CSM cohort before and after decompressive surgery.

10.2 Overview of the thesis

The literature review was divided into three chapters. The first of these, Chapter 2, presented an overview of the aetiology, pathophysiology and clinical signs and symptoms of CSM. This review highlighted the difficulties in diagnosing CSM due to its variable presentation and MRI findings. The emergence of surgery as the mainstay of treatment was discussed. The goal of surgery in most cases was to prevent further deterioration in neurological function (Rao et al., 2006). Prediction of post-operative outcome was limited by the absence of evidence on the natural history of CSM, lack of RCTs comparing different surgical approaches, and inadequate follow-up after surgery in the published studies. The review highlighted the need for quantitative, sensitive outcome measures, such as 3DGA, over standardised follow-up intervals to improve knowledge on the effect of surgery in CSM.

Chapter 3 focused on gait impairment as one of the primary symptoms of CSM. Previous research had identified some of its features. People with CSM tended to walk more slowly than HCs, with prolonged double support duration and shorter stride length (Kuhtz-Buschbeck et al., 1999, Maezawa et al., 2001, Lee et al., 2011). Kinematic analysis identified reduced ROM at the knee in early stage disease. Reduced ankle plantarflexion and knee hyperextension in stance were features of more severe CSM (Maezawa et al., 2001, Kim et al., 2010, Lee et al., 2011). Although these earlier studies contributed to the understanding of gait in CSM, the literature review identified a number of gaps in current knowledge. These were 1) absence of kinetic and EMG analysis, 2) lack of matched
speed comparisons, 3) follow-up assessments at non-standardised intervals following surgery, and 4) lack of concurrent assessment of outcome measures of CSM severity, HRQOL and functional mobility.

Evidence for the role of 3DGA in other pathologies was reviewed in Chapter 4. It was shown that kinetic analysis determined compensatory strategies for weakness of specific lower limb muscles in children with myelomeningocele (Gutierrez et al., 2005). EMG-based measures of the timing of muscle activation and co-activation identified key neuromuscular adaptations underpinning the recovery of mobility after stroke (Buurke et al., 2008), and showed that people with orthopaedic problems used similar strategies to compensate for weakness (Heiden et al., 2009). A locomotor-based measure of spasticity objectively quantified abnormal responses to muscle lengthening in CP and stroke, bridging the gap between passive measures of spasticity and its effect on gait (Crenna, 1999, Lamontagne et al., 2001). Such studies had contributed to the understanding of the biomechanical and neuromuscular mechanisms that influenced gait in these conditions, and had implications for rehabilitation and outcome assessment. The methods and findings of these studies, and the gaps in the understanding of gait in CSM identified in Chapter 3, provided the basis for the aims and objectives of the thesis.

The methods of the thesis were developed in Chapter 5 and described in Chapter 6. Of particular interest was the validation of a novel method to determine the timing of muscle activation from EMG signals in gait. This “double threshold method”, based on the Teager Kaiser Energy Operator function, built on the work of previous authors in the field. It showed 87.5% agreement with visual interpretation of muscle activation, the highest accuracy of the four methods tested. The selection of 3DGA and EMG parameters for the thesis was based on their use in previous studies in neurological conditions, however information on their test-retest reliability in CSM was lacking. It was decided to test the reliability of a wide range of possible parameters, and select those with sufficient repeatability for use in the remainder of the thesis.

The results and discussion of the thesis were divided into its component studies, the reliability study in Chapter 7, the cross-sectional study in Chapter 8, and the experimental study of post-operative changes in CSM in Chapter 9. The reliability study showed that most TSPs, kinematic and kinetic parameters were sufficiently reliable for clinical and research purposes. Variation in scores from test to retest was considered to result from a combination of marker placement error and intrinsic variability in the gait patterns of the participants. EMG data yielded more variable reliability. Timing parameters were generally reliable, although BF showed a larger SEM of than RF, TA and MG. Amplitude parameters showed poor reliability with the MVC normalisation method. The peak dynamic method (PDM) showed higher, but still variable, reliability. The PDM method was therefore chosen for use in the cross-sectional and experimental studies, while
acknowledging the limitation that it diluted inter-individual variability due to its lack of a quantifiable physiological reference. Finally, of the parameters used in the LSMS, the LVT and critical time of EMG onset during lengthening showed better reliability than the slope criterion of Lamontagne et al. (2001). It was decided to retain the direction of the slope, positive or negative, as an outcome measure, but not the slope gradient. The reliability study aided in the interpretation of the results of the cross-sectional and experimental studies, as it estimated the change in each parameter that would be required to exceed measurement error.

The results of the cross-sectional study were presented in Chapter 8. The CSM participants had a significantly slower comfortable walking speed. Stride length and single support duration were reduced compared to HCs at both comfortable and matched speeds. Kinematic and kinetic data showed multiple differences between groups at comfortable speed. At matched speed, significant decreases persisted in the CSM cohort in peak ankle plantarflexion and peak knee flexion in swing. Non-significant reductions in ankle power generation and knee power absorption peaks were noted. The CSM group showed prolonged EMG activation duration of BF, RF and TA, and prolonged RF–BF co-activation, at both speeds. Of interest was the finding that timing parameters changed little in HCs from comfortable to matched speed, indicating that the timing of muscle activation as measured by EMG is innate and not speed dependent. The LSMS found evidence of increased velocity-related sensitivity to lengthening in TA, but not in RF, BF or MG. Many of the findings provided evidence for paresis as a cause of impairment in gait in CSM. The evidence for spasticity as a contributing feature was less convincing, though this was interpreted within the limitations of the LSMS. Participants in the study were not assessed under conditions that would challenge balance and stability, and therefore impairment of proprioception as a contributing feature could not be fully evaluated. Impaired proprioception was suggested by the reduced single support duration, however were no differences in step width or medio-lateral GRF, variables that might indicate instability (Judge et al., 1996a). PCA revealed more differences in the PC representing momentum generation and propulsion, than in the PC representing stability in stance. It was therefore postulated that paresis was the main contributory impairment to gait in CSM.

Chapter 9 presented the results of the experimental study. Pre to post-operative 3DGA and EMG analysis of gait in 13 CSM participants found no changes in TSPs or kinematics. Kinetic analysis showed statistically significant post-operative increases in K4 absorption power at terminal swing, A2 power generation in pre-swing, and ankle plantarflexor moment, although the first two of these variables had not reached the range of HC data. EMG analysis showed compensatory responses in the timing of TA, which had further prolonged its duration of activation from pre-operative assessment. The LSMS showed a significant increase in LVT of RF, indicating reduced sensitivity to
lengthening. PCA revealed small improvements in the first PC representing propulsion, but not in the second PC, representing stability in stance. The gait analysis findings were associated with significant improvements in CSM severity, functional mobility and HRQOL. Overall, the experimental study indicated that gait did not deteriorate post-operatively. The aim of surgery to stabilise function had therefore been achieved. The improvements in kinetic parameters suggested that power was absorbed and generated more effectively at key points in the GC. This was interpreted as an indicator of some neurological recovery. The improvements in the secondary outcome measures supported this trend of recovery.

10.3 Major contributions of the thesis

10.3.1 Reliability of 3DGA and EMG technology in analysing gait in CSM

Although 3DGA had been used previously to evaluate kinematics in CSM, its reliability in this population had not been established. The reliability study in this thesis contributed to this gap in the literature. It found that most 3DGA and EMG parameters had the psychometric properties to differentiate change from measurement error, and quantified the likely test-retest error ranges. It also highlighted those measures that were insufficiently reliable. The results have applicability to other neurological populations, particularly the findings pertaining to EMG. Several studies have used EMG measures of timing, amplitude and response to lengthening of muscle activity, without considering the influence of measurement error or intrinsic variability on the observed scores. Future studies are required to determine whether reliability is similar in other pathologies, however comparisons can be made between the population of this thesis and other conditions.

10.3.2 Identification of the key characteristics of gait in CSM

Previous studies of gait in CSM focused only on temporal-spatial and kinematic parameters, but not on the underlying biomechanical and neuromuscular strategies that produced movement during walking. The association between gait speed and many kinematic key parameters had not been considered. The current study identified several abnormalities in the CSM cohort that persisted when compared to HCs at matched speed. Generation of momentum from stance to swing was particularly affected, and this was confirmed by multivariate analysis using PCA. EMG analysis highlighted significant differences in the duration of muscle activation and co-activation in the lower limbs during gait. The study identified strong evidence for paresis as a contributing factor to gait impairment in CSM. This was the first study to describe the key characteristics of gait in CSM, and to ascertain its underlying contributory factors.
10.3.3 Understanding the effect of surgical decompression on gait in CSM

Literature on the effects of surgical decompression of the spinal cord in CSM has been mixed to date. Reports have stated that surgery aimed to prevent further neurological deterioration, and that improvement in function could be expected only in the minority of cases (Rao et al., 2006). However, a number of studies showed improvements in aspects of mobility such as gait speed (Singh et al., 2009). The current study was the first to evaluate changes in gait using a detailed gait analysis protocol involving three-dimensional motion analysis and EMG study of muscle activation patterns. Follow-up was conducted at standardised intervals, although final analysis included a six-month follow-up for three participants who could not complete the 12-month post-operative assessment. Secondary outcome measures of functional limitation and participation restriction were evaluated concurrently. The findings showed an improvement in secondary outcome measures, but no changes in gait speed, TSPs, or kinematic parameters. There were, however, significant changes in power generation and absorption at key phases of the GC, compensatory responses in muscle activation, and improvements in the principal component reflecting momentum generation and propulsion. The fact that these did not affect TSPs and kinematics could be explained by two possible mechanisms, 1) the increased power peaks were of insufficient magnitude to improve joint ROM, stride length and speed, and 2) the pre-operative gait pattern had become embedded in the CNS, despite the capacity for improved momentum generation and adaptation of EMG parameters. In either case, the experimental study concluded that surgery had impacted the biomechanical and neuromuscular features of gait impairment. Capacity for further improvement should be explored through rehabilitation strategies aimed at locomotor function.

10.4 Implications of the thesis

10.4.1 Implications for physiotherapists

This need for this thesis was prompted by a lack of evidence on expected outcomes following surgery in CSM, and the absence of definitive rehabilitation protocols or guidelines in managing the condition. The expectation that surgery would stabilise rather than improve gait impairment meant that the potential of patients to respond to post-operative rehabilitation was uncertain. Recently, there has been growing evidence on the ability to improve gait in people with SCI by neuroplasticity, neurological recovery and compensation (Dietz, 2010). This thesis identified key aspects of gait impairment in CSM, with many implications for physiotherapists. Firstly, the biomechanical and neuromuscular patterns underlying gait impairment have been described in greater detail, and therapists can improve their understanding of this condition as a result. Secondly, the role of paresis as a contributing factor implies that rehabilitation programmes should focus on
strengthening of the lower limb muscles. Finally, it is possible that a targeted post-operative rehabilitation programme could facilitate the translation of improved kinetics into faster walking with more normal kinematics. Future RCTs will be necessary to evaluate such programmes. However, until these emerge, the evidence suggests that there is potential for improved gait patterns in people with CSM following surgery. Locomotor strategies aimed at maximising the improved kinetics, harnessing the ability of the CNS to compensate for deficits, and preventing the hardwiring of pre-operative gait patterns into the CNS, should be trialled with individual patients to facilitate recovery.

**10.4.2 Implications for surgeons**

Surgeons face a significant problem in the lack of sensitive, quantitative published data on outcomes following surgical intervention for CSM (Jankowitz and Gerszten, 2006). In deciding the optimal management for any given patient, the risk of intra-operative complications must be weighed against the prospect of future neurological deterioration if surgery is not undertaken. The 3DGA and EMG assessment of gait in this thesis is a significant contribution to knowledge of post-operative changes. The findings should inform the pre-operative decision-making process by providing surgeons with greater clarity on the aspects of gait that can be affected by surgery. Furthermore, the evidence for further potential improvements in gait, outlined in Section 10.4.1 above, suggests that surgeons should encourage their patients to pursue active rehabilitation following surgery. The significant post-operative improvements in the secondary outcome measures of severity of CSM, functional mobility and HRQOL are encouraging, as they suggest that the potential benefits of surgery are multi-dimensional. Finally, the lack of evidence to implicate spasticity as a causative factor of gait impairments suggests that people with CSM may be unlikely to benefit from anti-spasmodic medication as a means of improving their gait. This is in line with expert opinion on the nature of spasticity (Dietz, 2003).

**10.4.3 Implications for people with CSM**

Individuals with CSM are faced with the uncertainty of future progressive neurological deterioration due to ongoing degenerative processes in the cervical spine. Surgery is often presented as the only option to prevent this deterioration. Its goal to stabilise rather than improve neurological function, coupled with the risk of major surgical complications, creates a situation of potentially undesirable outcomes regardless of whether operative or conservative management is chosen. This thesis confirms the stabilisation of a gait deficit at one year following surgery, even with the inclusion of participants who experienced complications. Furthermore, changes in kinetics and EMG suggest that further improvement in gait is possible with targeted rehabilitation intervention, in line with recent studies on the recovery of mobility in incomplete SCI. The findings indicate that the post-
operative course for people with CSM may be more optimistic than previously expected, and suggest that they should pursue more active rehabilitation following surgery.

10.5 Limitations of the thesis

10.5.1 Statistical power for gait data

The study had 90% statistical power to detect a difference of 0.1 m/s at the 0.05 significance level with 13 participants in the experimental study, and 13 participants in each group in the cross-sectional study. This was in keeping with the recommendation that studies using multiple outcome measures should have a minimum power of 90% to detect change in the primary measures (Borm et al., 2006). The power calculation was based on gait speed because it was the most widely reported measure of gait in CSM, has a clear relationship with functional performance (Judge et al., 1996b), and is a predictor of many outcomes (Bohannon and Williams Andrews, 2011). The variance of other gait parameters, particularly in EMG, is not widely known. The power of the study to detect change in kinematic, kinetic or EMG variables cannot be assumed, and it is possible that some type two statistical errors occurred in the analysis.

The participants in the experimental study had greater variance than expected. Three of 13 participants experienced complications that affected their recovery from surgery, leading to a larger than expected standard deviation of the pre- to post-operative difference in gait speed. All small sample sizes are vulnerable to the effects of heterogeneity, so it is possible that type two errors may have occurred. However, the participants in this study represented a large proportion of the eligible population who were approached on a continuous prospective basis over a two-year period. Furthermore, the rate of complications in the current study was in keeping with larger international studies (Furlan et al., 2011). These facts suggest that the participants can be considered representative of the wider population of people with CSM.

