

Electromyographic characteristics of gait impairment in cervical spondylotic myelopathy

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Abstract

Aim Gait impairment in cervical spondylotic myelopathy (CSM) is characterised by a number of kinematic and kinetic abnormalities. Surface electromyography (EMG) can evaluate the contributions of individual muscles to a movement pattern and provide insight into the underlying impairments that characterise an abnormal gait. This study aimed to analyse EMG signals from major lower limb muscles in people with CSM and healthy controls during gait.

Methods Sixteen people with radiologically confirmed CSM and 16 matched healthy controls participated in gait analysis. Surface EMG was recorded during walking from four lower limb muscles bilaterally. The timing of muscle activation, relative amplitudes of each burst of activity and baseline activation during gait, and the muscles' responses to lengthening as a measure of spasticity were compared using previously validated methods of EMG analysis.

Results Compared to healthy controls, people with CSM had prolonged duration of activation of biceps femoris (12.5 % longer) and tibialis anterior (12.4 %), prolonged co-activation of rectus femoris and biceps femoris (5.14 %), and impaired scaling of the amplitude of rectus femoris and biceps femoris. Muscle activation in response to lengthening was similar between groups.

Conclusion The results provide evidence for paresis as a contributory factor to gait impairment in CSM, indicated by impaired amplitude and the need for proximal co-activation to compensate for lack of distal power generation. Poor proprioception may have contributed to prolonged activation of tibialis anterior. Analysis of muscle responses to lengthening suggested that spasticity was not an important contributor. These findings have implications for the assessment and rehabilitation of gait impairment in CSM.

Keywords Cervical myelopathy · Gait · Rehabilitation · Electromyography

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Introduction

Gait impairment is a primary symptom of cervical spondylotic myelopathy (CSM). Our laboratory previously demonstrated that people with CSM exhibited significant abnormalities in several kinematic and kinetic gait parameters, particularly at the knee and ankle [1]. These features reflected fundamental differences in the motor strategies adopted by people with CSM as a result of their neurological deficits. Limited propulsion, possibly due to paresis of the distal lower limb muscles with some compensation by the proximal hip musculature, was hypothesised as the underlying impairment [1].

Analysis of the electromyography (EMG) signals generated during gait allows for direct interpretation of the

biological signals responsible for muscle activation [2]. EMG can evaluate the contributions of individual muscles to a gait pattern, providing greater insight into the underlying cause of a deficit. Moorthy et al. [3] captured EMG signals from eight lower limb muscles during gait in six people with CSM. All muscles appeared to show prolonged duration of activation and delayed onset in relaxation, suggesting a problem with co-activation or spasticity; however, signals were only interpreted visually with no quantitative analysis. A more complete analysis of EMG, including objective measurement of the timing and amplitude of activity, is lacking in CSM at present. Such parameters are critical in the interpretation of neurological gait disorders [2].

Surface EMG studies in other neurological conditions have successfully measured features of muscular activation including timing, amplitude and response to lengthening. In healthy individuals, the timing of the onset and offset of muscle activation occurs in a predictable manner, appropriate to the tasks of each gait cycle phase [4]. Alterations in this temporal pattern could reflect a primary pathology, such as spasticity causing inappropriate muscle activation in response to stretch [5] or weakness preventing a normal burst of activity. Prolonged muscle activation or co-activation could also be a secondary compensation to provide stability where strength or balance is impaired [6]. The second parameter, EMG amplitude, can indicate whether a muscle's level of activation is appropriately scaled to the demands of the motor task. EMG cannot directly measure or infer muscle force [7]. Instead, the amplitude of a muscle's EMG signal during a burst of activity relative to its baseline amplitude indicates the ability to selectively recruit and de-recruit that muscle when required [8]. Finally, spasticity is of particular interest in the CSM gait as is often considered to be the underlying impairment [9]. Studies on cerebral palsy [10] and stroke [5] have combined the interpretation of EMG with kinematic analysis of muscle length to determine whether inappropriate contraction of a muscle in response to stretch is a feature and to quantify its severity.

