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Gait impairment in cervical spondylotic myelopathy: comparison with age- and gender-matched healthy controls

Ailish Malone · Dara Meldrum · Ciaran Bolger

Abstract

Introduction Gait impairment is a primary symptom of cervical spondylotic myelopathy (CSM); however, little is known about specific kinetic and kinematic gait parameters. The objectives of the study were: (1) to compare gait patterns of people with untreated CSM to those of age- and gender-matched healthy controls; (2) to examine the effect of gait speed on kinematic and kinetic parameters.

Materials and methods Sixteen patients with CSM were recruited consecutively from a neurosurgery clinic, and 16 healthy controls, matched to age (±5 years) and gender, were recruited for comparison. Patients and controls underwent three-dimensional gait analysis using a Vicon® motion analysis system, at self-selected speed over a 10-m track. Controls were also assessed at the speed of their CSM match.

Results At self-selected speed, the CSM group walked significantly more slowly, with shorter stride lengths and longer double support duration. They showed significant decreases in several kinematic and kinetic parameters, including sagittal range of motion at the hip and knee, ankle plantarflexion, anteroposterior ground reaction force (GRF) at toe-off, power absorption at the knee in loading

response and terminal stance, and power generation at the ankle. At matched speed, the CSM group showed significant decreases in knee flexion during swing, total sagittal knee range of motion, peak ankle plantarflexion and anteroposterior GRF.

Conclusion and implications The findings suggested that people with CSM have significant gait abnormalities that have not been previously reported. In particular, there are key differences in the motor strategies used in the terminal stance phase of gait that cannot be explained by speed alone.

Keywords Cervical myelopathy · Gait · Gait analysis · Biomechanics

Introduction

Gait impairment is a primary symptom of cervical spondylotic myelopathy (CSM). In contrast to the substantial body of literature on gait patterns in neurological conditions such as cerebral palsy [1] and stroke [2], little is known about the kinematic and kinetic characteristics of gait in CSM. There is evidence that people with CSM have a slower gait speed, prolonged double support duration and reduced cadence compared to healthy controls (HCs) [3–6]. Previous studies also identified reduced knee flexion during swing in the early stages of the disease, and in more severe disease, decreased ankle plantarflexion at the terminal stance and reduced knee flexion during loading response [3, 6–8]. However, these studies evaluated a limited number of parameters. A greater insight could be attained through the systematic analysis of a larger number of discrete parameters at specific points in the gait cycle across three planes of motion [9]. Furthermore, no previous

A. Malone (✉)
Physiotherapy Department, Beaumont Hospital,
Dublin 9, Ireland
e-mail: ailishmcd@gmail.com

A. Malone · D. Meldrum
School of Physiotherapy, Royal College of Surgeons in Ireland
(RCSI), Dublin, Ireland

C. Bolger
Department of Neurosurgery, Beaumont Hospital,
Dublin, Ireland

studies evaluated joint moments and powers from kinetic data, information that could significantly enhance the understanding of a gait deficit in spinal cord pathology [10].

An important consideration in the interpretation of kinematic and kinetic data is the inter-dependence of many variables. Gait speed is known to influence lower limb kinematics, with faster speeds generally associated with larger sagittal range of motion (ROM) [9]. Previous studies of the CSM gait compared patient and control groups at different speeds [6, 8, 11] or at enforced speeds [3]. It is important that participants should be assessed at self-selected gait speeds to evaluate natural walking performance; however, there is also a need to match the speeds of CSM and control participants to avoid confounding effects on kinematics [12]. Otherwise, it cannot be known whether reduced joint excursions are the cause or the effect of slower speed.

The aims of this study were:

1. to compare gait patterns of people with untreated CSM with age and gender-matched HCs;
2. to examine the effect of speed on kinematic and kinetic features of the CSM gait.

Methods

Participants

Approval was obtained from a local ethics committee. Participants with CSM were consecutively recruited from a neurosurgical clinic. The following inclusion criteria were applied: (1) aged 18 years or over; (2) able to give informed consent; (3) able to mobilise at least 10 m without assistance of another person; (4) clinical and radiological evidence of CSM. CSM was diagnosed in the presence of one or more of the symptoms such as clumsy hands, numb hands or feet, lower limb weakness, unsteady gait, and bilateral upper or lower limb paraesthesiae, and one or more of the following signs on examination: lower limb spasticity, unsteady gait, hyperreflexia, upgoing plantars, clonus, weakness in a corticospinal distribution and positive Hoffman's sign. The diagnosis was confirmed by evidence on magnetic resonance imaging of cord compression due to spondylosis or intrinsic signal change.

Patients were excluded if they were affected by any of the following: (1) severe respiratory or cardiac disease hindering safe mobilisation; (2) history of neurological disorders with persistent deficit; (3) symptomatic musculoskeletal problems affecting gait; (4) tandem lumbar spine stenosis; (5) previous surgical decompression for CSM. Severity of myelopathy was measured using the Nurick

classification [13] and the modified Japanese orthopaedic association scale [14].

