Increased spinal reflex excitability is associated with enhanced central activation during voluntary lengthening contractions in human spinal cord injury

Hyosub E. Kim,1,2 Daniel M. Corcos,3 and T. George Hornby1,2,4
1Graduate Program in Neuroscience, University of Illinois at Chicago, Chicago, Illinois; 2Sensory Motor Performance Program, Rehabilitation Institute of Chicago, Chicago, Illinois; 3Department of Physical Therapy & Human Movement Sciences, Northwestern University, Chicago, Illinois; and 4Department of Physical Therapy, University of Illinois at Chicago, Chicago, Illinois

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Kim HE, Corcos DM, Hornby TG. Increased spinal reflex excitability is associated with enhanced central activation during voluntary lengthening contractions in human spinal cord injury. J Neurophysiol 114: 427–439, 2015. First published May 13, 2015; doi:10.1152/jn.01074.2014.—This study of chronic incomplete spinal cord injury (SCI) subjects investigated patterns of central motor drive (i.e., central activation) of the plantar flexors using interpolated twitches, and modulation of soleus H-reflexes during lengthening, isometric, and shortening muscle actions. In a recent study of the knee extensors, SCI subjects demonstrated greater central activation ratio (CAR) values during lengthening (i.e., eccentric) maximal voluntary contractions (MVCs), compared with during isometric or shortening (i.e., concentric) MVCs. In contrast, healthy controls demonstrated lower lengthening CAR values compared with their isometric and shortening CARs. For the present investigation, we hypothesized SCI subjects would again produce their highest CAR values during lengthening MVCs, and that these increases in central activation were partially attributable to greater efficacy of Iα-motoneuron transmission during muscle lengthening following SCI. Results show SCI subjects produced higher CAR values during lengthening vs. isometric or shortening MVCs (all \(P < 0.001\)). H-reflex testing revealed normalized H-reflexes (maximal SOL H-reflex-to-maximal M-wave ratios) were greater for SCI than controls during passive (\(P = 0.023\)) and active (i.e., 75% MVC; \(P = 0.017\)) lengthening, suggesting facilitation of Iα transmission post-SCI. Additionally, measures of spinal reflex excitability (passive lengthening maximal SOL H-reflex-to-maximal M-wave ratio) in SCI were positively correlated with soleus electromyographic activity and CAR values during lengthening MVCs (both \(P < 0.05\)). The present study presents evidence that patterns of dynamic muscle activation are altered following SCI, and that greater central activation during lengthening contractions is partly due to enhanced efficacy of Iα-motoneuron transmission.

spinal cord injury; H-reflex; eccentric contractions; central activation ratio

DAMAGE TO DESCENDING MOTOR pathways is the primary factor contributing to deficits in strength and voluntary activation of muscle following motor incomplete spinal cord injury (SCI). At the level of the muscle, significant atrophy, alterations in fiber phenotypes, and increased fatigability affect force-generating capacity (Cope et al. 1986; Martin et al. 1992; Shields 1995). However, reflex pathways and intrinsic neuronal properties at spinal levels below the lesion are also severely affected by SCI (Faist et al. 1994; Murray et al. 2010; Nielsen et al. 2007; Thompson et al. 2011b), and questions remain as to how these changes affect central activation of muscle (i.e., activation due to central nervous system processes, including both descending drive and sensory inputs) (Kent-Braun 1997). Specifically, there is little information regarding patterns of central activation post-SCI during dynamic (shortening and lengthening) contractions, in which diverse sensory inputs to spinal inter- and motoneurons are activated. This gap in knowledge may partly result from the common practice of investigating strength and muscle activation post-SCI using isometric testing, where muscle length and joint angle remain constant. While isometric measures are important, functional movement is composed of dynamic contractions. The investigation of dynamic muscle contractions post-SCI will therefore improve understanding of patterns of central activation and the neural control of functional movements.

In a recent study of dynamic contractions of the knee extensors (KEs), SCI subjects demonstrated a pattern of central activation markedly different from that of healthy noninjured adults (Kim et al. 2015). SCI subjects produced greater central activation ratio (CAR) values and KE electromyographic (EMG) activity during lengthening maximal voluntary contractions (MVCs) than during isometric and shortening MVCs. CAR values were quantified by providing supramaximal muscle stimulation during an MVC and calculating the ratio of voluntary torque to peak superimposed torque (Kent-Braun and Le Blanc 1996). Although healthy controls demonstrated higher CAR values than SCI subjects across all contraction types, during lengthening MVCs their measures of central activation (i.e., CAR values and KE EMG) were depressed compared with during isometric or shortening MVCs. Previous studies in different muscle groups have also reported that healthy adults demonstrate deficits in central drive during lengthening MVCs, whether quantified by EMG amplitude (Pinninger et al. 2000; Westing et al. 1991) or through use of evoked twitches (Babault et al. 2001; Beltman et al. 2004; Loscher and Nordlund 2002). In contrast, with regards to SCI, the generalizability of our initial finding of greater central activation during lengthening MVCs is unknown.

One hypothesized mechanism contributing to the increased agonist muscle activity during lengthening MVCs observed in...
SCI subjects was enhanced activation of homonymous motoneurons from stretch reflex inputs (Ia excitation). Stretch reflex hyperexcitability, or spasticity, is thought to affect 65–78% of the chronic SCI population (Maynard et al. 1990; Skold et al. 1999). There are many potential mechanisms underlying spasticity following SCI, including increased activity in type II fibers (Eriksson et al. 1996), decreased reciprocal inhibition (Crone et al. 2003), and increased motoneuron excitability (Gorassini et al. 2004). However, the main excitatory component of spasticity comes from Ia afferents (Nielsen et al. 2007). Therefore, as the agonist is being forcibly stretched during a lengthening contraction, increased efficacy at Ia-α motoneuron synapses may serve to further excite the motor pool.