The aims of this study were: (1) to measure the timing of muscle activation in people with CSM compared to healthy controls during gait; (2) to quantify the relative amplitude of the EMG signal during bursts of muscle activity and relaxation; and (3) to determine the extent to which inappropriate muscle activation in response to lengthening, namely spasticity, is a feature of gait in CSM.

Methods

Participants

Approval was obtained from the ethics (medical research) committee of the university teaching hospital where the

study took place. 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. 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.

Each CSM participant was matched to a healthy control of the same age (± 5 years) and gender. Healthy controls were recruited from a local population and had no symptomatic musculoskeletal, neurological or respiratory impairment that would hinder gait analysis. Controls were analysed at the same [± 0.1 metre/second (m/s)] gait speed as their matched CSM participants to avoid any confounding effect of speed on EMG data. All participants gave informed consent.

Gait analysis

Participants underwent three-dimensional gait analysis using a Vicon[®] 250 Motion Analysis system (Oxford Metrics Ltd., Oxford, UK), Kistler force plate (Kistler Group, Winterthur, Switzerland) and Motion Lab Systems MA-300 EMG system (Motion Lab Systems Inc, Baton Rouge, LA, USA). Surface EMG signals were recorded from the following muscles bilaterally: rectus femoris (RF), biceps femoris (BF), tibialis anterior (TA) and medial head of gastrocnemius (MG), using double-differentiated pre-amplified stainless steel electrodes with a common mode rejection ratio of 100 decibels (dB) at 65 Hertz (Hz). The skin underlying the electrode was shaved and cleaned to improve electrode–skin contact and reduce impedance. Electrode placement followed the guidelines of SENIAM [11]. A reference electrode was placed over C7. Signals were collected across a bandwidth of 20–500 Hz with a signal-to-noise ratio of at least 50 dB, amplified with a gain range of 2,000–13,200 and sampled into a PC at 1,000 Hz using a 32-channel DI-720 analogue to digital convertor with 12-bit resolution (DATAQ Instruments Inc., Akron, Ohio, USA). A resting signal from the participants resting in supine and a maximal contraction signal from a manual muscle test were recorded from each muscle to confirm optimal detection. EMG data were recorded during ten gait trials at self-selected walking speed over a 12-m walkway. The full gait analysis protocol has been described previously [1]. Data were stored on a PC in C3D format.

Data processing

EMG signals from one left and right gait cycle from each trial were imported into MATLAB[®] (Mathworks Inc., Natick, MA, USA) and filtered with a fourth-order Butterworth low-pass filter of 400 Hz and a second-order Butterworth high-pass filter of 25 Hz, applied in forward and reverse directions to remove motion artefact [12]. Activation of each muscle was detected from the EMG signal in MATLAB using a double-threshold method (DTM) shown to be valid and reliable in the CSM population [13]. The amplitude of each EMG signal was extracted by calculating the root mean square (RMS) of the signal over a 30-ms window with 20-ms overlap [14]. Due to significant inter-participant variability in RMS amplitude [15], signals were normalised by expressing the RMS amplitude at each time point as a percentage of the maximum amplitude obtained during gait [16]. The mean normalised amplitude of each burst of muscle activity, detected by the DTM algorithm as a period of at least 50 ms when the muscle was active, and the mean normalised amplitude of the signal at baseline during gait, defined as the time when the algorithm detected no muscle activity, were extracted (Fig. 1).

Spasticity during gait was determined by the response of each muscle to stretch. Muscle length and lengthening velocity were calculated from kinematic data using a method described by Winter [17]. A muscle's resting length was defined as its length in the anatomical position, with hips, knees and ankles at 0° of flexion. Its length at each point during gait was a function of the angle of the joints it crossed, its angle of pennation and three or six mathematical constants, expressed in relation to its resting length. One key lengthening phase was examined for each muscle: BF in terminal swing, MG in mid stance, TA in pre swing and RF in pre swing. Two parameters were extracted to describe spasticity: (1) the muscle's lengthening velocity at the point of onset of EMG activity during the lengthening phase, termed the lengthening velocity threshold (LVT) [10] and (2) the time of EMG activity onset, expressed as a percentage of gait cycle time (Fig. 2). A spastic muscle would be expected to contract prematurely and at a lower LVT than one with normal tone [10].