Each CSM participant was matched to an HC of the same age (±5 years) and gender. HCs were recruited from a local population of colleagues and had no symptomatic lower limb injuries, neurological disorders, or cardiovascular or respiratory impairment that would hinder gait analysis. All participants gave informed consent.

Gait analysis

Three-dimensional gait analysis (3DGA) was conducted using a VICON® 250 five-camera Motion Analysis system (VICON, Oxford, UK) and an integrated Kistler multi-component force plate (Kistler, Winterthur, Switzerland). Anthropometric data were collected according to a standard protocol. Fifteen 25-mm reflective markers were applied to anatomical landmarks on the lower limbs using the modified Helen Hayes method [15]. A knee alignment device (Motion Lab Systems Inc, Baton Rouge, USA) was used to aid the identification of the knee joint axis.

Each assessment consisted of a static trial for calibration and a warm-up trial to familiarise the participant with the protocol, followed by a number of barefoot walking trials. Data were captured at a frequency of 50 Hz. Participants were instructed to walk at self-selected comfortable speed along a 12-m overground walkway. The assessment continued until ten trials, comprising five left and right force plate strikes, with good quality data had been achieved. Breaks between walking trials were provided at participants' request to avoid fatigue.

HCs then completed a second assessment at the walking speed of the individual CSM participants to whom they were matched. Trials were timed using a stopwatch. The goal speed was indicated by verbal feedback at the end of each trial. 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.

Gait data were processed using VICON Workstation® software. Marker trajectories were reconstructed and filtered using the Woltring routine [16] with a mean standard error of 15 mm². VICON Plug-in Gait® was used to calculate motion at the lower limb joints in three planes. Gait cycle events (heel strike and toe-off) were identified automatically from force plate data for gait cycles where force was recorded, and by using frame-by-frame monitoring of the heel, ankle and toe trajectories, where force was not recorded. The average of ten captured trials was used to represent the gait pattern of each participant under each condition (comfortable speed only for CSM, comfortable and matched speed for HC). Data from CSM participants' more affected lower limbs were analysed and

Table 1 Kinematic and kinetic key points extracted from 3DGA data

Joint	Description of key point
Pelvis	Peak pelvic tilt
	Range of pelvic tilt
	Average pelvic tilt
	Peak pelvic obliquity
	Range of pelvic obliquity
	Range of pelvic rotation
Hip	Hip position in the sagittal plane at initial contact
	Peak hip flexion
	Peak hip extension
	Total range of hip excursion in the sagittal plane
	Peak hip abduction in swing
	Total range of hip excursion in the frontal plane
	Peak hip internal rotation
	Total range of hip rotation
Knee	Knee position at initial contact
	Peak knee flexion in stance, loading response
	Peak knee flexion in swing
	Peak knee extension in mid-stance
	Total range of knee excursion in the sagittal plane
Ankle	Ankle position at initial contact
	Peak ankle dorsiflexion in stance
	Peak ankle dorsiflexion in swing
	Peak ankle plantarflexion
Ground reaction force (GRF) (N/kg)	Peak mediolateral GRF
	Peak negative antero-posterior GRF (braking)
	Peak positive antero-posterior GRF (propulsion)
	First vertical GRF peak, loading
	Minimum value of GRF during stance
	Second vertical GRF peak, propulsion
Moments (Nm/kg)	Peak hip flexor moment
	Peak hip extensor moment
	Peak hip abductor moment
	Peak knee flexor moment
	Peak knee extensor moment
	Peak ankle plantarflexor moment

Table 1 continued

Joint	Description of key point
Powers (W/kg)	Peak concentric hip extensor power during loading response (H1)
	Peak eccentric hip flexor power during mid-stance (H2)
	Peak concentric hip flexor power during terminal stance (H3)
	Peak eccentric knee extensor power during loading response (K1)
	Peak concentric knee extensor power during mid-stance (K2)
	Peak eccentric knee extensor power in terminal stance (K3)
	Peak eccentric knee flexor power, terminal swing (K4)
	Peak eccentric ankle power in loading response through to mid-stance (A1)
	Peak concentric ankle plantarflexor power at terminal stance (A2)

W watts, *kg* kilograms, *N* Newtons, *Nm* Newton metre

Statistical analysis

A sample size calculation was performed in Stata (Stata-Corp, Texas, USA). 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 per second (m/s) with a standard deviation of 0.11 m/s.

Two analyses were conducted. The first analysis compared CSM with HC participants at comfortable speed, and the second compared the pairs at matched speed. The distribution of each variable was examined using quantile–quantile and box plots. Normally distributed data were analysed using two-tailed paired *t* tests. The Wilcoxon signed-rank test analysed variables that were not normally distributed.

Results

Sixteen participants with CSM and 16 HCs were recruited between December 2008 and December 2010. Table 2 shows their characteristics.

