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People with Degenerative Cervical Myelopathy Have Impaired Reactive Balance during Walking

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Abstract

Background: People with degenerative cervical myelopathy are known to have impaired standing balance and walking abilities, but less is known about balance responses during walking.

Research question: The aim of this project was to assess reactive balance impairments during walking in people with degenerative cervical myelopathy (PwDCM). We hypothesized that center of mass motion following perturbations would be larger in PwDCM and gluteus medius electromyographic amplitude responses would be decreased in PwDCM.

Methods: Reactive balance responses were quantified during unanticipated lateral pulls to the waist while treadmill walking. Walking biomechanics data were collected from 10 PwDCM (F=6) and 10 non-myelopathic controls (F=7) using an 8 camera Vicon System (Vicon MX T-Series). Electromyography was collected from lower limb muscles. Participants walked on an instrumented treadmill and received lateral pulls at random intervals and in randomized direction at 5% and 2.5% body mass. Participants walked at 3 prescribed foot placements to control for effects of the size of base of support.

Results: As compared with controls, the perturbation-related positional change of the center of mass motion (ΔCOM) was increased in PwDCM ($p=0.001$) with similar changes in foot placement ($p>0.05$). Change in gluteus medius electromyography, however, was less in PwDCM than in controls ($p<0.001$).

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All authors declare there are no conflicts of interest to report.

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Significance: After experimentally controlling step width, people with mild-to-moderate degenerative cervical myelopathy at least 3 months following cervical spine surgery have impaired reactive balance during walking likely coupled with reduced gluteus medius electromyographic responses. Rehabilitation programs focusing on reactive balance and power are likely necessary for this population.

Keywords

Reactive Balance; Gait; Balance; Dynamic Balance; Posture; Spondylosis; Cervical Spondylotic Myelopathy; Balance Perturbation; Cervical Compressive Myelopathy

INTRODUCTION

Degenerative cervical myelopathy (DCM) is a spinal cord disease resulting from vertebral compression of the cervical spinal cord, impacts approximately 2% of adults. [1–4]. Importantly, people with DCM (PwDCM) have an elevated risk for falls even after surgical decompression due to chronic damage to sensory and motor pathways causing delayed and reduced-amplitude evoked potentials[5–14]. This results in increased postural sway and larger and delayed responses to standing perturbations[10, 14, 15]. Reactive balance during walking, however, may be more contextually appropriate for understanding fall risk, but has not been extensively studied in DCM[16].

Assessment of lateral reactive balance during walking is a growing area of research in several patient populations [17–19]. Reactive balance is typically defined as “the ability to control balance in response to mechanical disturbances” [20]. Relevant variables of interest in response to discrete perturbations in walking are the movement of the center of mass (COM), margin of stability (MOS), step placement, and gluteus medius activity [21, 22]. These variables reflect change in body position within the base of support and neuromuscular control [17, 22–30]. Therefore, a comprehensive assessment of reactive balance during walking would involve the COM, MOS and step placement as global measures and the gluteus medius activity as a muscular controller of lateral stability.

Therefore, the purpose of this study was to quantify impaired reactive balance in PwDCM as compared to controls by measuring kinematic and EMG responses to unexpected lateral perturbations. We hypothesized that PwDCM would have larger COM motion following perturbations during walking than controls. Secondarily, we hypothesized that exaggerated ΔCOM motion would be accompanied by reduced or delayed electromyographic (EMG) responses, especially of the gluteus medius. Likewise, we hypothesized that MOS and foot placement would change more in PwDCM relative to controls. Tertiarily, we hypothesized that ΔCOM motion responses would correlate with clinical measures of balance and myelopathic severity.