The inter-dependence of many gait parameters also deserves consideration from a statistical point of view. Each variable was considered of benefit in the description and analysis of gait impairment, and as a result, multiple comparisons were made using paired t tests. The possibility of a chance finding of significance due to a type one error cannot be out-ruled. The strong correlations between individual gait variables precluded the use of Bonferroni adjustments (Perneger, 1998). The problem of a possible chance finding was addressed by evaluating the relationships between gait variables that were found to be significantly different, with the rationale that a chance finding of significance would not be associated with significant differences in its contributing variables. Multivariate analysis was also employed to focus the discussion to the principal components of gait that showed greatest deviation.
10.5.2 Protocol deviations

The interval between pre-operative assessment and surgical intervention could not be standardised among participants, as this was beyond the control of the PI. Some participants experienced delays in their planned admission to hospital. The mean interval from pre-operative assessment to surgery was 2.2 months. The possibility of further deterioration in gait during this time, not captured by the pre-operative assessment, cannot be out-ruled. If a further deterioration had occurred, it is possible that the true changes in gait following surgery were under-represented.

Three participants were unavailable for follow-up at the 12-month post-operative assessment. Their six-month data were used for the pre- to post-operative analysis. Further change could have occurred between six and 12 months. However, graphs of gait data of the 10 participants who completed the protocol suggested that the greatest improvement occurred in the first six months, with smaller improvements thereafter. This temporal trend was not tested for significance due to the already high volume of hypothesis tests, though similar findings were reported in a previous study (Singh et al., 2009).

10.6 Recommendations for future studies

10.6.1 Evaluation of proprioceptive challenges and community ambulation

The thesis focused on the analysis of overground barefoot walking at comfortable gait speed to indicate the natural preferences of the locomotor system. Functional walking requires nervous system control for three tasks, 1) equilibrium during propulsive movement, 2) basic reciprocal movement strategy, and 3) adaptation to behavioural goals and external constraints (Behrman et al., 2006). The current study did not evaluate the third task. The subjective improvement in gait reported by the participants, and the reduction in falls incidence, could reflect an improvement in walking ability associated with activities of daily living and the ability to adapt the motor program to varying demands. Future studies could address this issue by including gait analysis of a range of gait speeds, tandem gait, and obstacle avoidance (van Hedel et al., 2007). This would evaluate the ability to control centre of mass in more unpredictable conditions (Schrager et al., 2008), giving a greater indication of instability and proprioceptive deficits in CSM. Evaluation of the sensory, vestibular and visual components of balance using posturography is also recommended, as this technology has shown the ability to discriminate the causes of balance deficits (Yardley et al., 1998). Finally, future studies should assess the ability to ambulate within the community, a skill that is not adequately predicted by gait speed (Lord and Rochester, 2005).
10.6.2 Rehabilitation of CSM

The literature review identified a lack of evidence to guide the rehabilitation of people with CSM. There is a need for studies of rehabilitation intervention in this population, given that many patients will be limited by a gait impairment that, based on the evidence of the experimental study, shows potential to improve after surgery. The cross-sectional study deduced that paresis was the most evident contributory factor, suggesting that strengthening programmes may be beneficial. Rehabilitation should also specifically target locomotor function. Adjuncts such as body weight supported treadmill training have shown promise in incomplete SCI (Lucareli et al., 2011). The current study showed evidence for biomechanical and neuromuscular changes following surgery that had not translated into improved gait speed or kinematics. Targeted locomotor training could bridge this gap, resulting in more positive outcomes for people with CSM.

10.6.3 Analysis of neuromuscular control using EMG

The thesis focused on the evaluation of neuromuscular control using EMG measures of timing, amplitude, and response to lengthening. Apart from TKEO, little processing was applied to the EMG signals. Measures of timing showed good discriminative validity and were repeatable and accurate compared to visual interpretation. However, measures of amplitude in particular showed limited ability to distinguish people with CSM from HCs, due to the dilution of inter-participant variation by normalisation. Visual interpretation suggests that much detail is contained within the EMG signal. The challenge is to remove clinically meaningful information from the signal in a manner that facilitates comparison between different individuals and on different test days. Recent studies of more complex EMG processing techniques, such as wavelet decomposition of signals into time-frequency space and subsequent PCA, have shown promising results. These methods have the potential to provide new information on the nature of muscle adaptation to a CNS lesion (Wakeling et al., 2007). As with any technique, test-retest reliability of the parameters obtained in wavelet transformation or other complex EMG processing should be established, prior to their application in clinical and research practice.

10.7 Conclusion

The thesis aimed to address the lack of knowledge on gait impairment in CSM, and to evaluate changes in gait following decompressive surgery. The chosen outcome measures, 3DGA and EMG, were found to be sufficiently reliable for use in clinical and research practice in the evaluation of gait impairment in CSM. Comparison with age- and gender-matched HCs, including a matched speed analysis and PCA, indicated key deficits in gait in CSM, particularly in relation to generation of momentum at terminal stance and pre-swing. Compensatory EMG responses of prolonged activation and co-
activation were evident. The deficits were considered to be due primarily to paresis. Repeat analysis at six and twelve months following surgery indicated that decompression of the spinal cord had succeeded in stabilising the gait deficit. Some kinetic features of gait improved significantly following surgery, though these had not translated into temporal-spatial and kinematic changes. There may be potential for further improvement in gait in CSM through rehabilitation protocols focused on strengthening, as well as the targeting of locomotor strategies to maximise the improved kinetics and facilitate adaptation within the CNS to a more normal gait pattern.
References


Patrick, E. & Ada, L., 2006. The Tardieu Scale differentiates contracture from spasticity whereas the Ashworth Scale is confounded by it. *Clinical Rehabilitation*, 20, 173-82.


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Appendices
### Equipment Trolley

<table>
<thead>
<tr>
<th>Name</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measuring tape</td>
<td>cloth, 1.5m in length</td>
</tr>
<tr>
<td>Calipers</td>
<td>measuring up to 14cm widths</td>
</tr>
<tr>
<td>Single-use disposable razors</td>
<td>1 per subject trial</td>
</tr>
<tr>
<td>Alcohol swabs</td>
<td>(Sterets) 3-4 per subject</td>
</tr>
<tr>
<td>Black kohl pencil</td>
<td>for surface marking</td>
</tr>
<tr>
<td>Leucopore fixing tape</td>
<td>(Mueller) 2.5cm width</td>
</tr>
<tr>
<td>3mm Screwdriver</td>
<td>to alter gain settings on the EMG system</td>
</tr>
<tr>
<td>tibial torsion device</td>
<td>for measurement of tibial torsion</td>
</tr>
</tbody>
</table>

### EMG

16 Channel (MA-300-16) EMG Back Pack Unit (Motion Lab Systems), plus gait waistcoat
8 pre-amplified surface electrodes, (Motion Lab Systems)
Ground electrode (Motion Lab Systems)

### Marker placement

- Double-sided adhesive tape (2.5 cm, Motion Lab Systems)
- Scissors
- 10 Vicon markers and 5 wands
- Knee alignment devices (Motion Lab Systems)

### Other

- Wooden step with rubber cover (21cm high, to allow more accurate visualisation of Vicon markers)
- Full length mirror
- Height-adjustable plinth plus 2 pillows.
Anthropometric Measurements

Height
Measured with a stadiometer. The head is positioned in the Frankfurt Plane, a standard plane for orientation of the head. It is established by an imaginary line passing through the right tragion (the front of the ear) and the lowest point of the right eye socket. Subject is barefoot. Measure the vertical distance from the floor to the vertex (the highest point on the head in the mid-sagittal plane).

Weight
Calculated using Seca digital measuring scales, and record in kilogrammes. Subject is barefoot.

Leg length
Subject lies supine and is asked to bridge and swing pelvis from side to side and then lower pelvis. Both lower limbs are then extended. Pen mark (X) placed on each ASIS - located by palpating caudally to cranially on anterior hip to find inferior aspect of ASIS. The most inferior bony point is marked. Leg length is measured from the mark to the inferior aspect of the medial malleolus. It is measured in cm to nearest 0.1cm

Inter-ASIS distance
Subject supine, knees extended, measure distance from each marker point on the ASIS using measuring tape, measured in cm to nearest 0.1cm

Knee width
Subject in supine lying, knee flexed (75–80°)
Lateral knee: Palpate lateral epicondyle and locate popliteal groove, which is a groove just below the lateral epicondyle. Mark position with skin surface marker.
Medial knee: Palpate the medial femoral condyle, locate the medial collateral ligament and place a mark where the midpoint of the medial collateral ligament intersects with the joint line. Mark this point with a skin surface marker, the knee joint axis is downwards and medially. Knee width is measured with a callipers, one arm is placed on the medial and the other on the lateral knee joint mark. This is measured in cm to nearest 0.1cm, recording the average of three measurements.
Double–sided tape is applied to the medial and lateral aspects of the knee to allow better attachment of the knee alignment device during Vicon marker placement.

Ankle width
Subject in supine lying, knee flexed (75–80°). Measured with callipers, one arm on the most medial point of the medial malleolus and one arm on the most lateral point of the lateral malleolus. This the widest part of the ankle joint. Measured in cm to nearest 0.1cm, recording average of three measurements to nearest 0.1cm

Tibial Torsion
With the subject supine, rotate the thigh and shank (around the long axis of the thigh and shank) until the knee axis lies horizontal (if you have marked the knee axis on the medial and lateral side then these marks should now be the same vertical height above the couch surface).
Sit at the end of the couch facing the plantar surface of the feet.
With the preferred hand place the device tips up on the malleoli marks, adjusting the width of the tips of the device with the fingers through the two ring-holes on each of the legs. Ensure that the device is held with the plane of the dial-face vertical allowing the angle indicating arm to hang freely under gravity. The degree of tibial torsion is indicated by the angle indicating arm when the device is held in this position. It is a measure of the angular difference between the knee axis and the ankle axis in the vertical plane. It is otherwise
expressed as the amount of rotation around the long axis of the shank between the projection of the knee flexion/extension and the bimalleolar (ankle) axes onto the plane perpendicular to the long axis of the shank.

Fig 1: Measurement of Tibial Torsion

Procedure for SEMG electrode Placement

Equipment
- 16 Channel (MA-300-16) EMG system (Motion Lab Systems), plus gait waistcoat
- 8 Active surface electrodes, pre-amplified and labeled for each muscle and corresponding channel.
- Ground electrode (Motion Lab Systems)
- Leucopore fixing tape (1.25cm & 2.5cm widths) Alcohol swabs (sterets),
- Single-use disposable razors
- Black kohl pencil for surface marking
- Measuring tape

Protocol
Subject dressed appropriately in shorts, supine on plinth for limb measurements and anterior surface electrode marking.

1. Check all attachments to MA-300 desk top unit and ensure all leads are in place.
2. Attach SEMG electrodes to their specific channels (1-8) in the MA-300 before attaching to patient.
3. Bring the wires of the 8 leads down the sides of the patient’s shorts (4 on the left and 4 on the right side). Rest the MA-300 Back Pack Unit on the subject’s torso.
4. Clean all electrodes with an alcohol swab and leave to dry with contact area facing upwards.
5. Identify location for electrode placement on muscle (see below: “Electrode Placement”). Locate muscle bulk to be tested (e.g. gastrocnemius), and engage muscle by resisting its action (resist ankle plantarflexion). Mark borders of the location for the electrode with skin marker.
6. Prepare the skin by shaving the area for the electrode until all hair is removed.
7. Wipe area with alcohol swab 6 times forward and back and leave to dry.
8. Secure the body of the electrode to the muscle using Micropore surgical tape or Leucotape. Secure the wire with a separate strip of tape approximately 2cm from the base of the electrode.

9. Connect the black transmission wire from the MA-300 desktop unit to the backpack unit and turn on the desktop unit. Ensure green “power” light is glowing at front of desktop unit.

10. Note the gain of each electrode channel.

11. Open “Windaq USB0” application and check the quality of the output signal from the 8 channels, firstly at rest, and secondly with the subject moving the lower limbs to activate the corresponding muscle channels.
   - If interference is noted on a channel’s output when the corresponding muscle is at rest:
     - Remove electrode from skin, shave and swab the area again, leave to dry and re-apply electrode. If the skin is very dry, hydrate the area using Eko-Gel™ ultrasound gel after shaving and cleaning to minimise static interference from the skin surface.
     - Disconnect and re-connect the electrode to its corresponding channel.
     - Re-check all wires and connections to the MA-300 desktop unit.
     - Ensure ground electrode is in contact with an electrically neutral area (e.g. bony prominence).
   - When the subject moves the lower limbs, ensure each channel is registering an electrical signal with muscle activity. If no signal is observed:
     - Ensure electrode is correctly connected to its channel by removing and re-connecting if necessary.
     - Increase the gain on the corresponding channel and repeat contraction.
     - Check that electrode is in contact with skin and is over the muscle belly.
     - Remove the electrode from its channel and test in another channel.
     - If all of the above measures fail to elicit an electrical signal with muscle contraction, remove the electrode, mark as faulty, and use a spare electrode.
   - If the signal from a channel appears “saturated”, reduce the gain of the channel on the MA-300 Back Pack Unit using a 3mm screwdriver.

**Electrode Placement**

1. **Rectus Femoris (knee extensor and hip flexor)**
   - Patient supine.
   - Activate muscle by resisting knee extension and hip flexion.
   - Measure distance from ASIS to the superior border of the patella, place electrode halfway between these surface markings parallel to the orientation of the fibres.

2. **Tibialis Anterior (ankle dorsiflexor)**
   - Patient supine.
   - Activate muscle by resisting ankle dorsiflexion.
   - Measure the distance between the lateral joint line of the knee and the medial malleolus.
   - Place electrode just lateral to medial shaft of tibia at one third distance from the knee to the ankle marker (over area of greatest muscle bulk).

3. **Biceps Femoris (knee flexor, hip extensor and lateral rotator of tibia in relation to femur)**
   - Patient prone.
   - Activate muscle by resisting further flexion of knee held in flexion and slight lateral rotation.
   - Palpate muscle bulk and place electrode parallel to muscle fibres midway between gluteal fold to the lateral knee on posterior-lateral aspect of thigh.

4. **Gastrocnemius Medial Head (ankle plantarflexor)**
   - Patient prone.
Activate muscle by resisting plantarflexion of the ankle. Measure distance between the medial knee joint line and the medial malleolus. Apply electrode one third distance from the knee, 2cm medial to the midline of the posterior aspect of the leg. Apply parallel to muscle fibre orientation over area of greatest muscle bulk.

5. **Ground electrode** (green) is applied to the right shoulder of the subject and attached to the back of the EMG recorder.

