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Muscle synergies reveal impaired trunk muscle coordination strategies in individuals with thoracic spinal cord injury

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ABSTRACT

Spinal cord injury (SCI) can result in paralysis of trunk muscles, which can affect sitting balance. The objective of this study was to analyze trunk muscle coordination of individuals with thoracic SCI and compare it to able-body individuals. A total of 27 individuals were recruited and subdivided into: (a) high thoracic SCI; (b) low thoracic SCI; and (c) able-body groups. Participants were seated and asked to lean their trunk in eight directions while trunk muscle activity was recorded. Muscle coordination was assessed using the non-negative matrix factorization (NMF) method to extract muscle modules, which are the synergistic trunk muscle activations, and their directional activation patterns. Our results showed that individuals with SCI used less muscle modules, more co-contractions, and less directional tuning, compared to able-bodied people. These results suggest impaired and simplified muscle coordination due to the loss of supraspinal input after SCI. Observed variability in muscle coordination within SCI groups also suggests that other mechanisms such as spasticity and muscle stretch reflexes or individual factors such as experience and training contributed to the postural muscle synergies. Overall, muscle coordination deficits revealed impaired neuromuscular strategies which provide implications for rehabilitation of trunk muscles during sitting balance after SCI.

1. Introduction

A spinal cord injury (SCI) at, and above, the thoracic-level could cause paresis or paralysis of the trunk muscles which can impair sitting balance (Gauthier et al., 2013), as well as the ability to stand and walk (Fox et al., 2013; Hayes et al., 2014). Due to the paralysis of the postural trunk muscles, individuals with thoracic SCI sometimes develop new compensatory muscle activation patterns using innervated, non-postural muscles to control sitting balance (Seelen et al., 1998). However, this is often not sufficient to compensate for the impairment and stability in SCI individuals remains suboptimal (Gauthier et al., 2013; Potten et al., 1999). Individuals with low thoracic SCI have mild trunk impairment usually characterized by the deficiency of some trunk muscles, whereas individuals with high thoracic SCI typically have a higher level of impairment, including the inability to use the abdominal muscles (Gauthier et al., 2013; Potten et al., 1999). Trunk is a complex neuromuscular system with multiple degrees of freedom and many muscles acting to achieve stability (Bergmark, 1989). The nervous system controls groups of muscle simultaneously during complex movement, rather than controlling each muscle individually (Ting and Chvatal, 2010). However, identifying synergistic muscle coordination is not obvious from individual muscle activation patterns recorded from a large number of muscles during multiple trials, especially after neurological injuries such as SCI.

Non-negative matrix factorization (NMF) is a method that can be used to analyze such synergistic neuromuscular activations (Lee and Seung, 1999; Ting and Chvatal, 2010), which are difficult to observe from individual muscle activation patterns. The NMF algorithm performs a linear extraction that can separate muscle activations into muscle modules and their recruitment patterns. Muscle modules are composed of synergistic muscle activations, called muscle synergies, which are the building blocks of muscle

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coordination used by the central nervous system to simplify construction of motor behaviours (Ting and Chvatal, 2010). Muscle coordination can be characterized by the number of extracted muscle modules, as well as their composition and the activation patterns. Such analysis has been used to describe muscle coordination during standing postural control (Torres-Oviedo et al., 2006; Torres-Oviedo and Ting, 2007), locomotion (Chvatal and Ting, 2012; Clark et al., 2010; Fox et al., 2013; Hayes et al., 2014; Rodriguez et al., 2013; Zelik et al., 2014), and arm reaching (Cheung et al., 2012). It has been utilized in analyzing changes in the muscle coordination after neurological disorders in individuals with SCI (Fox et al., 2013; Hayes et al., 2014), stroke (Cheung et al., 2012; Clark et al., 2010) and Parkinson's disease (Rodriguez et al., 2013). After neurological disorders, the number of muscle modules was typically reduced, implying impaired muscle coordination. Among individuals with SCI, there was a general loss of muscle synergies that were utilized (Fox et al., 2013; Hayes et al., 2014), whereas stroke patient muscle synergies merged together (Clark et al., 2010), and individuals with Parkinson's disease had altered activation patterns (Rodriguez et al., 2013) during walking. After SCI, there was also an overall increase in muscle co-activations (Chvatal et al., 2013; Hayes et al., 2014), and a general loss of directional tuning (Chvatal et al., 2013).

To date, there is no clear understanding of how trunk muscle activation synergies change after thoracic SCI. This would allow us to reveal, in a quantitative way, the underlying building blocks of the trunk muscle impairment after thoracic SCI. Therefore, the objective of this study was to quantify muscle coordination of individuals with high and low thoracic SCI during seated leaning and compare it to able-bodied individuals. We hypothesized that muscle coordination will be impaired in individuals with thoracic SCI, and to a higher degree in individuals with high thoracic SCI compared to individuals with low thoracic SCI.