METHODS

Participants

Ten PwDCM (F=6) and 10 non-myelopathic controls (F=7) participated in this study. PwDCM were recruited from a single multi-disciplinary spine clinic. PwDCM were

included if they had been diagnosed by a spine surgeon with DCM with previous anterior or posterior decompression surgery >3 months. We recruited post-surgical participants to decrease the likelihood of between-day test session variability. Participants were screened by a spine surgeon or physician assistant. General inclusion criteria were: > 18 years old and able to give information consent. General exclusion criteria were: diagnosis of neurological disease other than DCM, inability to stand or walk unassisted, uncorrected visual or vestibular impairments, or uncontrolled cardiovascular conditions. As such, all participants were rather high functioning with mild myelopathy at time of testing. Upon screening, no recruited participants needed to be excluded based on this criterion. Controls were recruited through re-contacting participants from other studies in our lab, through fliers, and word of mouth. This study was conducted in accordance with the Declaration of Helsinki and approved by the Marquette University Institutional Review Board; protocol number HR-3387. All participants provided informed consent.

Procedures

Participants walked on an instrumented treadmill (Bertec Corporation, Columbus, OH) while receiving 8 lateral waist pulls per step width condition (48 pulls per participant) at random number of step intervals (over 10–40 gait cycles) and in random directions (right/left) to assess reactive balance during walking (Fig 1). Briefly, participants were perturbed using a cable pulley system attached via harness to the participants waist and controlled by a servomotor (Kollmorgen, Radford, VA). This system has been previously described [14, 31]. Participants walked on the treadmill at 3 step widths using visual feedback(12cm, 19cm, and 33cm) [32], and at 70% overground walking speed to allow for safe walking. Participants walked with arms across their chest to prevent arm-cable contact. Overground comfortable walking speed for all participants was assessed from the 10-Meter Walk Test (described below).

Clinical Measures

Prior to balance testing, clinical measures of the modified Japanese Orthopedic Association scale (mJOA), 10-meter walk test, Berg Balance Scale (BBS), and Functional Gait Assessment (FGA) were collected. The mJOA is a myelopathy-specific clinical scale involving 6 items of upper and lower extremity motor function, upper, trunk, and lower extremity sensory function, and sphincter function [33]. The 10-meter Walk Test[34] involves a participant walking along a 10-meter walkway at comfortable and fastest self-selected speeds. Time to complete the middle 6 meters of walking was collected with a manual stop-watch and converted to speed. The BBS is a multi-dimension balance scale involving standing in various postures, seated to standing transitions, functional reaching, stepping, etc. and has been recommended for assessments of DCM [34]. The FGA is a 10 item walking scale that involves walking while performing various balance challenging tasks including head turns, and walking backwards, in tandem and with eyes closed [35].

Outcome Measures

Postural control was quantified by motion capture using a Plug-In-Gait marker model (Vicon Nexus, Oxford, UK) using 8 Vicon cameras (sampled at 100Hz). The primary outcome measure was whole body change in ΔCOM motion in response to perturbations. This was

defined as the largest value from the 3 gait cycles following the onset of pull minus the average of 5-gait cycles prior to the perturbation. COM was estimated using an 8 segment body model (trunk, pelvis, bilateral thighs, shanks and feet) derived from the Plug-In-Gait model [36]. Foot placement in response to perturbations was an additional interpretive kinematic variable and was calculated as the mediolateral distance between the heel markers at heel strike [31]. The largest value from the 3 gait cycles following the onset of pull was used to quantify the response to account for multi-step responses. Change in minimum margin of stability (ΔMOSmin, see calculation below) was obtained similar to ΔCOM motion measurements by extracting the minimum MOS for each of 5 gait cycles prior to pull onset and then used as the baseline. The largest deviation from baseline was considered ΔMOSmin. EMG data (Motion Lab Systems, Baton Rouge, LA) were collected at 1000Hz from the gluteus medius, rectus femoris, medial hamstrings, medial gastrocnemius, and tibialis anterior muscles bilaterally. Prior to surface EMG placement by a single athletic trainer, according to SENIAM guidelines based on anatomical landmarks, the skin was abraded and cleaned with alcohol. EMG signals were referenced to the right side radial styloid process. Kinetic data from the instrumented treadmill and cable pull forces from separate s-shaped force transducers were sampled at 1000Hz.

