

Clinical Study

# Characterizing gait abnormalities in patients with cervical spondylotic myelopathy: a neuromuscular analysis

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## Abstract

**BACKGROUND CONTEXT:** Gait impairment is a hallmark of cervical spondylotic myelopathy (CSM). It has been shown to affect quality of life but has not been well defined. Further electromyographic (EMG) characterization of the gait cycle may help elucidate the true neuromuscular pathology with implications on prognosis and rehabilitation techniques.

**PURPOSE:** This study compares neuromuscular activity in patients with CSM to that of healthy age-matched controls.

**STUDY DESIGN:** Nonrandomized, prospective, concurrent control cohort study.

**METHODS:** Neuromuscular activity was measured in 40 patients with symptomatic CSM during a series of over-ground gait trials at a self-selected speed before surgical intervention. External oblique, multifidus, erector spinae, rectus femoris, semitendinosus, tibialis anterior, medial gastrocnemius, and medial deltoid were assessed. Identical measurements were taken in 25 healthy control patients. Differences in time of muscle onset, peak EMG, time to peak EMG, and integrated electromyography (iEMG) were assessed using one-way ANOVA.

**RESULTS:** There were no significant differences between patients with CSM and healthy controls with respect to time of muscle contraction onset. Peak EMG muscle activity was significantly higher in the medial deltoid of patients with CSM (39.3% vs. 23.3% sMVC,  $p=.042$ ), but no other differences were seen in the remaining muscles tested. They also demonstrated significantly longer time to peak EMG muscle activity compared with controls in 5 of the 8 muscles tested, including the multifidus (20.2 vs. 16.8 ms,  $p=.050$ ), erector spinae (18.2 vs. 8.9 ms,  $p<.001$ ), semitendinosus (26.3 vs. 22.4 ms,  $p=.037$ ), tibialis anterior (14.7 vs. 11.0 ms,  $p=.050$ ), and medial deltoid (24.2 vs. 9.2 ms,  $p<.001$ ). Compared with controls, patients with CSM demonstrated significantly higher iEMG activity in the semitendinosus (586.5% vs. 272.5 sMVC,  $p=.047$ ) and medial deltoid (87.62% vs. 22.5% sMVC,  $p=.008$ ).

**CONCLUSIONS:** The onset of muscle activity is not delayed in CSM patients, but many key muscles take longer to fully contract. This produces a situation in which patients with CSM are unable to fully fire their muscles with sufficient speed to maintain a normal gait. The core and lower extremity muscles do not contract with increased peak amplitude in response, but the deltoid and hamstring muscles are more active, suggesting compensatory activity as patients attempt to maintain balance. The end result is less efficient ambulation. These findings provide a more nuanced understanding of gait in individuals suffering from CSM and may have implications on rehabilitation protocols. © 2019 Elsevier Inc. All rights reserved.

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## Introduction

Cervical spondylotic myelopathy (CSM) is a degenerative condition of the cervical spine leading to a spectrum of neurological dysfunction [1]. It is the most common etiology of spinal cord dysfunction in patients over the age of 50, with symptoms varying from subtle and nearly asymptomatic to overtly disabled [1]. The typical natural history includes a stepwise decline in neurological function, and spontaneous regression of symptoms is rare [2]. CSM affects patients' quality of life and ability to work [3–5], and it has been shown to place patients at higher risk for hip fractures [6]. Despite CSM being first described in the early 1950s [1], the absence of a pathognomonic sign and the heterogenous presentation of this disease can make the diagnosis difficult.

Both Gorter et al. and Lunsford et al. reported that subtle gait disturbance is usually the first symptom of CSM [7,8]. The gait disturbance in CSM is generally described as having a broad-base, shortened stride length, decreased velocity, and prolonged double support [9]. The existing literature on this gait abnormality is inconsistent—some of studies describe it as spastic [10–12], whereas others suggest a paretic component [13,14]. These conflicting descriptions highlight the difficulty of characterizing the gait cycle in patients with CSM and the need for improved understanding. A more sensitive and objective means to diagnose, study, and prognosticate would therefore be beneficial.

Electromyography (EMG) has become a useful tool for understanding normal and pathologic gait cycles. Surface EMG (sEMG) is especially useful because it is noninvasive and is able to detect both the timing and intensity of superficial muscle activation [15–17]. This information can be analyzed to determine a variety of specific parameters. The time of muscle contraction onset quantifies when muscle electrical activity begins [17–19]. Peak EMG amplitude is the highest value of motor unit activity during the gait cycle and indicates the maximum extent to which a given muscle fires [17,18]. Time to peak EMG measures the duration from the beginning of the gait cycle to maximal contraction [17–19]. Integrated EMG (iEMG) is a summation of all muscle activity over a single gait cycle and represents the total muscle activation over that time period [18].