2. Methods

2.1. Participants

A total of 27 individuals were recruited into: (a) high thoracic SCI (n = 8; neurological injury level T1-T7); (b) low thoracic SCI (n = 11; neurological injury level T8-T12); and (c) able-body (n = 8) subgroups (Table 1). Individuals with SCI were recruited if they had a motor and/or sensory complete or an incomplete injury (American Spinal Injury Association (ASIA) Impairment Scale (AIS) A to D) at the thoracic-level (T1-T12). Moreover, individuals with SCI were eligible to participate if they had the ability to independently maintain erect short-sitting position with their hands on their thighs, their feet on the floor, and had an activity tolerance of at least 60 min. Participants were excluded if they had secondary musculoskeletal complications that may affect trunk control, had pressure sores on their buttocks, and visual, vestibular, or auditory conditions which may limit their ability to perform the experiment. Able-bodied individuals were recruited if they had no history of neurological or musculoskeletal impairment that could affect their sitting balance, and visual, vestibular, or auditory conditions which may limit their ability to perform the experiment. All participants gave written informed consent in accordance with the Declaration of Helsinki. The experimental

Table 1
Participants' demographic information and clinical evaluations, including the American Spinal Injury Association Impairment Scale (AIS) and the American Spinal Injury Association (ASIA) motor and sensory scores, for the high thoracic SCI (HighTh), low thoracic SCI (LowTh), and able-body (AB) groups.

Group	Subject	Age (years)	Height (m)	Weight (kg)	Neurological level of injury	AIS	ASIA motor (/100)	ASIA sensory (/224)	Time since injury (years)
HighTh	1	45.8	1.8	80.0	T7	A	50	117	12.1
	2	36.5	1.7	67.9	T6	A	50	53	6.9
	3	53.3	1.8	74.8	T1	C	68	177	0.4
	4	37.0	1.8	79.3	T6	D	81	194	12.9
	5	53.2	1.8	84.7	T4	A	50	–	27.9
	6	50.2	1.7	70.2	T7	B	52	88	5.5
	7	60.7	1.7	62.3	T5	A	50	92	38.3
	8	39.4	1.8	84.8	T6	B	50	166	9.0
Mean		49.5	1.8	75.3			55.7	124.9	13.2
SD		9.3	0.1	7.7			11.2	49.3	14.6
LowTh	1	34.6	1.9	–	T12	C	56	162	9.2
	2	35.4	1.8	101.7	T11	C	54	182	2.8
	3	29.7	1.9	73.0	T12	A	50	154	11.9
	4	41.7	1.5	66.5	T8	B	50	134	11.0
	5	67.1	1.8	84.9	T11	D	75	163	0.1
	6	24.1	1.8	54.0	T11	A	66	160	0.1
	7	53.9	1.8	110.7	T9	A	50	132	20.0
	8	37.6	1.9	83.5	T10	A	50	138	2.4
	9	57.1	1.8	85.3	T12	A	55	168	25.0
	10	61.2	1.9	102.4	T10	A	50	140	6.3
	11	54.0	1.7	73.6	T12	B	63	172	10.2
Mean		45.1	1.8	83.6			56.3	155	9.0
SD		14.1	0.1	17.6			8.3	16.8	8.0
AB	1	31.9	1.7	57.9					
	2	59.8	1.8	68.9					
	3	42.8	1.8	78.1					
	4	50.2	1.9	76.2					
	5	27.8	1.7	54.8					
	6	54.8	1.9	71.0					
	7	43.5	1.7	100.8					
	8	34.2	1.8	93.6					
Mean		44.5	1.8	75.2					
SD		12.8	0.1	15.9					

procedures were approved by the institutional Research Ethics Committee.

2.2. Experimental protocol

Participants were seated upright on a chair without back support, with their feet on the ground, and their hands on their thighs (Fig. 1). Participants were instructed to refrain from using their hands for support. During the experiment, participants were asked to lean their trunk as far as possible, at a self-selected speed, and return to the starting position within 15 s. Participants had to lean in one of eight directions, in a random order, separated at 45° (Fig. 1). A computer screen was used to show the direction being tested and the trajectory to follow (Gauthier et al., 2012, 2013). Each of the eight directions was tested twice for a total of 16 trials. Prior to the experiment, participants were familiarized with the task. At all times, an experimenter remained close to ensure safety.

