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Training of isometric force tracking to improve motor control of the wrist after incomplete spinal cord injury: A case report

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Abstract

Objective: Upper limb function is a high priority for people with cervical spinal cord injury (SCI). This case report describes an application of technology to activate spared neural pathways and improve wrist motor control.

Case Description: A 73-year-old man with chronic incomplete C5 SCI completed 24 training sessions over 92 days. Each session included 2 maximal contractions, 6 test trials and 10 training trials of a visuomotor force tracking task. The participant attempted to match a sinusoidal target force curve, using isometric wrist flexor and extensor contractions. Electromyography (EMG) and force signals were recorded.

Outcomes: Error was elevated initially and improved with training, similarly during extension and flexion phases of the force tracking task. Improvement in both phases was associated with greater flexor activation in flexion phases and greater extensor relaxation in flexion phases. Error was not related to EMG modulation during extensor phases. Small improvements in active range of motion, grip force, spasticity, touch sensation and corticospinal excitability were also observed.

Conclusions: Motor skill training improved motor control after incomplete SCI, within the range of residual force production capacity. Performance gains were associated with specific adjustments in muscle activation and relaxation, and increased corticospinal excitability.

Keywords

spinal cord injury; motor control; upper extremity; neuroplasticity; case study

INTRODUCTION

Recovery of upper limb movement ability is a high priority for people with incomplete tetraplegia after spinal cord injury (SCI) (Collinger et al, 2013; Simpson et al, 2012). Clients and clinicians seek to maximize recovery by activating spared neural pathways, to preserve

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Declaration of Interest

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residual function and to induce neural adaptations (Little, Ditunno, Steins, and Harris, 1999; Vining et al, 2017). Evidence-based restorative approaches include massed practice of functional motor skills, somatosensory stimulation, and functional electrical stimulation, all of which improve upper limb function and are associated with increased corticospinal excitability (Gomes-Osman and Field-Fote, 2015; Lu, Battistuzzo, Zoghi, and Galea, 2015; McGie, Zariffa, Popovic, and Nagai, 2015). Specific changes in muscle activation patterns that underlie improvements in upper limb motor control after SCI are not well understood, however. Further insight into neuromotor adaptations may help to maximize effectiveness of rehabilitation for people with partial paralysis.

Integration of technology into the field of rehabilitation continues to expand opportunities for quantitative assessments and novel, personalized interventions (Pierella et al, 2017). In this case report, we implemented a technology-based intervention for focused motor control training after SCI, and examined neural adaptations corresponding to performance gains, using electromyography (EMG) and transcranial magnetic stimulation (TMS). By quantifying performance error and muscle activity during an isometric visuomotor tracking task (Carey, 1990; Lindberg et al, 2012), we aimed to help an individual with incomplete tetraplegia regain control over his wrist flexors and extensors, and to reveal neural adaptations associated with improved performance.

CASE DESCRIPTION

The participant was a 73-year-old right-handed man who sustained an incomplete SCI in a bicycling accident 2.7 years previously. He was diagnosed with a C3-C6 spinal cord contusion and underwent posterior spinal fusion from C2 - T1. The injury resulted in central cord syndrome and tetraplegia, with partial paralysis and impaired sensation affecting his upper limbs more than his lower limbs. As assessed using the International Standards for Neurological Classification of SCI, his Neurological Level of Injury was C5, and his ASIA Impairment Scale grade was C (Kirshblum et al, 2020). He was hospitalized in acute care for 8 days and in a rehabilitation unit for the following 128 days. After being discharged to his home, he received physical therapy and occupational therapy on an outpatient basis, and daily assistance of a home health aide. Those services continued while he participated in this intervention, without notable changes in the frequency, goals, or other interventions received.

The participant used a power wheelchair as his primary means of mobility, and operated it using a joystick with his right hand. He also used his right upper limb to interact with an iPad mounted on the left side of his wheelchair, and to feed himself with adapted utensils. He wore wrist splints bilaterally to prevent excessive flexion. He expressed an interest in trying novel strategies to maximize recovery of movement and function, especially for the right wrist. Pre-existing medical conditions included type 2 diabetes, peripheral neuropathy affecting both feet, atrial fibrillation, left clavicle fracture and right rotator cuff injury more than 10 years before the SCI. Within the previous year, he had received injections of onabotulinumtoxinA to his left wrist flexors, bilateral pectorals, and bilateral forearm pronators. He had not received any onabotulinumtoxinA injections to his right wrist flexors,

extensors, or hand muscles. He was taking baclofen, escitalopram, dabigatran, metoprolol, pregabalin, empagliflozin, and metformin consistently.

For comparison, ten healthy individuals performed the wrist motor control outcome measure that was used, with their non-dominant side, on one occasion. They each demonstrated normal upper limb movement and sensation, and had no history of musculoskeletal or neurological conditions affecting either upper limb. Five males and 5 females, ages 50.8 ± 13.4 years participated (mean, SD). All work was approved by the Institutional Review Board of the University of Iowa (Approval #201712733) and met the requirements of the Health Insurance Portability and Accountability Act. All participants gave written informed consent.