Perturbation Control

Perturbations were controlled using custom LabVIEW (National Instruments, Austin, Texas, USA) code modified from a previous study [31] to deliver a single lateral waist pull during stance phase. Pulls began at detection of heel strike and continued until the contra-lateral heel strike. This was done to accentuate the motion of the COM in phase with the velocity of the COM [31] over the stance phase of the respective gait cycle. Detection of heel strikes were automatically recorded using zero-crossings of the velocity of the center of pressure collected from the instrumented treadmill. A running average of step time was calculated to estimate the duration of stance phase to ensure proper pull phasing such that peak pull would occur during single limb support. Pulls were delivered at variable inter-step intervals (10–40 gait cycles) and during left/right steps randomly at intensities of 5% and 2.5% body mass.

Visual Feedback

To control for step width, the participant’s center of pressure and target foot placements were projected onto a wall in front of the participants, but not on the treadmill belt. Participants practiced walking with visual feedback until they expressed familiarity with the paradigm. Root mean squared error of step width was not different between groups ($p>0.05$).

Data Processing

Kinematic markers were labelled in Vicon Nexus 1.8.5 (VICON, Oxford, UK), low pass filtered at 6Hz and exported to Matlab (The MathWorks, Natick, MA) for processing. Custom Matlab scripts were written to calculate the mediolateral position of the COM using a weighted average of body segment positions as previously validated [36]. MOSmin was quantified as

$$MOSmin = BOS - \left(COM + vCOM * \sqrt{\frac{l}{g}}\right)$$

where BOS is the lateral boundary of the base of support (the lateral malleolus), vCOM is the velocity of the COM, l is the height of the center of mass, and g is the gravitational constant thus MOS measures the distance between the COM and BOS [26]. Further, MOSmin normalizes COM motion within the base of support. Heel strike and toe off events were calculated using a custom Matlab script as previously done [31], using a combined approach of vertical ground reaction force with a 15N threshold or the angle between the sacrum and toe markers in the sagittal plane if foot crossing to the opposite belt occurred [37]. ΔCOM motion was calculated by further high-pass filtering the COM signal at 0.1Hz using a 4th order zero-phase Butterworth filter and time normalizing to 0–100% of the gait cycle based on heel-strike to heel-strike. Next, peak COM position of the zero-mean high-pass filtered signal in the direction of the pull was calculated for the preceding 5 steps and averaged (baseline). This was subtracted from the high-pass filtered peak COM position of the first 3 steps following onset of waist pulls. The largest positive difference from baseline of the 3 post-pull steps was considered ΔCOM motion. Finally, we extracted the change in foot placement following the same method as COM. Whole body posture is presented in Fig 2 for a representative PwDCM and non-myelopathic control from start of pull, +500ms, and +1000ms.

Peak EMGs from each pre-pull gait cycle were extracted and averaged. The peak EMG of the baseline was then subtracted from post-pull EMG peaks to identify the change in peak EMG amplitude (ΔEMG amplitude). For ΔEMG values, we also extracted the number of gait cycles (delay) following the onset of the perturbation to reach peak value (i.e. pull+0, pull+1, pull+2) which were averaged within each participant. EMG data were filtered with a 4th order, zero phase, band pass filter between 30Hz and 450Hz. A notch filter at 59–61Hz was employed to remove line noise. EMG data were then full-wave rectified and smoothed with a 4th order zero phase low pass filter at 6Hz. EMG data were normalized to the maximal value obtained from each respective muscle from the initial 15 seconds of each respective (foot placement*pull magnitude) combination. EMG amplitude was time normalized to 0–100% of a gait cycle to allow comparisons against EMG activity in unperturbed walking. This was done because PwDCM cannot fully activate their muscles [33, 38] which would result in an overestimation of percent activation if EMG were normalized to MVC values. Normalization was done on a per-condition basis to account for systematic changes in EMG across different foot placements.