It is conceivable that EMG might someday be used to quantify CSM severity preoperatively to inform the decision of if and when to proceed with surgery and it could be used postoperatively to benchmark outcomes and identify muscle units in need of strengthening. The purpose of this study was to measure the neuromuscular activity of major muscles in patients with CSM before surgical intervention and compare this profile to that of healthy age-matched controls. Although

the limited existing literature has focused solely on lower extremity EMG, the inclusion of core and upper extremity muscle activity as well as a more robust cadre of EMG measures will more accurately define the neuromuscular pathology in CSM patients, with implications on prognosis and rehabilitation.

## Methods

### Subjects

The sample size needed in this study to approach 80% statistical power was estimated from the data of previously examined lifting literature [9–14,20–23]. A large effect size index of  $f=0.40$  was estimated. With a desired power of 80% ( $1-\beta=0.80$ ) and desired  $\alpha=0.05$ , this effect size index would require a minimum sample size of 26 per group [24]. Data were collected from 40 patients with CSM and 25 healthy volunteers. Both group demographic was statistically similar ( $p<.050$ ; Table 1). To be included in the study, patients had to be diagnosed with CSM by a fellowship-trained spine surgeon. Diagnosis was based on history and physical exam findings of cervical myelopathy (eg, loss of fine motor coordination, gait disturbance, hyperreflexia, and long-track signs) with corresponding advanced imaging demonstrating cervical spinal cord compression. Patients were excluded if they had a history of prior spine or major lower extremity surgery, a history of treatment for lower extremity degenerative joint disease, a body mass index greater than 35, a primary neurological disorder other than CSM (eg, diabetic neuropathy or multiple sclerosis), an inability to stand without assistance, or the potential to be pregnant. Because variability of up to 7% can be observed when comparing EMG findings between younger and older people [25], patients below the age of 50 and those older than 70 were also excluded from the study. Healthy controls between the ages of 50 and 70 years old were recruited from the general population and were excluded from the healthy cohort if they had any previous history of neck pain, back pain, or known lower extremity degenerative joint disease or spinal pathology that had previously required treatment.

Table 1  
Patients demographic characteristics (Mean±SD)

	CSM (N=40)	Controls (N=25)	p Value
Gender % (females)	53	52	>.050
Age (years)	60.4±9.3	56.1±8.9	>.050
Height (m)	1.67±0.1	1.73±0.3	>.050
Weight (kg)	84.85±18.2	72.04±9.4	>.050
BMI	30.65±6.1	27.22±3.4	>.050

*Preparatory procedures*

For each test subject, EMG electrodes were placed bilaterally on the skin overlying the external oblique, multifidus at the level of L5, erector spinae at the level of L1, rectus femoris, semitendinosus, tibialis anterior, and medial gastrocnemius. During a pilot study, some patients with CSM ambulated with increased abduction of the arms so the decision was made to place a sEMG electrode over the medial deltoid to capture motor activity at this location as well. The skin at the recording sites was cleaned with alcohol, shaved if necessary, and then lightly abraded to reduce impedance. After electrode placement, the subjects performed submaximum voluntary contraction (sMVC) of each muscle group to establish a baseline measure for subsequent normalization during functional activities [18].

*Testing procedures and data acquisition*

Each subject performed a series of 5 10-meter gait trials at a comfortable self-selected speed. EMG (Delsys, Inc, Natick, MA, USA) data were recorded simultaneously at 2000 Hz and wirelessly transmitted to the sampling computer using a 16 channel Delsys Trigno unit. The Trigno bandwidth was 200–500 Hz with a signal-to-noise ratio of 1  $\mu$ V root mean square baseline noise. The signal amplification was preset at 2000 times, with an impedance of 10 M $\Omega$  and a common-mode rejection ratio of 100 dB. The EMG data was band-pass filtered between 20 and 450 Hz with a fourth-order, no pass zero-phase-lag Butterworth filter and then fully wave rectified. The filtered EMG data was normalized to the sMVC previously described and further analyzed to identify muscle magnitude and muscle timing onset. The mean normalized amplitude of each burst of muscle activity was detected by double-threshold method algorithm as a period of at least 50 ms while muscle was active [18]. For the purposes of the study, only the right side was considered for analysis of muscular activity during the gait cycle.