Intervention

The participant attended training sessions twice weekly for 3 months. Impairments and activity limitations were assessed within a 2-week period before and after the intervention. In addition, measures obtained from the training task itself were used to track changes in performance and muscle activation patterns.

Motor Control Training and Testing

An isometric force tracking task and a custom-designed positioning device were developed to train and quantify wrist motor control. As shown in Figure 1, adjustable vertical posts stabilized the forearm near the elbow and the wrist, and the hand was attached to a force transducer with a strap across the palm, just proximal to the metacarpal heads (80/20 Inc., Columbia City, IN, Model SSM-NS-250, Interface, Inc., Scottsdale, AZ). Output from the force transducer was digitized, calibrated, displayed in real time and recorded, using a data acquisition device connected to a computer that was running a custom-written LabVIEW program (DAQ Model USB 6002, National Instruments, Inc., Austin TX). Force signals were filtered forward and backward using a second order Butterworth low pass filter with a cutoff frequency of 6 Hz.

EMG was recorded from wrist flexors and extensors using a wireless EMG system (Trigno, Delsys, Inc., Natick, MA). Each sensor included the Delsys parallel silver bar technology, a fixed inter-electrode distance of 10 mm, and a bandwidth of 20-450 Hz. Standard Trigno sensors were adhered to the skin over the muscle bellies of the flexor carpi radialis (FCR) and extensor carpi radialis longus (ECR) muscles. The FCR electrode was placed along a line from the medial epicondyle of the humerus to the radial styloid process, one third of the distance from the epicondyle, as measured with the forearm supinated (Jabre, 1981). The ECR electrode was placed along a line from the lateral epicondyle of the humerus to the radial styloid process, one third of the distance from the epicondyle, as measured with the forearm pronated (Chow et al, 1999). Since the FCR and ECR electrodes likely also recorded activity from adjacent synergistic muscles, recordings are subsequently referred to as recordings from wrist flexors and wrist extensors. Synchronized EMG and force data were collected at 4000 Hz and saved to files for offline analysis. EMG signals were demeaned, full wave rectified, and filtered forward and backward using a second order Butterworth low pass filter with a cutoff frequency of 10 Hz.

At the beginning of each session, the participant performed maximal voluntary isometric contractions (MVICs) of the right wrist flexors and extensors, two trials each, while force was displayed on a monitor placed at eye level, 70 cm from the participant's eyes. Each effort lasted 3-5 seconds, and MVIC trials were separated by at least 1 minute of rest. Each training session then included a set of 6 test trials with varying frequency and force requirements, followed by 10 training trials with stable conditions. During each trial, 6 cycles of a sinusoidal curve were gradually drawn on the monitor from left to right, indicating the target force that the participant was asked to match using isometric contraction of his wrist muscles. Force exerted by the participant was also displayed, providing real time visual feedback (Figure 1). Frequency and amplitude of the sinusoidal curve were varied to alter the requirements of the task. The 6 test trials consisted of 3 frequencies (0.10, 0.15, and 0.20 Hz), each performed at $\pm 10\%$ and $\pm 20\%$ of the largest wrist extension force measured during MVIC trials on the same day. Each training trial was performed with a frequency of 0.15 Hz and a force range of $\pm 10\%$ of the largest wrist extension MVIC force. Test trials were separated by 30 seconds, training trials were separated by at least 15 seconds, and a 3-minute rest occurred at the conclusion of the test, before the training trials began. During each participant's first session, three practice trials were performed before the test trials, to familiarize the participant with the task and to verify their understanding.

Data Analysis

Data were analyzed using custom-written MATLAB programs (MathWorks, Natick, MA). For MVIC trials, maximum values were determined for wrist extension and wrist flexion force and EMG. For motor control test trials, performance was quantified separately for the extension phases and flexion phases of each trial, based on the peaks and valleys of the target force curve, as shown in Figure 2. The primary measure of motor control was error, calculated as the root mean square of the participant's exerted force compared to the target force, according to the following equation, where n represents the number of data points in a trial.

$$Error = 2\sqrt{\frac{\sum_{i=1}^n (Target\ force\ i - Exerted\ force\ i)^2}{n}}$$

For each trial, error was averaged over the first 5 extension phases, and over the first 5 flexion phases, and was expressed as a percentage of the target force range for that trial. Lower error values indicate better motor control.

Measures of EMG modulation were analyzed to examine changes in muscle activation and relaxation patterns, and their associations with error. Data from the healthy participants showed that extensor EMG gradually increased throughout the extension phase, peaked around the transition from extension to flexion, and decreased during the flexion phase. Likewise, flexor EMG gradually increased during the flexion phase and decreased during the extension phase. Thus the percent modulation from the first third of each phase to the last third of the phase was calculated for each muscle, according to the following equation,

where nEMG is the average of the filtered EMG signal expressed as a percentage of the maximum EMG signal recorded for that muscle during MVIC trials on the same day (Wagner, Dromerick, Sahrman, and Lang, 2007).

$$EMG \% Modulation = \left(\frac{(nEMG \text{ last third} - nEMG \text{ first third})}{nEMG \text{ first third}} \right) \times 100$$

Positive EMG modulation values represent muscle activation, and negative values represent relaxation.