**Order of Placement of electrodes**

**Supine**
- Left Rectus Femoris Channel 1
- Left Tibialis Anterior Channel 5
- Right Rectus Femoris Channel 2
- Right Tibialis Anterior Channel 6

**Prone**
- Right Biceps Femoris Channel 4
- Right Gastrocnemius Channel 8
- Left Biceps Femoris Channel 3
- Left Gastrocnemius Channel 7

**MVIC (Maximum Voluntary Isometric Contraction)**

MVIC recordings are taken from each muscle in turn, since the patient is already in prone, left biceps femoris and left gastrocnemius are tested first followed by the right side. The patient is asked to hold the limb in position and contract *as hard as you can* for 3 seconds against the testers applied resistance. Each subject is allowed a practice trial on each muscle before recordings are taken using the Workstation programme. SEMG gain channels are usually set to 1 for MVIC, but should be increased if the signal is too small to be readable at this setting when observed in Windaq.

**Prone**
- **Biceps Femoris**: knee in 70 degrees flexion, tester applies strong extension force just proximal to the tendo-achilles. Subject resists and attempts to flex knee maximally *“bend your knee, hard as you can”*

- **Gastrocnemius**: knee in 70 degrees flexion and ankle in mid-plantarflexion. Patient is asked to push foot and toes up into testers hand at the ball of the foot; tester applies a dorsiflexion force and instructs *“push your foot up, hard as you can”*

The subject is asked to sit over the edge of the bed (taking care that electrode leads and tape are not pulled or displaced). The subject is allowed a practice MVIC trial of each of the anterior muscles before recording takes place. The subject is asked to stabilise with their hands at the side of the bed with the back of the knees against the edge.

**Sitting**
- **Rectus Femoris**: Participant sits with thighs supported, hands placed at edge of plinth for support, knees in 90° flexion, feet clear of floor. Participant flexes hip so that thigh clears the bed, tester places one hand on lower tibia proximal to ankle joint, participant attempts to strongly extend knee while maintaining flexed hip. Instruction is *“hold your knee up and straighten your leg as hard as you can”*

- **Tibialis Anterior**: ankle in 5-10 degrees dorsiflexion and slight inversion. The tester attempts to pull the foot out of this position and the subject tries to keep their foot in this position. Pressure is applied by the tester at the dorsum of the foot at the level of the metatarsal heads. Avoid stress on the wires by making sure that the wires are not taut when the limb fully extends.
Protocol for Vicon Marker Placement

Equipment:
15 lightweight retro-reflective markers 2cm in diameter (Vicon) (includes 5 wand markers)
Knee Alignment Device (KAD) x2 (Motion Lab Systems)
Double sided-adhesive tape (2.5cm) (Motion Lab Systems)
Black kohl pencil for surface marking
Full length mirror

Protocol:
Subject standing, on wooden box with rubber grip. The extra height of the subject allows for more accurate placement of the foot markers and alignment of the ankle, knee and thigh markers

1. **Forefoot Markers:** Attach to the heads of the 2nd metatarsal bones. To assist in identifying them, ask the subject flex their toes.

2. **Ankle Joint Markers:** Attach directly over the lateral malleoli.

3. **Heel Markers:** Attach over the os calcis at the same height as the forefoot marker. A caliper (which was used to measure knee widths) can be used to measure the distance from the uppermost point of the forefoot and heel markers to the floor (Figure 2). These should be equal. The mirror should also be used to check that the markers are in alignment along transverse plane.

   ![Figure 2: Photograph demonstrating how a calipers is used to ensure that the retro reflective markers of the forefoot and heel are placed at the same height.](image)

4. **ASIS:** Markers are placed over each anterior superior iliac spine, left then right. These have been marked previously leg-length measurement (on the iliac crest palpate caudal to cranial, the marker is placed over the most anterior part).

5. **Knee Joint Axis:** The axis is determined using a knee alignment device (KAD) and the markers for the lateral and medial femoral condyles. The KAD is attached with the subject standing away from the bed. The horizontal wands are aligned parallel to the floor. The back of the KAD is adjusted so that it gradually falls into the back of the calf whilst tightening the lateral side of the device and letting the back of it to rest on the back of the subject’s leg. The subject is then asked to actively flex and extend each knee in turn; the location of the device is altered by the principal tester until the flexion/extension wand shows minimum movement.

6. **Thigh Markers:** The shorter wand markers are placed at the lateral aspect of the thigh, in alignment with the greater trochanter (hip joint centre) and the lateral knee joint axis marker. A full length mirror is placed 2 metres from the subject to aide the examiner to accurately line up the markers. This is done firstly on the left side, then the right. The left thigh marker is lower than the right.

7. **Shank Markers:** The longer wand markers are used for the shank; these are aligned with the ankle joint markers and the knee joint markers, again using the mirror for reference. The left thigh marker is lower than the right.
8. **Sacral Marker**: The sacral wand marker is placed at the midpoint between the skin dimples formed by the PSIS (level of S2)

**Data Capture**

**Static Trial**: The subject stands quietly on the force platform (at the centre of the walkway) with KADs attached facing towards the workstation. Subject is asked to place arms across chest for the calibration and to remain as still as possible with knees extended fully. Up to three seconds of video data is recorded.

The KADs are removed and the lateral joint markers are placed over the points where the KADs had been attached - there should be some temporary marks on the skin where the arms of the KAD’s were attached to the lateral knee. A second static trial is then taken with the lateral joint markers in place of the KADs.

**Gait Analysis**

Once the subject is ready for testing, he/she will have a trial walk down the walkway towards the workstation, without recording. During this trial walk:

- The outputs of the electrodes are checked and gains are increased or decreased to ensure a readable signal which is not saturated (aim for amplitude of one Volt at the signal’s maximum point during the gait cycle).
- The cameras are checked so that they are detecting all the reflective markers whilst the subject is walking over the forceplate.

The subject begins the walk from different point of the walkway good heel strike is achieved. Coloured start positions allow the subject to start at the correct position. To achieve sufficient data, 5 good strikes are required on each foot. Poor strikes are discarded, thus more than 14 walks may be required to achieve desired number of strikes. The assistant advises the subject of the point to start at. At this point the subject should be unaware of the forceplate. If the subject knows that the forceplate is there, then he/she may change the gait pattern in an attempt to strike the forceplate – this may lead to inaccurate and inconsistent results.

**References**


Appendix 5.2: Butterworth filter implemented in MATLAB

function mf = Butterworth(signal)
% Butterworth(signal) calculates the filtered EMG signal (musclef) for a single muscle using set filtering parameters decided in PhD Development of Methodology
% Ailish McDermott, 18 Aug 2010, RCSI Movement Lab
% fs = 1kHz
% Signal is a 1-D vector
% Butterworth takes the raw EMG data and filters using a 4th order Butterworth low pass filter with 400 Hz cut-off frequency followed by a 2nd order high-pass filter with a 25 Hz cut-off frequency
% All filters are dual pass (i.e. applied in forward and backward directions to avoid phase distortion) and zero lag
% Note: If fs is not = 1Khz, then divide HP cut and LP cut by new fs (in kHz)
% -----------------------------------------------------
% Butterworth filters low-pass and high-pass ------
[B1 A1]=butter(4,0.4,'low'); % 4th order LP filter, LP cut = 400 Hz
[B2 A2]=butter(2,0.025,'high'); % 2nd order HP filter, HP cut = 25 Hz
% ------ Butterworth filters low-pass and high-pass -----
% Resulting Filter ---------------------------------
b=cconv(B1,B2); % cconv function to convolve vectors B1 and B2
a=cconv(A1,A2); % cconv function to convolve vectors A1 and A2
mf=filtfilt(b,a,signal);
end
**Appendix 5.3: Scaling of gains in the MA-300 system**

### MA-300 EMG scale calculation for volts at skin surface

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Preamplifier gain 20
Desk Top Unit gain 2

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**MA-300 Gain switch settings for a GEN_SCALE of ............ 1.000000**

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Source: www.c3d.org
function TKEO = tkeo(signal)

% Applies Teager Kaiser Energy Operator to a signal and returns the output
% Created by Ailish McDermott, RCSI Movement Lab 02-Nov-2010
% Last modified 10-Nov-2010

mod_signal = [zeros(1,1); signal; zeros(1,1)]; % zero pads start and end of signal
signal_nplus1 = [zeros(2,1); signal]; % Creates vector of n+1 values for signal
signal_nminus1 = [signal; zeros(2,1)]; % Creates vector of n-1 values for signal
signal_tkeo1 = (mod_signal.^2) - (signal_nplus1.*signal_nminus1);
TKEO_long = abs(signal_tkeo1); % zero padded version of original signal
a = length(TKEO_long);
TKEO = TKEO_long(2:a-1); % returns signal to its original length
Appendix 5.5: MATLAB script for implementation of Resting Threshold Method and comparison to visual interpretation of muscle activation timing

function rest_threshold_method(th)
% ------------------------ %
% Part of the validation of EMG timing
% This function represents Method 1-3, the testing of various
% thresholds, th (1, 7 and 15) above resting EMG signal TKEO
% Compares against a pre-defined standard from visual
% interpretation of the signal
% Created by Ailish Malone (McDermott), RCSI Movement Lab, 06-Apr-2011
% Inputs: Threshold
% Outputs: logical "pass" (1) or "fail" (0) values to output
% matrices, ON and OFF, the result
% Folder "TimingValidation" contains files 'On_Result.mat' and
% 'Off_Result.mat', the results of visual interpretation
% ----------------------- %
root = '/Users/ailish/Documents/MATLAB/TimingValidation/';
load([root,'On(Result.mat']);
load([root,'Off_Result.mat']);
for n = 1:12
  % first load file, apply TKEO and filter
  load([root,'TA',int2str(n),'.mat']);
sig = tkeo(rtaf);
[B,A] = butter(2,0.05,'low');
sig2 = filtfilt(B,A,sig);
% Now find the resting signal and apply threshold algorithm
  load([root,'TA_rest',int2str(n),'.mat']);
  rtaf = Butterworth(rta);
  rest_tkeo = tkeo(rtaf);
  rest_mean = mean(rest_tkeo);
  rest_sd = std(rest_tkeo);
  threshold = rest_mean + (th*rest_sd);
% Now find points where gait signal exceeds threshold
  sig_on = sig2 > threshold;
% Now see where threshold is exceeded for > 24 / 25 data points
  TIME = [sig_on(end-12:end); sig_on; sig_on(1:12)];
t = length(sig_on);
  R = zeros(t,1);
  for k = 1:t
    R(k) = sum(TIME(k:k+24));
  end
  ACTIVE = R > 23;
  % Now we have our ON values - save before moving on to test
  against visual interpretation
  folder = '/Users/ailish/Documents/MATLAB/TimingValidation/TestResults/';
  outputfile = [folder,'TA_th',int2str(th),',',int2str(n),'.mat'];
  eval(['save ',outputfile,' ACTIVE sig2 sig rest_mean
  rest_sd']);
% Now compare to visual interpretation
  StandardFile = [root,'VisualResult_TA',int2str(n),'.mat'];
  load(StandardFile);
  tc = b - a;
  test = sum(ACTIVE(a:b));
  if test == tc + 1
    result = 1;
  end
end
else result = 0;
end
% Now output this result to ON matrix in On_Result.mat
if th == 1
    ON(n+1,2) = result;
elseif th == 7
    ON(n+1,3) = result;
else ON(n+1,4) = result;
end
ResultOutput1 = [root,'On_Result.mat'];
eval(['save ',ResultOutput1,' ON']);
% Repeat above for OFF matrix
if sum(ACTIVE(c:d)) == 0
    result2 = 1;
else result2 = 0;
end
if th == 1
    OFF(n+1,2) = result2;
elseif th == 7
    OFF(n+1,3) = result2;
else OFF(n+1,4) = result2;
end
ResultOutput2 = [root,'Off_Result.mat'];
eval(['save ',ResultOutput2,' OFF']);
end
clear all
end
Appendix 5.6: MATLAB script for implementation of Double Threshold Method and comparison to visual interpretation of muscle activation

% double_threshold_method
% A script file to test the accuracy of the "double threshold method", incorporating slope and amplitude parameters of the TKEO modified signal, in determining timing of muscle activation compared to visual interpretation as a "gold standard"
% Written by Ailish Malone, 14-Apr-2011
% Last modified 14-Apr-2011
% Inputs = signal from file 'TA',int2str(p),'.mat' [p = 1:12]
% Outputs = logical "pass" (1) or "fail" (0) values to output matrices, ON and OFF
% --------------------------- %
root = '/Users/ailish/Documents/MATLAB/TimingValidation/';
load([root,'On_Result.mat']);
load([root,'Off_Result.mat']);
for n = 1:12
    load([root,'TA',int2str(n),'.mat']);
    sig = tkeo(rtaf);               % Apply TKEO to filtered gait data
    [B,A] = butter(2,0.05,'low');   % Get vectors for dual-pass filter for smoothing
    sig2 = filtfilt(B,A,sig);       % Smooths the signal
    slope = gradient(sig2);         % Gets the slope of the smoothed signal
    sig_on = abs(slope) > 1e-6;     % Creates logical index where the absolute value of the slope is above a threshold
    th = (max(sig2))*0.03;          % Sets a threshold at 3% maximum value of signal
    sig_on2 = sig2 > th;            % Finds points where sig2 exceeds threshold value
    % ------------------------ %
    % Now put sig_on and sig_on2 side by side and sum the values
    % Create new logical matrix where both sig_on and sig_on2 declare 1, i.e.
    % both slope and amplitude exceed threshold
    isiton = [sig_on sig_on2];      % Creates matrix of both "on" conditions in rows for summing columns
    both_on = sum(isiton);          % Sums the columns of the 2 row vectors
    timeon = both_on';              % Transposes the resulting vector back to a column instead of a row
    changetime = timeon == 2;       % Creates a new logical vector where both conditions are satisfied for slope and threshold
    % ------------------------ %
    % Now see if both conditions are satisfied for at least 24 of 25
    % consecutive samples
    % May need to change value of 23 if it leads to false positives
    % 25 consecutives led to false off signals where there was a slight blip in
    % contraction
    TIME = [changetime(end-12:end); changetime; changetime(1:12)];
    % Appends the last 12 values to the beginning and first 12 values to end to create new vector TIME
% %--------------------------- %
t = length(changetime); % Gets the number of data points for new matrix containing 25 consecutive ON values
R = zeros(t,1); % Initialises a matrix that will contain logical values of TIME
for k = 1:t
    R(k) = sum(TIME(k:k+24)); % Sums each consecutive set of 25 data points
end
ACTIVE = R > 23; % If 24 of 25 consecutive points are satisfying the on condition, then consider muscle to be on (allows for blips in middle of contraction)
% ------------------------ %
% Now we have our ON values - save before moving on to test against visual interpretation
folder = '/Users/ailish/Documents/MATLAB/TimingValidation/TestResults/';
outputfile = [folder,'TA_dtm',int2str(n),'.mat'];
eval(['save ', outputfile, ' ACTIVE sig2 sig']);
% Now test against visual interpretation %
StandardFile = [root,'VisualResult_TA',int2str(n),'.mat'];
load(StandardFile);
% First test against ON values
tc = b - a;
test = sum(ACTIVE(a:b));
if test == tc + 1
    result = 1;
else result = 0;
end
% Now output this result to ON matrix in On_Result.mat
ON(n+1,5) = result;
ResultOutput1 = [root,'On_Result.mat'];
eval(['save ', ResultOutput1, ' ON']);
% Now test against OFF values %
if sum(ACTIVE(c:d)) == 0
    result2 = 1;
else result2 = 0;
end
% Now output this result to OFF matrix in Off_Result.mat
OFF(n+1,5) = result2;
ResultOutput2 = [root,'Off_Result.mat'];
eval(['save ', ResultOutput2, ' OFF']);
end
clear all
Appendix 6.1. Approval letter from Beaumont Hospital Ethics (Medical Research) Committee

Ethics (Medical Research) Committee - Beaumont Hospital
Notification of ERC/IRB Approval

Investigator: Ms. Ailish McDermott (Physiotherapist)
REC reference: 08/52
Protocol Title: Gait Impairment in cervical spondylotic myelopathy: Analysis, impact on function, and effect of surgical intervention

Ethics Committee Meeting date: 27th June 2008
Final Approval Date: 7th August 2008
From: Ethics (Medical Research) Committee - Beaumont Hospital, Beaumont, Dublin 9

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Professor Alice Stanton
ERC/IRB – Convenor’s Signature
Approval # 8, dated 22nd September 2011*
Following your recent consultation with Professor Ciaran Bolger, consultant neurosurgeon at Beaumont Hospital, I would like to inform you about a research project currently taking place in collaboration with Professor Bolger and the Royal College of Surgeons in Ireland.