2.3. Data acquisition and processing

Bilateral surface electromyography (EMG) was recorded from five thoracohumeral muscle groups that were identified as relevant for sitting among individuals with SCI (Potten et al., 1999; Seelen et al., 1998) and could be recorded reliably. Disposable EMG electrodes (Ag/AgCl; 10 mm diameter) were placed 2 cm apart on the: (1) lumbar erector spinae (L3), 3 cm lateral to the L3 spinous process; (2) low thoracic erector spinae (T9), 3 cm lateral to the T9 spinous process; (3) high thoracic erector spinae (T3), 3 cm lateral to the T3 spinous process; (4) trapezius (Tr), midway between the C7 spinous process and the lateral aspect of the acromion; and (5) latissimus dorsi (LD), 2 cm inferior and lateral to the inferior angle of the scapula. A reference electrode was placed over the clavicle. The data were acquired using two Noraxon Telemyo 900 portable EMG recording systems (Noraxon Inc., USA) with a pre-amplification gain of 2000 and a frequency response of 10–500 Hz. Signals were sampled at 1200 Hz and stored on a computer using a custom LabVIEW program (National Instruments, USA).

All EMG recordings were high-pass filtered at 30 Hz using a fifth-order, Butterworth filter to remove the electrocardiogram artifact (Hof, 2009). Each signal was referenced by subtracting it from its mean. The signals were then full-wave-rectified, low-pass filtered at 10 Hz using a fourth-order Butterworth filter (Zelik et al., 2014), and down-sampled to 200 Hz. To allow comparisons between subjects, EMG responses for each muscle were normalized to the maximum activation over all the trials (Hayes et al., 2014). The EMG responses in the 15 s window during each leaning trial were then analyzed.

2.4. Muscle coordination analysis

Trunk muscle coordination was analyzed using the NMF method (Lee and Seung, 1999; Ting and Chvatal, 2010) to extract the muscle modules (i.e., synergistic muscles activations) and directional activations patterns (i.e., recruitment pattern of muscle modules for different directions) for each subject. First, the recorded EMG data for each subject, $\boldsymbol{M}$, was organized as a matrix with j rows (corresponded to five muscles recorded bilaterally; $j = 10$) and s columns (corresponding to the number of samples; $s = 48{,}000 = 8$ directions $\times$ 2 trials/direction $\times$ 15 s/trial $\times$ 200 samples/s) (Fig. 2). Each muscle was scaled to unit variance to equally weigh the EMG activity across muscles, although the scaling was removed after the extraction (Chvatal and Ting, 2012). The NMF algorithm was then used to decompose the EMG input data, $\boldsymbol{M}$, into muscle modules, $\boldsymbol{W}$, and their directional activation patterns, $\boldsymbol{C}$. The NMF algorithm performs the decomposition of $\boldsymbol{M}$ by randomly initializing non-negative matrices $\boldsymbol{W}$ and $\boldsymbol{C}$, and iteratively updating their organization to minimize the error between the original data (i.e., $\boldsymbol{M}$) and the reconstructed data (i.e., $\boldsymbol{W.C}$), according to Eq. (1), until sufficient variability in data is accounted for (Ting and Chvatal, 2010).

$$\boldsymbol{M} = \sum_{i=1}^{n} \boldsymbol{W_i} \cdot \boldsymbol{C_i} + \boldsymbol{error} \quad (1)$$

Despite random initialization, the NFM algorithm performed robustly (Atoufi et al., 2014; Ting and Chvatal, 2010), and a single extraction was used to obtain the muscle modules (Chvatal and Ting, 2012; Clark et al., 2010; Rodriguez et al., 2013; Torres-Oviedo et al., 2006; Torres-Oviedo and Ting, 2007; Zelik et al., 2014). For each n, which can vary from 1 to 10 (i.e., the number of muscles), the goodness of fit was calculated as variance accounted for (VAF), which was defined as the uncentred Pearson correlation coefficient between the original and the reconstructed data (Torres-Oviedo et al., 2006). The optimal number of muscle modules, n_{syn}, was computed as the smallest n that accounted for

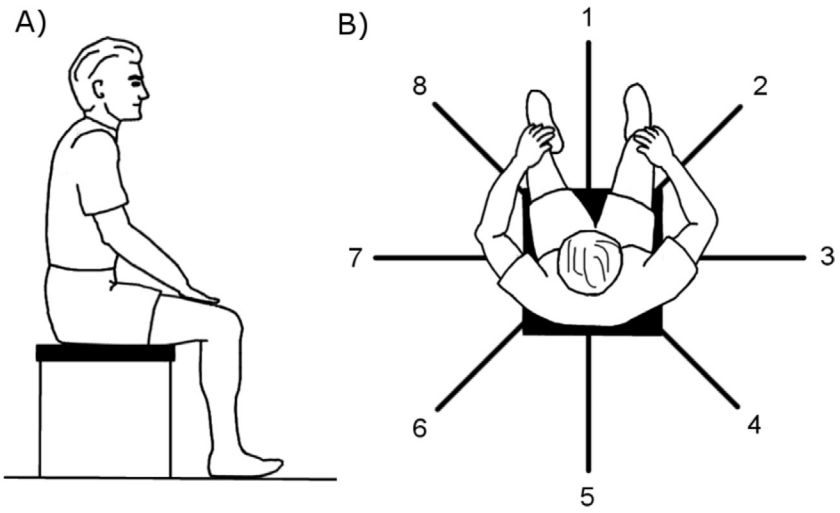

Fig. 1. Experimental setup illustrating the posture of the subject during performance of the experimental tasks: (A) side view; and (B) top view with the indicated directions in which the subjects had to lean.