*Statistical analyses*

The average of the 5 tests for each study subject was used for further analysis. One-way ANOVA was used to determine differences in gait patterns in CSM patients

compared with healthy controls. All statistical tests were used in an exploratory analysis comparing both groups ( $\alpha=0.05$ ). Statistical analyses were conducted using SPSS, Version 23.0 (IBM, Inc., Chicago, IL, USA).

**Results**

Patients with CSM had an average Nurick grade of  $1.86 \pm 1.11$ . All dependent variables were assessed for distribution normality using the Shapiro-Wilk test (p value range: .04 to .82 and W statistic range: 0.73–0.97 for CSM patients; p value range: .03 to .88 and W statistic range: 0.77–0.97 for controls).

There was no difference between the patients with CSM and healthy controls with respect to time of muscle contraction onset in any muscles tested (Table 2). Peak EMG muscle activity was significantly higher in the medial deltoid (39.3% vs. 23.3% sMVC,  $p=.042$ ) of patients with CSM compared with healthy controls. No difference in peak muscle activity was seen in patients with CSM versus healthy controls in the remaining musculature tested (Table 2).

For patients with CSM, time to peak EMG muscle activity was significantly longer in 5 of the 8 muscles tested, including the multifidus (20.2 vs. 16.8 ms,  $p=.050$ ), erector spinae (18.2 vs. 8.9 ms,  $p<.001$ ), semitendinosus (26.3 vs. 22.4 ms,  $p=.037$ ), tibialis anterior (14.7 vs. 11.0 ms,  $p=.050$ ), and medial deltoid (24.2 vs. 9.2 ms,  $p<.001$ ). No difference between groups was observed in time to peak activity in the external oblique, rectus femoris, or medial gastrocnemius muscles (Fig. 1).

Compared with controls, patients with CSM demonstrated significantly less iEMG of the external oblique (72.0% vs. 152.6% sMVC,  $p=.032$ ). CSM patients also had significantly more iEMG of the semitendinosus (586.5% vs. 272.5% sMVC,  $p=.047$ ) and medial deltoid (87.6% vs. 22.5% sMVC,  $p=.011$ ). The remaining muscles tested did not show any significant difference in activation (Fig. 2).

**Discussion**

In this study, surface EMG was used to detect neural impulses and muscle activity in patients with CSM and compare them to healthy controls. Differences were

Table 2  
Peak EMG and time to onset in tested muscles of patients with cervical spondylotic myelopathy compared with healthy controls

	External oblique	Multifidus	Erector spinae	Rectus femoris	Semi-tendinosus	Tibialis anterior	Medial gastroc	Medial deltoid
<b>Peak EMG (% of sMVC)</b>								
CSM	33.54 $\pm$ 12.23	31.80 $\pm$ 11.76	36.50 $\pm$ 9.76	35.86 $\pm$ 18.70	23.87 $\pm$ 12.17	50.08 $\pm$ 10.00	46.25 $\pm$ 21.77	39.30 $\pm$ 11.04
Healthy	44.58 $\pm$ 0.02	43.60 $\pm$ 15.84	27.25 $\pm$ 6.05	29.85 $\pm$ 12.09	21.01 $\pm$ 9.06	50.23 $\pm$ 13.11	32.87 $\pm$ 11.14	23.25 $\pm$ 6.021
p value	.589	.617	.244	.519	.435	.808	.395	.042*
<b>Time to onset (% of gait cycle)</b>								
CSM	3.32 $\pm$ 0.80	3.44 $\pm$ 0.89	3.37 $\pm$ 0.87	3.45 $\pm$ 0.80	3.44 $\pm$ 0.87	3.44 $\pm$ 0.86	3.53 $\pm$ 0.85	3.41 $\pm$ 0.91
Healthy	3.43 $\pm$ 1.16	3.53 $\pm$ 1.12	3.52 $\pm$ 1.16	3.52 $\pm$ 1.11	3.49 $\pm$ 1.15	3.43 $\pm$ 1.16	3.65 $\pm$ 1.07	3.58 $\pm$ 1.12
p value	.666	.747	.554	.753	.861	.941	.646	.521

CSM, cervical spondylotic myelopathy; EMG, electromyography.

\* Denotes a significant difference between patients with CSM and healthy control subjects.