Data were averaged across the 6 test trials in each session. Linear regression was used to identify significant changes over the course of the intervention days in all variables, and the relationships between error and EMG modulation. Statistical significance was assumed if $p < 0.05$. Correlations were considered weak if $r > 0.3$ and < 0.5 , moderate if $r = 0.5$ and < 0.7 and strong if $r = 0.7$.

Other Impairment Level Outcome Measures

Passive and active range of motion into wrist flexion and wrist extension were measured on the right side, in accordance with the method described by Norkin and White (2107) and da Silva Camassuti et al. (2015). Muscle tone was assessed at the elbow and wrist bilaterally, using the Modified Ashworth Scale (MAS) (Bohannon and Smith, 1987). Maximal grip and pinch forces were measured bilaterally using a Jamar grip dynamometer with a palmar grip and a Jamar pinch gauge with a key grip (Lafayette Instrument, Lafayette, IN) (Mathiowetz, Weber, Volland, and Kashman, 1984; Mathiowetz et al, 1985). Semmes-Weinstein monofilaments for the hand were used to assess touch sensation on the pad of the index finger bilaterally (Bell-Krotoski, 1991).

The upper extremity portion of the Fugl-Meyer Assessment (FMA-UE) was administered on the right side (Gladstone, Danells, and Black, 2002). The FMA is one of the most widely used measures of motor impairment, and although it was developed for people with hemiparesis after stroke, its usefulness for people with SCI has been demonstrated (Prochazka and Kowalczewski, 2015; Yu et al, 2020). It was included with particular interest in the “Wrist” subscale, which assessed the participant’s ability to achieve and maintain an extended wrist position against gravity and resistance.

To assess corticospinal connectivity, excitability of the wrist flexor and extensor muscles was examined using TMS to elicit motor evoked potentials (MEPs) (MagPro X100 with MagOption, MagVenture, Inc, Alpharetta, GA) (Groppa et al, 2012; Kleim, Kleim, and Cramer, 2007). EMG electrode placements were the same as those described above for the motor control task. Using a neuronavigation system (Brainsight, Rogue Research Inc., Montreal, Quebec, Canada) and a model brain image, an 8 x 6 grid with 1 cm spacing was placed over the left lateral cerebral hemisphere. During the pre-training assessment, each grid site was stimulated twice with a figure-8 TMS coil (Model C-B60, MagVenture), using a monophasic pulse and intensity 100% of the maximum stimulator output (MSO). The coil was held tangential to the head with the handle oriented posterolaterally 45 degrees

to the midline, resulting in posterolateral-to-anteromedial induced currents in the cerebral cortex. After this procedure failed to elicit an MEP with the participant resting or with a low level of background muscle activity, a circular coil with an outer diameter of 110 mm was used to deliver stronger and less focal stimulation (Model C-100, MagVenture). The circular coil was held tangential to the head with its center positioned at the vertex, placing its large ring-shaped magnetic field over the motor cortex. With the participant at rest, stimulus intensity was increased in increments of 10% MSO after every 3 stimuli, from 60% to 100%. Fatigue and discomfort limited the number of stimulations delivered using the circular coil, and prevented additional testing with each muscle in an active state. Inter-stimulus intervals of 5-10 seconds were maintained throughout the TMS procedures. A 300 ms epoch of EMG data corresponding to each TMS pulse was collected through a custom-written LabVIEW program and displayed on a monitor. Data were sampled at 2000 Hz and saved to files for offline analysis. Peak-to-peak MEP amplitudes were determined from unfiltered EMG signals in the 10 to 60 ms post-stimulus time interval.

Activity Level Outcome Measures

The Action Research Arm Test (ARAT) was administered on the right side (Lyle, 1981; Yozbatiran, Der-Yeghiaian, and Cramer, 2008). The ARAT is a performance-based assessment of upper limb functioning, which includes 19 items covering grasping, gripping, pinching and gross arm movements.

The participant also completed the Capabilities of Upper Extremity questionnaire (CUE), which assesses upper extremity functional limitations in individuals with tetraplegia (Marino, Shea, and Stineman, 1998; Oleson and Marino, 2014). The 32 items were administered by interview and scored by the participant on a 0 to 4 scale, where 0 indicated complete difficulty or inability, and 4 indicated no difficulty. In addition to the total score, left and right sub-scores were calculated.

Two weeks after the last training session, the participant was asked to describe any changes he noticed in his ability to move his right wrist and any activities that were either easier or more difficult than they were before the intervention. He was also asked to rate his right wrist movement ability on a 5-point scale, where 1 meant "Much worse than before", 3 meant "About the same as before" and 5 meant "Much better than before".

OUTCOMES

The participant attended 24 training sessions over 92 days. No adverse events occurred. On the first day, his maximum extension force was 21% of the average reference value from the healthy participants (Table 1), and his maximum flexion force was 8% of the average reference value. Maximum wrist extensor and wrist flexor EMG signals during MVIC trials increased over time and were strongly correlated with intervention days (Figure 3). Maximum wrist extension force did not change, however, and there was a significant but small increase in maximum wrist flexion force (Figure 3).