Prior to the present study, this hypothesis regarding spinal reflex excitability, specifically Ia-α motoneuron transmission during muscle lengthening, had yet to be investigated in subjects with SCI. However, it has been repeatedly demonstrated in healthy adults that the H-reflex, an electrophysiological measure of Ia-α motoneuron transmission (Capaday 1997; Zehr 2002), is depressed during muscle lengthening compared with during isometric and shortening muscle actions. Specific depression of the H-reflex during lengthening has been reported in healthy adults for passive rotations (Duclay et al. 2011; Duclay and Martin 2005; Pinniger et al. 2001; Romanò and Schieppati 1987), submaximal contractions (Nordlund et al. 2002), and MVCs (Duclay et al. 2011; Duclay and Martin 2005). Potential spinal mechanisms underlying this phenomenon include presynaptic inhibition of Ia afferent terminals and homosynaptic postactivation depression (HPAD). HPAD is a reduction in neurotransmitter release in previously activated afferent fibers (Curtis and Eccles 1960), and it likely plays a larger role during passive stretches than during active contractions (Pinniger et al. 2001). Interestingly, some have also linked these same mechanisms to decreased central activation during lengthening MVCs (Duchateau and Baudry 2014; Duchateau and Enoka 2008).

Although modulation of spinal reflex excitability via the H-reflex technique has been studied in SCI patients during gait (Fung and Barbeau 1994; Yang et al. 1991), there is little data on how Ia-α motoneuron transmission is specifically modulated during even simple, controlled single-joint dynamic contractions. The importance of assessing H-reflexes during dynamic changes to muscle length in SCI subjects is highlighted by the potential relationship between the specific modulation of Ia excitation and control of lengthening contractions described for healthy adults (Duchateau and Baudry 2014; Duchateau and Enoka 2008). Hence, the aims of this study were to investigate patterns of central activation of the plantar flexor (PF) muscles using interpolated twitches, and modulation of soleus (SOL) H-reflexes during lengthening, isometric, and shortening muscle actions. Our primary objectives were to test the generalizability of our initial findings from the KEs, and to investigate the hypothesis that specific modulation of spinal reflex excitability post-SCI may contribute to distinct patterns of central activation. The PFs were chosen as a model muscle due to their importance in locomotion and the relative ease of eliciting H-reflexes from SOL (Zehr 2002). We hypothesized that subjects with SCI would demonstrate increased central drive during lengthening MVCs, and that specific gains in motor output could be partly attributed to a relative increase in efficacy of Ia-α motoneuron transmission compared with healthy adults.

**METHODS**

**Subjects**

Eight men with chronic (>1 yr) motor incomplete SCI (mean age: 49 yr; range: 31–66 yr) were recruited from the outpatient clinics of the Rehabilitation Institute of Chicago. Seven healthy, uninjured men (mean age: 40 yr; range: 30–54 yr) reporting regular participation in at least moderate levels of physical activity also volunteered to serve as control subjects.

Subjects with SCI were classified as either C or D using the American Spinal Injury Association Impairment Scale (AIS). AIS grades C and D indicate some preservation of motor function below the neurological level (i.e., lowest segment where motor and sensory function is normal on both sides), with either less than half (AIS C) or at least half (AIS D) of the key muscles below the neurological level being able to generate movement against gravity throughout their respective joint range of motion (ROM) (Maynard et al. 1997). Inclusion criteria for SCI subjects included minimum passive ankle ROM from −5° (dorsiflexion) to +25° (plantar flexion; 0° equals neutral ankle) without pain and a minimum AIS Lower Extremity Motor Score of 2 (scale: 0–5) (Marino and Graves 2004) in at least one limb during clinical examination, indicating “active movement, full ROM with gravity eliminated.” Findings from clinical testing were confirmed by visualization of torque and EMG signals during experimental testing.

Exclusion criteria included medical history of multiple central nervous system lesions, history of lower limb peripheral nerve injury, orthopedic injury, which would limit PF contractions, or use of anti-spasticity medications at the time of this study. All SCI and control subjects were familiar with the testing apparatus and had prior experience with electrical stimulation. Informed, written consent was provided by all subjects, and all procedures were conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of Northwestern University.

**Clinical Examination**

Two licensed physical therapists performed all clinical examinations. Testers examined strength (Lower Extremity Motor Score range: 0–5) (Marino and Graves 2004), clinical signs of spastic motor behaviors of the PFs and dorsiflexors (DFs) using a variation of the Modified Ashworth Scale technique (Kim et al. 2015; Thompson et al. 2011a), and other spastic motor behaviors, including spasms, using the Spinal Cord Assessment Tool for Spastic Reflexes (Benz et al. 2005). Patellar and Achilles deep tendon reflexes were also tested and scored using the Medical Research Council grading scale (Thijs et al. 1998). All SCI subjects demonstrated some form of spastic motor behaviors, and over half reported being community ambulators (see Table 1).

**Study Design**

**Experiments A and B** were performed on 2 nonconsecutive days, separated by at least 72 h with the order of experiments pseudorandomized. Experiment A assessed muscle mechanical properties and central activation of the PFs. Experiment B investigated modulation of passive and active SOL H-reflexes during lengthening, isometric, and shortening conditions. All eight SCI and seven control subjects participated in all experimental procedures and were instructed to not perform any strenuous exercise for 48 h before testing sessions.