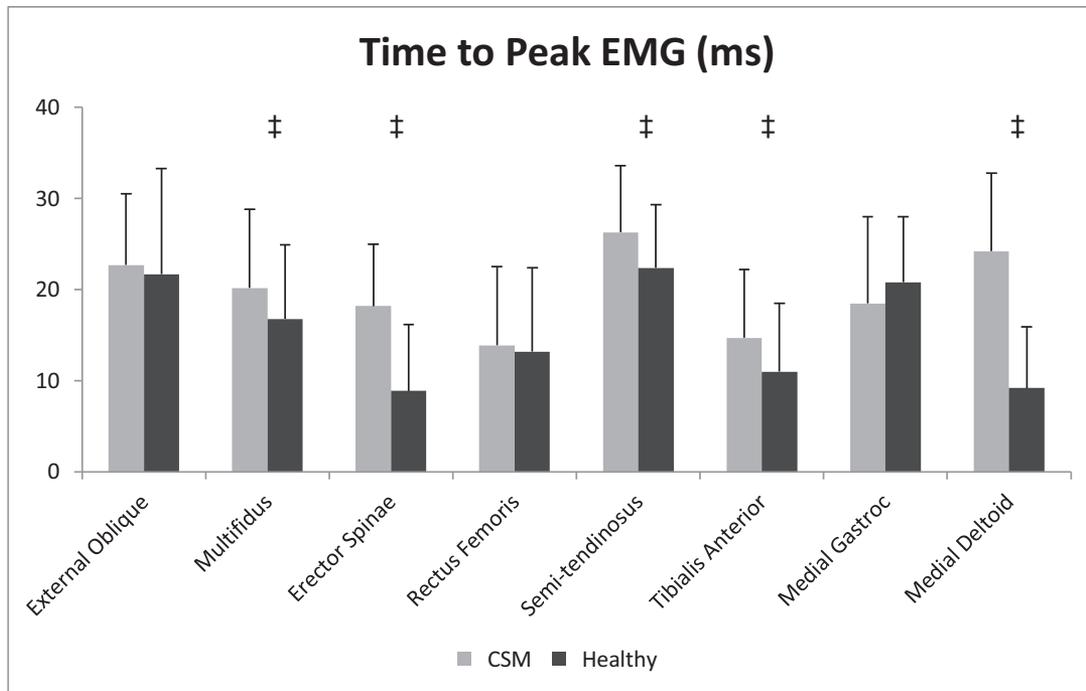


Fig. 1. Time to peak EMG activity (ms) in key muscles during gait in patients with CSM compared with healthy controls. The symbol, ‡, denotes significantly different time to peak EMG activity between patients with CSM and healthy control patients ( $p < .05$ ). CSM, cervical spondylotic myelopathy; EMG, electromyography.

observed in the motor control strategies used by these two groups.

The time of muscle contraction onset did not differ significantly between patients with and those without CSM and

peak EMG values in the core and lower extremity muscles were similar between the CSM and healthy control groups. However, time to peak EMG, which represents how long it takes for full muscle recruitment to occur, was significantly