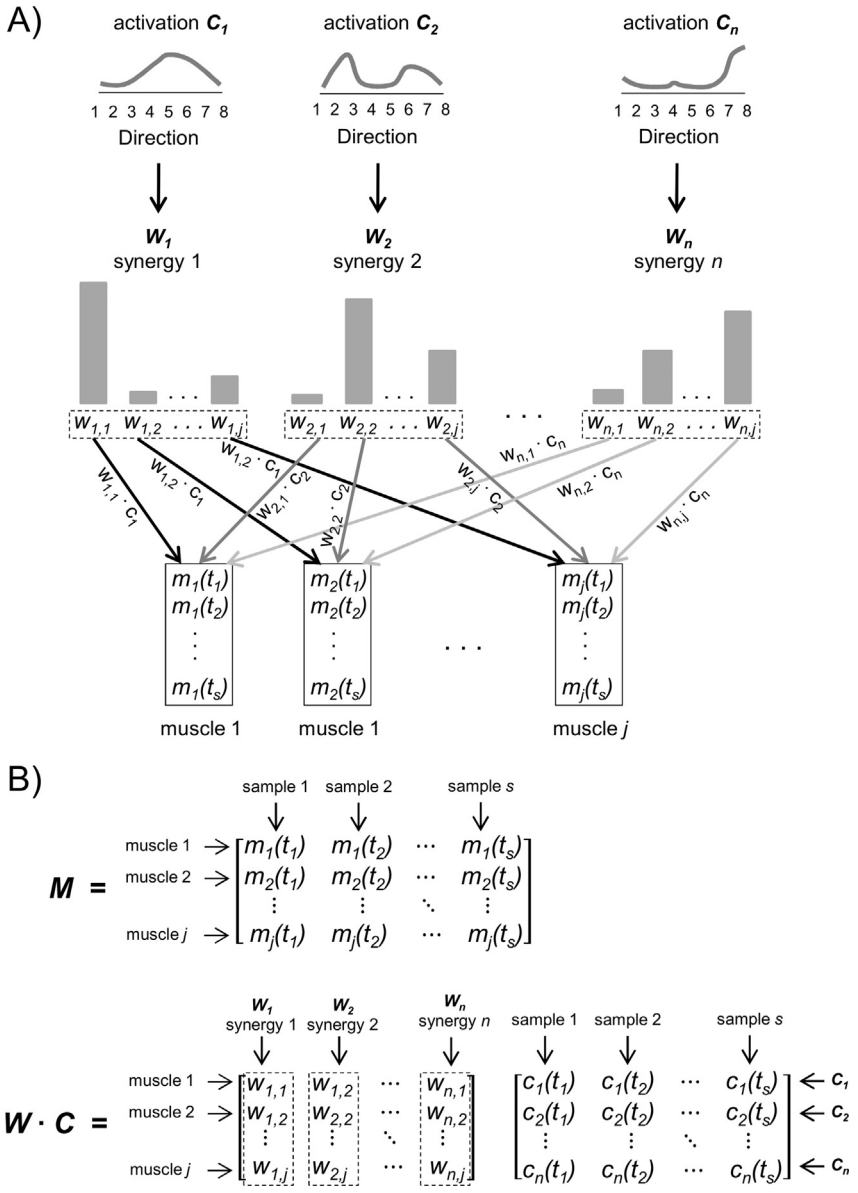

Fig. 2. Block diagram explaining muscle coordination analysis using non-negative matrix factorization (NMF) method: (A) Visual illustration of the NMF analysis showing that the observed input data for each muscle ($\boldsymbol{m_j}$) which can be decomposed into the n distinct muscle synergy modules ($\boldsymbol{W_n}$), each of which is activated by the corresponding directional activation pattern ($\boldsymbol{C_n}$); and (B) Matrix representation of the NMF analysis showing the observed input data ($\boldsymbol{M}$) and the extracted muscle synergy modules ($\boldsymbol{W}$) and their directional activation pattern ($\boldsymbol{C}$).

>90% of VAF for the overall data containing all muscles and all directions (Torres-Oviedo et al., 2006), and the smallest n for which 80% of active muscle accounted for >80% of VAF for data of each muscle per direction (Zelik et al., 2014). The extracted muscle modules, $\boldsymbol{W}$, are composed of n_{syn} distinct muscle synergy vectors (i.e., $\boldsymbol{W_1}$, $\boldsymbol{W_2}$,…,$\boldsymbol{W_n}$), each scaled by the corresponding directional activation pattern, $\boldsymbol{C}$ (i.e., $\boldsymbol{C_1}$, $\boldsymbol{C_2}$,…,$\boldsymbol{C_n}$) (Fig. 2). For each subject, the composition of muscle modules and their activation patterns were further analyzed.