Temporal–spatial parameters

The mean gait speed of CSM participants was 1.12 m/s, significantly slower than HCs' mean speed of 1.49 m/s ($p < 0.0001$). This slower speed resulted from a shorter mean stride length of 1.19 m compared to 1.45 m ($p = 0.0001$), and a lower mean cadence of 113 compared to 122 steps per min ($p = 0.005$). CSM participants spent a

compared to data from the same limb of their HC matches. The more affected lower limb was determined by subjective questioning. Temporal–spatial parameters and kinematic and kinetic key points, listed in Table 1, were selected based on clinical significance in a neurological population [17] and the results of a reliability study of 3DGA in CSM [18].

Table 2 Characteristics of participants

CSM									HC		
Case	Gender	Age (years)	Height (m)	Weight (kg)	Duration of symptoms (months)	Nurick	mJOA	MRMI	Age (years)	Height (m)	Weight (kg)
02	F	68	1.52	85.2	12	3	8	34	68	1.69	74.4
03	M	57	1.66	80.3	12	3	11	38	59	1.76	78.4
04	F	50	1.61	57.7	5	1	14	40	45	1.67	62.8
05	M	35	1.93	100.7	24	2	10	40	33	1.76	85.5
06	F	43	1.46	80.2	48	3	12	39	41	1.73	67.1
07	F	46	1.5	56.4	60	3	13	39	41	1.67	61.5
08	F	63	1.6	70.4	36	3	9	39	58	1.71	71.7
11	M	53	1.56	54.6	108	2	10	39	53	1.73	76
12	F	51	1.72	74.8	48	2	10	40	55	1.63	57.5
13	M	74	1.69	61.5	420	3	12	38	73	1.71	63.9
15	F	73	1.64	54.1	10	2	10	40	68	1.56	61.3
16	F	48	1.72	89.8	48	1	13	40	50	1.59	74.5
18	M	47	1.67	78.7	12	3	11	39	52	1.89	89.5
19	M	64	1.83	91.1	180	2	14	40	65	1.8	81
20	M	54	1.79	96.3	18	3	10	39	54	1.79	69.7
22	M	58	1.66	75.6	10	4	12	39	62	1.66	94.2
Mean/*median*		55.25	1.66	75.5	*36*	*3*	*11*	*39*	54.8	1.71	73.1

Italic values to indicate that the median is reported rather than the mean

mJOA modified Japanese orthopaedic association score, *MRMI* modified rivermead mobility index, *kg* kilograms, *M* male, *F* female

shorter proportion of the gait cycle (GC) in single limb support (36.7 % compared to 39.9 % GC duration, $p < 0.0001$).

At matched speed, stride length (CSM 1.19 m, HC 1.27 m, $p = 0.03$) and single leg support (HC 36.7 %, CSM 37.6 % GC, $p = 0.049$) remained significantly lower in CSM participants. The full results are shown in Table 3.

Kinematics

Kinematic data are provided in Fig. 1 and Table 4. At comfortable speed, CSM participants had reduced range of pelvic obliquity (CSM 6.34°, HC 8.78°, $p = 0.003$) and reduced total sagittal plane excursion at the hip (CSM 44.3°, HC 49.1°, $p = 0.004$). At the knee, CSM participants showed lower 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 (HC 59.9°, CSM 51.9°, $p = 0.004$). Ankle plantarflexion was also reduced in CSM (11.3°, HC −18.4°, $p = 0.013$).

At matched speed, CSM participants showed reduced knee flexion in swing (HC 54.6°, CSM 48.6°, $p = 0.006$) and total sagittal plane excursion (HC 56.8°, CSM 51.9°, $p = 0.03$). At the ankle, dorsiflexion in stance (HC 16.2°, CSM 14.3°, $p = 0.02$) and peak plantarflexion at pre-swing (HC −16.6°, CSM −11.3°, $p = 0.02$) were also lower in CSM participants.

Kinetics

Figure 2 and Table 5 show the results of the kinetic analysis. At comfortable speed, vertical ground reaction force (GRF) during loading was significantly lower in CSM ($p = 0.0004$). Antero-posterior GRF was also lower 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, $p < 0.0001$). CSM participants had reduced peak hip flexor moments (HC −0.81 Nm/kg, CSM −0.62 Nm/kg, $p = 0.02$) and reduced peak knee extensor moments (HC 0.56 Nm/kg, CSM 0.27 Nm/kg, $p = 0.0005$). Peak ankle plantarflexor moments were also lower in CSM (HC 1.6 Nm/kg, CSM 1.41 Nm/kg, $p = 0.0007$). Analysis of net power showed reduced hip power absorption in mid-stance, H2 (HC −0.96 W/kg, CSM −0.61 W/kg, $p = 0.004$), and hip power generation in pre-swing, H3 (HC 1.71 W/kg, CSM 1.0 W/kg, $p = 0.0001$). Knee power absorption peaks were also lower in CSM throughout the GC. Ankle power generation at pre-swing, A2, was higher in HC (4.85 N/kg) than in CSM (2.82 N/kg, $p = 0.0001$).