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Table 1. Subject demographics

| Subject No. | Age, yr | Years Post-SCI | Neurological Level | AIS Class | LEMS: PF/DF | Total | Ambulation Level | Assistive Device | DTRs: Ptl/Ach | MAS: PF/DF | SCATS: C/F/E |
|-------------|---------|----------------|--------------------|-----------|-------------|-------|-----------------|------------------|--------------|------------|-------------|-----------|
| SCI-1       | 51      | 29             | C5–6               | D         | 2/3         | 19    | Community       | Canes            | + + + / + + +  | 2/1         | 1/1/2       |            |
| SCI-2       | 66      | 12             | C3–4               | D         | 4/2         | 17    | Household       | Crutches          | + + / +         | 1/0         | 2/2/1       |            |
| SCI-3       | 31      | 4              | C7                  | D         | 5/5         | 25    | Community       | None             | + + + / + + +  | 0/0         | 3/1/0       |            |
| SCI-4       | 41      | 16             | C4–6               | D         | 2/1         | 14    | Community       | Walker            | + + + / + + +  | 2/0         | 3/2/3       |            |
| SCI-5       | 56      | 13             | C2–4               | C         | 4/5         | 20    | Household       | Walker            | + + + / + + +  | 0/0         | 3/0/0       |            |
| SCI-6       | 51      | 18             | C5–7               | D         | 5/5         | 25    | Community       | None             | + + + / 0       | 2/0         | 0/3/3       |            |
| SCI-7       | 52      | 7              | C6–7               | C         | 2/4         | 20    | Community       | Canes            | + + + / + + +  | 0/0         | 1/1/3       |            |
| SCI-8       | 51      | 4              | T2–4               | C         | 2/3         | 17    | Household       | Walker            | + + + / + + +  | 2/0         | 3/2/3       |            |

SCI, spinal cord injury; AIS, American Spinal Injury Association Impairment Scale; LEMS, Lower Extremity Motor Scores (scale: 0–5; maximum total = 25); PF, plantar flexor; DF, dorsiflexor; DTRs, deep tendon reflexes, Ptl/Ach, patellar/achilles (Medical Research Council Scores: 0, absent; +, hypoactive; + +, normal; + + +, hyperactive; + + + +, clonus); MAS, Modified Ashworth Scores; SCATS, Spinal Cord Assessment Tool for Spastic Reflexes (C, clonus; F, flexor; E, extensor; scale: 0–3).

Mechanical Data

For all experiments, subjects were seated in the adjustable height testing chair of an isokinetic dynamometer system (System 3: Biodex Medical Systems, Shirley, NY) with hips flexed 75°, and the knee joint of the tested limb flexed 40° (0° = full extension). We chose a comfortable bent knee position (Pierrot-Deseilligny and Burke 2005) easily attainable by all SCI subjects, and one which would reduce contributions of the gastrocnemius to plantar flexion torque (Cresswell et al. 1995). Therefore, consistent with prior studies of dynamic H-reflexes in the PFs, the focus of this study was on SOL activation and reflex modulation (Duclay and Martin 2005; Nordlund et al. 2002). The right lower limb of all control subjects was used for testing, whereas for SCI subjects, whichever limb met inclusion criteria (see Subjects) was chosen for testing. In cases where both legs met inclusion criteria, the more affected side was tested. The tested foot was secured to a footplate coupled to a 6 degrees-of-freedom load cell. The ankle axis of rotation was aligned with the center axis of the load cell. Subjects were secured by belts across the abdomen as well as the tested thigh, to further stabilize the leg (Todd et al. 2004). Total ankle ROM was 30° (−5° to +25°) for all dynamic testing (exceptions noted below), while isometric trials were performed at midrange with the ankle at a constant angle of 10° plantar flexion. For H-reflex testing during submaximal contractions, a shorter 10° ROM centered around the same constant angle of 10° plantar flexion (i.e., +5 to +15°) was used for all tested SCI subjects and controls. Based on pilot data which were collected, subjects with SCI had difficulty maintaining constant levels of SOL activation during 30° of movement, but were able to successfully do so during 10° of motion. Moreover, based on a previous study (Pinniger et al. 2001) and pilot data, this range was judged sufficient to observe distinct patterns of spinal reflex modulation consistent with those observed during a 30° rotation. Care was taken during all test sessions to ensure the same posture was maintained during all test trials, especially during H-reflex testing, to keep consistent vestibular influences on motor pool excitability (Zehr 2002).

To remove torques caused by the weight of the tested limb (Fillyaw et al. 1986; Nelson and Duncan 1983), 5/s passive trials through the tested range were recorded prior to the test trials. The slow speed during passive trials was chosen to ensure spastic reflexes would not be elicited, with EMG visually inspected online and later analyzed offline to ensure negligible muscle activity [i.e., EMG signal indistinguishable from baseline noise with EMG root mean square (RMS) < 3 μV] (Konrad 2005) throughout the whole movement. During offline analysis, curve-fitting of these passive torque signals was performed using a third-degree polynomial (cubic function). These position-varying values resulting from the weight of the limb were subsequently subtracted from all test trials to obtain active torque values. Torque signals were low-pass filtered at 200 Hz and collected at 2,000 Hz. Surface EMG signals were recorded using active bipolar electrodes (Delsys, Boston, MA). The SOL electrode was placed in the middorsal line of the lower leg, 5 cm distal to where the two heads of the gastrocnemius join the Achilles tendon (Duclay et al. 2011; Duclay and Martin 2005). To measure antagonist coactivation, an additional electrode was placed over the belly of the tibialis anterior (TA) muscle, one-third of the distance on the line from the fibular head to the medial malleolus (Hermens et al. 2000). All EMG signals were amplified (×1,000), band pass filtered (20–450 Hz), and sampled at 2,000 Hz simultaneously with the torque and position data.