The aim of the project is to examine walking patterns in people who are experiencing symptoms related to “wear and tear” in the neck which causes pressure on the spinal cord. (The medical term for this is “cervical spondylotic myelopathy”). These symptoms may include clumsiness or awkwardness of the hands, changes in feeling in the hands or feet, or inability to walk as far or with the same ease as normal. If you are experiencing any of these symptoms, and if you are currently awaiting surgery to your neck as a result, it is likely that you will be eligible to take part in this project.

So what would taking part involve? In brief, a detailed assessment of your walking pattern would be conducted using a VICON™ motion analysis system. This would take place in the Physiotherapy Movement Laboratory in the Royal College of Surgeons in Ireland, St. Stephen’s Green, Dublin 2. Using this technology, we can examine your walking pattern in detail, measuring many aspects such as your walking speed, the position of your feet as you walk, the forces going through each of your joints, the movement available at your joints, and the activity in your muscles. The assessment is painless and involves simply attaching some markers to your legs, which the computer then records as you walk along a walkway. The protocol would require you to attend for [one / two] initial assessments – [less than a week apart from one another] – and then a [NUMBER] assessment six months after your neck surgery and a [NUMBER] assessment twelve months after your surgery to evaluate any changes in your walking pattern as a result of the surgery. The results of your assessments would all be provided to you on a CD, which we hope would be of interest and benefit to you and to the healthcare professionals involved in your care.
At present, there is very little research on why the symptoms relating to this condition develop, or on how they change over time. We hope that, by recruiting as many people as possible to this project, we will improve knowledge and understanding of the condition among healthcare professionals, so that patients will get the best possible care. The project is funded by the Health Research Board (www.hrb.ie).

I wish to emphasise that participation in this research project is purely voluntary, and you are under no obligation to take part. If you decide to take part, you have the right to withdraw from the study at any stage if you no longer wish to continue your participation. Your care at Beaumont Hospital will not be affected in any way.

If you think you may be interested in taking part in this study, or if you would like further information, I would be delighted to hear from you. Please do not hesitate to contact me by telephone at 085-8336094 or 01-8092526, or by e-mail to ailishmcdermott@rcsi.ie.

Alternatively, please complete the response slip and send it to me in the stamped addressed envelope enclosed.

I look forward to hearing from you.

Yours sincerely,

__________________________________
Ailish McDermott BSc (Physio), MISCP
Senior Physiotherapist, Neurosurgery
HRB Clinical Research Fellow
Beaumont Hospital
Dublin 9.
# Participant Information Leaflet

**Protocol Title:**

| Gait Impairment in Cervical Spondylotic Myelopathy: Analysis, Impact on Function, and Effect of Surgical Intervention |

**Principal Investigator’s Name:** Ailish McDermott  
**Principal Investigator’s Title:** Senior Physiotherapist  
**Telephone No. of Principal Investigator:** 018092526 / 085 8336094

You are being invited to take part in a clinical research study carried out at Beaumont Hospital and the Royal College of Surgeons in Ireland. Before you decide whether or not you wish to take part, you should read the information provided below carefully and if you wish discuss it with your family, friends or GP. Take time to ask questions – do not feel rushed or under any obligation to participate. You should clearly understand the risks and benefits of participating in this study so that you can make a decision that is right for you – this process is known as Informed Consent.

You are not obliged to take part in this study and failure to participate will have no effect on your future care. You may change your mind at any time (before the start of the study or even after you have commenced the study) for whatever reason without having to justify your decision and without any negative impact on your care.

**WHY IS THIS STUDY BEING DONE?**

This study is being done because we know from experience and previous research that patients who suffer from compression of the spinal cord in their neck as a result of “wear and tear” (the condition called “cervical spondylotic myelopathy” in the title of the study) experience difficulties with walking. They may feel unsteady, the legs may feel stiff or weak, or they may not be able to walk as far or with the same ease as before. We hope that, by examining in more detail how patients with this condition walk, doctors and physiotherapists will better understand why these problems come about. We also hope it will help us to understand how walking patterns may change after having surgery. Finally, this study will help physiotherapists and doctors plan how best to treat patients with this condition after surgery.

**WHO IS ORGANISING AND FUNDING THIS STUDY?**

The Principal Investigator of this study is Ailish McDermott, Senior Physiotherapist. The Health Research Board has provided funding for the study to Ailish McDermott, who has registered for a PhD degree with the Royal College of Surgeons in Ireland.
HOW WILL IT BE CARRIED OUT?
This research study commenced in October 2008. It will last for three years. Patients who have symptoms like unsteady walking or clumsy hands, and who have been told by their surgeon that they have spinal cord compression due to degenerative changes or “wear and tear” of the joints in the neck, will be asked to participate in the study. Participants will have an analysis of their walking conducted in the Human Movement Laboratory in the Physiotherapy Department of the Royal College of Surgeons in Ireland, 123 St. Stephen’s Green, Dublin 2.

WHAT WILL HAPPEN TO ME IF I AGREE TO TAKE PART?
You will be given appointments to attend the Movement Laboratory in the Royal College of Surgeons in Ireland (RCSI), where an analysis of your walking will be carried out. If you are going to have surgery on your neck, the researcher will try to do two analyses of your walking about a month or less before you have your operation. The assessment in RCSI will take about two hours. Measurements will be taken of your height, weight, and the width of the joints in your leg. The range of movement and “stiffness” of the muscles and joints in your legs will be measured. You will have small electrodes (about 2cm square) attached to four muscles in your leg, and you will have markers placed on the joints of your leg. This is not painful, they are simply attached to your skin, though we will need to shave a small area of hair to allow the electrodes to stick properly. You will then be asked to walk along a 10 metre walkway a number of times while your walking pattern is recorded by the an infrared camera system (VICON). Next, you will be asked to walk a small flight of stairs if you are able to do this. Finally, you will be given a questionnaire asking you about your health. This is called the “Short Form 36”. You may already have completed this questionnaire in the clinic in Beaumont within the past month. If so, we will use the results from the clinic and not ask you to complete it again. We may need you to come back to have a second, shorter assessment (about two hours), one week or less after the first assessment. This is required to ensure that the recordings from the infrared camera system are reliable, and are not greatly altered by slight variations that may occur between visits. It is important to ensure that the research is of high quality. We will then call you back for your third assessment about six months after you have had your operation, and for a fourth and final assessment about one year after your operation. These assessments will be exactly like the first one.

BENEFITS:
Taking part in the study will allow the Principal Investigator to gather detailed information on your walking pattern. The results of the assessments will all be explained to you. If you wish, we can also share this information with your doctor, physiotherapist, or other healthcare professional, to improve their understanding of your condition, or to help them decide which treatment is of most benefit to you. If you would like your own copy of the results, we will give them to you on a CD. Your participation will benefit others in the future with the same condition as you, because health care professionals will improve their knowledge of cervical spondylotic myelopathy, and the treatment that might be needed for it.

RISKS:
There are three potential risks in the study:

1. You may have a skin reaction to the sticky electrodes. The risk of this is low because the adhesive on the electrodes is hypoallergenic, however please inform the researcher if you have sensitive skin, or if you have had a reaction to something similar in the past. If this happens, the reaction is likely to be small, with some redness and itchiness of the skin where the electrode was placed. We will ask your doctor in Beaumont to take a look at the skin and give you some cream if needed.

2. In order to gather sufficiently accurate information, we may require you to walk along the walking track as many as ten times, which will mean you will have walked about 100 metres. If your muscles are weak, you may become tired. If this happens, please tell the Principal Investigator who will ensure you are given enough rest between walks.
3. If your walking is unsteady, and you have had recent falls or need a walking aid or stick, there is a risk that you might fall during the test. This risk is no greater than your risk of falling while walking in your usual environment because the sensing equipment does not interfere with the way you walk. Please tell the Principal Investigator if you have had falls recently, if you need a walking aid or stick, or if you need someone to walk beside you. Remember, you can walk as slowly as you like during the test and take rest breaks between walks on the 10 metre track.

WILL THERE BE ANY ADDITIONAL COSTS INVOLVED?
If you incur travel expenses to come to your appointment in the Royal College of Surgeons, please give your receipt to the Principal Investigator who will organise a refund.

YOUR RESPONSIBILITIES AS A PARTICIPANT
9. To attend for assessment of your walking pattern at the appointment times given, or to give the researcher adequate notice, i.e. more than 24 hours, if you need to change the appointment.
10. To inform the researcher of any factors which may affect your ability to walk for the required length of time e.g. new medical conditions such as angina, a chest infection causing you to be breathless, or an injury to your leg.
11. If you are a woman of child-bearing age, to inform the researcher if you are pregnant.
12. To inform the researcher if you are currently on medication, or if you are currently having physiotherapy, or any other treatment for your condition.

OUR RESPONSIBILITIES TO YOU AS INVESTIGATORS
We will ensure that our assessment of your walking pattern is carried out safely, accurately, and following all protocols and procedures to minimise the risk to you, the participant, and to ensure that the quality of information we provide to you afterwards is of the highest possible quality. We will ensure that the results of your walking test are made available to you and any health care professionals involved in your care, if you wish. If you would like to know the results of the study when it is finished, we will organise a CD copy for you.

CONFIDENTIALITY ISSUES
When we take records of your assessment in the laboratory, we will not keep your name or any other details with this data. Instead, you will be given a “unique identifier” which will be coded on a secure hard drive and available only to the Principal Investigator. The document with this coded information will be destroyed as soon as the study is completed. The data from the laboratory, with details of your walking assessment, will be kept for a period of five years after the study is completed, because it may be included in future studies. If you wish to consent to this study only, and do not wish to consent to have your data included in possible future studies, please inform the researcher who will ensure your data are deleted after this study.
We will contact your GP to advise them of your participation in this study with your consent. If you wish for us to provide a copy of the results of your walking test to your GP, we will provide these with your consent.
We may also need to contact your physiotherapist to find out how many physiotherapy treatment sessions you have had, again with your consent. We will provide a copy of the results of your walking test to your physiotherapist if you would like us to do so.
You may refuse, however, to have your GP or physiotherapist contacted, and this will not affect your participation in the study or your future care.
The Principal Investigator may need to access your medical chart for details of your previous history and your treatment for your current condition.
IF YOU REQUIRE FURTHER INFORMATION
If you have any further questions about the study, now or any future time, please contact
the Principal Investigator:
Ailish McDermott,
Senior Physiotherapist,
Beaumont Hospital,
Dublin 9.
Phone: 01-8092526 / 085-8336094.

If you wish to withdraw from the study you may do so at any time without justifying your decision and your future treatment will not be affected.
Appendix 6.4: Consent form for CSM participants

CONSENT FORM

Protocol Title:


Please tick the appropriate answer.

I confirm that I have read and understood the Patient Information Leaflet dated 23/02/2010 attached, and that I have had ample opportunity to ask questions all of which have been satisfactorily answered. Yes  No

I understand that my participation in this study is entirely voluntary and that I may withdraw at any time, without giving reason, and without this decision affecting my future treatment or medical care. Yes  No

I understand that my records may be viewed by individuals with delegated authority from Ailish McDermott, principal investigator. Yes  No

I understand that my identity will remain confidential at all times. Yes  No

I am aware of the potential risks of this research study. Yes  No

I have been given a copy of the Patient Information Leaflet and this Consent form for my records. Yes  No

I understand that the principal investigator, Ailish McDermott, may contact my General Practitioner to inform them of my participation in this study and my results. Yes  No

I understand that the results of this study may be shared with my Chartered Physiotherapist to assist with my physiotherapy treatment. Yes  No

I understand that the principal investigator, Ailish McDermott, may contact my Chartered Physiotherapist regarding my treatment to date. Yes  No
FUTURE USE OF ANONYMOUS DATA:
I agree that I will not restrict the use to which the results of this study may be put. I give my approval that unidentifiable data concerning my person may be stored or electronically processed for the purpose of scientific research and may be used in related or other studies in the future. (This would be subject to approval by an independent body, which safeguards the welfare and rights of people in biomedical research studies - the Beaumont Hospital Ethics (Medical Research) Committee).

Yes  No

Participant _____________________________
Signature                          Date                 Name in block capitals

Witness _____________________________
Signature                          Date                 Name in block capitals

To be completed by the Principal Investigator or his nominee.

I the undersigned, have taken the time to fully explained to the above participant the nature and purpose of this study in a manner that he/she could understand. I have explained the risks involved, the experimental nature of the treatment, as well as the possible benefits and have invited him/her to ask questions on any aspect of the study that concerned them.

Signature: _____________________________
Name in Block Capitals: _____________________________
Qualification: _____________________________
Date: _____________________________

3 copies to be made: 1 for patient, 1 for PI and 1 for hospital records.
Dr. ........
Address

Re: Participant’s Name and address

Dear Dr. ,

I am conducting a study to identify the main problems with mobility and gait in cervical spondylotic myelopathy.