Statistical Analysis

All statistical analyses were conducted in SPSS v 26 (IBM, Inc.). The primary outcome variable was ΔCOM motion following perturbations. The secondary mechanistic variables of interest were ΔMOSmin, ΔFoot placement and gluteus medius ΔEMG amplitude following perturbations. Interpretive clinical variables were walking speed, mJOA, BBS, and FGA scores. Primary and secondary outcome variables were compared across group, pull magnitudes, and prescribed foot placement with a linear mixed effect model. Group,

magnitude, and foot placement were all considered fixed effects for the statistical model. Because the distributions of ΔCOM motion, Δgluteus medius amplitude, and foot placement were skewed, these data were natural log transformed prior to statistical modeling. Additionally, ΔMOSmin data were negative and skewed, so these data were inverted then natural log transformed. Comfortable speed and fast walking speed collected from the 10-meter walk test were compared using a linear mixed effects model with speed and group as fixed effects. BBS and FGA were only analyzed for correlation with ΔCOMsway and comfortable walking speed. A sample size calculation was performed based on the initial 5 participants from each group, resulting in a recommended sample size of 6 per group. Therefore, we recruited 10 per group to account for potential dropout. Alpha was set a priori at 0.05 with Sidak correction for multiple comparisons for all tests. A single outlier was identified in the 19cm, 2.5% pull condition for ΔCOM motion or ΔMOSmin (>mean+2.5*standard deviation). Removal of this outlier did not impact statistical results.

RESULTS

Ten PwDCM (6F/4M, 58.80±12.37 years old) and 10 non-myelopathic controls (7F/3M, 55.78±10.09 years old) participated in this study. Characteristics of PwDCM and controls are presented in Table 1.

Center of Mass Motion

ΔCOM motion following waist pulls in walking was larger in PwDCM than in non-myelopathic controls ($F_{1,107.74}$=12.24, p=0.001, Fig 2, Panels B vs C and PwDCM bars vs controls in panels D & E). Further, between group differences remained after normalizing for gait speed (p=0.003).

There was a main effect of pull magnitudes, whereby larger pull magnitudes ($F_{1,107.74}$=168.01, p<0.001, Fig. 2, panel D > panel E) and narrower step widths ($F_{2,84.39}$=4.37, p=0.016, esp. Fig 2 panel D 12cm vs 19cm vs 33cm) resulted in larger sway values. There was a magnitude * step width interaction ($F_{2,84.39}$=6.91, p=0.002) whereby ΔCOM motion values did not differ across step widths in 2.5% pulls (Fig 2E) but increased from 33cm to 19cm to 12cm in 5% pulls (Fig 2D).

Margin of Stability

PwDCM experienced a greater negative ΔMOSmin than controls (F1,84.44=6.05, p=0.016, Fig 3). Larger pull magnitudes ($F_{1,84.44}$=58.54, p<0.001, Fig 3 panel A vs B) and narrower foot placements ($F_{1,75.81}$=5.06, p=0.009, Fig 3, 12cm vs 19cm vs 33cm bars) resulted in larger reductions of MOSmin. There was a magnitude * foot placement interaction (F1,75.81=6.47, p=0.003) whereby ΔMOSmin values did not differ across foot placements in 2.5% pulls but increased from 33cm to 19cm to 12cm in 5% pulls.

Gluteus Medius Amplitude and Latency

PwDCM responded to lateral waist pulls with reduced amplitude modulation of gluteus medius EMG (Fig 4) and took more steps before reaching peak gluteus medius response (Supplementary Fig 2). Gluteus medius ΔEMG was lower in PwDCM than non-myelopathic

controls (main effect of group, $F_{1,107.66}$=16.34, p<0.001, Fig 4 E–F). PwDCM needed more steps, on average, to achieve peak gluteus medius amplitude (Supplementary Fig 2; $F_{1,107.88}$=5.10, p=0.026).