There were fewer differences between CSM and HC participants at matched speed; however, some findings persisted. The acceleration component of antero-posterior GRF remained reduced in CSM (HC −1.9 N/kg,

Table 3 Temporal–spatial parameters of CSM and HC participants

Variable	CSM data		Comfortable speed comparison							Matched speed comparison							SEM[a]
	CSM		HC		Difference		95 % Confidence intervals		p value	HC		Difference		95 % Confidence intervals		p value	
	Mean	SD	Mean	SD	Mean	SD				Mean	SD	Mean	SD				
Cadence (steps/min)	113.22	10.40	122.97	8.62	−9.75	12.05	−16.17	−3.33	0.006	103.91	12.02	9.31	9.00	4.52	14.11	0.0009	1.70
Double support (s)	0.28	0.06	0.20	0.03	0.08	0.06	0.05	0.11	0.0001	0.30	0.07	−0.02	0.03	−0.03	0.00	0.07	0.01
Single support (s)	0.39	0.04	0.39	0.02	0.00	0.04	−0.02	0.02	0.9	0.44	0.04	−0.05	0.04	−0.07	−0.02	0.0005	0.01
Double support duration (% GC)	26.06	3.82	20.32	2.03	5.74	4.14	3.54	7.95	0.0001	24.94	3.32	1.12	2.84	−0.40	2.64	0.14	
Single support duration (% GC)	36.66	2.06	39.86	0.85	−3.20	1.99	−4.26	−2.13	<0.0001	37.56	1.66	−0.90	1.67	−1.79	0.00	0.049	
Foot off (% GC)	62.71	2.19	60.18	1.33	2.53	2.81	1.03	4.03	0.003	62.49	1.76	0.21	2.01	−0.85	1.28	0.7	0.81
Gait speed (m/s)	1.12	0.24	1.49	0.18	−0.36	0.24	−0.49	−0.24	<0.0001	1.11	0.21	0.02	0.07	−0.02	0.06	0.3	0.02
Opposite foot contact (% GC)	49.56	1.25	50.26	0.59	−0.69	1.57	−1.53	0.14	0.09	50.27	0.65	−0.71	1.41	−1.46	0.04	0.06	1.01
Opposite foot off (% GC)	12.91	2.14	10.40	1.13	2.51	2.33	1.27	3.75	0.0006	12.69	1.73	0.22	1.54	−0.61	1.04	0.6	0.77
Step length (m)	0.59	0.10	0.73	0.06	−0.14	0.11	−0.19	−0.08	0.0001	0.64	0.06	−0.05	0.07	−0.09	−0.01	0.02	0.01
Step time (s)	0.54	0.05	0.49	0.03	0.05	0.06	0.02	0.08	0.002	0.58	0.06	−0.04	0.05	−0.07	−0.01	0.007	0.02
Step width (m)	0.17	0.04	0.16	0.03	0.01	0.04	−0.01	0.03	0.4	0.16	0.04	0.01	0.05	−0.02	0.03	0.5	0.01
Stride length (m)	1.19	0.19	1.45	0.11	−0.26	0.20	−0.37	−0.16	0.0001	1.27	0.13	−0.08	0.14	−0.16	−0.01	0.03	0.02
Stride time (s)	1.07	0.11	0.98	0.06	0.09	0.11	0.03	0.15	0.006	1.17	0.13	−0.10	0.10	−0.15	−0.05	0.0008	0.02