(Participant’s name) has been recruited to the study. The details are as follows:

Title of the study:

Co-Investigators
Prof. Ciaran Bolger, consultant neurosurgeon, Beaumont Hospital
Ms. Dara Meldrum, lecturer in physiotherapy, Royal College of Surgeons in Ireland
Ms. Louise Keating, lecturer in physiotherapy, Royal College of Surgeons in Ireland.

This study involves (participant's name) undergoing gait analysis in the Human Movement Laboratory, RCSI School of Physiotherapy, prior to undergoing surgery for cervical spondylotic myelopathy. The analysis will be repeated at six months post-surgery to evaluate change. (Participant's name) will also be assessed on a simple physiotherapy score of mobility, the Modified Ashworth Scale for measurement of spasticity, and a quality of life questionnaire (Medical Outcomes Study Short Form 36). The results of the gait analysis will be collated with these measures to evaluate the effect of gait impairment on quality of life and functional mobility.

Should you have any queries, I can be contacted at 01-8092526.

Yours sincerely,

________________________________
Ailish McDermott BSc, MISCP
Senior Physiotherapist, Neurosurgery
HRB Clinical Research Fellow
Beaumont Hospital
Appendix 6.6: Data collection sheet for CSM participants

DATA COLLECTION SHEET
Gait Impairment in Cervical Spondylotic Myelopathy: Analysis, Impact on Function, and Effect of Surgical Intervention

Subject Unique Identifier

Date of Assessment

Assessment Number

Section 1. Background Data.
1.1 Age at time of assessment
1.2 Pre-op duration of symptoms
1.3 Date of surgery
1.4 Surgical Approach Anterior Posterior
1.5 Level of Surgery
1.6 Details of Surgery
1.7 Current Symptoms
Section 2. Screening Questions.

2.1 Past medical history

2.2 Current Medications

2.3 Level of mobility outdoors

2.4 Number of falls in past six months: Stumbles / near misses:

2.5 Participant's own copy of gait results on CD?

2.6 Previous skin reactions to electrodes or similar adhesive material.

2.7 Intensity of physiotherapy treatment post surgery

<table>
<thead>
<tr>
<th>INPATIENT number of sessions</th>
<th>Average time per session</th>
</tr>
</thead>
<tbody>
<tr>
<td>OUTPATIENT number of sessions</td>
<td>Average time per session</td>
</tr>
<tr>
<td><strong>TOTAL THERAPY TIME (hours)</strong></td>
<td></td>
</tr>
</tbody>
</table>

Additional Information
Section 3. Severity of Myelopathy.

3.1. Nurick Score.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Signs or symptoms of root involvement, but without evidence of spinal cord disease.</td>
</tr>
<tr>
<td>1</td>
<td>Signs of spinal cord disease but no difficulty in walking.</td>
</tr>
<tr>
<td>2</td>
<td>Slight difficulty in walking, which did not prevent full-time employment.</td>
</tr>
<tr>
<td>3</td>
<td>Difficulty in walking which prevented full time employment, or the ability to do all housework, but which was not so severe as to require someone’s help to walk.</td>
</tr>
<tr>
<td>4</td>
<td>Able to walk only with someone else’s help or with the aid of a frame.</td>
</tr>
<tr>
<td>5</td>
<td>Chairbound or bedridden.</td>
</tr>
</tbody>
</table>

3.2. Modified Japanese Orthopaedic Association Score

<table>
<thead>
<tr>
<th>Subsection</th>
<th>Description</th>
<th>Score</th>
<th>Score Given</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor dysfunction of the upper extremity.</td>
<td>Unable to feed oneself.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Unable to use knife and fork; able to eat with spoon.</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Able to use knife and fork with much difficulty.</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Able to use knife and fork with some difficulty.</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No dysfunction.</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Motor dysfunction of the lower extremity.</td>
<td>Unable to walk.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Can walk on flat floor with walking aid.</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Can walk up and / or down stairs with handrail.</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lack of stability and smooth gait.</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No dysfunction.</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Sensory Deficit: Upper Limb.</td>
<td>Severe sensory loss or pain.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Mild sensory loss.</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No dysfunction.</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Sensory Deficit: Lower Limb.</td>
<td>Severe sensory loss or pain.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Mild sensory loss.</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No dysfunction.</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Sensory Deficit: Trunk.</td>
<td>Severe sensory loss or pain.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Mild sensory loss.</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No dysfunction.</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Sphincter dysfunction.</td>
<td>Unable to void.</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Marked difficulty in micturition (retention).</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Difficulty in micturition (frequency, hesitation).</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No dysfunction.</td>
<td>3</td>
<td></td>
</tr>
</tbody>
</table>
### Section 4. Functional Mobility.

#### 4.1. Modified Rivermead Mobility Index (MRMI)

<table>
<thead>
<tr>
<th>Item</th>
<th>Task</th>
<th>Score</th>
</tr>
</thead>
</table>
| 1    | Turning Over.  
Please turn over from your back to your side. | R  | L |
| 2    | Lying to Sitting.  
Please sit up on the edge of the bed. |   |   |
| 3    | Sitting Balance.  
Please sit on the edge of the bed (10 seconds). |   |   |
| 4    | Sitting to Standing.  
Please stand up from your chair (<15 seconds). |   |   |
| 5    | Standing.  
Please remain standing (10 seconds). |   |   |
| 6    | Transfers.  
Please go from the plinth to the chair and back again. | R  | L |
| 7    | Walking Indoors.  
Please walk 10 metres in your usual way. |   |   |
| 8    | Stairs.  
Please climb up and down this flight of stairs in your usual way. | R  | L |
|      | TOTAL |   |   |

### SCORING SYSTEM

- 0  Unable to perform
- 1  Assistance of two people
- 2  Assistance of one person
- 3  Requires supervision or verbal instruction
- 4  Requires an aid or an appliance
- 5  Independent
Section 5.  Spasticity

5.2  Ashworth Scale

<table>
<thead>
<tr>
<th>Muscle Group</th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rectus femoris</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Biceps femoris</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adductor magnus / longus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gastroc / soleus</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**SCORING SYSTEM**

0 = No increase in muscle tone.
1 = Slight resistance, i.e. catch and release OR minimal resistance at end of ROM.
1+ = Slight resistance, i.e. catch and release AND minimal resistance throughout remainder (less than half) of ROM.
2 = More marked resistance throughout range, but affected part easily moved.
3 = Considerable increase in tone, passive movement difficult
4 = Affected part rigid in flexion or extension.

Section 6.  Quality of Life.

6.1.  Medical Outcomes Study Short Form 36 (SF-36) Score.

<table>
<thead>
<tr>
<th>Component</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical functioning</td>
<td></td>
</tr>
<tr>
<td>Role functioning - physical</td>
<td></td>
</tr>
<tr>
<td>Bodily pain</td>
<td></td>
</tr>
<tr>
<td>General health</td>
<td></td>
</tr>
<tr>
<td>Vitality</td>
<td></td>
</tr>
<tr>
<td>Social functioning</td>
<td></td>
</tr>
<tr>
<td>Role functioning - emotional</td>
<td></td>
</tr>
<tr>
<td>Mental health</td>
<td></td>
</tr>
<tr>
<td>Health transition</td>
<td></td>
</tr>
<tr>
<td>PHYSICAL COMPONENT SUMMARY</td>
<td></td>
</tr>
<tr>
<td>MENTAL COMPONENT SUMMARY</td>
<td></td>
</tr>
</tbody>
</table>
### Section 7. Anthropometric Data.

<table>
<thead>
<tr>
<th>Section</th>
<th>Measurement</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>6.1</td>
<td>Height (cm)</td>
<td></td>
</tr>
<tr>
<td>6.2</td>
<td>Weight (kg)</td>
<td></td>
</tr>
<tr>
<td>6.3</td>
<td>ASIS to ASIS (cm)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Left</td>
</tr>
<tr>
<td>6.4</td>
<td>Leg length (cm)</td>
<td></td>
</tr>
<tr>
<td>6.5</td>
<td>Knee width (cm)</td>
<td>Trial 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Trial 2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Trial 3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>AVERAGE</td>
</tr>
<tr>
<td>6.6</td>
<td>Ankle width (cm)</td>
<td>Trial 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Trial 2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Trial 3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>AVERAGE</td>
</tr>
<tr>
<td>6.7</td>
<td>Tibial torsion (degrees)</td>
<td>Trial 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Trial 2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Trial 3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>AVERAGE</td>
</tr>
</tbody>
</table>
Section 8. Gait Analysis.

8.1. Surface EMG Settings.

<table>
<thead>
<tr>
<th>Section</th>
<th>Channel</th>
<th>Gain (MVIC)</th>
<th>Section</th>
<th>Channel</th>
<th>Gain (CGA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>8.1</td>
<td>1</td>
<td></td>
<td>8.9</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>8.2</td>
<td>2</td>
<td></td>
<td>8.10</td>
<td>2</td>
<td></td>
</tr>
<tr>
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8.2. Trial Records.

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Appendix 6.7: MATLAB function to extract key gait parameters from
a time-normalised average gait cycle

function keygaitparams(p,k,type)
% A function to run through a mat file AvgGaitData(p).(k).mat and
extract key points for statistical analysis
% Created by Ailish McDermott, 19-Jan-2011
% Last modified 19-Jan-2011
% p = patient number (healthy or CSM)
% k = assessment number
% type = whether patient is a healthy control or not (0 if no, 1 if yes)
% Change variables ROOT and FILEPATH if changing from MAC to
Windows or vice versa
% ------------------------- %

root = '/Users/ailish/Documents/MATLAB/Polygon/';
if type == 1
    filepath = [root,'HC/HC',int2str(p),'/Ax',int2str(k)];  %
Filename depends on whether data is from a healthy control or CSM
participant
else filepath = [root,'CSM/',int2str(p),'/Ax',int2str(k)];
end

inputfile =
    [filepath,'/AvgGaitData',int2str(p),'.',int2str(k),'.mat'];
load(inputfile);                % Loads file of interest

% Get kinematic variables %
lpt_avg = mean(lpeltilt);       % Mean pelvic tilt during GC
rpt_avg = mean(rpeltilt);
lpo_rn = max(lpelobl) - min(lpelobl);   % Range pelvic obliquity
rpo_rn = max(rpelobl) - min(rpelobl);
lpr_rn = max(lpelrot) - min(lpelrot);   % Range pelvic rotation
rpr_rn = max(rpelrot) - min(rpelrot);
lhipic = lhipfe(1,1);           % Hip position at IC
rhipic = rhipfe(1,1);
lhipext = min(lhipfe);          % Peak hip extension
rhipext = min(rhipfe);
lhtspe = max(lhipfe) - min(lhipfe);   % Hip total sagittal plane
excursion
rhtspe = max(rhipfe) - min(rhipfe);
lhaa_rn = max(lhipaa) - min(lhipaa);   % Range of hip motion in
frontal plane
rhaa_rn = max(rhipaa) - min(rhipaa);
lhr_rn = max(lhiprot) - min(lhiprot);   % Range of hip motion in
transverse plane
rhr_rn = max(rhiprot) - min(rhiprot);
lkneeic = lkneefe(1,1);         % Knee position at initial contact
rkneeic = rkneefe(1,1);
lkf_st = max(lkneefe(1:15)); % Peak knee flexion in stance
rkf_st = max(rkneefe(1:15));
lkext = min(lkneefe);          % Peak knee extension
rkext = min(rkneefe);
lkf1_sw = max(lkneefe(30:51)); % Peak knee flexion in swing
rkf1_sw = max(rkneefe(30:51));
lktspe = max(lkneefe) - min(lkneefe);   % Knee total sagittal
plane excursion
rktspe = max(rkneefe) - min(rkneefe);
lankic = lankdp(1,1);           % Ankle position at initial
contact
rankic = rankdp(1,1);
ladf_st = max(lankdp(1:30));  % Maximum ankle dorsiflexion in stance
radf_st = max(rankdp(1:30));
laf = min(lankdp);            % Peak ankle plantarflexion
raf = min(rankdp);
ladf_sw = max(lankdp(30:51)); % Peak ankle dorsiflexion in swing
radf_sw = max(rankdp(30:51));
% Now get kinetic parameters %
lgrfx_pk = min(lgrfx);          % Peak medio-lateral GRF (note: L is neg)
rgrfx_pk = max(rgrfx);
lgrfy1 = max(lgrfy);            % Peak braking antero-posterior GRF
rgrfy1 = max(rgrfy);
lgrfy2 = min(lgrfy);            % Peak propulsion AP GRF
rgrfy2 = min(rgrfy);
lgrfv1 = max(lgrfz);            % Peak vertical GRF
rgrfv1 = max(rgrfz);
lhmomf = min(lhipmomfe);        % Peak hip flexor moment
rhmomf = min(rhipmomfe);
lhmome = max(lhipmomfe);        % Peak hip extensor moment
rhmome = max(rhipmomfe);
lhmomab = max(lhipmomaa);       % Peak hip abductor moment
rhmomab = max(rhipmomaa);
lkmomf = min(lkneemom);         % Peak hip flexor moment
rkmomf = min(rkneemom);
lkmome = max(lkneemom);         % Peak knee extensor moment
rkmome = max(rkneemom);
lamomp = max(lankmom);          % Peak ankle plantarflexor moment
ramomp = max(rankmom);
lh1 = max(lhippower(6:20));     % H1 hip power
rh1 = max(rhippower(6:20));
lh2 = min(lhippower(20:35));    % H2 hip power
rh2 = min(rhippower(20:35));
lh3 = max(lhippower(25:51));    % H3 hip power
rh3 = max(rhippower(25:51));
lk1 = min(lkneepower(2:10));    % K1 knee power
rk1 = min(rkneepower(2:10));
lk2 = max(lkneepower(10:30));   % K2 knee power
rk2 = max(rkneepower(10:30));
lk3 = min(lkneepower(30:40));   % K3 knee power
rk3 = min(rkneepower(30:40));
lk4 = min(lkneepower(40:51));   % K4 knee power
rk4 = min(rkneepower(40:51));
lal = min(lankpower(5:25));     % A1 ankle power
ral = min(rankpower(5:25));
lal2 = max(lankpower);          % A2 ankle power
ra2 = max(rankpower);
% Save the output %
outputfile =
[filepath,'/KeyGaitParams',int2str(p),'.',int2str(k),'.mat'];
eval(['save ', outputfile, ' lpt_avg rpt_avg lpo_rn rpo_rn lpr_rn
rpr_rn lhipic rhipic lhipext rhipext lhtspe rhtspe lhaa_rn rhaa_rn
lhr_rn rhr_rn lknecic rknecic lkgf_st rkgf_st lkekt rkekt lklf_sw
rklf_sw lkttspe rkttspe lankic rankic ladjf_st radf_st lapf rapf
ladf_sw radf_sw lgrfx_pk rgrfx_pk lgfyl rgrfy1 lgrfy1 lgrfy2 rgrfy2
lgrfv1 rgrfv1 lhmomf rhmomf lhmome rhmome lhmomab rhmomab lkmomf
rkmomf lkmome rkpmome lamomp ramomp lh1 rh1 lh2 rh2 lh3 rh3 lk1 rk1
lk2 rk2 lk3 rk3 lk4 rk4 lal ral la2 ra2']);
% getemg2 = A script file to extract 8 signals arrays from a defined gait cycle, using Workstation to provide gait cycle on and off times.
% Written by Ailish McDermott; modified 19/08/10
% For use with CSM or healthy control data
% Input: filein = csv file to be read by MATLAB containing all the analog data from a single gait cycle; l1=frame of onset of left gait cycle;
% 12=frame of offset of left gait cycle; r1=frame of onset of right gait cycle; r2=frame of offset of right gait cycle. Frames are based on Workstation time bar which captures at 50 Hz.
% p = subject number
% k = session code (1=preop1,2=preop2, 3=postop 6 months; 4= postop 1 year)
% k for healthy controls: 1 = comfortable speed, 2 = matched speed
% n = trial number (1-10)
% Output: 8 EMG channels.
%--------------------------------------------------

p = input('Please enter participant number ');  
k = input('Please enter assessment number ');  
n = input('Please enter trial number ');  
HC = input('Enter 1 if participant is a healthy control, 0 if not ');  
filein = input('Please enter filepath for reference csv file ');  
l1 = input('Enter frame for start of left gait cycle ');  
l2 = input('Enter frame for end of left gait cycle ');  
r1 = input('Enter frame for start of right gait cycle ');  
r2 = input('Enter frame for end of right gait cycle ');  