Examples of force and EMG data from the isometric force tracking task are shown in Figure 2, illustrating performance of the participant at the beginning and end of the intervention,

with representative examples from a healthy individual. On the first day, the participant's error was nearly twice the average reference value. Error decreased over time at similar rates in extension phases and flexion phases, and was strongly correlated with intervention days (Figure 4). By the 20th training session, which occurred 71 days after the first training session, the participant's error was similar to the average reference value.

EMG modulation variables were significantly correlated with intervention days only during the flexion phases (Figure 4). There was a moderate increase in flexor activation and a moderate increase in extensor relaxation during flexion phases. Percent modulation of the extensors (relaxation) reached the healthy reference value, but flexor activation remained severely diminished (modulation of approximately 30%, versus the average reference value of 463%). During extension phases, the flexors showed minimal relaxation, much less than the reference average, and there was no change over intervention days. Extensor activity more than doubled during extension phases, but the relationship between extensor activation and intervention days was weak and did not reach significance.

EMG modulation that occurred during the flexion phase was related to error during both phases (Figure 5). Greater activation of the flexors and greater relaxation of the extensors were both moderately associated with less error. In contrast, EMG modulation during the extensor phase was not significantly correlated with error during extension phases ($p > 0.10$), nor flexion phases ($p > 0.9$).

Before the intervention, TMS elicited no MEPs above the customary 50 μV threshold in the right wrist extensors (38, 38, and 36 μV) or flexors (24, 33, and 21 μV), even while using a circular coil and the maximum possible stimulus intensity (100% MSO) (Figure 6). After the intervention, the same protocol produced wrist extensor MEPs in 3 of 3 trials (99, 52, and 100 μV). Flexor responses remained slightly below threshold (46, 47, and 46 μV).

Pre-training and post-training scores on the measures of impairments and activity limitations are shown in Table 2. Active range of motion increased by 6 degrees for right wrist extension and 9 degrees for right wrist flexion, but remained diminished compared to the participant's normal passive range of motion. Modest improvements were also noted in the maximal palmar grip force on the right, and touch sensation on the right index finger. The participant gained 4 points on the FMA-UE, which is less than the 5-point minimal detectable change and 10-point minimal clinically important difference reported for people with hemiparesis (Shelton, Volpe, and Reding, 2001; Wagner, Rhodes, and Patten, 2008). Notably, the improved scores were observed on 4 of the 5 test items in the "Wrist" sub-scale. Spasticity at the right wrist was minimal before the intervention, and the change in Modified Ashworth Scale scores from 1 to 0 is not likely to be clinically significant. Scores on the ARAT and CUE questionnaire also did not change substantially.

Two weeks after the intervention, the participant provided his perspective. He stated: "*I note that I have more ability to flex and extend my right wrist. My wrist will remain straight rather than in a flexed position when I am in bed*" and "*I believe I am able to move my wrist more which may be helping my use of my fingers in the right hand*". On a 5-point scale,

he rated his right wrist movement ability as a 4, indicating it was “Somewhat better than before”.

DISCUSSION

This case report demonstrates the use of technology in rehabilitation, to enable focused training of specific muscle groups, and to reveal motor control strategies associated with a change in performance. Years after his incomplete SCI, the participant was able to improve control of the force production capacity that remained in his partially paralyzed wrist extensor and flexor muscles. He achieved error levels similar to those observed in healthy individuals operating within the same force range. This concurs with a prior study showing intact ankle motor control after incomplete SCI (van Hedel, Wirth, and Curt, 2010). Analysis of EMG modulation during extensor and flexor phases of the task identified two factors that were related to his performance gain: 1) increased flexor activation during flexion phases; and 2) increased extensor relaxation during flexion phases. This deep level of insight into motor control strategies, which are likely to vary across individual clients according to their unique pattern of impairments, may be crucial for the delivery of personalized, precision rehabilitation.

In this case, although the wrist extensors and flexors both showed diminished activation and might benefit from resistance training, improvements in flexor activation would likely have a greater impact on tasks involving precise wrist motor control, like driving a power wheelchair and using adapted eating utensils. Intervention to target wrist extensor relaxation also may be beneficial. The participant’s reduced extensor EMG modulation during flexion phases likely reflects motoneuron hyper-excitability and its influence on voluntary motor control. In the chronic stage after SCI, persistent inward currents in dendrites of motoneurons mediate repetitive discharge of the motoneuron, causing prolonged involuntary firing after volitional activation (D’Amico et al, 2014; ElBasiouny, Schuster, and Heckman, 2010; Heckman, Gorassini, and Bennett, 2005). In addition, mechanisms of spinal inhibition may be disrupted after SCI, including pre-synaptic inhibition, reciprocal inhibition and post-activation depression (Boorman, Lee, Becker, and Windhorst, 1996; ElBasiouny, Schuster, and Heckman, 2010; Faist, Mazevet, Dietz, and Pierrot-Deseilligny, 1994). In this case report, wrist extensor EMG modulation improved with training, suggesting that activity, in the form of motor skill training, may induce adaptive plasticity affecting one or more of these mechanisms. Neurofeedback strategies that increase awareness of antagonist activation may constitute a novel intervention approach for future investigation.