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

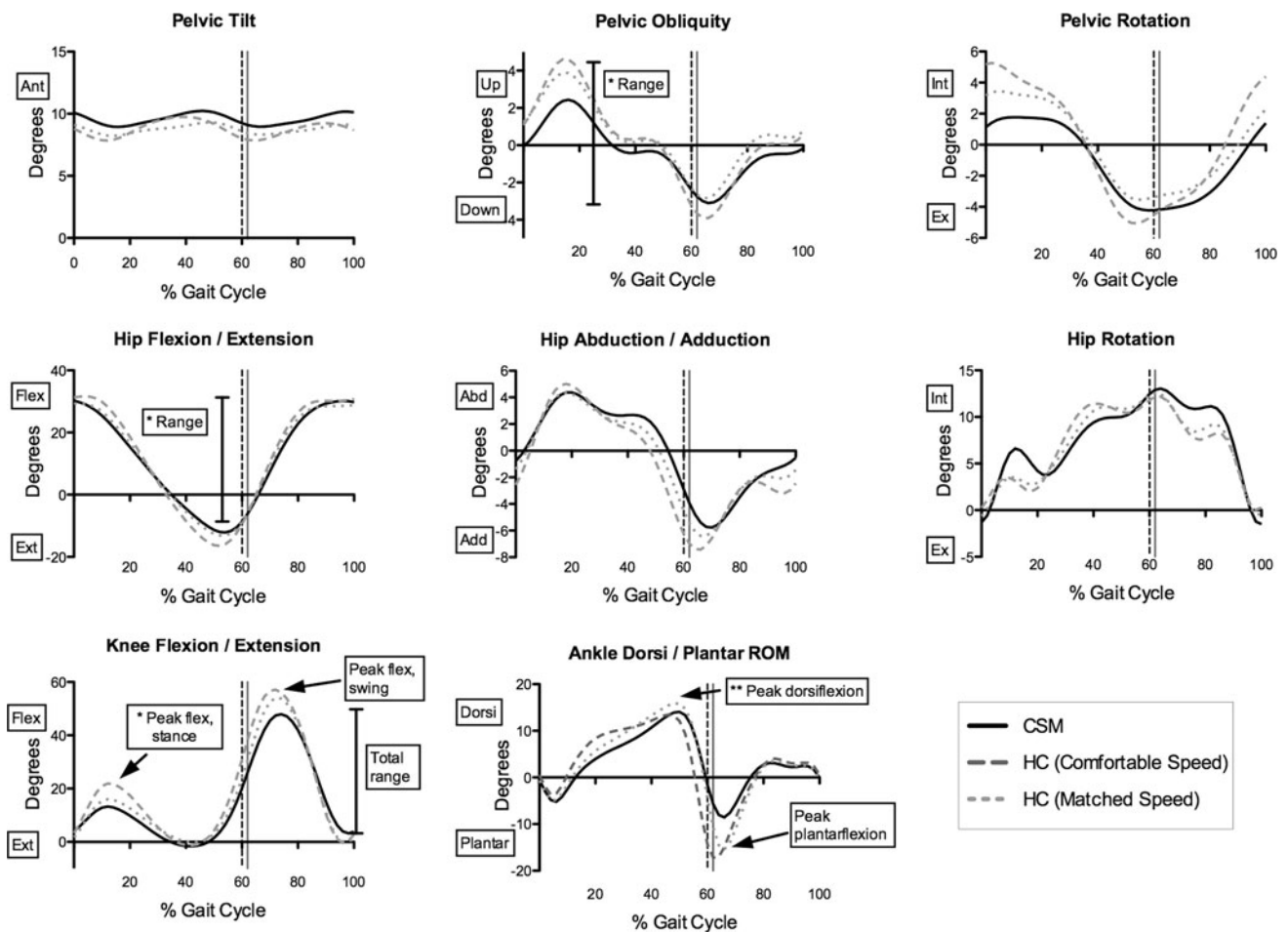[a] Standard error of measurement, calculated in a prior study [18]

Fig. 1 Kinematic curves of HC and CSM participants. Significant differences between HC (*dashed lines*) and CSM (*continuous line*) are indicated using *boxes* and *arrows*; *asterisks* denotes significant difference at comfortable speed only; *double asterisks* denotes significant difference at matched speed only. *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 CSM(*black*) and HC (*grey*)

CSM -1.6 N/kg, $p = 0.02$). CSM participants 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 CSM (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 lower absorption peaks at K1 and K4 in CSM. At the ankle, there was a non-significant trend towards higher power generation at the ankle at toe-off in HC (3.32 W/kg) compared to CSM (2.82 W/kg) ($p = 0.075$). All significant differences in temporal–spatial, kinematic and kinetic parameters exceeded previously calculated SEM values [18].

Discussion

This study was the first to examine a wide range of kinematic key points, moments and powers during gait in the CSM population, and to control for the confounding effects of gait speed when comparing to healthy individuals. The reliability of the primary outcome measure, three-dimensional gait analysis, was evaluated in a previous paper, ensuring that all statistically significant differences could be interpreted within the context of prior known measurement error [18]. In keeping with other studies [3, 4, 6–8], the current study found that CSM participants walked at significantly slower gait speeds, demonstrated difficulty in generating adequate stride length and spent less time in single support, suggesting either a lack of stability in single leg stance, weak