[numeric]=xlsread(filein);  % Specify filepath for .csv files containing gait cycle data
% time=numeric(:,1);    % Specify 1st column in numeric matrix to be time, in units of ms
[x1]=find(time==(l1*20));  
x2]=find(time==(l2*20));  % Find matrix references for start and end points of left gait cycle where x=row. *20 translates Workstation frame to EMG units (50 to 1000 Hz).
lrf=numeric(x1:x2,2);   % Define EMG for L rectus femoris as 2nd column, rows defined by GC
lbf=numeric(x1:x2,4);   % Define EMG for L biceps femoris as 4th column, rows x1-x2
lta=numeric(x1:x2,6);   % Define EMG for L tibialis anterior as 6th column, rows as above
lgm=numeric(x1:x2,8);   % Define EMG for L medial gastrocnemius, 8th column, rows as above
[x3]=find(time==(r1*20));  
x4]=find(time==(r2*20));  % Find matrix references for start and end points of right GC
rrf=numeric(x3:x4,3);   % Define EMG for R rectus femoris as 3rd column, rows defined by GC
rbf=numeric(x3:x4,5);   % Define EMG for R biceps femoris as 5th column, rows as above
rta=numeric(x3:x4,7);   % Define EMG for R tibialis anterior as 7th column, rows as above
rmg=numeric(x3:x4,9);   % Define EMG for R medial gastrocnemius as 9th column, rows as above

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if p < 10
    if HC == 0
        path = '08520';
        root = 'EMG0';
    else
        path = 'HC0';
        root = 'HCEMG0';
    end
else
    if HC == 0
        path = '0852';
        root = 'EMG';
    else
        path = 'HC';
        root = 'HCEMG';
    end
end
if n < 10
    stem = '.0';
else
    stem = '.';
end
savefile = ['C:\Documents and Settings\Vicon\My Documents\MATLAB\', path, int2str(p), '\', root, int2str(p), '\.0\', int2str(k), stem, int2str(n), '.mat'];
save(savefile, 'rrf', 'lrf', 'rbf', 'lbf', 'rta', 'lta', 'rmg', 'lmg');
clear all
function ON = emgtiming3(signal)

% A script file to determine muscle activation based on slope and amplitude of the EMG signal after it is modified by TKEO
% Applies the TKEO function to an already filtered signal
% Then smooths with a low-pass Butterworth
% Created by Ailish McDermott, RCSI Movement Lab, 16-Nov-2010
% Modified 18-Nov-2010
% Inputs: signal
% ----------------------- %

sig = tkeo(signal);    % Apply TKEO to filtered gait data
[B,A] = butter(2,0.05,'low'); % Get vectors for dual-pass filter
for smoothing
    sig2 = filtfilt(B,A,sig); % Smooths the signal
    slope = gradient(sig2); % Gets the slope of the smoothed signal
    sig_on = abs(slope) > 1e-6; % Creates logical index where the absolute value of the slope is above a threshold
    th = (max(sig2))*0.03;  % Sets a threshold at 3% maximum value of signal
    sig_on2 = sig2 > th;    % Finds points where sig2 exceeds threshold value

% Now put sig_on and sig_on2 side by side and sum the values
% Create new logical matrix where both sig_on and sig_on2 declare 1, i.e. both slope and amplitude exceed threshold

isiton = [sig_on sig_on2]'; % Creates matrix of both "on" conditions in rows for summing columns
both_on = sum(isiton); % Sums the columns of the 2 row vectors
timeon = both_on'; % Transposes the resulting vector back to a column instead of a row
changetime = timeon == 2; % Creates a new logical vector where both conditions are satisfied for slope and threshold

% Now see if both conditions are satisfied for at least 24 of 25 consecutive samples

TIME = [changetime(end-12:end); changetime; changetime(1:12)];
% Appends the last 12 values to the beginning and first 12 values to end to create new vector TIME

T = length(changetime); % Gets the number of data points for new matrix containing 25 consecutive ON values
R = zeros(T,1); % Initialises a matrix that will contain logical values of TIME
for n = 1:T
    R(n) = sum(TIME(n:n+24)); % Sums each consecutive set of 25 data points
end
ON = R > 23; % If 24 of 25 consecutive points are satisfying the on condition, then consider muscle to be on (allows for blips in middle of contraction)
T = 1:T;
[AX,H1,H2] = plotyy(T,sig,T,ON); % Plots signal and ON values on 2 y axes
set(AX(2),'ylim',[0 1.5]); % Sets limits of second y axis
hold on % Holds the graph
plot(sig2,'r') % Plots the smoothed signal for comparison (in red)
saveas(gcf,'muscleplot.fig');
hold off
% Now find where the changes occur at the beginning and end of
% each "on" period
% Sum each pair of consecutive values. If muscle is continuously
OFF then sum = 0; if continuously ON then sum = 2; if changing
then sum = 1
R2 = [ON(end); ON; ON(1)]; % Creates R2 by appending start and
end values of ON
for n = 1:length(ON)
    Alert(n) = sum(R2(n:n+1)); % Adds consecutive variables
end
CHANGE = Alert == 1;
Idx = find(CHANGE == 1); % Find indices where change is
signalled
percent = (Idx/length(ON))*100; % Gets percentage points in GC
that changes occur
save Idx ON percent
end
Appendix 6.10: MATLAB function for calculation of a signal’s Root Mean Square amplitude

%% DECLARATIONS AND INITIALIZATIONS

% Calculates windowed (over- and non-overlapping) RMS of a signal using the specified windowlength y = rms(signal, windowlength, overlap, zeropad)
% signal is a 1-D vector
% windowlength is an integer length of the RMS window in samples
% overlap is the number of samples to overlap adjacent windows (enter 0 to use non-overlapping windows)
% zeropad is a flag for zero padding the end of your data...(0 for NO, 1 for YES)
% ex. y=rms(mysignal, 30, 10, 1). Calculate RMS with window of length 30 samples, overlapped by 10 samples each, and zeropad the last window if necessary
% ex. y=rms(mysignal, 30, 0, 0). Calculate RMS with window of length 30 samples, no overlapping samples, and do not zeropad the last window
% Author: A. Bolu Ajiboye

function y = rms(signal, windowlength, overlap, zeropad)

delta = windowlength - overlap;

% CALCULATE RMS

indices = 1:delta:length(signal);
% Zeropad signal
if length(signal) - indices(end) + 1 < windowlength
    if zeropad
        signal(end+1:indices(end)+windowlength-1) = 0;
    else
        indices = indices(1:find(indices+windowlength-1 <= length(signal), 1, 'last'));
    end
end

y = zeros(1, length(indices));
% Square the samples
signal = signal.^2;

index = 0;
for i = indices
    index = index+1;
    % Average and take the square root of each window
    y(index) = sqrt(mean(signal(i:i+windowlength -1)));
end

Appendix 6.11: Equation to calculate muscle lengthening velocity and lengthening velocity threshold

The length of any given muscle, \( l_m \), at time \( t \) is expressed as:

\[
\frac{l_m(t)}{l_0} = 1 + a\theta_1(t) + b\theta_1^2(t) + c\theta_2(t) + d\theta_2^2(t) + e\theta_2^3(t) + f\theta_2^3(t)
\]  
(Eq. 1)

where \( l_m \) is the length change (shortening) of the muscle (origin to insertion), \( l_0 \) is the resting fibre length of the muscle when the body is in the anatomical position and all angles, \( \theta \), = 0, \( \theta_1 \) is the joint angle for a single joint muscle, in degrees, with flexor being positive, \( \theta_2 \) is the joint angle for the adjacent joint for a biarticulate muscle with flexor also being positive, and \( a–f \) are coefficients determined by the curve fit calculated by Winter and Scott (1991) and provided in Table 1.

When all joint angles \( \theta = 0^\circ \), \( l_m/l_0 = 1 \)

If a parallel fibre muscle is being analysed, the velocity of the muscle fibres \( v_f \) at a given time \( t \) in \( l_0/s \) is the same as the velocity of the length of the muscle:

\[
v_f(t) = \frac{d}{dt}\left(\frac{l_m(t)}{l_0}\right)
\]  
(Eq. 2)

where \( d/dt \) is the change in length of the muscle with respect to time, i.e. the first derivative

For a pennate muscle, the angle of pennation will change with respect to time as a result of the length changes calculated in Eq. 1. The muscle volume remains constant, therefore the pennation angle \( \alpha_p \) will change with time as follows:

\[
\alpha_p(t) = \tan^{-1}\left(\frac{\sin\alpha_0}{\cos\alpha_0 - (1 - \frac{l_m(t)}{l_0})}\right)
\]  
(Eq. 3)

where \( \alpha_0 \) is the angle of pennation of the muscle at resting length, provided in Table 1.

The active length of the muscle in \( l_0 \) is \( l_f(t) \):

\[
l_f(t)/l_0 = \sin\alpha_0/\sin\alpha_p(t)
\]  
(Eq. 4)

The velocity of the muscle fibres in \( l_0/s \) is:

\[
v_f(t) = \frac{d}{dt}(l_f(t)/l_0)
\]  
(Eq. 5)
Table 1. Coefficients relating muscle length change with joint angle

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<tr>
<th>Muscle</th>
<th>$a$</th>
<th>$b$</th>
<th>$c$</th>
<th>$d$</th>
<th>$e$</th>
<th>$f$</th>
<th>$\alpha_0$</th>
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<td>$-6.07 \times 10^{-3}$</td>
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<tr>
<td>MG</td>
<td>$1.22 \times 10^{-2}$</td>
<td>$-4.25 \times 10^{-5}$</td>
<td>$-6.12 \times 10^{-7}$</td>
<td>$-6.75 \times 10^{-3}$</td>
<td>$-9.16 \times 10^{-6}$</td>
<td>$-8.48 \times 10^{-8}$</td>
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<tr>
<td>RF</td>
<td>$1.63 \times 10^{-2}$</td>
<td>$-1.75 \times 10^{-5}$</td>
<td>$-3.5 \times 10^{-7}$</td>
<td>$-1.16 \times 10^{-2}$</td>
<td>$-6.06 \times 10^{-5}$</td>
<td>$6.36 \times 10^{-7}$</td>
<td>15</td>
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<tr>
<td>BF</td>
<td>$7.3 \times 10^{-3}$</td>
<td>$1.29 \times 10^{-4}$</td>
<td>$-8.52 \times 10^{-7}$</td>
<td>$-1.93 \times 10^{-3}$</td>
<td>$-9.26 \times 10^{-6}$</td>
<td>$1.15 \times 10^{-7}$</td>
<td>0</td>
</tr>
</tbody>
</table>

$\alpha_0$ = pennation angle at resting length, TA = tibialis anterior, MG = medial gastrocnemius, RF = rectus femoris, BF = biceps femoris, $a$, $b$, $c$ = coefficients applied to the hip angle (BF and RF) and ankle angle (MG and TA), $d$, $e$, $f$ = coefficients applied to knee angle (BF, RF, MG)