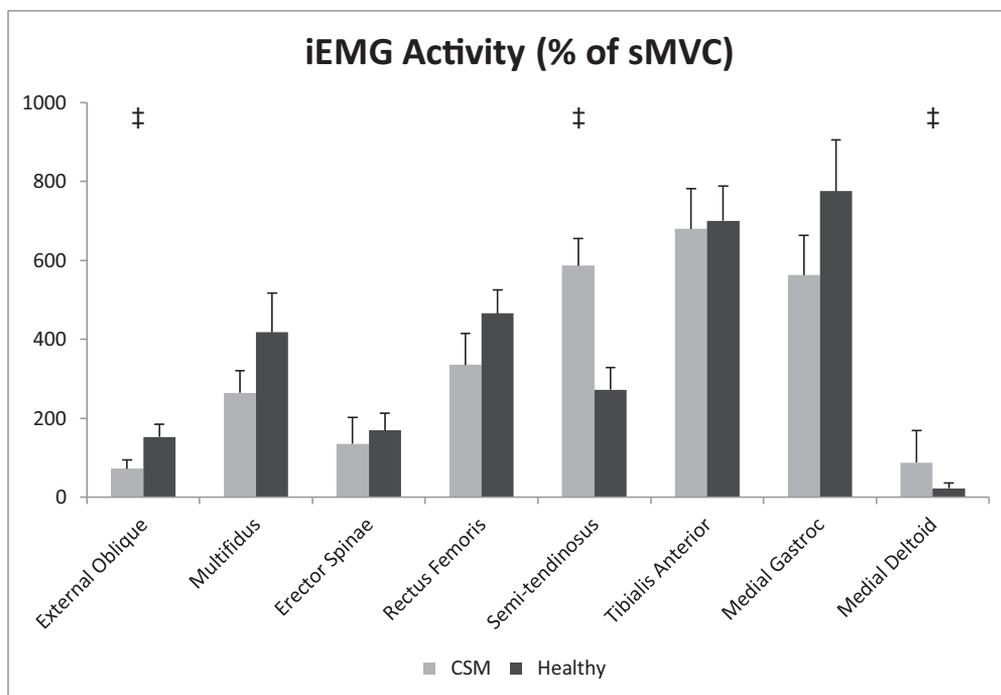


Fig. 2. Integrated EMG activity of key muscles during gait in patients with CSM compared with healthy control subjects. The symbol, ‡, denotes significantly different iEMG activity between patients with CSM and healthy control patients ( $p < .05$ ).

longer in a majority of the muscles tested. These findings suggest that initial neural impulses in patients with CSM are not delayed in reaching the motor units and that the muscles reach a similar degree of maximal contraction as in healthy patients, but the time it takes to fully recruit the muscles is protracted in the setting of CSM.

The gait pattern in CSM has been described as spastic [11,26], but recent work by Malone et al. [13] using sEMG reported normal motor responses to lengthening as well as impaired amplitude generation, suggesting that the disease process is one of paresis rather than spasticity. Authors also observed prolonged coactivation of proximal musculature (rectus femoris and biceps femoris). In contrast, the present study does not support the characterization of myelopathic gait as paretic. No significant differences in lower extremity peak EMG were found when comparing patients with CSM to healthy controls. However, in keeping with Malone et al., significantly higher iEMG values in the semitendinosus were observed herein. Prolonged activation of the biceps femoris generally is thought to be a compensatory response to lack of power generation from the distal lower extremity muscles; it allows patients with gait pathology to control the position of the pelvis and ultimately, balance [13,16,27]. Furthermore, the significantly decreased iEMG activity noted in the external oblique muscles of patients with CSM may be reflective of poor core muscle function and contribute to the instability and gait inefficiencies observed in these patients [27,28].

It is well known that CSM affects upper extremity motor function [29,30]. However, prior EMG studies investigating neuromuscular gait patterns have focused on the lower extremities [9,10,26,31], despite the fact that the upper extremities are known to contribute to locomotion and balance [32–34]. Medial deltoid EMG activity was measured in this study as a marker for upper extremity recruitment during gait. Significantly higher medial deltoid peak EMG and iEMG values were observed in myelopathic patients than in healthy controls, suggesting that the upper extremities are used in a compensatory manner in an attempt to improve balance in patients with CSM.

There were several limitations of this study. First, sEMG data were only collected from right-sided musculature, assuming symmetry bilaterally, in an effort to maximize the number of patients that could be tested within the confines of a limited research budget. However, it is possible that there are differences in motor function between patients' right and left sides. Although incorporation of medial deltoid parameters was an improvement upon the existing literature and demonstrated abnormal recruitment of this muscle, a complete analysis pattern of upper extremity muscle activation during myelopathic gait could not be conducted in the absence of data on other muscle groups. Frailty and sarcopenia were not measured in study subjects. An attempt was made to control for these variables using demographically similar cohorts, but it is possible that patients in the CSM cohort may have been less physically robust, which may have impacted the data. Finally, there are the inherent

limitations of EMG and associated data smoothing errors. Large intersubject variability was observed so uncertainty around reported averages should be noted.

In conclusion, the initiation of muscle activity is not slowed in CSM patients, but many key muscles take longer to reach a state of full contraction. This protracted recruitment produces a state of perpetual falling, in which patients with CSM are unable to fully fire their muscles with sufficient speed to maintain a normal gait. The core and lower extremity muscles do not contract with greater peak amplitude in response; but the deltoid and hamstring muscles are more active, suggesting compensatory activity as patients attempt to maintain balance. The end result is less efficient ambulation. These findings provide a more nuanced understanding of gait in individuals suffering from CSM and may have implications on rehabilitation protocols. Future studies comparing sEMG data in patients with CSM before and after surgical decompression will provide useful information regarding postoperative prognosis.

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