Table 4 Peak kinematic parameters of CSM and HC participants

Variable	CSM data		Comfortable speed comparison							Matched speed comparison							SEM[a]
	CSM		HC		Difference		95 % Confidence intervals		p value	HC		Difference		95 % Confidence intervals		p value	
	Mean	SD	Mean	SD	Mean	SD				Mean	SD	Mean	SD				
Pelvic obliquity range	6.3	3.2	8.8	2.8	-2.4	2.7	-3.9	-1.0	0.003	7.0	2.3	-0.7	2.3	-1.9	0.5	0.2	0.92
Pelvic rotation range	8.2	3.6	10.8	4.1	-0.6	3.2	-2.3	1.1	0.5	8.4	2.1	1.2	3.5	-0.7	3.0	0.2	1.14
Average pelvic tilt	9.4	5.9	8.8	4.2	0.6	7.5	-3.4	4.6	0.75	8.8	4.0	0.6	7.2	-3.2	4.5	0.74	2.57
Hip position at initial contact	30.3	6.9	31.3	5.6	-1.0	9.5	-6.0	4.1	0.7	29.3	5.3	1.0	9.4	-4.0	6.0	0.67	2.99
Peak hip extension	-13.4	6.3	-16.5	5.3	3.1	8.8	-1.6	7.9	0.17	-13.5	5.9	0.1	8.6	-4.5	4.7	0.97	3.19
Total sagittal plane excursion of hip	44.3	5.3	49.1	4.9	-4.8	5.7	-7.8	-1.8	0.004	43.7	3.2	0.6	4.2	-1.6	2.9	0.55	1.28
Range of hip abduction/adduction	12.0	3.2	13.0	3.0	-1.1	3.6	-3.0	0.9	0.27	11.4	2.6	0.5	3.0	-1.1	2.1	0.49	1.05
Range of hip rotation	19.1	10.1	16.2	4.8	2.9	11.6	-3.3	9.1	0.33	17.0	4.6	2.1	10.8	-3.6	7.9	0.45	3.66
Knee position at initial contact	4.2	6.0	3.3	3.7	0.9	6.3	-2.4	4.3	0.57	1.7	4.5	2.5	6.9	-1.2	6.1	0.17	3.11
Peak knee flexion in stance	13.7	6.4	22.1	6.9	-8.3	7.5	-12.3	-4.3	0.0005	16.2	7.9	-2.5	7.3	-6.4	1.4	0.19	3.67
Peak knee extension	-3.3	4.4	-2.4	4.4	-0.9	5.8	-4.0	2.1	0.52	-2.2	4.0	-1.1	5.3	-3.9	1.7	0.4	3.12
Peak knee flexion in swing	48.6	5.6	57.5	4.2	-8.9	8.1	-13.2	-4.6	0.0005	54.6	5.0	-6.0	7.5	-10.0	-2.0	0.006	3.32
Total sagittal plane excursion of knee	51.9	6.9	59.9	5.4	-8.0	9.4	-13.0	-3.0	0.004	56.8	5.2	-4.9	8.0	-9.2	-0.6	0.03	2.62
Ankle position at initial contact	-0.6	3.7	0.5	3.1	-1.1	4.2	-3.4	1.1	0.3	-0.7	3.5	0.1	4.0	-2.1	2.2	0.95	2.35
Peak ankle dorsiflexion in stance	14.3	2.6	14.4	2.7	-0.1	3.2	*	*	0.92*	16.2	2.5	-2.0	3.0	-3.6	-0.4	0.02	1.37
Peak ankle plantarflexion	-11.3	6.9	-18.4	7.6	7.1	10.0	1.7	12.4	0.013	-16.6	6.4	5.3	8.0	1.1	9.6	0.02	2.90
Peak ankle dorsiflexion in swing	6.2	4.2	4.7	2.5	1.5	4.1	-0.7	3.7	0.16	6.2	4.0	0.1	4.5	-2.3	2.5	0.96	2.00

All variables are measured in degrees

SD standard deviation

* Denotes a non-normally distributed variable with p value calculated using Wilcoxon signed-rank test; confidence interval not calculated

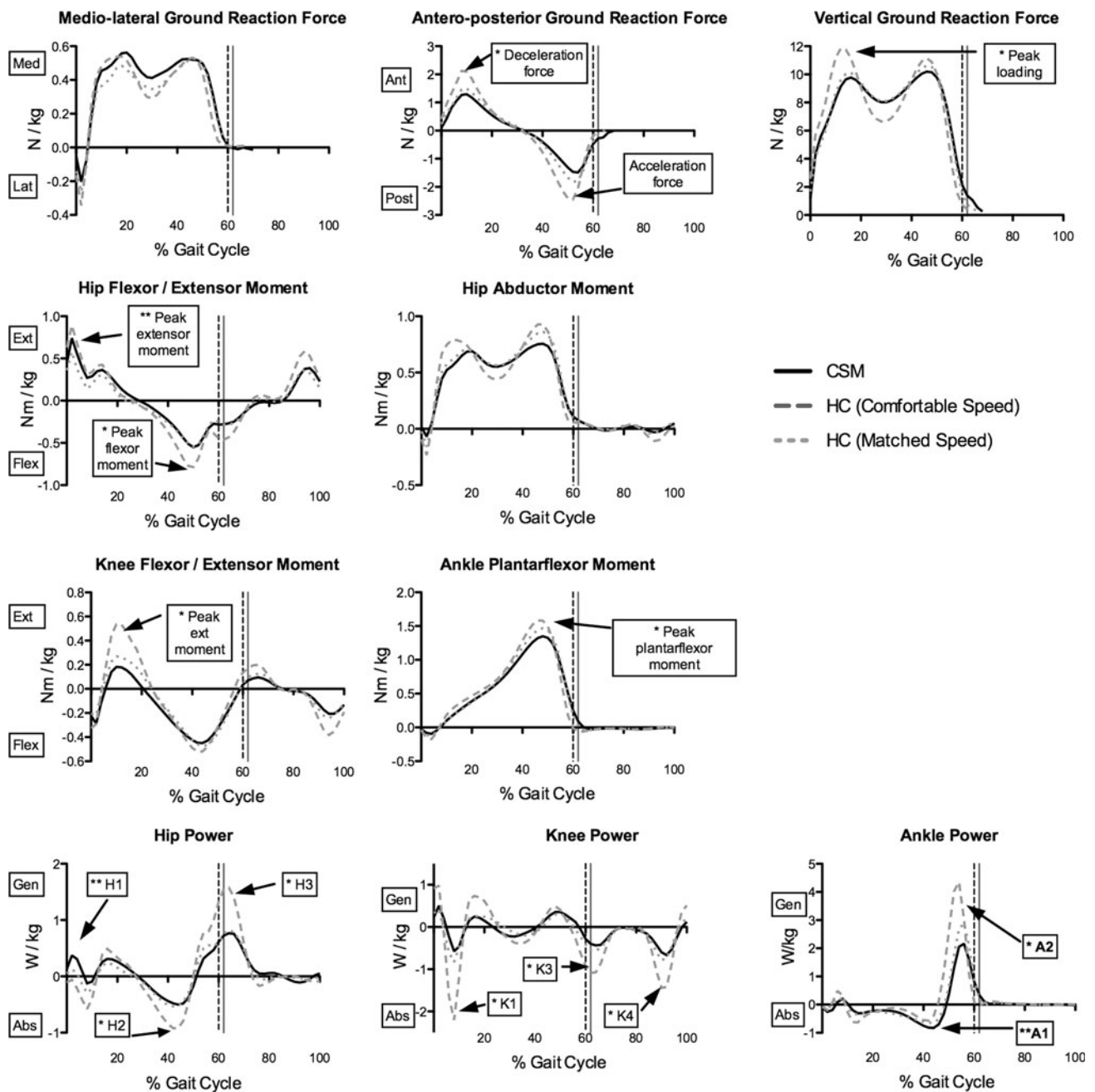[a] Standard error of measurement, calculated in a prior study [18]