This case report adds to the accumulating evidence revealing neural adaptations in people with SCI, and illustrates the potential to increase corticospinal excitability through training (Beekhuizen and Field-Fote, 2005; Gomes-Osman and Field-Fote, 2015; Jurkiewicz et al, 2007). The participant demonstrated no wrist flexor or extensor MEPs before the training, yet he was able to increase his wrist motor control, without a concomitant change in maximum force production, and after training he demonstrated MEPs above threshold in his wrist extensors. This finding, combined with a prior study showing intact cortical representations in people with minimal voluntary muscle activation after SCI, suggests that

presence versus absence of an MEP may not be a reliable predictor of rehabilitation potential for people with incomplete SCI (Cortes et al, 2017).

Improvement on the training task was accompanied by modest positive changes in other impairment level outcomes, but no substantial gains on the activity level tests. This likely reflects the focused nature of the intervention, which targeted the right wrist specifically, in contrast to the many factors that contribute to overall upper limb function. For certain individuals, however, improved motor control at a specific joint may enable critical functions and allow achievement of individualized goals, which may or may not be reflected in standardized outcome measure scores.

This case report demonstrates an application of technology in rehabilitation, to train and quantify wrist motor control. Outcomes suggest that motor skill training improved precise motor control after incomplete SCI, within the range of this individual's residual force production capacity. Performance gains were associated with specific adjustments in muscle activation and relaxation, and increased corticospinal excitability.

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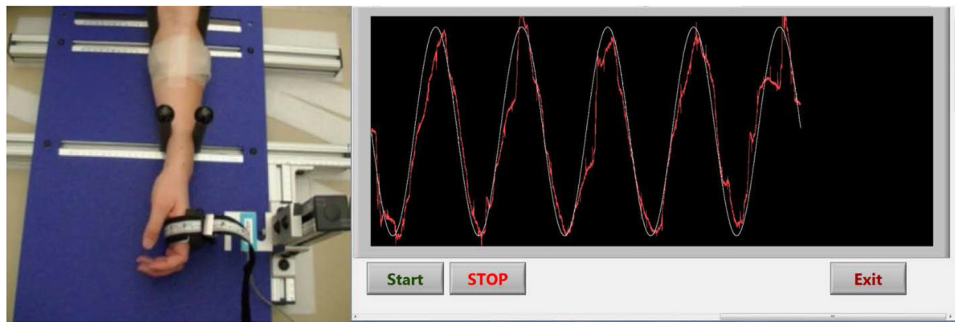


Figure 1. Isometric Visuomotor Force Tracking Task

The custom-designed positioning device stabilized the forearm and hand, with neutral forearm supination/pronation and neutral wrist flexion/extension. Using isometric wrist flexion and extension contractions, the participant attempted to match a target force that appeared as a white line on a screen in front of him. The participant's exerted force was displayed as a red line, providing visual feedback in real time.