The number of active muscles per synergy was calculated for each subject, for each extracted muscle synergy, as an indicator of co-activations by analyzing the composition of $\boldsymbol{W_n}$. Each muscle module, $\boldsymbol{W_n}$, was a vector containing the normalized muscle activation levels (Fig. 2). Active muscles in a module were defined as the muscles whose normalized weight value exceeded 0.3 (Zelik et al., 2014). For each muscle module, the number of active muscles could vary from 10 (i.e., high co-activation of muscles) to 1 (i.e., no co-activations).

The directional specificity coefficient of each muscle synergy was calculated for each subject, for each extracted muscle synergy, as an indicator of directional tuning by analyzing the activation pattern, $\boldsymbol{C_n}$. Two repeated directional trials were averaged to obtain the activation pattern for each of the eight directions. The activation pattern for each direction was then normalized by the maximum activity direction, such that the values ranged from 0 to 1 (Fig. 3). The directional specificity coefficient of each muscle synergy was obtained by summing the normalized recruitment coefficients of all eight directions, such that they could vary from 1 (very directionally specific; muscle module recruited in only one direction) to 8 (not directionally specific; muscle module recruited in all eight directions equally).

2.5. Statistical analysis

Comparisons between high thoracic SCI, low thoracic SCI, and able-body subgroups were performed using the non-parametric