Statistical analysis

Data were checked visually and quantitatively for normal distribution. Normally distributed data for CSM participants and healthy controls were compared using paired *t* tests. Non-parametric data were compared with Wilcoxon signed-rank tests. All statistics were performed in Stata (StataCorp, Texas, USA) with significance at $p < 0.05$.

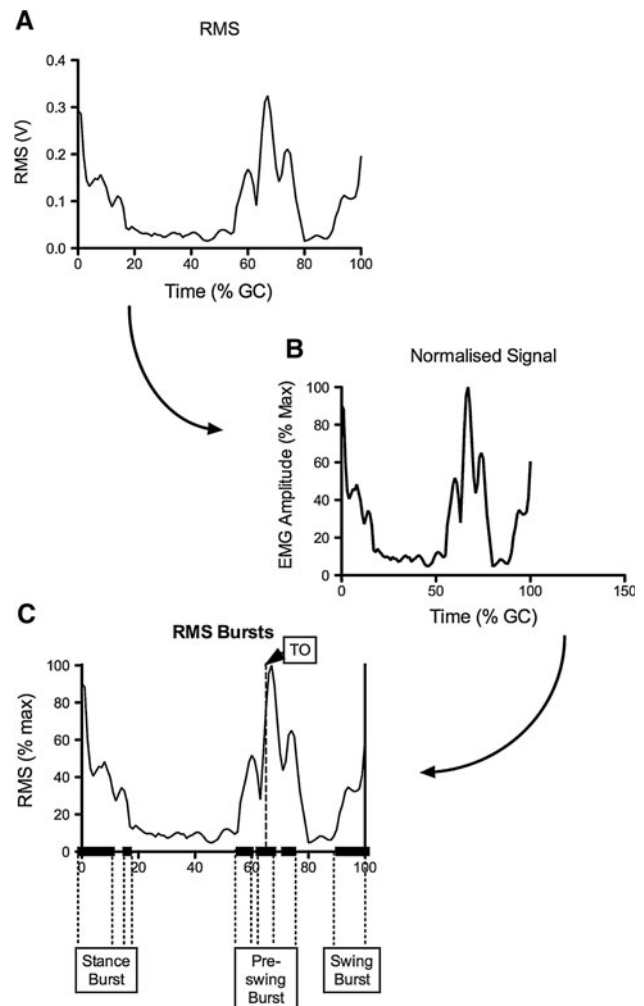


Fig. 1 **a** Root-mean-square amplitude of rectus femoris muscle over a gait cycle from one participant with CSM, **b** normalisation and **c** extraction of amplitude over bursts of muscle activation. *TO* toe off, *RMS* root mean square, *V* Volts, *GC* gait cycle, *% max* percentage of maximum RMS amplitude

Results

Sixteen participants with CSM (eight female), mean age 55 years (range 35–73), were recruited between December 2008 and December 2010. Participants reported symptoms of myelopathy for a median of 36 months (range 5 months–35 years). The median mJOA score was 11 (range 8–13) and the median Nurick score was 3 (range 1–4). All participants tested positive for one or more of the following clinical signs of CSM: hyperreflexia, clonus, upgoing plantar responses, increased tone and positive Hoffman's sign. Participants were matched to 16 healthy controls of the same gender and age. The mean difference in age between patients and controls was 0.4 years (range 5 years). There were no significant differences in weight or height.