Stimulation

For both experiments, percutaneous stimulation of the posterior tibial nerve was delivered in the form of a rectangular pulse (1 ms) via a constant-current stimulator (model DS7, Digitimer, Welwyn Garden City, UK) to elicit maximal twitches and M-waves during Experiment A, and H-reflexes and M-waves during Experiment B. All procedures involved placing the cathode (10-mm diameter, Ag-AgCl, Noraxon) into the popliteal fossa and the anode (2-in. diameter, Tyco Uni-Patch, Wabasha, MN) over the patella. A custom-made hand-held cathode was used to locate the stimulation site providing the greatest amplitude response. Once located, the stimulating electrode was fully secured to this site using athletic wrapping tape. For Experiment A, single and paired pulses (10-ms interpulse interval) were used to evoke maximal twitches. Maximal stimulus intensity was determined by progressively increasing the stimulus intensity while subjects remained passive until both the maximal M-wave (Mmax) and torque responses reached a plateau. To further ensure complete depolarization of motor axons, the maximal stimulus intensity was increased by an additional 20% and used for the remainder of testing. For Experiment B, single pulses were used to elicit SOL H-reflexes and M-waves. For all experimental sessions, custom-designed LabVIEW software (National Instruments, Austin, TX) was written to trigger stimulations as the ankle passed 10° plantar flexion (midrange) to make valid comparisons of central activation across isometric and dynamic contractions, and also to avoid changes in H-reflex size due to changing muscle lengths. Stimulation at consistent muscle lengths also helped control for potential contributions from type II sensory fibers to differential activation patterns and H-reflex modulation, as their activity primarily encodes for muscle length as opposed to the velocity of muscle length changes (Matthews 1972).

Experimental Protocol

All experimental procedures lasted ~1.5–2 h.

Experiment A. Experiments began with assessment of muscle mechanical properties by recording mechanical responses of the PFs to three single pulses and three paired pulses of supramaximal elec...
trical stimuli delivered to the posterior tibial nerve. Subjects were then asked to perform several submaximal isometric PF contractions to confirm their comfort with the testing apparatus, as well as their understanding of the required task. Next, supramaximal paired electrical stimuli (120% Mmax stimulation intensity; 10-ms interpulse interval) were manually delivered by the tester during the torque plateau (Miller et al. 1999) of three isometric MVCs. Individual isometric MVCs lasted 3–5 s. We chose to use paired stimuli during MVCs without comparison to potentiated resting twitches. This method is capable of producing comparable central activation values to those utilizing potentiated resting twitches (Klass et al. 2005; Morse et al. 2005) and has the advantage of not requiring extra stimulations, which are particularly fatiguing for patients. Following isometric PF MVCs, subjects performed three dorsiflexion isometric MVCs. Next, shortening MVCs followed by lengthening MVCs were performed at +20°/s and −20°/s (+” and “−” indicate shortening and lengthening velocities, respectively). The order for the different contraction types was predetermined to reduce the risk of confounding the results due to possible muscle damage from lengthening contractions (Enoka 1996; Klass et al. 2005). Similar to the isometric condition, subjects were provided time to perform submaximal trials to ensure proper task procedures, prior to performing three MVCs for each contraction type. For each dynamic contraction, prior to the start of movement, subjects were instructed to produce maximal isometric torque and to sustain

Fig. 1. Representative data during plantar flexor (PF) maximal voluntary contractions (MVCs). Comparison of central activation patterns between representative healthy control (A) and spinal cord injury (SCI) (B) subjects is shown. A: the healthy control subject shows near maximal central activation across all contraction types (i.e., small evoked twitch). B: the SCI subject demonstrates overall deficits in central activation across contraction types, but generates greater volitional torque and soleus (SOL) electromyogram (EMG) during lengthening MVCs. TA, tibialis anterior.
maximal effort throughout the entire ROM (Fig. 1). Hence, each dynamic MVC departed from a 2- to 3-s isometric MVC (Fig. 1). Subjects were given strong verbal encouragement during all MVCs, and >2 min rest was provided between all trials (Gandevia 2001). In cases where CAR values for the three trials were not within 10% of each other, additional MVCs were performed. Less than 5% of experimental sessions required performance of additional MVCs.

**Experiment B. Isometric recruitment curves.** To test baseline spinal reflex excitability at rest, passive isometric recruitment curves were performed first. Stimulus intensities inducing maximal SOL H-reflexes (Hmax) and Mmax were carefully recorded. Prior to building isometric recruitment curves, estimates of H-reflex threshold and Mmax were made for each subject by adjusting the stimulus intensity settings and visually examining responses on an oscilloscope. This preliminary step was quickly performed to determine the appropriate step increases in intensity level for each subject to have ~10 intensity levels for each recruitment curve. Depending on the individual, increments of 0.5–2.0 mA were used for all recruitment curves. Four stimuli, with each pulse separated by 10 s, were delivered at every intensity level. Next, four stimuli at supramaximal intensity (120% of Mmax stimulus intensity) were delivered, and the mean value of the responses was taken as resting isometric Mmax. For both isometric and dynamic (below) H-reflex testing, if there was excessive variability in either Hmax or Mmax (i.e., coefficient of variation was > 7.5%), four additional trials were performed. After completion of the isometric recruitment curve, subjects performed three isometric PF MVCs with no superimposed stimulation. The purpose of these MVCs was to determine the SOL RMS associated with the highest peak torque to set the target range for the active portion of testing. Following the PF MVCs, subjects performed two DF isometric MVCs. All MVCs were separated by >2 min rest.