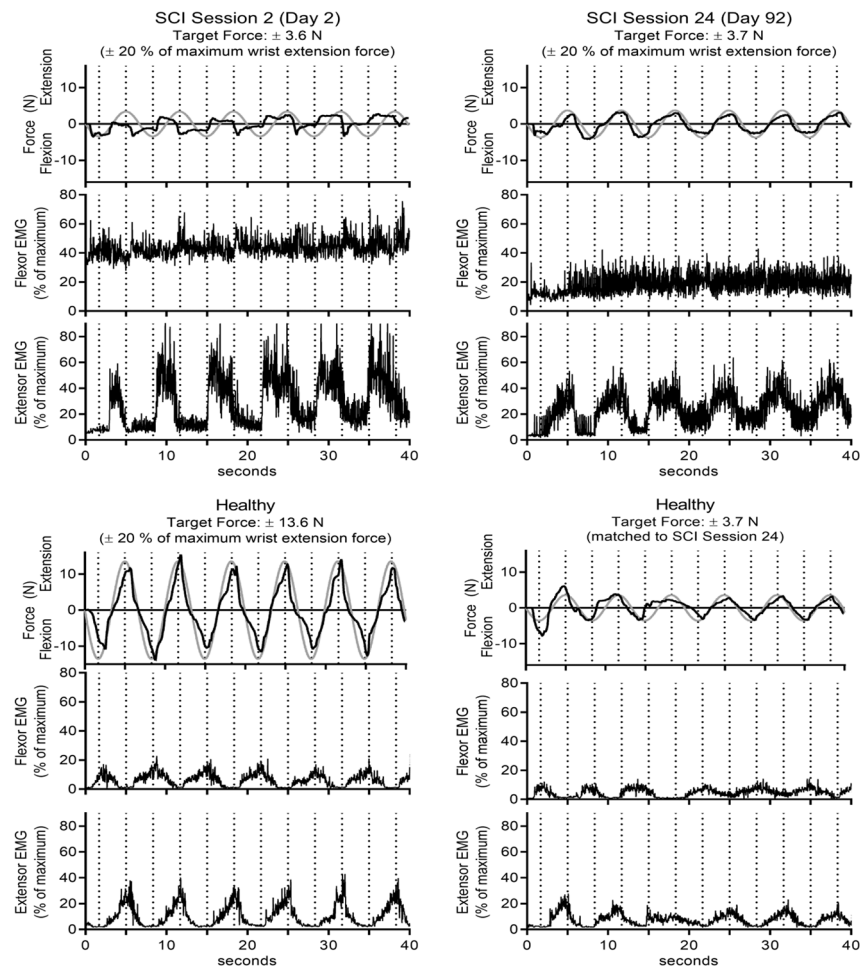


Figure 2. Example Data

Examples of force and EMG recorded during performance of the visuomotor force tracking task. The top two panels show data for the participant with SCI during his 2nd and 24th training sessions. The bottom two panels show data for a healthy individual performing the task with a target force range $\pm 20\%$ of their maximum wrist extension force (bottom left) and the same person with a target force range matched to the participant's target range (bottom right). Vertical dashed lines separate the task into extension phases with an upward slope of the target force trace, and flexion phases with a downward slope of the target force trace.

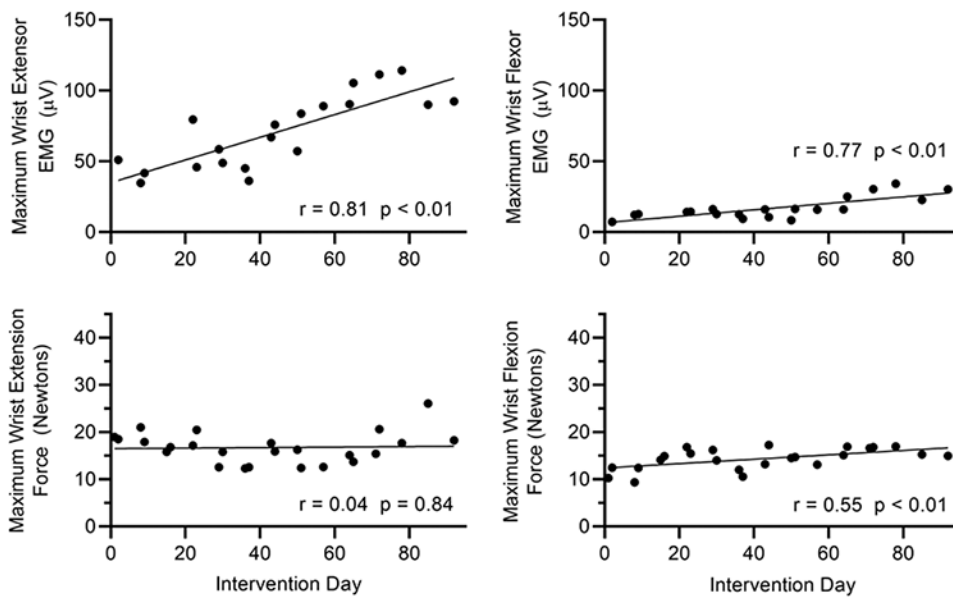


Figure 3. Force and EMG during Maximum Voluntary Isometric Contractions

Maximum EMG amplitudes recorded from the participant's wrist extensors (top left) and wrist flexors (top right) increased and were strongly correlated with study days. A small increase in wrist flexor maximum force also occurred and was moderately correlated with study days (bottom right). Maximum wrist extensor force did not change significantly (bottom left).