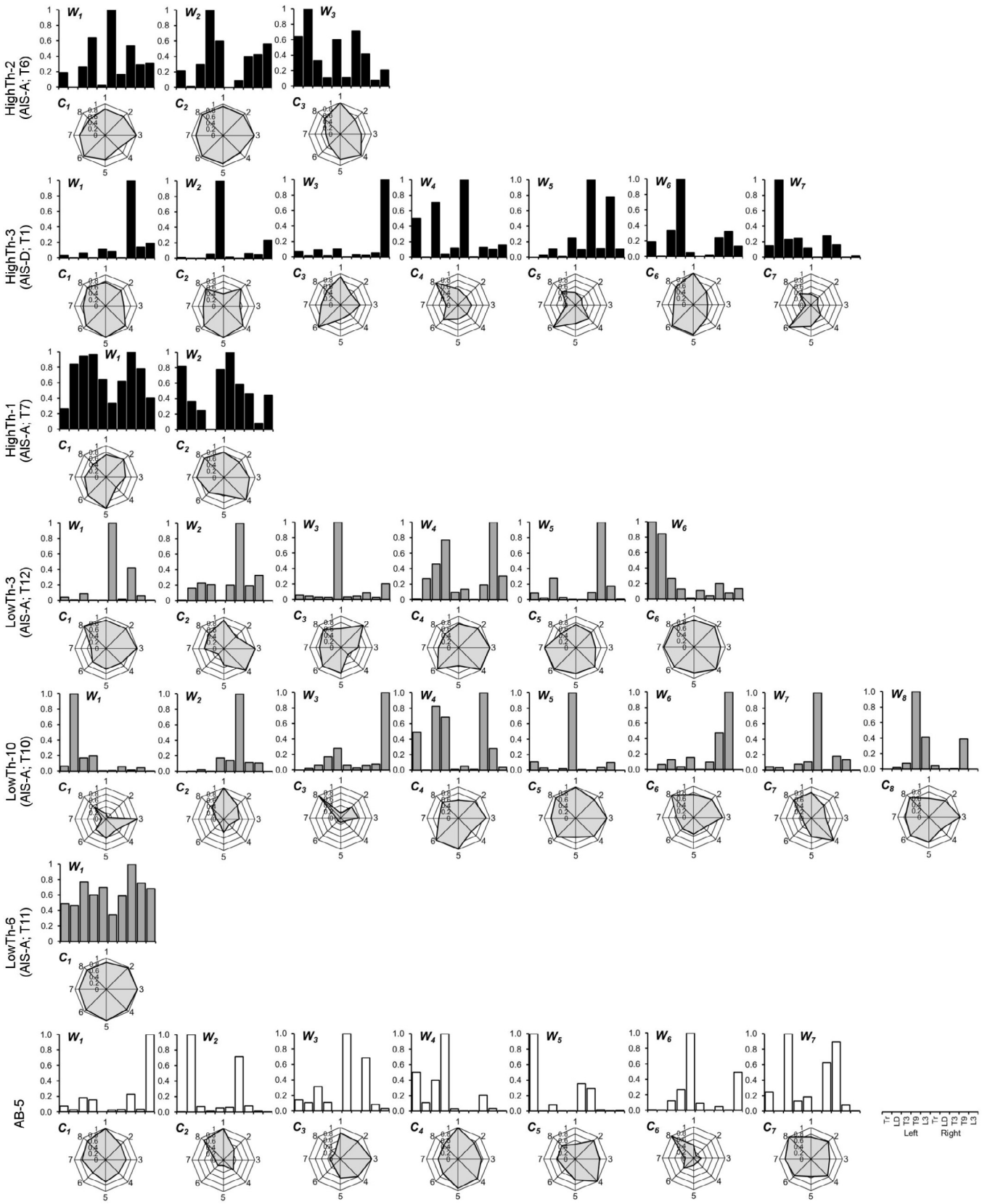

Fig. 3. Muscle synergy modules ($\boldsymbol{W_n}$) and the corresponding directional activation patterns ($\boldsymbol{C_n}$) showing the high thoracic spinal cord injury (SCI) individuals (HighTh) using black bars, low thoracic SCI individuals (LowTh) using gray bars, and able-body individuals (AB) using white bars. The figure shows representative individuals in the HighTh group (HighTh-2), LowTh group (LowTh-3) and AB group (AB-5), as well as two other individuals in the HighTh group (HighTh-3 and HighTh-1) and LowTh group (LowTh-10 and LowTh-6) who had different responses to illustrate the heterogeneity of muscle coordination among individuals with SCI. Indicated are the American Spinal Injury Association Impairment Scale (AIS) and the neurological injury levels for each SCI individual. Muscle modules and directional activations patterns are normalized in each module to the maximum muscle activity and maximum directional activation level, respectively.

Kruskal-Wallis one-way analysis of variance (ANOVA) test with Dunn's test for post hoc multiple comparisons when a significant difference was found on the Kruskal-Wallis test. Non-parametric tests were chosen because the sample size remains relatively small and the Shapiro-Wilk test suggested that all identified measures were not normally distributed. Significance level was set to $p < 0.05$.

3. Results

3.1. Participant groups

The mean age (Kruskal-Wallis one-way ANOVA, $H_2 = 0.447$, $p = 0.800$), mean weight ($H_2 = 1.763$, $p = 0.414$), and mean height ($H_2 = 2.183$, $p = 0.336$) of participants in the high thoracic SCI, low thoracic SCI, and able-bodied groups were not significantly different. Moreover, the AIS ($H_1 = 0.018$, $p = 0.893$), ASIA motor score ($H_1 = 0.241$, $p = 0.623$), ASIA sensory score ($H_1 = 0.740$, $p = 0.390$), and the time since injury ($H_1 = 0.987$, $p = 0.322$) of participants in the high thoracic SCI and low thoracic SCI groups were not significantly different. This suggests that the groups were matched.

3.2. Muscle coordination analysis

Fig. 3 shows the extracted muscle synergy modules (i.e., $\boldsymbol{W_n}$) and the corresponding directional activation patterns (i.e., $\boldsymbol{C_n}$) for the representative individuals in each subgroup as well as individuals in the high thoracic SCI and low thoracic SCI groups who had different responses from the representative subjects.

3.3. Differences between groups

The number of extracted muscle synergy modules, n_{syn}, was significantly different between the groups (Kruskal-Wallis one-way ANOVA, $H_2 = 7.961$, $p = 0.019$). Specifically, it was significantly lower in the high thoracic SCI group compared to the able bodied group (post hoc Dunn's test, $Q = 10.812$, $p = 0.005$). There were no differences between the able-bodied group and the low thoracic SCI group ($Q = 3.949$, $p = 0.823$), and between the high thoracic SCI group and the low thoracic SCI group ($Q = 6.864$, $p = 0.173$) (Fig. 4A).

The number of active muscles per synergy was significantly different between the groups ($H_2 = 6.460$, $p = 0.040$). Specifically, it was significantly higher in the high thoracic SCI group compared to the able bodied group ($Q = 18.980$, $p = 0.041$), and compared to the low thoracic SCI group ($Q = 22.010$, $p = 0.014$). There were no differences between the able-bodied group and the low thoracic SCI group ($Q = 3.029$, $p = 0.700$) (Fig. 4B).

The directional specificity coefficient was significantly different between the groups ($H_2 = 8.705$, $p = 0.013$). Specifically, it was significantly higher in the low thoracic SCI group ($Q = 21.819$, $p = 0.007$), and the high thoracic SCI group ($Q = 22.307$, $p = 0.020$), compared to the able-bodied group. There were no differences between the low thoracic SCI group and the high thoracic SCI group ($Q = 0.488$, $p = 0.958$) (Fig. 4C).