**Dynamic recruitment curves.** Following isometric recruitment curves, dynamic recruitment curves were performed to investigate the effects of passive movement on H-reflex modulation. The order of passive shortening and lengthening recruitment curves was randomized among subjects. The ankle began at either −5° (shortening) or +25° (lengthening) and was held in that position for 10 s and then moved at 20°/s through the ROM. This moderate speed was chosen to ensure muscle stretch would not elicit reflex activity and increase background EMG levels during passive testing (Pierrot-Deseilligny and Burke 2005). Electrical stimulation of the tibial nerve was triggered as the ankle passed +10° (isometric testing angle). Similar to previous investigations of dynamic H-reflex modulation (Duclay et al. 2011; Duclay and Martin 2005), after reaching the end position, the ankle was then held for 4 s prior to being returned to the start position. Once returned, subjects were asked to perform a submaximal isometric contraction for 1 s to obtain similar thixotropic effects that may have resulted from the preceding change in muscle length between conditions (Duclay and Martin 2005; Prose et al. 1993).

**Active H-reflexes.** To investigate modulation of spinal reflexes during high-intensity volitional contractions, H-reflexes and M-waves were recorded during 75% MVC isometric and dynamic contractions. The purpose of recording H-reflexes during voluntary contractions was to draw inferences as to how specific modulation of Ia-α motoneuron transmission may contribute to distinct patterns of central activation during MVCs in SCI subjects. However, during pilot data collections, we observed greater SOL EMG during lengthening MVCs compared with during isometric or shortening MVCs. As background EMG levels need to be consistent across conditions to make valid comparisons of Ia-α motoneuron transmission across tasks (Pierrot-Deseilligny and Burke 2005; Zehr 2002), we chose an appropriately high-intensity contraction where EMG levels could be consistently maintained during all contraction types. For all active trials, the level of SOL activation was set to 75% of a maximal isometric effort (described above) using real-time visual feedback of the SOL RMS on a computer monitor placed ~6 ft. in front of the subject. When subjects were within ±10% of the target level (65–85% MVC level), a large green light on the monitor would flash on. In the case of isometric contractions, this condition would need to be maintained for ~1 s prior to manual delivery of the stimulation by the experimenter. For the dynamic conditions, subjects were asked to maintain this level of effort in the start position prior to their ankle being rotated at 20°/s by the dynamometer. Subjects were all instructed to “keep the green light on” during movement by maintaining a constant level of SOL RMS. A series of familiarization trials were performed prior to all test trials until consistency with maintaining a constant level of SOL activation was achieved. For each muscle action type, stimulus intensity to elicit passive Hmax intensity was used in six trials followed by three trials using passive Mmax intensity. Greater than 90 s rest was provided between contractions. For the Hmax and Mmax trials, if after the initial attempts less than three trials met the criteria, enough additional trials were performed until three fell within the correct range. Similar to CAR testing, however, there were few instances where extra trials were required. The isometric condition was always first, and the ordering of shortening and lengthening conditions was counterbalanced among all subjects.

**Data Analysis**

Data were collected and analyzed using custom-designed LabVIEW software (National Instruments, Austin, TX). 

**Torque.** Torque signals were low-pass filtered at 25 Hz using a fourth-order, zero-phase lag Butterworth filter. For Experiment A, maximal constant angular torques at 10° were obtained prior to stimulation under isometric, shortening, and lengthening conditions.

**CAR values.** CAR values were calculated using the following formula: CAR = peak MVC torque/(peak MVC torque + superimposed torque) (Kent-Braun and Le Blanc 1996). This ratio was then multiplied by 100 to provide a percentage of central activation. Throughout the text, “CAR value(s)” refer(s) to this calculated percentage of central activation. For isometric MVCs, the torque produced immediately preceding stimulation onset was taken to be the peak MVC torque. For dynamic MVCs, as the torque-time curve was linear for at least 50 ms preceding stimulation, the peak MVC torque that would have been reached at the time of the peak superimposed torque was determined by linear extrapolation of the slope from the 50 ms of the prestimulus measured torque (Babault et al. 2001; Gandevia et al. 1998; Kim et al. 2015; Klass et al. 2005).

**Muscle mechanical properties.** Peak torque, contraction time (time from stimulus onset to peak torque), and half relaxation time (time from peak torque to half of peak torque value) were measured from resting twitch responses to single pulse and paired pulse stimulations.

**EMG.** SOL and TA RMS over the 200-ms period preceding stimulation were used for all analyses of prestimulus muscle activity. For Experiment B, to set target levels for the active H-reflexes, the 200-ms SOL RMS surrounding the maximal peak torque of three trials was used. For both experiments, the TA RMS over 200 ms surrounding peak torque during the strongest DF isometric MVC trial was used to determine normalized TA values (Knutson et al. 1994; Pinniger et al. 2000, 2003).

**Evoked potentials.** Passive Hmax, submaximal M-wave evoked at Hmax, and Mmax values were calculated based on the means of peak-to-peak amplitudes of four responses. Analysis of active H-reflex testing was performed by calculating the mean Hmax and Mmax responses of each subject from the three trials where SOL EMG activity was closest to the 75% MVC target. Hmax-to-Mmax ratios (Hmax/Mmax) were calculated for each subject and used for comparisons of group data.