There were main effects of pull magnitude ($F_{1,107.74}$=27.01, p<0.001, Fig 4 E > F) and step width ($F_{2,74.13}$=3.84, p=0.026). There were no interactions for gluteus medius ΔEMG (p>0.05). There was a main effect of step width ($F_{2,69.70}$=3.98, p=0.023), but no significant main effect of pull magnitude ($F_{1,107.88}$=3.81, p=0.054). Overall, participants needed fewer steps to reach peak gluteus medius ΔEMG in 12cm step width than in 19cm step width conditions. There were no significant interactions for number of steps (p>0.32).

Foot Placement

Change in foot placement following perturbations was larger in 5% perturbations than 2.5% perturbations (pooled 5%=0.05±0.03m, pooled 2.5%=0.03±0.01m; $F_{1,82.84}$=15.83, p<0.001). There were no other interactions or any other main effects (p≥0.37 for all tests).

Clinical Correlations

mJOA was associated with 10MWT and FGA scores (Table 2). ΔCOM motion was not significantly associated with mJOA, 10mwt, BBS, or FGA scores (Table 2). Additionally, gluteus medius ΔEMG during narrow step widths at 2.5% pull magnitudes correlated with gait speed, BBS, and FGA (r=0.67, r=0.73, r=0.70, all p<0.05). Gluteus medius ΔEMG and gait speed, BBS, and FGA during all other conditions were non-significant (p>0.05).

Discussion

To our knowledge, this is the first study to quantify reactive balance deficits during walking in PwDCM. Balance impairments during walking manifested as increased ΔCOM motion (Fig 2), greater ΔMOSmin (which accounts for ΔCOM within the base of support) -(Fig 3) and reduced (Fig 4) and delayed (Supp. Fig 2) gluteus medius responses in response to lateral perturbations. Additionally, current measures of myelopathic severity do not strongly correlate with reactive balance.

Degenerative Cervical Myelopathy and Balance

Our findings demonstrate that PwDCM, albeit variable in time post decompression, have reactive balance deficits during walking. These findings are an important extension of previous works that have observed standing balance impairments pre- and post-surgery [10, 11, 14, 15, 39, 40]. These suggest that perturbation-based balance training during walking may be necessary to improve fall risk [11].

Neuromuscular Mechanisms of Balance Impairment in DCM

In this study, PwDCM had larger COM excursions and smaller and delayed modulations of gluteus medius muscle activity. The gluteus medius is the primary muscular contributor to decelerate the COM in the frontal plane during walking [27–30]. Therefore, delays and deficits in activation likely contribute to impaired reactive balance during walking. In PwDCM, previous work has shown delayed gluteus medius responses following standing lateral perturbations [14]. This suggests delayed corticospinal and/or dorsal column

transmission [12, 13, 41]. We found, however, that PwDCM have reduced EMG amplitude and delayed EMG over larger, multi-step, scales possibly suggesting alterations in motor strategy through improper scaling of responses or reduced power output. Therefore, training to increase rate of force development may improve fall risk in PwDCM.

Reactive Balance and Clinical Scales

Our findings suggest that reactive balance responses during walking do not correlate with mJOA scores, however, 10MWT and FGA do. Thus, the mJOA is possibly more related to walking speed and internally generated perturbations than reactive balance. The mJOA, therefore, may not sufficiently capture the complexity of balance impairments [22, 42]. Reactive balance testing may, therefore, be important for balance assessments in this population.

Limitations

Our ability to generalize findings is limited by a low sample size. Nevertheless, we have demonstrated residual reactive balance impairments in this sample of PwDCM, albeit some differences were small magnitude. Additionally, this study is limited by a relatively high functioning heterogeneous cohort of PwDCM measured post-decompression. Subsequent large studies are needed to replicate our findings pre- to post-operative improvements in reactive balance across a representative range of functional capabilities. Finally, we controlled foot placement and had participants walk with arms across their chest which may have altered their balance response.