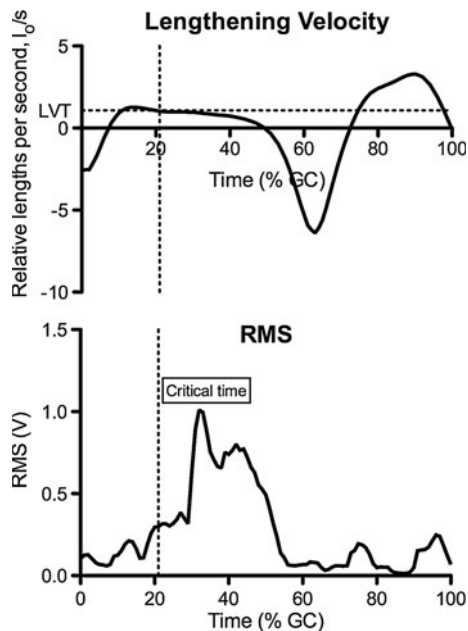


Fig. 2 Muscle lengthening velocity for medial gastrocnemius and calculation of lengthening velocity threshold and time of muscle activation during lengthening. *LVT* lengthening velocity threshold, the lengthening velocity at which muscle activation occurs. In this case, *LVT* = 1.1 relative lengths per second. Visual interpretation of the graph indicates that the onset of muscle activation on EMG does not occur at peak lengthening velocity, indicating subjectively that this muscle allows some yield before contracting and suggesting a normal (non-spastic) response. Critical time = time of onset of muscle activation during lengthening; in this graph critical time = 21 %. Data are from a representative participant with CSM

Analysis of the timing of muscle activation in gait (Table 1) found that the duration of activation of BF and TA were significantly prolonged in CSM. The magnitude of the differences in activation time, 12.5 % longer for BF and 12.4 % for TA, exceeded the standard errors of measurement (SEM) of 9.3 and 5.5 % for BF and TA, respectively [13]. The activation duration of MG and RF showed a non-significant trend of longer activation time in CSM of 8.5 % for RF ($p = 0.07$) and 4.9 % for MG ($p = 0.14$) and exceeded the SEM for RF (5.5 %) and MG (4.4 %) [13]. Co-activation between RF and BF was significantly longer in CSM (14.4 %) compared to controls (9.3 %) ($p = 0.013$).

Table 2 shows the results for the amplitude of EMG activity in gait. CSM participants showed statistically higher normalised amplitudes during the inactive phases of RF (CSM 17.8 % of maximal amplitude, control 13.2 %, $p = 0.02$) and BF (CSM 15.5 %, healthy control 11.7 % $p = 0.01$), but no differences in the amplitude during bursts of activity.

In relation to spasticity, the time of onset of RF and TA activity during lengthening occurred significantly earlier in CSM than in healthy controls (Table 3). *LVTs* were not

lower in CSM, indicating that these muscles were not firing prematurely as a pathological response to stretch.

Discussion

This study aimed to analyse EMG signals from major lower limb muscles in people with CSM and healthy controls during gait. Participants had moderately severe CSM for a median of 36 months, an important prognostic indicator in predicting outcome [18]. We identified key abnormalities in muscle activity during gait, particularly the prolonged activation of RF and TA, and co-activation of BF and RF. Prolonged muscle activation has been attributed to impaired corticospinal activity and defective motor commands [19]. Timing abnormalities could therefore be a direct consequence of spinal cord pathology.

Recently, research has provided evidence for timing abnormalities as adaptive compensatory strategies. One paper studied a cohort of people with a variety of orthopaedic conditions, including total hip replacement, club foot, tibial fracture and bony rotational deformities. Prolonged duration of muscle activity during gait was found in 66 % of the lower limbs studied [6]. These participants had normal neurology and therefore the prolonged activation was attributed to increased proximal stabilisation to compensate for weakness. In another study, the non-paretic limb of people with stroke was found to demonstrate greater co-activation than the paretic side, a finding considered to reflect adaptive behaviour by the non-paretic limb to help maintain postural stability [20]. It is possible that people with CSM used a co-activation strategy for similar reasons.

Our EMG analysis found that prolonged activation was more evident in the proximal thigh muscles, while most kinematic and kinetic abnormalities occurred at the knee and ankle [1]. This may be explained by the need for greater proximal co-activation 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 power generated at loading response to facilitate forward translation of the trunk over the supporting limb where the contra-lateral pre-swing phase has failed to generate adequate propulsion [21]. The prolonged activation of TA was not accompanied by co-activation with MG. Its isolated prolonged activation may have been a strategy to increase the stability of the ankle during stance due to impaired proprioception and joint position sense, previously identified in CSM [22].