Fig. 2 Kinetic curves of CSM and HC participants. Significant differences between HC (*dashed lines*) and CSM (*continuous line*) are indicated using *boxes* and *arrows*; *asterisks* denotes significant difference at comfortable speed only; *double asterisks* denotes significant difference at matched speed only. *Med* medial, *Lat* lateral, *N* Newtons, *kg* kilograms, *Nm* Newton metres, *Ant* anterior, *Post* posterior, *Ext* extensor, *Flex* flexor, *W* Watts, *kg* kilograms, *Gen* generation, *Abs* absorption, *H* Hip power peak, *K* knee power peak, *A* ankle power peak. *Vertical dashed line* indicates toe-off for both groups

contralateral push off at pre-swing, premature cessation of swing due to hyperactivity of the hamstrings or limited hip extension on the stance leg [19].

Kinematic and kinetic analyses provided further insight into the underlying movement patterns. Peak hip extension showed a mean reduction of 3° in CSM compared to HC. This did not reach statistical significance; however, it caused a significant reduction in total sagittal plane range

at the hip. 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 [20]. This may have contributed to the shorter step lengths and reduced single support duration of the CSM cohort.

Table 5 Peak kinetic parameters of CSM and HC participants

Variable	CSM data		Comfortable speed comparison							Matched speed comparison							SEM[a]
	CSM		HC		Difference		95 % Confidence intervals		p value	HC		Difference		95 % Confidence intervals		p value	
	Mean	SD	Mean	SD	Mean	SD				Mean	SD	Mean	SD				
Medio-lateral GRF	0.66	0.19	0.59	0.19	0.07	0.27	-0.07	0.21	0.3	0.59	0.19	0.07	0.27	-0.07	0.21	0.3	0.05
AP GRF, deceleration	1.39	0.46	2.10	0.53	-0.71	0.61	-1.03	-0.38	0.0003	1.53	0.45	-0.13	0.44	-0.37	0.10	0.24	0.16
AP GRF, acceleration	-1.60	0.52	-2.53	0.41	0.93	0.59	0.61	1.24	<0.0001	-1.90	0.45	0.30	0.45	0.05	0.54	0.02	0.10
Vertical GRF	10.58	0.74	12.23	1.42	-1.65	1.57	b	b	0.0004b	10.82	0.57	-0.24	0.60	-0.56	0.08	0.12	0.40
Hip extensor moment	0.78	0.20	0.88	0.22	-0.10	0.21	-0.21	0.01	0.075	0.61	0.23	0.16	0.23	0.04	0.29	0.01	0.122
Hip flexor moment	-0.62	0.27	-0.81	0.23	0.19	0.28	0.04	0.34	0.02	-0.58	0.27	-0.04	0.26	-0.18	0.10	0.5	0.088
Hip abductor moment	0.84	0.18	0.98	0.22	-0.14	0.30	-0.30	0.02	0.08	0.90	0.21	-0.06	0.25	-0.19	0.07	0.3	0.069
Knee extensor moment	0.27	0.18	0.56	0.26	-0.30	0.27	-0.44	-0.15	0.0005	0.33	0.22	-0.06	0.21	-0.18	0.05	0.25	0.056
Knee flexor moment	-0.52	0.11	-0.57	0.14	0.06	0.17	-0.03	0.14	0.2	-0.51	0.16	0.00	0.18	-0.10	0.09	0.95	0.058
Ankle plantarflexor moment	1.41	0.21	1.60	0.12	-0.18	0.18	-0.28	-0.09	0.0007	1.48	0.13	-0.07	0.17	-0.16	0.02	0.12	0.053
Hip power generation, H1	0.51	0.29	0.58	0.41	-0.07	0.45	-0.31	0.17	0.5	0.32	0.25	0.19	0.36	0.00	0.38	0.05	0.19
Hip power absorption, H2	-0.61	0.27	-0.96	0.39	0.35	0.41	0.13	0.56	0.004	-0.57	0.31	-0.05	0.28	-0.20	0.10	0.5	0.10
Hip power generation, H3	1.00	0.45	1.71	0.47	-0.71	0.54	-1.00	-0.42	0.0001	0.94	0.45	0.06	0.40	-0.15	0.28	0.54	0.12
Knee power absorption, K1	-0.70	0.87	-2.10	1.36	1.40	1.47	0.62	2.18	0.002	-0.84	0.56	0.15	0.73	-0.24	0.53	0.43	0.42
Knee power generation, K2	0.59	0.28	0.97	0.33	-0.38	0.39	-0.58	-0.17	0.0015	0.56	0.25	0.03	0.31	-0.13	0.19	0.7	0.17
Knee power absorption, K3	-0.68	0.42	-1.21	0.50	0.53	0.63	0.20	0.87	0.004	-0.66	0.44	-0.02	0.50	-0.28	0.25	0.9	0.11
Knee power absorption, K4	-0.70	0.27	-1.56	0.63	0.86	0.69	0.49	1.22	0.0002	-0.84	0.44	0.14	0.41	-0.08	0.36	0.19	0.12
Ankle power absorption, A1	-0.99	0.34	-0.92	0.24	-0.07	0.29	-0.22	0.09	0.37	-0.80	0.24	-0.19	0.31	-0.36	-0.02	0.03	0.12
Ankle power generation, A2	2.82	1.34	4.85	1.19	-2.03	1.56	-2.86	-1.20	0.0001	3.32	1.09	-0.50	1.04	-1.06	0.06	0.075	0.36