**Statistical Analysis**

All data in the text are presented as means ± SD, and all figures use SE of the mean. Statistical analyses were performed using SPSS (IBM, Armonk, NY). A mixed-model, repeated-measures ANOVA.
was used to compare within-subject (muscle action type: lengthening, isometric, and shortening) and between-subject (group variable: SCI vs. control) effects, as well as interactions for all dependent variables (i.e., measures of central activation, EMG, evoked responses). When there was statistical significance in the ANOVAs, post hoc analyses using the Bonferroni correction were used to locate specific differences across the muscle action types within each group. In addition, $t$-tests were utilized to compare between-group differences for each muscle action type (lengthening, isometric, and shortening), as well as muscle mechanical properties. Correlations between variables were determined using Pearson product moments and Spearman rho coefficients as appropriate. $P$ values \( < 0.05 \) were considered significant, except when using the Bonferroni correction, in which case significance was found at \( P < 0.017 \).

RESULTS

Experiment A

Torque. Regardless of the contraction type, SCI subjects demonstrated significantly reduced peak volitional torques compared with controls during PF MVCs (Fig. 1), with a significant group effect when analyzing peak constant angular torques \( [F(1,13) = 56.57, P < 0.001] \). Specifically, SCI subjects generated peak torque values of 32.2 ± 9.7, 21.1 ± 6.9, and 13.7 ± 7.6 Nm across lengthening, isometric, and shortening MVCs, respectively. Across matched conditions, controls produced 106.0 ± 26.6, 92.9 ± 28.3, and 79.1 ± 25.6 Nm. A significant effect was also observed for contraction type \( [F(2,12) = 24.97, P < 0.001], \) with the highest peak torques observed during lengthening MVCs, followed by isometric and then shortening MVCs. There was no interaction between group and contraction type \( [F(2,12) = 0.78, P = 0.478] \).

Normalizing lengthening and shortening peak torques to peak isometric values revealed both increases in lengthening torque and reductions in shortening torque were more pronounced for SCI subjects than controls. Specifically, there was a significant effect for contraction type \( [F(2,12) = 17.20, P < 0.001] \) and a significant interaction between group and contraction type \( [F(2,12) = 4.15, P = 0.043] \). SCI subjects increased their normalized torque during lengthening MVCs 40.7 ± 22.4% more than controls \( (P = 0.041) \), while their normalized shortening torques were 22.3 ± 9.8% less than controls \( (P = 0.041) \).

Central activation. All eight SCI subjects produced their highest CAR values during lengthening MVCs. Representative

![Fig. 2. Evoked twitches and group data from central activation ratio (CAR) trials.](image)

A: isolated evoked twitches from the SCI subject’s MVC trials in Fig. 1B. Smallest evoked twitch was observed during the lengthening MVC. B: SCI subjects demonstrate overall deficits in central activation, but generate greater central activation during lengthening MVCs than during isometric or shortening MVCs. C: group comparisons of normalized SOL EMG values reveal SCI subjects generate increases in SOL muscle activity during lengthening MVCs compared with isometric or shortening MVCs. Normalized SOL values for SCI subjects were also ~45% greater than that of controls during lengthening MVCs. D: SCI subjects demonstrated greater coactivation of the antagonist TA muscle than controls during PF MVCs \( (P < 0.05) \). However, no differences in TA activity across contraction types were observed in either group \( (P > 0.05) \). DF, dorsiflexor; MVIC, maximum volitional isometric contraction. Values are means ± SE. Asterisks not associated with brackets refer to significant between groups differences (i.e., SCI vs. control). \( **P < 0.01 \), \( ***P < 0.001 \).
Experiment B

Passive H-reflexes. As seen in Fig. 3, modulation of passive H-reflexes across the different muscle action types contrasted between SCI and controls. For normalized Hmax amplitudes (Hmax/Mmax), there was a main effect for group \([F(1,13) = 5.133, P = 0.041]\). This was due to an overall increase in spinal reflex excitability in the SCI group, as Hmax/Mmax collapsed across muscle action types were 30.9 ± 13.6% higher in SCI than controls. A significant effect for muscle action type \([F(2,12) = 14.92, P < 0.001]\) was also observed for Hmax/Mmax, but there was no group-by-muscle action type interaction \([F(2,12) = 1.439, P = 0.275]\). Post hoc analyses revealed that controls demonstrated significantly depressed lengthening Hmax/Mmax values compared with isometric and shortening Hmax/Mmax values (both \(P < 0.001\)). In contrast, SCI subjects’ lengthening Hmax/Mmax were reduced only compared with shortening Hmax/Mmax \((P = 0.005)\). Hmax/Mmax values of SCI subjects were significantly higher than that of controls during passive muscle lengthening \((P = 0.023)\), but were not different during isometric \((P = 0.088)\) muscle actions. There was a trend for group differences during shortening conditions \((P = 0.066)\).

For each muscle action type, the M-wave recorded at Hmax stimulus intensity was normalized to Mmax. The test M-wave amplitudes across muscle action types was consistent for both SCI and controls \((P > 0.05); \text{range: } 19–21\% \text{ of Mmax}, \text{indicating stable stimulus conditions. In addition, examination of prestimulus SOL and TA EMG revealed muscle activity levels were negligible and not different across muscle action types or between groups (all } P > 0.05\). Examination of Mmax amplitudes, however, revealed a main effect for group \([F(1,13) = 5.04, P = 0.043]\), but there was no effect observed for action type \([F(2,12) = 1.08, P = 0.371]\) and no interaction \([F(2,12) = 2.76, P = 0.103]\). Collapsed across muscle action types, Mmax was 2.28 ± 0.72 mV for SCI subjects vs. 3.33 ± 1.02 mV for controls.