EMG amplitude was measured to indicate whether muscle recruitment was of adequate intensity. There were no differences in EMG amplitude of the muscle bursts of

Table 1 Duration of muscle activation and co-activation of CSM and healthy control participants

| Muscle | CSM | | Control | | Difference | | Confidence interval | <i>p</i> value | |
|--------------------------------------|-------|-------|---------|-------|------------|-------|---------------------|----------------|--------|
| | Mean | SD | Mean | SD | Mean | SD | | | |
| Rectus femoris | 32.97 | 18.22 | 24.45 | 6.50 | 8.53 | 17.22 | −0.65 | 17.70 | 0.07 |
| Biceps femoris | 31.81 | 10.96 | 19.30 | 6.52 | 12.51 | 9.58 | 7.40 | 17.61 | 0.0001 |
| Tibialis anterior | 42.16 | 9.86 | 29.76 | 10.88 | 12.40 | 15.64 | 4.06 | 20.74 | 0.006 |
| Medial gastrocnemius | 31.75 | 9.68 | 26.81 | 8.03 | 4.95 | 12.75 | −1.85 | 11.74 | 0.14 |
| Rectus–biceps co-activation | 14.44 | 8.73 | 9.30 | 3.79 | 5.14 | 7.26 | 1.27 | 9.01 | 0.013 |
| Tibialis–gastrocnemius co-activation | 7.47 | 7.91 | 3.93 | 3.60 | 3.54 | 9.15 | −1.34 | 8.41 | 0.14 |

Activation times are expressed as a percentage of gait cycle duration

SD standard deviation

Table 2 Amplitude of muscle activity bursts and baseline phases during gait

| Muscle burst | CSM | | Control | | Difference | | Confidence interval | <i>p</i> value | |
|---------------------|-------|-------|---------|-------|------------|-------|---------------------|----------------|-------|
| | Mean | SD | Mean | SD | Mean | SD | | | |
| RF loading response | 56.15 | 9.82 | 57.25 | 6.39 | −1.10 | 7.80 | −5.26 | 3.06 | 0.58 |
| RF pre swing | 45.70 | 15.86 | 38.10 | 14.57 | 7.6 | 17.99 | * | * | 0.58* |
| RF swing | 19.34 | 12.21 | 30.06 | 10.56 | −10.72 | 25.93 | * | * | 0.11* |
| RF baseline | 17.86 | 6.08 | 13.28 | 3.18 | 4.59 | 6.69 | 1.02 | 8.16 | 0.02 |
| BF stance | 47.63 | 11.10 | 53.99 | 8.32 | −6.36 | 11.12 | −11.20 | 1.11 | 0.1 |
| BF swing | 43.87 | 12.88 | 38.49 | 13.35 | 5.38 | 15.38 | −2.82 | 13.57 | 0.18 |
| BF baseline | 15.55 | 4.03 | 11.70 | 3.29 | 3.85 | 5.54 | 0.90 | 6.80 | 0.014 |
| TA stance | 55.78 | 8.68 | 58.66 | 11.81 | −2.88 | 18.28 | −12.62 | 6.86 | 0.54 |
| TA swing | 47.73 | 6.72 | 42.44 | 7.84 | 5.29 | 11.34 | −0.75 | 11.33 | 0.08 |
| TA baseline | 15.91 | 4.23 | 13.35 | 3.11 | 2.56 | 5.19 | −0.21 | 5.33 | 0.07 |
| MG stance | 57.35 | 6.97 | 58.93 | 5.10 | −1.58 | 9.31 | −6.54 | 3.38 | 0.51 |
| MG baseline | 13.29 | 3.42 | 10.93 | 5.33 | 2.37 | 6.68 | −1.20 | 5.93 | 0.18 |

Amplitude is expressed as a percentage of 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 asterisk denotes that the groups were compared using the Wilcoxon signed-rank test