GRFs are reported in Newtons per kilogram, moments in Newton metres per kilogram, and powers in watts per kilogram

SD standard deviation, AP antero-posterior, GRF ground reaction force, SEM standard error of measurement

[a] SEM calculated in a previous study [18]

[b] Denotes non-normally distributed data tested with a Wilcoxon signed-rank test; confidence intervals were not calculated in this case

CSM participants showed reduced knee flexion in both stance and swing. Power absorption peaks at loading response and initial swing were also reduced, confirming that the losses of knee flexion in stance and swing were associated with reduced eccentric activity.

Peak ankle plantarflexion at the pre-swing phase was lower in CSM, and associated with reduced plantarflexor moments and peak ankle power generation. The plantarflexors provide most of the body's support during pre-swing [21]. The loss of their concentric power burst at this point would have limited the propulsion of the leg into swing [22]. This lack of propulsion was not compensated by an increase in the H3 hip power generation peak, as noted in other populations [23]. An effective swing phase, and therefore stride length, depends on the generation of sufficient momentum at the toe-off phase of gait [22], and the lack thereof could have contributed to the CSM participants' shorter stride lengths and reduced single support times.

The matched speed condition was designed to distinguish true differences between CSM and HC gait patterns from speed-dependent differences. Without controlling for speed, a difference in a kinematic or kinetic parameter could simply restate the fact that people with CSM walk more slowly, rather than providing insight into the causes of the slower gait [2]. The abnormal features at matched speed indicated that there were fundamental changes in the biomechanical strategies of gait in CSM. For example, single support time was significantly shorter in CSM than in HC participants at matched speed, supporting the hypothesis that people with CSM lacked either stability in stance or adequate propulsive power in pre-swing [22].

Reductions in peak ankle dorsiflexion, plantarflexion and knee flexion in swing further supported the hypothesis of inadequate propulsion [24]. Similar patterns have been reported in the paretic limbs of people with stroke [2]. Furthermore, the increase in hip power generation and extensor moment at loading response in CSM is a known compensatory mechanism for a disruption of forward progression during stance [22]. It indicated that hip extensors were activated more intensely to transfer the trunk over the supporting limb, compensating for a lack of momentum that would usually be present from pre-swing of the previous stride.

This study identified the key biomechanical strategies underlying gait impairment in CSM. Clinically, 3DGA offers the potential for sensitive, detailed and reliable evaluation of change following surgery for CSM, as well as detailed monitoring of disease progression and the opportunity to develop rehabilitation strategies based on key gait impairments identified by this study. Our study included only participants with clear clinical and radiological evidence of CSM. Given the difficulties in diagnosing early-stage or uncertain CSM [25], 3DGA could play a role in informing the diagnostic process through a detailed evaluation of gait in people with suspected, but unconfirmed disease.

Conclusion

This study identified and described key features of gait in CSM that were independent of speed and could be attributed to the biomechanical effects of this neurological impairment. Limited propulsion, possibly due to paresis of the distal lower limb muscles with some compensation by the proximal hip musculature, was hypothesised as a likely impairment. Future studies should examine the effect of decompressive surgery on these gait features. The role of 3DGA in identifying sub-clinical changes in gait in early or uncertain CSM should also be determined.

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