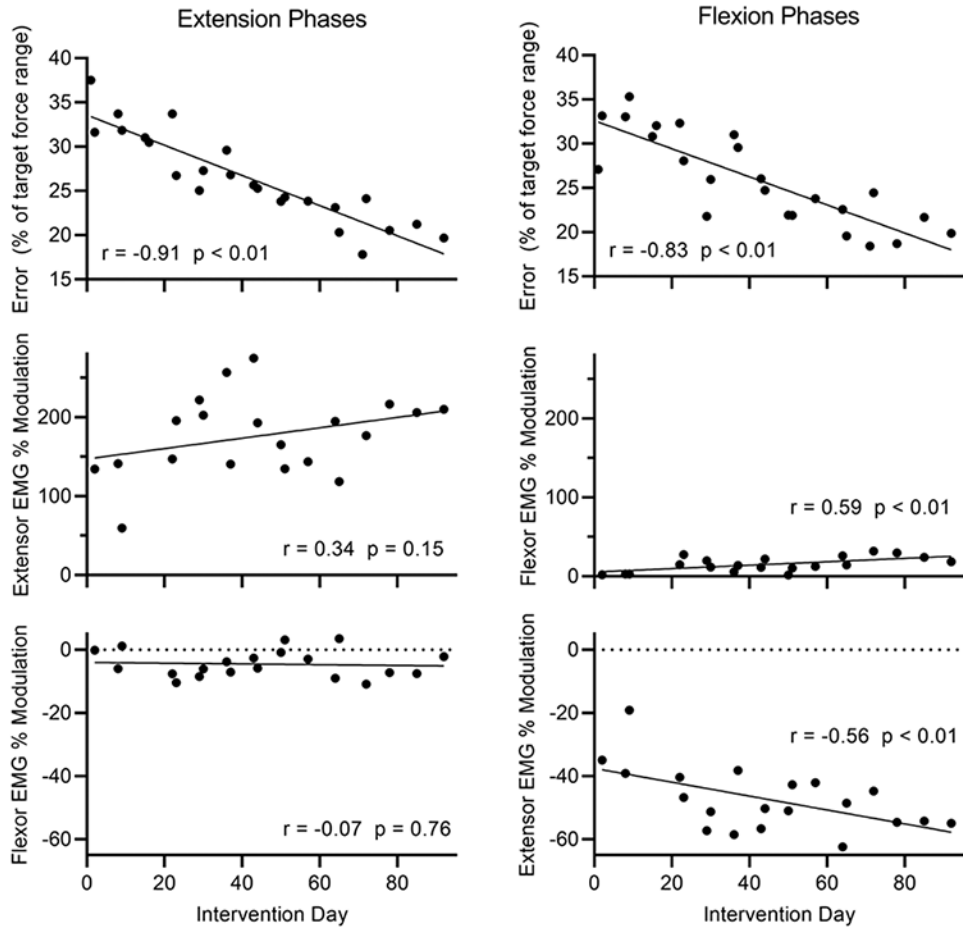


Figure 4. Changes across Intervention Days

Error during the visuomotor force tracking task was strongly related to intervention days, and decreased similarly in extension phases of the task (top left) and in flexion phases (top right). Flexor activation during flexion phases was small throughout the training, but the increase over intervention days was significant (middle right). Extensor activation during extension phases tended to increase over intervention days, but the relationship was weak and did not reach significance (middle left). Wrist extensor relaxation during flexion phases improved over the course of the training, and was moderately correlated with intervention days (bottom right). There was no change in relaxation of the wrist flexors during extension phases (bottom left).

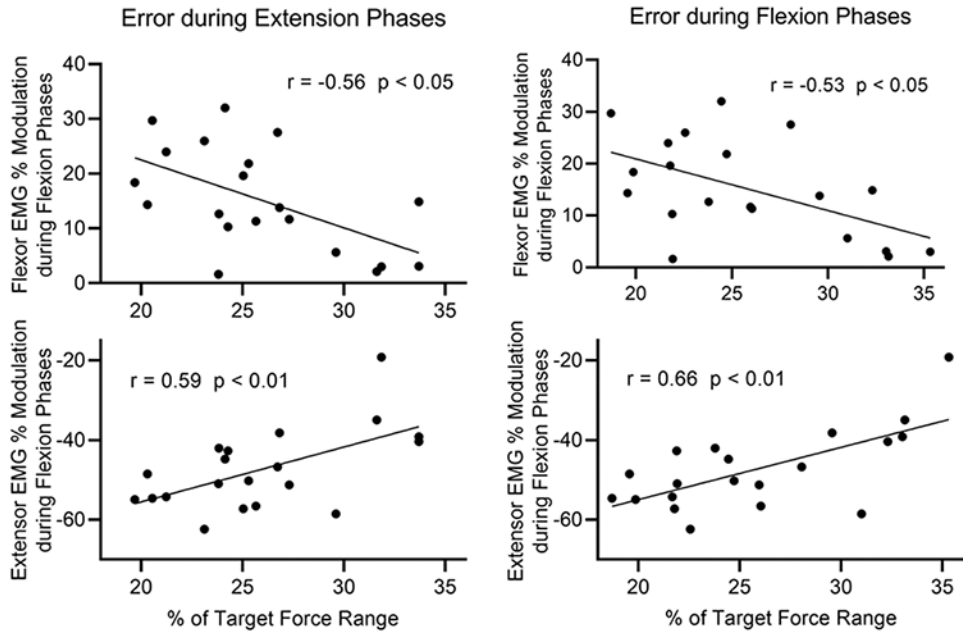


Figure 5. Predictors of Error

Each data point represents one intervention day. Error during both phases of the visuomotor force tracking task was moderately related to EMG modulation that occurred during flexion phases. Greater flexor activation and greater extensor relaxation during flexion phases were moderately associated with less error during the extension phases (left top and bottom), and during the flexion phases (right top and bottom).