Table 3 Locomotor-specific measure of spasticity

| Measure | CSM | | Control | | Difference | | Confidence interval | <i>p</i> value | |
|--------------------|-------|------|---------|------|------------|------|---------------------|----------------|--------|
| | Mean | SD | Mean | SD | Mean | SD | | | |
| RF LVT (l_0/s) | 2.78 | 1.12 | 3.26 | 1.20 | −0.49 | 1.46 | −1.26 | 0.29 | 0.2 |
| RF Onset (% GC) | 84.08 | 6.70 | 89.40 | 5.07 | −5.32 | 8.06 | −9.62 | −1.02 | 0.02** |
| BF LVT (l_0/s) | 1.47 | 0.43 | 1.11 | 0.40 | 0.36 | 0.51 | 0.09 | 0.63 | 0.01* |
| BF Onset (% GC) | 79.49 | 3.86 | 82.76 | 5.53 | −3.26 | 7.64 | −7.33 | 0.81 | 0.11 |
| TA LVT (l_0/s) | 1.02 | 0.71 | 1.44 | 0.94 | −0.42 | 0.96 | −0.93 | 0.10 | 0.1 |
| TA Onset (% GC) | 56.83 | 5.24 | 61.27 | 4.27 | −4.45 | 7.21 | −8.29 | −0.61 | 0.03 |
| MG LVT (l_0/s) | 0.50 | 0.71 | 0.71 | 0.49 | −0.21 | 0.76 | −0.61 | 0.20 | 0.29 |
| MG Onset (% GC) | 20.39 | 9.13 | 18.33 | 4.43 | 2.06 | 9.02 | −2.75 | 6.87 | 0.38 |

Lengthening velocity threshold (LVT) is expressed as relative lengths per second, l_0/s

Critical time of EMG onset during lengthening (t_c) is expressed as a percentage of GC duration, % GC

RF rectus femoris, *BF* biceps femoris, *TA* tibialis anterior, *MG* medial gastrocnemius

* LVT was significantly lower in healthy controls than in CSM participants

** Statistically significant

activity; however, amplitude normalisation may have diluted inter-individual differences. Signals are attenuated by subcutaneous fat, variability in skin impedance and environmental factors such as humidity and temperature [23] and there is no standardised method to determine whether each individual's peak amplitude is high or low compared to normal. However, a significant finding was that the mean baseline amplitude outside activity bursts of BF and RF was higher in CSM. This suggests an inability to scale down the intensity of a muscle's output when its activation was not required, and provides an indication of impaired motor control [8].

The gait pattern associated with CSM is classically described as a “spastic pattern”. 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 [24]. If stretch reflex hyperexcitability contributed to the abnormally prolonged muscle activation, it would be expected that EMG activity would occur at a lower LVT in CSM than in healthy controls, indicating reduced stretch receptor threshold. No evidence of such a phenomenon was found. Dietz (2003) commented that while patients with neurological impairment may exhibit clinical signs of spasticity, the manifestations in gait may be very different [25]. Our findings support this observation: all participants with CSM demonstrated clinical evidence of spasticity, yet these features were not detected during gait.

There are a number of limitations to this method of evaluating spasticity during gait. It cannot distinguish between voluntary and reflexive activation on lengthening. An earlier activation could be an adaptive response to eccentrically control lengthening, rather than a mal-adaptive reflexive response, as may be the case for BF in terminal swing. A further limitation is the lack of discrimination between lengthening of the muscle belly versus its tendon, an important distinction in cerebral palsy [26]. Evaluation of spasticity during gait is a complex task. We are not aware of a method that distinguishes between voluntary and reflexive muscle activation or between tendon and muscle lengthening, while retaining specificity to gait and avoiding the need for unnatural stimuli [5]. Our findings suggest that hyper-excitability of the stretch reflex in CSM did not contribute to the abnormal kinematic and kinetic patterns in CSM, nor was it the cause of excessively prolonged muscle activity during gait.

Conclusion

Gait in CSM is characterised by prolonged duration of activation of RF and TA, prolonged co-activation of RF

and BF and impaired scaling of the amplitude of RF and BF. Spasticity, or hyper-excitability of the stretch reflex, was not a significant contributory feature. Although the interpretation of EMG during gait remains complex and challenging, the findings of impaired selectivity of recruitment and proximal co-activation to compensate for lack of power generation distally indicated that paresis was a likely underlying impairment. Impaired stability due to poor proprioception was suggested by the prolonged activation of TA. These findings have implications for the assessment and rehabilitation of gait in CSM.

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Conflict of interest None.

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