4. Discussions

4.1. Common muscle coordination deficits after thoracic SCI

The number of muscle modules used by individuals with high thoracic SCI was lower compared to able-bodied people (Fig.4A), consistent with the results in adults (Hayes et al., 2014) and children (Fox et al., 2013) with incomplete SCI, as well as people with stroke (Clark et al., 2010), and Parkinson's disease (Rodriguez et al., 2013) during walking. This neuromuscular impairment is likely due to damage in neural control caused by the SCI, which prevents descending neural signals from recruiting different muscle synergies (Cheung et al., 2012; Hart and Giszter 2004). Impaired ascending sensory information, driving the motor coordination at the spinal cord level, is also likely contributing to the impairment. These results are consistent with reports suggesting that individuals with high thoracic SCI have more compromised trunk control compared to individuals with low thoracic SCI (Potten et al., 1999; Seelen et al., 1998).

Our study also showed that the direction specificity coefficient was higher in individuals with thoracic SCI compared to able-bodied people (Fig. 4C), which implies less directional tuning of muscle synergies. Able-bodied individuals have clear directional tuning in the trunk (Milosevic et al., 2012, 2016) and the lower-limb muscles (Torres-Oviedo et al., 2006; Torres-Oviedo and Ting, 2007). On the contrary, no clear directionality was observed in cats after spinal transections (Chvatal et al., 2013). The trunk system has many biomechanical degrees of freedom (i.e., numerous directions in which it can move), requiring directional tuning (Bergmark, 1989). Therefore, reduction in the directional tuning of muscle recruitment after thoracic SCI is indicative of compromised neuromuscular control.

Finally, our results showed that the number of active muscles per synergy was higher among individuals with high thoracic SCI compared to individuals with low thoracic SCI and able-bodied people (Fig. 4B), which implies more co-contractions. Increased

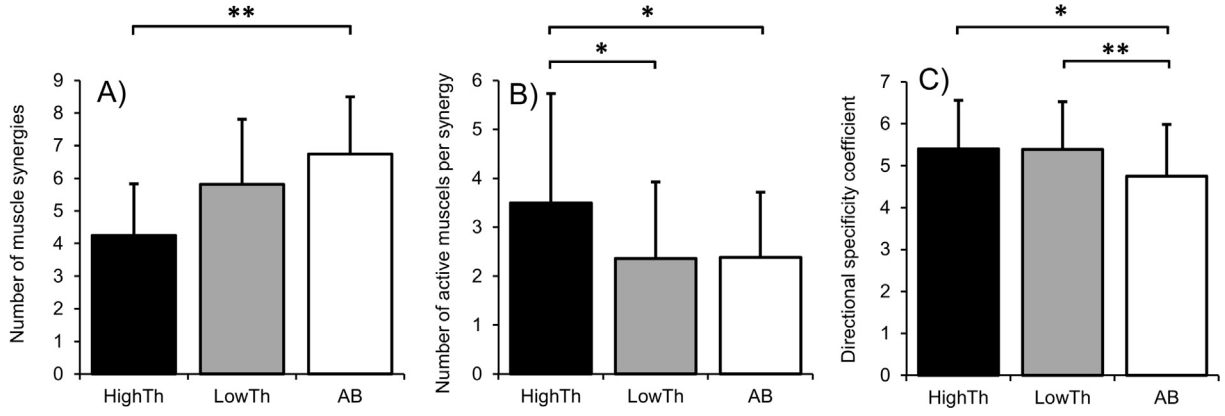

Fig. 4. Comparison of the high thoracic SCI group (HighTh), shown in black, low thoracic SCI group (LowTh), shown in gray, and able-bodied (AB) groups, shown in white to compare: (A) number of muscle synergies; (B) number of active muscles per synergy; and (C) directional specificity coefficient. The figure shows the mean and standard deviation (SD) for each outcome variable. Legend: $^{*}p < 0.05$ and $^{**}p < 0.01$.

co-contraction of muscles was shown in people after SCI during locomotion (Fox et al., 2013; Hayes et al., 2013), and in animals after spinal cord transections (Chvatal et al., 2013). Basic science studies have also shown that a reduction in the descending signals resulted in higher co-activation of muscles (Hart and Giszter, 2004). Increased co-contractions could also be a results of spasticity after SCI (Chvatal et al., 2013). Therefore, higher co-activations could be a result of an altered balance control strategy to stiffen the trunk (Milosevic et al., 2015), but also an automatic consequence of injury, which affects descending neural signals.

Our investigation utilized the NMF algorithm to extract the muscle modules from neuromuscular activations of multiple trunk muscles during seated leaning. The muscle modules reflect consistent patterns of muscle coordination, which are not obvious from individual muscle activation patterns (Ting et al., 2015). Moreover, the NMF analysis allows examination of muscle coordination independent of amplitude and timing of activations (Chvatal et al., 2013). While the motor control of able-bodied individuals was capable of coordinating many trunk muscles, this ability was impaired after SCI. Specifically three patterns of trunk muscle coordination impairment were revealed, including reduced ability to recruit distinct muscle synergies, decreased directional specificity of muscle recruitment, and increased co-activations. Consistent to our hypothesis, the results indicate that trunk muscle coordination was impaired and simplified after thoracic SCI, and to a higher degree in those with high thoracic SCI. It was previously shown that trunk muscles are controlled by simple polysynaptic reflexes, which could be modulated by supraspinal input (Milosevic et al., 2016). Our current results showed that trunk muscle synergies are impaired by the loss of the supraspinal input after SCI, suggesting that trunk muscle synergies are at least partially organized in the higher centers.