Source

Appendix 6.12: MATLAB function to calculate locomotor specific measure of spasticity

function lengthening_velocity_locomotor_spasticity(p,k,n,hc,q)
% A function to determine the lengthening velocities, lengthening velocity thresholds for EMG activation and slope of relationship between lengthening velocity and EMG for 8 lower limb muscles
% Inputs: p = patient number, k = assessment number, n = trial number, hc = healthy control (1 = yes), q = reliability assessment not conducted (1 = true)
% ------------------
% DEFINE FILES TO BE LOADED
if hc == 0
    if q == 1
        polygonfile = 
['/Users/ailish/Documents/MATLAB/Polygon/CSM/',int2str(p),'/Ax',int2str(k+1),'/Trial',int2str(n),'.mat'];
        else polygonfile = 
['/Users/ailish/Documents/MATLAB/Polygon/CSM/',int2str(p),'/Ax',int2str(k),'/Trial',int2str(n),'.mat'];
    end
    rmsfile = 
['/Users/ailish/Documents/MATLAB/RMS/RMS_Normalised/EMG_RMS_Normal',int2str(p),'.',int2str(k),'.',int2str(n),'.mat'];
    timingfile1 = 
['/Users/ailish/Documents/MATLAB/TimingTests/LRF',int2str(p),'.',int2str(k),'.',int2str(n),'.mat'];
    timingfile2 = 
['/Users/ailish/Documents/MATLAB/TimingTests/RRF',int2str(p),'.',int2str(k),'.',int2str(n),'.mat'];
    timingfile3 = 
['/Users/ailish/Documents/MATLAB/TimingTests/LBF',int2str(p),'.',int2str(k),'.',int2str(n),'.mat'];
    timingfile4 = 
['/Users/ailish/Documents/MATLAB/TimingTests/RBF',int2str(p),'.',int2str(k),'.',int2str(n),'.mat'];
    timingfile5 = 
['/Users/ailish/Documents/MATLAB/TimingTests/LTA',int2str(p),'.',int2str(k),'.',int2str(n),'.mat'];
    timingfile6 = 
['/Users/ailish/Documents/MATLAB/TimingTests/RTA',int2str(p),'.',int2str(k),'.',int2str(n),'.mat'];
    timingfile7 = 
['/Users/ailish/Documents/MATLAB/TimingTests/LMG',int2str(p),'.',int2str(k),'.',int2str(n),'.mat'];
    timingfile8 = 
['/Users/ailish/Documents/MATLAB/TimingTests/RMG',int2str(p),'.',int2str(k),'.',int2str(n),'.mat'];
    else polygonfile = 
['/Users/ailish/Documents/MATLAB/Polygon/HC/HC',int2str(p),'/Ax',int2str(k),'/Trial',int2str(n),'.mat'];
    rmsfile = 
['/Users/ailish/Documents/MATLAB/RMS/RMS_Normalised/HCEMG_RMS_Normal',int2str(p),'.',int2str(k),'.',int2str(n),'.mat'];
    timingfile1 = 
['/Users/ailish/Documents/MATLAB/TimingTests/HCLR',int2str(p),'.',int2str(k),'.',int2str(n),'.mat'];
% ending
timingfile2 = ['/Users/ailish/Documents/MATLAB/TimingTests/HCRRF',int2str(p),'.','int2str(k),'.','int2str(n),'.mat'];
timingfile3 = ['/Users/ailish/Documents/MATLAB/TimingTests/HCLBF',int2str(p),'.','int2str(k),'.','int2str(n),'.mat'];
timingfile4 = ['/Users/ailish/Documents/MATLAB/TimingTests/HCRBF',int2str(p),'.','int2str(k),'.','int2str(n),'.mat'];
timingfile5 = ['/Users/ailish/Documents/MATLAB/TimingTests/HCLTA',int2str(p),'.','int2str(k),'.','int2str(n),'.mat'];
timingfile6 = ['/Users/ailish/Documents/MATLAB/TimingTests/HCRTA',int2str(p),'.','int2str(k),'.','int2str(n),'.mat'];
timingfile7 = ['/Users/ailish/Documents/MATLAB/TimingTests/HCLMG',int2str(p),'.','int2str(k),'.','int2str(n),'.mat'];
timingfile8 = ['/Users/ailish/Documents/MATLAB/TimingTests/HCRMG',int2str(p),'.','int2str(k),'.','int2str(n),'.mat'];
end

load(polygonfile);

rfo = round(RFO);  % RFO = right opposite foot off
lfo = round(LFO);  % LFO = left opposite foot off
load(rmsfile);  % rmsfile contains root mean square amplitude

% INDIVIDUAL MUSCLE CALCULATIONS
% Left rectus femoris

load(timingfile1);

a = 1.63e-2;  % Constants from Winter & Scott (1991)
b = -1.75e-5;
c = -4.5e-7;
d = -1.16e-2;
e = -6.06e-5;
f = 6.36e-7;
pennation_angle = 15;
t = linspace(0,LStrTime,51);
time = t';
lrf_length = 1 + (a.*(LHipFE) + (b.*(LHipFE.^2)) + (c.*(LHipFE.^3))
+ (d.*(LKneeFE) + (e.*(LKneeFE.^2)) + (f.*(LKneeFE.^3)));
pennation_angle_wrt_time = 
atan(d(sind(pennation_angle)./((cosd(pennation_angle)) - (1 -
(lrf_length)))));
lrf_active_length = 
sind(pennation_angle)./sind(pennation_angle_wrt_time);
lrf_lengthening_velocity = 
interp1(lrf_lengthening_velocity1,lrf_lengthening_velocity1,xi);  % OUTPUT VARIABLE
lrf_slope_time = find(lrf_lengthening_velocity > 0);

percent = [0 percent 100];
else
percent_round = round(percent);
try
    lrf_threshold_options = find(percent_round >=
        min(lrf_slope_time));
    if mod(lrf_threshold_options(1),2) == 1     % Ensures that
        first indicator is an “on” value not an “off” value
        lrf_critical_time =
        percent_round(min(lrf_threshold_options));
    else lrf_critical_time =
        percent_round(lrf_threshold_options(2));
    end
    lrf_lengthening_velocity_threshold =
    lrf_lengthening_velocity(lrf_critical_time);   % Output variable
catch
    lrf_critical_time = NaN;
    lrf_lengthening_velocity_threshold = NaN;
end
X =
    lrf_lengthening_velocity(min(lrf_slope_time):max(lrf_slope_time));
Y = lrf_rms_norm(min(lrf_slope_time):max(lrf_slope_time));
p_slope = polyfit(X,Y,1);
lrf_response_slope = p_slope(1);
%-----------------------%
% Right rectus femoris
% load(timingfile2);
t = linspace(0,RStrTime,51);
time = t';
rrf_length = 1 + (a.*RHipFE) + (b.*(RHipFE.^2)) + (c.*(RHipFE.^3))
    + (d.*RKneeFE) + (e.*(RKneeFE.^2)) + (f.*(RKneeFE.^3));
pennation_angle_wrt_time =
    atand(sind(pennation_angle)./((cosd(pennation_angle)) - (1 -
    (rrf_length))));
rrf_active_length =
    sind(pennation_angle)./sind(pennation_angle_wrt_time);
rrf_lengthening_velociti ty1 =
    interp1(1:length(rrf_lengthening_velocity1),rrf_lengthening_velocity1,xi);   % OUTPUT VARIABLE
rrf_slope_time = find(rrf_lengthening_velocity > 0);
if ON(Idx(1)) == 0
    percent = [0 percent 100];
else
end
percent_round = round(percent);
try
    rrf_threshold_options = find(percent_round >=
        min(rrf_slope_time));
    if mod(rrf_threshold_options(1),2) == 1     % Ensures that
        first indicator is an on value not an off value
        rrf_critical_time =
        percent_round(min(rrf_threshold_options));
    else rrf_critical_time =
        percent_round(rrf_threshold_options(2));
    end
    rrf_lengthening_velocity_threshold =
load(timingfile3);
a = 7.3e-3;
b = 1.29e-4;
c = -8.52e-7;
d = -1.93e-3;
e = -9.26e-6;
f = 1.15e-7;
t = linspace(0,LStrTime,51);
time = t';
lbf_length = 1 + (a.*LHipFE) + (b.*(LHipFE.^2)) + (c.*(LHipFE.^3))
+ (d.*LKneeFE) + (e.*(LKneeFE.^2)) + (f.*(LKneeFE.^3));
lbf_active_length = lbf_length;
lbf_lengthening_velocity1 = diff(lbf_length)./diff(time);
% Interpolate lbf_lengthening_velocity to 101 data points for
% comparison
% with EMG
xi = 1:(length(lbf_lengthening_velocity1)-1)/(101-1):length(lbf_lengthening_velocity1);
lbf_lengthening_velocity = interp1(1:length(lbf_lengthening_velocity1),
lbf_lengthening_velocity1,xi);   % OUTPUT VARIABLE
lbf_slope_time = find(lbf_lengthening_velocity > 0);
for w = 1:length(lbf_slope_time)
    if lbf_slope_time(w) < lfo
        lbf_slope_time(w) = NaN;
    end
end
if ON(Idx(1)) == 0
    percent = [0 percent 100];
else
    percent_round = round(percent);
try
    lbf_threshold_options = find(percent_round >=
min(lbf_slope_time));
    if mod(lbf_threshold_options(1),2) == 1     % Ensures that
first indicator is an on value not an off value
        lbf_critical_time = percent_round(min(lbf_threshold_options));
    else lbf_critical_time = percent_round(lbf_threshold_options(2));
end
    lbf_lengthening_velocity_threshold = lbf_lengthening_velocity(lbf_critical_time);   % Output variable
catch
    lbf_critical_time = NaN;
    lbf_lengthening_velocity_threshold = NaN;
end
X = lbf_lengthening_velocity(min(lbf_slope_time):max(lbf_slope_time));
Y = lbf_rms_norm(min(lbf_slope_time):max(lbf_slope_time));
p_slope = polyfit(X,Y,1);
lbf_response_slope = p_slope(1);
%
% ------------------------ %
% Right biceps femoris
%
load(timingfile4);
t = linspace(0,RStrTime,51);
time = t';
rbf_length = 1 + (a.*RHipFE) + (b.*(RHipFE.^2)) + (c.*(RHipFE.^3))
+ (d.*RKneeFE) + (e.*(RKneeFE.^2)) + (f.*(RKneeFE.^3));
rbf_active_length = rbf_length;
rbf_lengthening_velocity1 = diff(rbf_length)./diff(time);
% Interpolate rbf_lengthening_velocity to 101 data points for
% comparison
% with EMG
xi = 1:(length(rbf_lengthening_velocity1) -1)/(101-1):length(rbf_lengthening_velocity1);
rbf_lengthening_velocity = interp1(1:length(rbf_lengthening_velocity1),rbf_lengthening_velocity1,xi); % OUTPUT VARIABLE
rbf_slope_time = find(rbf_lengthening_velocity > 0);
for w = 1:length(rbf_slope_time)
    if rbf_slope_time(w) < rfo
        rbf_slope_time(w) = NaN;
    end
end
if ON(Idx(1)) == 0
    percent = [0 percent 100];
else
end
end
percent_round = round(percent);
try
    rbf_threshold_options = find(percent_round >=
    min(rbf_slope_time));
    if mod(rbf_threshold_options(1),2) == 1
        % Ensures that
        first indicator is an on value not an off value
        rbf_critical_time =
        percent_round(min(rbf_threshold_options));
    else rbf_critical_time =
        percent_round(rbf_threshold_options(2));
    end
    rbf_lengthening_velocity_threshold =
    rbf_lengthening_velocity(rbf_critical_time); % Output variable
catch
    rbf_critical_time = NaN;
    rbf_lengthening_velocity_threshold = NaN;
end
X =
lbf_lengthening_velocity(min(lbf_slope_time):max(lbf_slope_time));
Y = lbf_rms_norm(min(lbf_slope_time):max(lbf_slope_time));
p_slope = polyfit(X,Y,1);
lbf_response_slope = p_slope(1);
%
% ------------------------ %
% Left tibialis anterior
%
load(timingfile5);
a = -6.07e-3;
b = 5.86e-5;
c = 4.5e-7;
pennation_angle = 8;
t = linspace(0,LStrTime,51);
time = t';
lta_length = 1 + (a.*LAnkDP) + (b.*(LAnkDP.^2)) +
(c.*(LAnkDP.^3));
pennation_angle_wrt_time =
atand(sind(pennation_angle)./((cosd(pennation_angle)) - (1 -
(lta_length))));
lta_active_length =
sind(pennation_angle)./sind(pennation_angle_wrt_time);
lta_lengthening_velocity1 = diff(lta_active_length)./diff(time);
% Interpolate lta_lengthening_velocity to 101 data points for
comparison
% with EMG
xi = 1:(length(lta_lengthening_velocity1)-1)/(101-
1):length(lta_lengthening_velocity1);
lta_lengthening_velocity =
interp1(1:length(lta_lengthening_velocity1),lta_lengthening_veloci
ty1,xi); % OUTPUT VARIABLE
lta_slope_time = find(lta_lengthening_velocity > 0);
loppfo = round(LOppFO);
for w = 1:length(lta_slope_time)
    if lta_slope_time(w) < loppfo
        lta_slope_time(w) = NaN;
    else
        end
end
if ON(Idx(1)) == 0
    percent = [0 percent 100];
else
    end
end
percent_round = round(percent);
try
    lta_threshold_options = find(percent_round >=
min(lta_slope_time));
    if mod(lta_threshold_options(1),2) == 1   % Ensures that
first indicator is an on value not an off value
        lta_critical_time =
percent_round(min(lta_threshold_options));
    else lta_critical_time =
percent_round(lta_threshold_options(2));
    end
    lta_lengthening_velocity_threshold =
lta_lengthening_velocity(lta_critical_time); % Output variable
catch
    lta_critical_time = NaN;
    lta_lengthening_velocity_threshold = NaN;
end
X =
lta_lengthening_velocity(min(lta_slope_time):max(lta_slope_time));
Y = lta_rms_norm(min(lta_slope_time):max(lta_slope_time));
p_slope = polyfit(X,Y,1);
lta_response_slope = p_slope(1);
% % ----------------------- %
% Right tibialis anterior
% load(timingfile6);
t = linspace(0,RStrTime,51);
time = t';
rtta_length = 1 + (a.*RAnkDP) + (b.*(RAnkDP.^2)) +
(c.*(RAnkDP.^3));
pennation_angle_wrt_time = atand(sind(pennation_angle).(cosd(pennation_angle)) - (1 - (rta_length))));
rta_active_length = sind(pennation_angle).sind(pennation_angle_wrt_time);
rta_lengthening_velocity1 = diff(rta_active_length).diff(time);

% Interpolate rta_lengthening_velocity to 101 data points for comparison

% with EMG

xi = 1:length(rta_lengthening_velocity1)/(101-1):length(rta_lengthening_velocity1);
rta_lengthening_velocity = interp1(1:length(rta_lengthening_velocity1),rta_lengthening_velocity1,xi);  % OUTPUT VARIABLE
rta_slope_time = find(rta_lengthening_velocity > 0);
roppfo = round(ROppFO);
for w = 1:length(rta_slope_time)
    if rta_slope_time(w) < roppfo
        rta_slope_time(w) = NaN;
    else

end
end

if ON(Idx(1)) == 0
    percent = [0 percent 100];
else
end

percent_round = round(percent);
try
    rta_threshold_options = find(percent_round >= min(rta_slope_time));
    if mod(rta_threshold_options(1),2) == 1  % Ensures that first indicator is an on value not an off value
        percent_round(min(rta_threshold_options));
    else rta_critical_time = percent_round(rta_threshold_options(2));
    end
    rta_lengthening_velocity_threshold = rta_lengthening_velocity(rta_critical_time);  % Output variable
catch
    rta_critical_time = NaN;
    rta_lengthening_velocity_threshold = NaN;
end

X = rta_lengthening_velocity(min(rta_slope_time):max(rta_slope_time));
Y = rta_rms_norm(min(rta_slope_time):max(rta_slope_time));
p_slope = polyfit(X,Y,1);
rta_response_slope = p_slope(1);