Conclusions

This study provides evidence that reactive balance in PwDCM is impaired during walking in response to unpredictable lateral waist pulls relative to people without myelopathy, and this does not depend on increased compensatory step width. Furthermore, gluteus medius amplitude responses were reduced possibly due to motor delays in PwDCM. These results highlight the possible need for reactive balance training during walking that seeks to improve the magnitude and timing of gluteus medius responses as an alternative interventions that emphasize increasing baseline step width.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Following surgical decompression, people with degenerative cervical myelopathy have residual balance impairments during walking.
- Impairments exist regardless of pre-perturbation step width.
- People with degenerative cervical myelopathy have a blunted hip abductor muscle response following perturbation

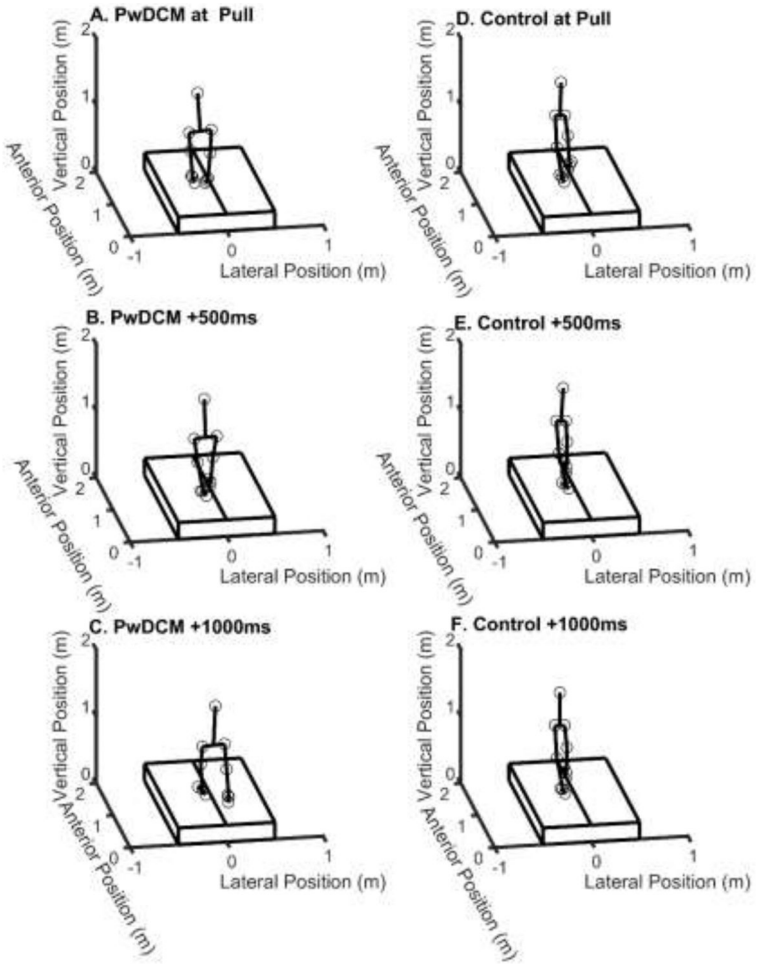

Fig 1.

Still images of a PwDCM (A-C) and control (D-G) from pull start (A/D), +500ms (B/E) after the pull, and +1000ms (C/G) after the onset of the pull. Both pulls were to the right. Note the larger shift in position of C) to the right (pull direction) relative to other cases.