4.2. Subject-specific neuromuscular assessments

Using the muscle coordination analysis, shown in Fig. 3, it seems that individuals with thoracic SCI in our study used higher activations of non-postural muscles. For instance, subject HighTh-2 had two of three muscle modules that were characterized with high activation of latissimus dorsi and the trapezius muscles (i.e., $\boldsymbol{W_1}$ and $\boldsymbol{W_3}$). Similarly, subject LowTh-3 subject had three of six muscle modules with high reliance on the non-postural muscles (i.e., $\boldsymbol{W_1}$, $\boldsymbol{W_2}$ and $\boldsymbol{W_6}$). Therefore, consistent to previous reports (Potten et al., 1999; Seelen et al., 1998), it seems that neuromuscular synergies after thoracic SCI shift towards using innervated, non-postural trunk muscles to compensate for impaired muscle coordination.

Results in Fig. 3 also illustrate muscle coordination heterogeneity among individuals with SCI. Representative high thoracic SCI subject, HighTh-2 (AIS-A; T6 injury), had higher co-contractions and non-specific muscle tuning. Another subject in the same group, HighTh-3 (AIS-D; T1 injury), had responses characterized by more muscle modules, which implied a more robust postural control, along with less co-contractions and more directional tuning (similar to able-body subject, AB-5). Subject HighTh-1 (AIS-A; T7 injury) had the most impaired muscle coordination characterized with two muscle modules, which had high co-contraction levels and low directional tuning. Such heterogeneity also existed in the low thoracic SCI group. For instance, subject LowTh-10 (AIS-A; T10 injury) had eight muscle modules characterized with low co-contractions. Despite similar clinical assessments (Table 1), subject LowTh-6 (AIS-A; T11 injury) used one muscle module and the responses were characterized with high co-contractions. Although it was previously shown that the number of postural muscle synergies and directional tuning were generally reduced after complete spinal cord transections in cats, some animals had increased number of modules (Chvatal et al., 2013). Altogether, these results imply that different mechanisms, such as spasticity or muscle stretch reflexes, also contribute to the postural muscle synergies. Some variability in neuromuscular responses is expected in able-bodied individuals (Milosevic et al., 2012). However, specialized muscle modules characterized by the activation of one muscle, which acts in one specific direction (e.g., LowTh-3), or non-specialized modules with high co-contraction and low directional tuning (e.g., LowTh-6) could indicate dysfunction (Ting et al., 2015).

Spinal cord injury is a highly heterogeneous, affecting both ascending and descending pathways in a non-linear manner. The NMF muscle module extraction can identify the underlying neuromuscular elements, which are shaped by experience, training, and individual movement styles (Ting et al., 2015). Therefore, such analysis offers insights for development of customized rehabilitation strategies tailored to individual patients.

4.3. Clinical implications

Based on our results from the muscle coordination analysis, we propose that rehabilitation should be directed towards increasing the complexity of neuromuscular control through training that includes repetitive, multi-directional seated leaning initiated rapidly after the SCI. Such training could improve trunk muscle coordination through activity-dependent plasticity (Curt et al., 2004). In addition, we propose that training programs for improving trunk function should focus on optimizing the development of new muscle synergies to facilitate effective sitting balance in combination with maximizing the functional contributions of the residual neurons for individuals with incomplete SCI (Curt et al., 2004). Lastly, we suggest that training should be tailored to each patient and guided by neuromuscular assessments.

4.4. Study limitations

As numerous factors affect sitting balance after SCI (Gauthier et al., 2013; Seelen et al., 1998), future studies with larger, representative sample sizes are warranted to systematically investigate the relationship between neuromuscular coordination deficits and clinical measures as well as postural performance. This will identify the key determinants and best predictors of muscle coordination impairment. Moreover, future studies should record the activity of abdominal muscles (e.g., rectus abdominis, internal or external obliques, and transversus abdominis), to gain a better understanding of the muscle coordination in individuals with SCI. Lastly, assessing muscle coordination during challenging sitting tasks (e.g., sitting on a wobble board) may also lead to a better understanding of the muscle coordination in this population.

5. Conclusions

We demonstrated impaired trunk muscle coordination, typically characterized with less muscle modules, more co-contractions, and lower directional tuning, in individuals with thoracic SCI during seated leaning. Muscle coordination deficits revealed impaired neuromuscular strategies that provide implications for rehabilitation of trunk muscles and sitting balance after thoracic SCI.

Conflict of interest statement

There are no known conflicts of interest associated with this publication.

Financial disclosure statement

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