% -------------------%
% Left medial gastroc
% load(timingfile7);
pennation_angle = 8;
a = 1.22e-2;
b = -4.25e-5;
c = -6.12e-7;
d = -6.75e-3;
e = -9.16e-6;
f = -8.48e-8;
t = linspace(0,LStrTime,51);
time = t';
lmg_length = 1 + (a.*LAnkDP) + (b.*(LAnkDP.^2)) + (c.*(LAnkDP.^3)) + (d.*LKneeFE) + (e.*(LKneeFE.^2)) + (f.*(LKneeFE.^3));
pennation_angle_wrt_time = atand(sind(pennation_angle)./(cosd(pennation_angle)) - (1 - (lmg_length)))));
lmg_active_length = sind(pennation_angle)./sind(pennation_angle_wrt_time);
lmg_lengthening_velocity1 = diff(lmg_active_length)./diff(time);
% Interpolate lmg_lengthening_velocity to 101 data points for comparison
% with EMG
xi = 1:(length(lmg_lengthening_velocity1)-1)/(101-1):length(lmg_lengthening_velocity1);
lmg_lengthening_velocity = interp1(1:length(lmg_lengthening_velocity1),lmg_lengthening_velocity1,xi);   % OUTPUT VARIABLE
lmg_slope_time = find(lmg_lengthening_velocity > 0);
if ON(Idx(1)) == 0
    percent = [0 percent 100];
else
    percent_round = round(percent);
    try
        lmg_threshold_options = find(percent_round >= min(lmg_slope_time));
        if mod(lmg_threshold_options(1),2) == 1     % Ensures that first indicator is an on value not an off value
            lmg_critical_time = percent_round(min(lmg_threshold_options));
        else
            lmg_critical_time = percent_round(lmg_threshold_options(2));
        end
        lmg_lengthening_velocity_threshold = lmg_lengthening_velocity(lmg_critical_time);   % Output variable
    catch
        lmg_critical_time = NaN;
        lmg_lengthening_velocity_threshold = NaN;
    end
end
X = lmg_lengthening_velocity(min(lmg_slope_time):max(lmg_slope_time));
Y = lmg_rms_norm(min(lmg_slope_time):max(lmg_slope_time));
p_slope = polyfit(X,Y,1);
lmg_response_slope = p_slope(1);

% Right medial gastroc
%----------------------
load(timingfile8);
t = linspace(0,RStrTime,51);
time = t';
rmg_length = 1 + (a.*RAnkDP) + (b.*(RAnkDP.^2)) + (c.*(RAnkDP.^3)) + (d.*RKneeFE) + (e.*(RKneeFE.^2)) + (f.*(RKneeFE.^3));
pennation_angle_wrt_time = atand(sind(pennation_angle)./(cosd(pennation_angle)) - (1 - (rmg_length))));
rmg_active_length = sind(pennation_angle)./sind(pennation_angle_wrt_time);
rmg_lengthening_velocity1 = diff(rmg_active_length)./diff(time);
% Interpolate rmg_lengthening_velocity to 101 data points for comparison
% with EMG
xi = 1:(length(rmg_lengthening_velocity1)-1)/(101-1):length(rmg_lengthening_velocity1);
rmg_lengthening_velocity =
interp1(1:length(rmg_lengthening_velocity1),rmg_lengthening_velocity1,xi); % OUTPUT VARIABLE
rmg_slope_time = find(rmg_lengthening_velocity > 0);
if ON(Idx(1)) == 0
    percent = [0 percent 100];
else
end
percent_round = round(percent);
try
    rmg_threshold_options = find(percent_round >= min(rmg_slope_time));
    if mod(rmg_threshold_options(1),2) == 1 % Ensures that first indicator is an on value not an off value
        rmg_critical_time = percent_round(min(rmg_threshold_options));
    else
        rmg_critical_time = percent_round(rmg_threshold_options(2));
    end
    rmg_lengthening_velocity_threshold = rmg_lengthening_velocity(rmg_critical_time); % Output variable
catch
    rmg_critical_time = NaN;
    rmg_lengthening_velocity_threshold = NaN;
end
X = rmg_lengthening_velocity(min(rmg_slope_time):max(rmg_slope_time));
Y = rmg_rms_norm(min(rmg_slope_time):max(rmg_slope_time));
p_slope = polyfit(X,Y,1);
rmg_response_slope = p_slope(1);
%
% Save output
if hc == 0
    outputfile = 
    ['/Users/ailish/Documents/MATLAB/RESULTS/Spasticity/Trials/Lengthening_Scores',int2str(p),'.',int2str(k),'.',int2str(n),'.mat'];
else
    outputfile = 
    ['/Users/ailish/Documents/MATLAB/RESULTS/Spasticity/Trials/Lengthening_Scores_HC',int2str(p),'.',int2str(k),'.',int2str(n),'.mat'];
end
eval(['save ',outputfile,' lrf_critical_time
    lrf_lengthening_velocity_threshold lrf_response_slope
    lrf_lengthening_velocity lrf_active_length rrf_critical_time
    rrf_lengthening_velocity_threshold rrf_response_slope
    rrf_lengthening_velocity rrf_active_length lbf_critical_time
    lbf_lengthening_velocity_threshold lbf_response_slope
    lbf_lengthening_velocity lbf_active_length rbf_critical_time
    rbf_lengthening_velocity_threshold rbf_response_slope
    rbf_lengthening_velocity rbf_active_length lta_critical_time
    lta_lengthening_velocity_threshold lta_response_slope
    lta_lengthening_velocity lta_active_length rta_critical_time
    rta_lengthening_velocity_threshold rta_response_slope
    rta_lengthening_velocity rta_active_length lmg_critical_time
    lmg_lengthening_velocity_threshold lmg_response_slope
    lmg_lengthening_velocity lmg_active_length rmg_critical_time
    rmg_lengthening_velocity_threshold rmg_response_slope
    rmg_lengthening_velocity rmg_active_length']);
clear all
Appendix 7.1: Calculation of sample size for reliability study

Method of Streiner and Norman (2008)

Calculation of the required sample size begins with an estimate of the likely intraclass correlation, $R$, and the desired confidence interval around $R$. The standard error, SE, is half the confidence interval. The number of observations for use in the study, $k$, must also be known.

First compute:

$$R^- = R - SE$$

(Eq. 1)

Then calculate the $z$ scores of $R$ and $R^-$ as follows:

$$z_R = \frac{1}{2}\ln((1 + (k - 1)R) / (1 - R))$$

and

$$z_{R^-} = \frac{1}{2}\ln((1 + (k - 1)R^-) / (1 - R^-))$$

(Eq. 2)

SE of the $z$ scores is then computed as follows:

$$SE = z_R - z_{R^-}$$

(Eq. 3)

From a previous equation derived by Streiner and Norman (2008):

$$SE(z_R) = \sqrt{k / (2(k - 1)(n - 2))}$$

(Eq. 4)

Therefore, squaring and cross multiplying, the sample size $n$ can be calculated:

$$n = 2 + \frac{k}{(2(k - 1)( z_R - z_{R^-})^2)}$$

(Eq. 5)

Source

Appendix 8.1: Healthy controls information leaflet

Protocol Title: Controls Information Leaflet

Gait Impairment in Cervical Spondylotic Myelopathy: Analysis, Impact on Function, and Effect of Surgical Intervention

Principal Investigator’s Name: Ailish McDermott
Principal Investigator’s Title: Senior Physiotherapist
Telephone No. of Principal Investigator: 018092526 / 085 8336094

You are being invited to take part in a clinical research study carried out at Beaumont Hospital and the Royal College of Surgeons in Ireland. Before you decide whether or not you wish to take part, you should read the information provided below carefully and if you wish discuss it with your family, friends or GP. Take time to ask questions – do not feel rushed or under any obligation to participate. You should clearly understand the risks and benefits of participating in this study so that you can make a decision that is right for you – this process is known as Informed Consent.

You are not obliged to take part in this study and failure to participate will have no effect on your future care. You may change your mind at any time (before the start of the study or even after you have commenced the study) for whatever reason without having to justify your decision and without any negative impact on your care.

WHY IS THIS STUDY BEING DONE?

This study is being done because we know from experience and previous research that patients who suffer from compression of the spinal cord in their neck as a result of “wear and tear” (the condition called “cervical spondylotic myelopathy” in the title of the study) experience difficulties with walking. They may feel unsteady, the legs may feel stiff or weak, or they may not be able to walk as far or with the same ease as before. We hope that, by examining in more detail how patients with this condition walk, doctors and physiotherapists will better understand why these problems come about. We are recruiting a population of healthy people to give us a “baseline” with which to compare the walking patterns of the people with the condition cervical spondylotic myelopathy. As a healthy control, your data will be compared to a patient of the same age and gender.

WHO IS ORGANISING AND FUNDING THIS STUDY?

The Principal Investigator of this study is Ailish McDermott, Senior Physiotherapist. The Health Research Board has provided funding for the study to Ailish McDermott, who has registered for a PhD degree with the Royal College of Surgeons in Ireland.
HOW WILL IT BE CARRIED OUT?

This research study commenced in October 2008. It will last for three years. Patients who have symptoms like unsteady walking or clumsy hands, and who have been told by their surgeon that they have spinal cord compression due to degenerative changes or “wear and tear” of the joints in the neck, will be asked to participate in the study. Healthy controls, of the same age and gender, will then be recruited to provide an accurate reflection of a normal walking pattern. All participants will have an analysis of their walking conducted in the Human Movement Laboratory in the Physiotherapy Department of the Royal College of Surgeons in Ireland, 123 St. Stephen's Green, Dublin 2.

WHAT WILL HAPPEN TO ME IF I AGREE TO TAKE PART?

You will be given an appointment to attend the Movement Laboratory in the Royal College of Surgeons in Ireland (RCSI), where an analysis of your walking will be carried out. The assessment in RCSI will take between one and a half and two hours. Measurements will be taken of your height, weight, and the width of the joints in your leg. The range of movement and “stiffness” of the muscles and joints in your legs will be measured. You will have small electrodes (about 2cm square) attached to four muscles in your leg, and you will have markers placed on the joints of your leg. This is not painful, they are simply attached to your skin, though we will need to shave a small area of hair to allow the electrodes to stick properly. You will then be asked to walk along a 10 metre walkway a number of times while your walking pattern is recorded by the motion analysis system (VICON). Next, you will be asked to walk a small flight of stairs if you are able to do this.

BENEFITS:

Taking part in the study will allow the Principal Investigator to gather detailed information on your walking pattern. The results of the assessments will all be explained to you. If you would like your own copy of the results, we will give them to you on a CD.

Your participation will benefit patients with cervical spondylotic myelopathy, as it will allow their walking patterns to be compared to healthy people of the same age and gender who do not have the condition. This will help healthcare professionals to understand this condition more fully, and to provide the treatments that are best supported by the evidence.

RISKS:

This research study evaluates your normal walking pattern, and for healthy people, this should not pose any difficulty. There is one small risk in relation to the equipment used, and that is the risk of a skin reaction to the sticky electrodes. The risk of this is low because the adhesive on the electrodes is hypoallergenic, however please inform the researcher if you have sensitive skin, or if you have had a reaction to something similar in the past. If this happens, the reaction is likely to be small, with some redness and itchiness of the skin where the electrode was placed.

WILL THERE BE ANY ADDITIONAL COSTS INVOLVED?

If you incur travel expenses to come to your appointment in the Royal College of Surgeons, please give your receipt to the Principal Investigator who will organise a refund.

YOUR RESPONSIBILITIES AS A PARTICIPANT

1. To attend for assessment of your walking pattern at the appointment time given, or to give the researcher adequate notice if you need to change the appointment.
2. To inform the researcher of any factors which may affect your ability to walk for the required length of time e.g. new medical conditions such as angina, a chest infection causing you to be breathless, or an injury to your leg.
3. If you are a woman of child-bearing age, to inform the researcher if you are pregnant.
4. To inform the researcher if you are currently on medication, or if you are currently having physiotherapy, or any other treatment for your condition.

OUR RESPONSIBILITIES TO YOU AS INVESTIGATORS

We will ensure that our assessment of your walking pattern is carried out safely, accurately, and following all protocols and procedures to minimise the risk to you, the participant, and to ensure that the quality of information we provide to you afterwards is of the highest possible quality. We will provide you with the results of your walking test, which is yours to keep.

CONFIDENTIALITY ISSUES

When we take records of your assessment in the laboratory, we will not keep your name or any other details with this data. Instead, you will be given a “unique identifier” which will be coded on a secure hard drive and available only to the Principal Investigator. The document with this coded information will be destroyed as soon as the study is completed. The data from the laboratory, with details of your walking assessment, will be kept for a period of five years after the study is completed, because it may be included in future studies. If you wish to consent to this study only, and do not wish to consent to have your data included in possible future studies, please inform the researcher who will ensure your data are deleted after this study.

IF YOU REQUIRE FURTHER INFORMATION

If you have any further questions about the study, now or any future time, please contact the Principal Investigator:

Ailish McDermott,
Senior Physiotherapist
Beaumont Hospital,
Dublin 9.
Phone: 01-8092526 / 085-8336094.

If you wish to withdraw from the study you may do so at any time without justifying your decision and your future treatment will not be affected.
Appendix 8.2: Healthy controls consent form

CONSENT FORM – HEALTHY CONTROLS

Protocol Title:


Please tick the appropriate answer.

I confirm that I have read and understood the Controls Information Leaflet dated 23/02/10 attached, and that I have had ample opportunity to ask questions all of which have been satisfactorily answered.  Yes  No

I understand that my participation in this study is entirely voluntary and that I may withdraw at any time, without giving reason, and without this decision affecting my future treatment or medical care.  Yes  No

I understand that my identity will remain confidential at all times.  Yes  No

I am aware of the potential risks of this research study.  Yes  No

I have been given a copy of the Controls Information Leaflet, version 1, dated 23/02/10 and this Consent form for my records.  Yes  No

FUTURE USE OF ANONYMOUS DATA:

I agree that I will not restrict the use to which the results of this study may be put. I give my approval that unidentifiable data concerning my person may be stored or electronically processed for the purpose of scientific research and may be used in related or other studies in the future. (This would be subject to approval by an independent body, which safeguards the welfare and rights of people in biomedical research studies - the Beaumont Hospital Ethics (Medical Research) Committee.)  Yes  No

Participant_________________________  __________________________
Signature                      Date              Name in block capitals

Witness _________________________  __________________________
Signature                        Date  Name in block capitals

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To be completed by the Principal Investigator or his nominee.

I the undersigned, have taken the time to fully explained to the above participant the nature and purpose of this study in a manner that he/she could understand. I have explained the risks involved, the experimental nature of the treatment, as well as the possible benefits and have invited him/her to ask questions on any aspect of the study that concerned them.

Signature __________________ Name in block capitals ____________ Qualification ____________ Date ____________