Active H-reflexes. Representative data in Fig. 4, A and B, illustrates the marked differences in H-reflex modulation during active contractions in SCI and healthy control subjects. The depression of lengthening Hmax amplitude relative to isometric and shortening Hmax was still present in controls during volitional contractions. In SCI subjects, there was a disinhibition of the lengthening Hmax during active contractions. Also, as shown in Fig. 4, stimulating conditions for both groups were again stable during active Hmax testing, as M-waves elicited at Hmax stimulus intensities were consistent across contraction types for both groups \((P > 0.05)\).

Examination of Hmax/Mmax during 75% MVC contractions revealed a significant effect for contraction type \([F(2,12) = 7.49, P = 0.008]\) and a significant group-by-muscle action type interaction \([F(2,12) = 9.21, P = 0.004]\), but no group effect \([F(1,13) = 3.39, P = 0.088]\). Post hoc analyses showed that, during lengthening contractions, SCI subjects produced a 32 ± 11% larger Hmax/Mmax than controls \((P = 0.017)\). Similar to the passive conditions, however, isometric and shortening H-reflexes during active contractions were not different between groups \((P > 0.05)\). Controls again demonstrated a significant reduction in lengthening Hmax during 75% MVC contractions compared with isometric \((P = 0.002)\) and shortening Hmax \((P < 0.001)\), with no difference between isometric and shortening conditions \((P = 0.212)\).

In contrast to this behavior, SCI subjects demonstrated similar Hmax amplitudes across all contraction types \((all \ P > 0.05)\).
Consistent with prior reports of active H-reflexes, Mmax amplitudes were potentiated in both groups during 75% MVC contractions. There was only a main effect for group $[F\,(1,13) = 12.01, P = 0.004]$, with no effect for action type $[F\,(2,12) = 2.84, P = 0.098]$ and no interaction $[F\,(2,12) = 1.62, P = 0.238]$. Collapsed across muscle action types, mean Mmax values were $2.49 \pm 0.80 \text{ mV}$ for SCI subjects and $3.92 \pm 0.80 \text{ mV}$ for controls.

Correlations between measures of spinal reflex excitability and central motor drive. Correlation analysis revealed the relative size of the passive lengthening Hmax amplitude in SCI subjects was significantly correlated with increased agonist muscle activation during lengthening MVCs. Normalizing lengthening Hmax, SOL EMG, and CAR values to their respective isometric values revealed a positive linear relationship between lengthening Hmax amplitude and both lengthening SOL EMG ($r = 0.78; P = 0.022$) and CAR values ($r = 0.71; P = 0.049$) (Fig. 5). As a comparison, the shortening Hmax amplitudes have no relationship to either shortening SOL EMG or shortening CAR values ($r = 0.5, 0.06; P = 0.192, 0.888$, respectively). Similar normalization of lengthening Hmax during 75% MVC contractions to corresponding isometric Hmax amplitudes did not reveal any correlation to lengthening SOL EMG or CAR values ($r = 0.09, 0.11; P = 0.822, 0.804$, respectively).

Clinical correlations. Correlations between clinical measures of strength/spastic motor behaviors and normalized lengthening Hmax, SOL EMG, and CAR values were assessed using Spearman rank correlation coefficients. Clonus scores on the Spinal Cord Assessment Tool for Spastic Reflexes exam were positively correlated with normalized lengthening CAR values ($\rho = 0.93, P = 0.002$). No other correlations reached statistical significance (all $P > 0.05$).

DISCUSSION

The present study investigated patterns of central activation and spinal reflex excitability during lengthening, isometric, and shortening muscle actions in subjects with incomplete SCI. The main findings were that SCI subjects generated greater central activation of the PFs during lengthening MVCs compared with isometric or shortening MVCs, and that this may be partly due to increased efficacy of Ia-\(\alpha\) motoneuron transmission during lengthening contractions.

Greater Central Activation During Lengthening MVCs in Human SCI

Examination of PF mechanical properties revealed no loss in peak twitch torques post-SCI, consistent with previous reports of both human (Shields 1995) and rat (Lieber et al. 1986) SOL...
muscle following chronic SCI. However, SCI subjects demonstrated significant strength impairments during all MVCs, as weakness and paralysis post-incomplete SCI result largely from deficits in central activation (Hornby et al. 2009; Lin et al. 2012; Thomas et al. 1997). Despite such deficits, SCI subjects showed maximal increases of ~30% in CAR values and ~40% in SOL EMG during lengthening MVCs compared with isometric and shortening MVCs. These behaviors parallel those our laboratory previously observed in the KEs (Kim et al. 2015).

In contrast to SCI, similar CAR values were observed during all contraction types in healthy controls. This finding differs from previous interpolated twitch studies in lower limb muscles, such as the KEs, which report reduced central drive during lengthening MVCs in healthy adults (Babault et al. 2001; Beltman et al. 2004), but is consistent with those of another study that did not observe any contraction type effect on PF activation levels (Ekblom 2010). As similar voluntary activation levels during different contraction types have been reported for the DFs as well (Klass et al. 2005), there is strong

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Fig. 4. H-reflexes during 75% MVC contractions. A: during strong contractions, there is potentiation of H-reflexes, but specific depression of lengthening H-reflexes is still demonstrated by the control subject. B: there is a complete disinhibition of the lengthening H-reflex in this representative SCI subject. C: during voluntary contractions, SCI subjects continue to demonstrate a larger Hmax/Mmax than controls. In addition, there are no longer any differences in Hmax/Mmax across contraction types for the SCI group, whereas there is still depression of lengthening Hmax/Mmax for controls. Values are means ± SE. *P < 0.05. ***P < 0.001.