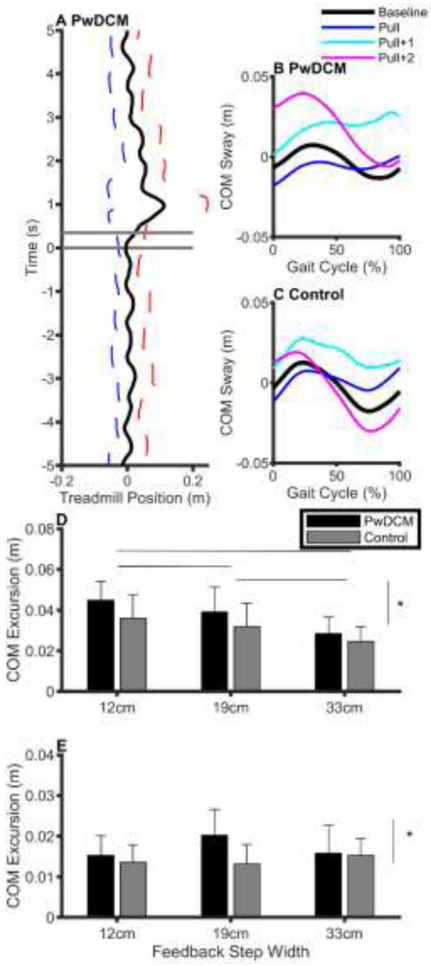

Fig 2.

Center of mass sway following lateral waist pulls. A) demonstrates a raw foot and COM position from a single pull response (ID #4). B) PwDCM (ID #4) single trial COM motion normalized to gait cycle. C) Control single trial COM trial normalized to gait cycle. D-E) aggregated data for 5% and 2.5% pulls respectively. Asterisks indicate main effect of myelopathy. Horizontal bars indicate significant differences between conditions.

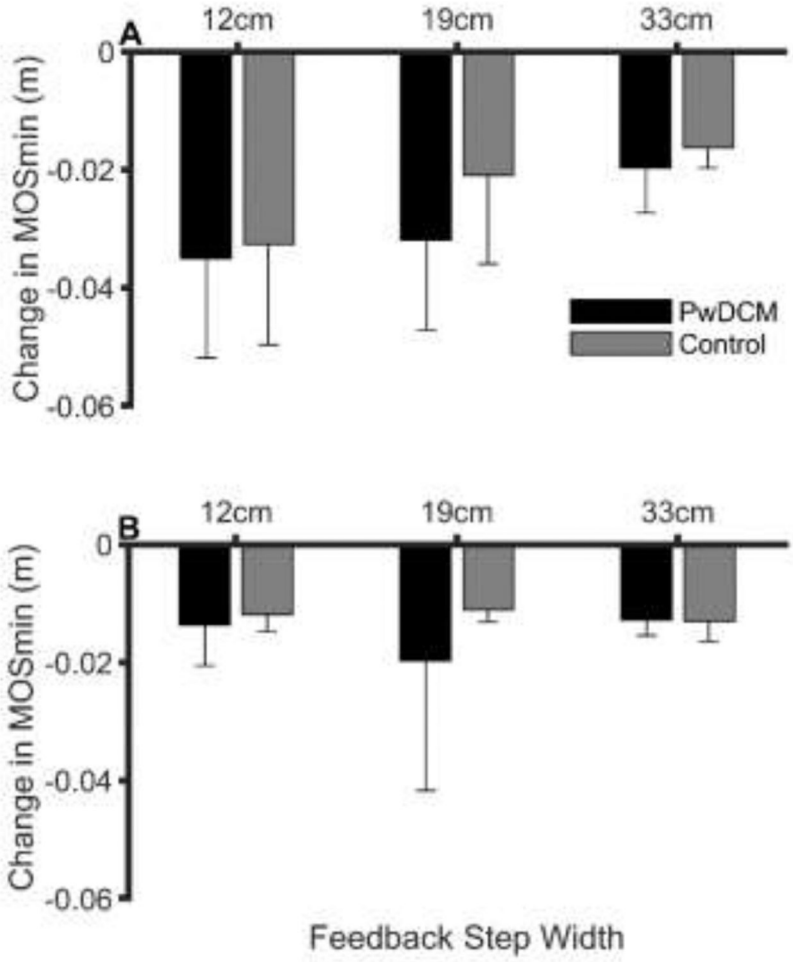

Fig 3.

Change in MOSmin following perturbations. Negative values mean MOS was changed to be closer to the lateral boundary of the base of support.