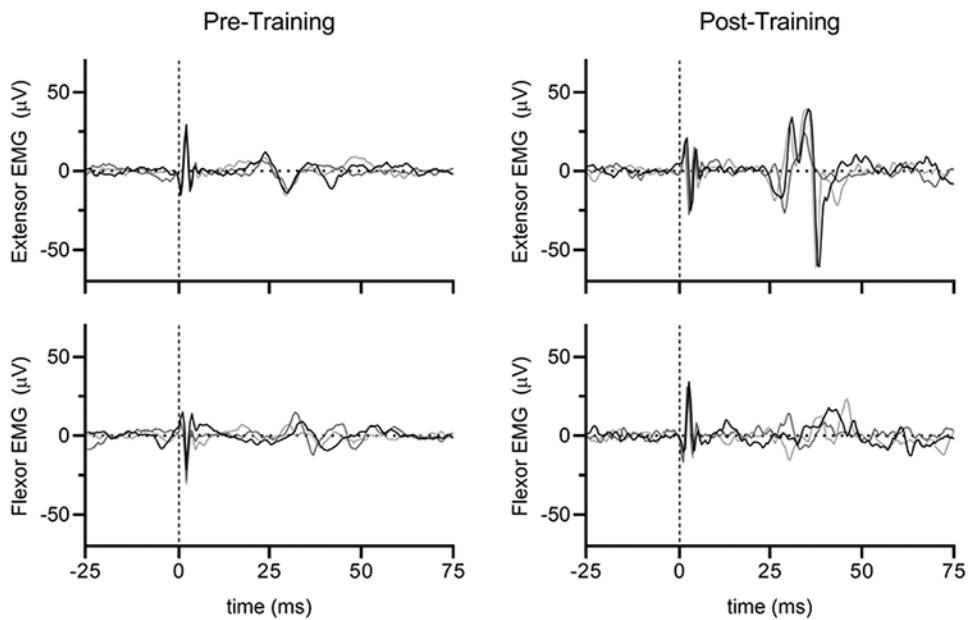


Figure 6. Motor Evoked Potentials

Muscle responses elicited by transcranial magnetic stimulation delivered over the participant's vertex using a circular coil and intensity 100% of the maximum stimulator output. At the beginning and end of the study, a single monophasic pulse was delivered 3 times, each separated by 5-10 seconds. Before training, no MEP > 50 μ V was recorded from the wrist extensors (top left) or wrist flexors (bottom left). After training, 3 of 3 wrist extensor responses exceeded the threshold (top right). Flexor responses remained < 50 μ V after training (bottom right).

Table 1.

Reference Values from Healthy Participants

MVICs	Wrist Extensors	Wrist Flexors
Maximum EMG (μV)	186 \pm 30	481 \pm 120
Maximum Force (Newtons)	90 \pm 8	128 \pm 14
Isometric Force Tracking Task	Extension Phases	Flexion Phases
Error (% of target force range)	17.3 \pm 2.9	19.5 \pm 3.5
Extensor EMG % Modulation	262 \pm 38	-53 \pm 8
Flexor EMG % Modulation	-56 \pm 17	463 \pm 133

Mean \pm SE; MVICs, maximal voluntary isometric contractions, EMG, electromyography

Table 2.

Measures of Impairments and Activity Limitations

	Before Intervention		After Intervention	
	Left	Right	Left	Right
Passive Range of Motion (degrees) ^a				
Wrist extension	nt	75	nt	75
Wrist flexion	nt	70	nt	70
Active Range of Motion (degrees) ^a				
Wrist extension	nt	32	nt	38
Wrist flexion	nt	25	nt	34
Modified Ashworth Scale ^b				
Wrist extensors	1	1	1	0
Wrist flexors	1	1	1	0
Elbow extensors	1	1	0	1
Elbow flexors	2	0	2	1
Maximal palmar grip force (kg) ^c				
	5.5	2.0	4.3	4.3
Maximal key-grip pinch strength (kg) ^c				
	3.5	2.0	4.3	2.0
Semmes-Weinstein monofilament test ^d				
Pad of the index finger	3.61	4.31	3.61	3.61
Fugl-Meyer Assessment of the Upper Extremity ^e				
Right side only	nt	23	nt	27
Action Research Arm Test ^f				
Right side only	nt	15	nt	14
Capabilities of Upper Extremity Questionnaire ^g				
Left and right item totals	21	22	23	22
Total score	44		45	

^aEstimates of normal wrist flexion and extension active and passive range of motion are between 60 and 75 degrees for each movement direction (da Silva Camassuti, 2015; Norkin and White, 2017)

^bMAS scores can range from 0, indicating no spasticity, to 4, indicating that the affected body part is rigid (Bohannon and Smith, 1987)

^cFor men ages 70-74 years, the average normative grip force value is 34.2 ± 9.8 kg (mean \pm SD) and the average normative key pinch value is 8.8 ± 1.1 kg. (Mathiowetz, Weber, Volland, and Kashman, 1984; Mathiowetz et al. 1985)

^dSize of the smallest monofilament that the participant could feel in at least 3 of 5 trials. People with intact sensation typically are able to feel the 2.83 monofilament size. Inability to feel the 3.61 monofilament indicates diminished protective sensation (Bell-Krotoski, 1991)

^eScores on the FMA-UE range from 0 to 66 points, with higher scores indicating less impairment (Gladstone, Danells, and Black, 2002)

^fScores on the ARAT range from 0 to 57 points, with higher scores indicating better upper limb functioning (Lyle, 1981; Yozbatiran, Der-Yeghiaian, and Cramer, 2008)

^gTotal scores on the CUE Questionnaire range from 0 to 128. Left and right sub-scores each range from 0 to 60 (Marino, Shea, and Stineman, 1998; Oleson and Marino 2014); nt, not tested