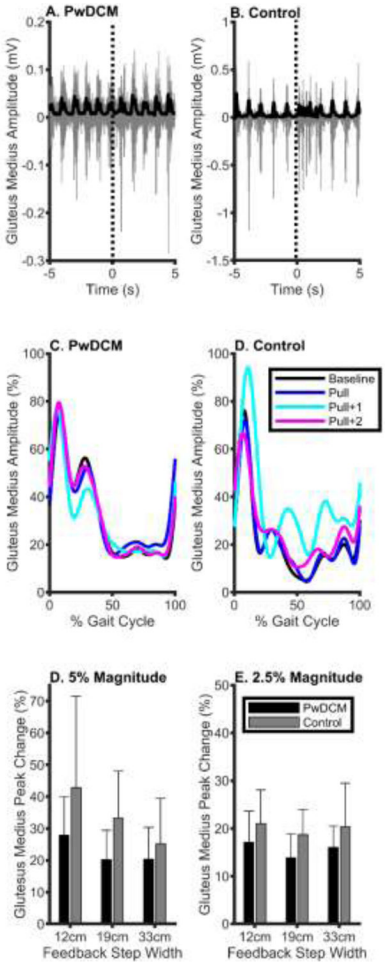

Fig 4.

Gluteus medius amplitude response following pulls. Panels A-B) demonstrates a filtered and envelope EMG from a PwDCM (ID #7) and control respectively. C) PwDCM (ID #7) single trial EMG amplitude normalized to gait cycle; amplitude normalized to 12cm steady state walking peak EMG. D) Control single trial EMG trial normalized to gait cycle; amplitude normalized to 12cm steady state walking peak EMG. E-F) aggregated data for 5% and 2.5% pulls respectively.

Table 1.

Characteristics of PwDCM.

ID	Age	Sex	Post Op (y)	Surgery	Approach	10mwt SS	10mwt FW	mJOA	Berg	FGA
1	65	F	13.00	PCF	POST	1.12	1.29	14	52	21
2	81	M	3.00	PCF	POST	1.25	1.65	15	43	15
3	46	F	2.50	ACDF	ANT	1.56	1.98	17	52	23
4	64	M	0.42	ACDF	ANT	1.07	1.49	10	48	17
5	64	F	0.33	PCF	POST	1.08	1.47	13	49	23
6	57	F	0.92	ACDF	ANT	1.61	2.27	16	53	27
7	34	F	0.83	ADR	ANT	1.22	1.80	16	55	29
8	56	F	7.00	ACDF	ANT	1.57	1.92	17	56	29
9	59	M	2.50	ADR	ANT	1.34	1.60	11	50	20
10	61	M	1.50	ACDF	ANT	1.57	2.22	17	55	28
Controls										
1	63	M				1.75	2.13		55	29
2	58	F				1.93	2.39		56	25
3	69	M				1.19	1.86		56	26
4	38	F				1.47	2.05		56	30
5	49	F				1.61	2.15		56	30
6	52	F				1.32	1.62		56	27
7	58	F				1.68	2.74		56	30
8	50	F				1.48	1.79		56	29
9	50	F				1.48	1.79		54	29
10	71	M				1.67	2.06		56	30

F=Female, M=Male. ACDF= anterior cervical discectomy and fusion, PCF=Posterior cervical fusion, ADR=anterior disc replacement. ANT=Anterior approach, POST=Posterior approach 10mwt SS=10 meter walk test self-selected comfortable. 10mwt FW=10 meter walk test fastest comfortable. mJOA= modified Japanese Association Scale, BBS=Berg Balance Test, FGA=Functional Gait Assessment.

Table 2.

Correlation coefficients and p-values for clinical/functional measures

	mJOA		**COMsway**		**10mwt C**		**Berg**	
	r	**p**	**r**	**p**	**r**	**p**	**r**	**p**
COMsway	0.38	0.28						
10mwt_C	**0.71**	**0.02**	0.26	0.47				
Berg	0.54	0.10	0.42	0.22	0.53	0.11		
FGA	**0.67**	**0.03**	0.49	0.15	0.57	0.09	**0.93**	**<0.01**