Fig. 5. Correlations in SCI subjects between spinal reflex excitability during passive muscle lengthening and central activation during lengthening MVCs. A: there is a significant positive correlation between passive lengthening Hmax/Mmax and SOL EMG during lengthening MVCs in SCI subjects when both are expressed as percentages of their corresponding isometric values. B: similarly, a significant positive correlation exists between lengthening Hmax/Mmax and lengthening CAR values.
evidence that the reduction of lengthening activation varies across muscle groups in healthy adults (Duchateau and Baudry 2014).

Consistent with the CAR data, controls also demonstrated similar levels of SOL and TA EMG across all contraction types. Interestingly, Pinniger and colleagues (2000) reported significant decreases in SOL EMG during lengthening compared with isometric and shortening MVCs, as well as greater coactivation of TA during lengthening MVCs compared with other contraction types. However, our EMG data are consistent with numerous other studies in which no effect of contraction type on EMG amplitudes in the agonist SOL and medial gastrocnemius, or the antagonist TA, were observed (Duclay et al. 2011, 2014; Duclay and Martin 2005; Hahn et al. 2012). Methodological differences may have contributed to the discrepant findings, as the current study and those of Duclay et al. had subjects fully supported and secured in a testing chair vs. subjects in the Pinniger et al. study, who performed contractions while kneeling on all four limbs. The importance of subject posture on central drive has been previously noted by others (Maffiuletti and Lepers 2003). In contrast to the variable findings in healthy adults, however, the large increase in central activation during lengthening MVCs in SCI subjects is not evident in any previous studies of unimpaired populations.

There are many potential sources for the increased central activation demonstrated by SCI subjects during lengthening MVCs. We hypothesized increased spinal reflex excitability, common among many SCI patients, may contribute to the observed increases in central activation. Specifically, stretch reflex hyperexcitability within agonist muscles may augment motoneuron activity during MVCs through primarily monosynaptic Ia-α motoneuron excitation (Knutsson et al. 1997). Alternatively, depending on the muscle group, stretch of spastic antagonist muscles may hinder muscle activation during shortening contractions through Ia-mediated reciprocal inhibition, as Knutsson and colleagues also reported.

In spite of increased levels of TA coactivation during all PF MVCs in SCI subjects compared with controls, there were no significant differences in TA activity across muscle contraction types for either group. Increased coactivation is thought to be due partly to impaired reciprocal inhibition and has been reported before in spastic SCI patients (Boorman et al. 1996; Crone et al. 2003). However, as there were similar levels of TA activity across all contraction types, our data do not support a role for varying levels of reciprocal inhibition to differences in central activation patterns. As only two individuals with SCI were included in the Knutsson study, our contrasting findings may be related to etiology-dependent differences in spastic motor behaviors (Burne et al. 2005; Woolacott and Burne 2006).

Spinal Reflex Excitability

To investigate the hypothesis that increased spinal reflex excitability in SCI subjects contributes to gains in lengthening MVCs, we examined H-reflex modulation across different muscle action types. Our control data during passive muscle actions are consistent with previous findings of depressed lengthening Hmax/Mmax (Duclay and Martin 2005; Pinniger et al. 2001; Románó and Schieppati 1987). Interestingly, SCI subjects demonstrated an overall increase in spinal reflex excitability at rest compared with controls, with the primary finding being differences between subject groups in H-reflex modulation during lengthening actions. SCI subjects demonstrated an ~30% higher lengthening Hmax/Mmax than healthy controls. As the normalized Hmax amplitude is an estimate of motor pool recruitment by Ia excitation (Pierrot-Deseilligny and Burke 2005; Zehr 2002), our data suggest the effectiveness of Ia transmission during muscle stretch is enhanced following SCI. It should be noted, however, that the authors of a prior study comparing only passive isometric SOL H-reflexes between SCI and controls did not find any differences in Hmax/Mmax between subject groups (Schindler-Ivens and Shields 2004). Although we observed a group effect when comparing Hmax/Mmax, we also did not find a significant difference during specific comparisons of isometric Hmax/Mmax between our SCI subjects and controls, consistent with the aforementioned report. Therefore, differences between their findings and those of the present study appear to be primarily due to the inclusion of dynamic muscle actions in the present study.

Many authors have posited presynaptic inhibition of Ia afferent terminals as a mechanism for the reduction in lengthening Hmax/Mmax observed in healthy adults (Abbruzzese et al. 1994; Duclay et al. 2011; Duclay and Martin 2005; Románó and Schieppati 1987). Since Ia firing is known to increase with muscle stretch (Brown and Matthews 1966), and voluntary lengthening contractions are coincident with greater muscle spindle afferent activity (Burke et al. 1978; Hulliger et al. 1985), inhibitory mechanisms are likely acting on Ia terminals or motoneurons, or possibly both, during muscle lengthening. Significantly, presynaptic inhibition of Ia afferents is thought to be diminished in many spastic patients, including those with multiple sclerosis (Nielsen et al. 1995) and SCI (Faist et al. 1994). The primary afferent depolarizing interneurons responsible for presynaptic inhibition are controlled in large part by descending tracts (Lundberg 1975). Therefore, following SCI, Ia pathways may be partially released from this presynaptic inhibition and contribute to the greater lengthening Hmax/Mmax observed. Furthermore, this reduction in the normal gating of Ia excitation by inhibitory interneurons post-SCI likely contributes to the development of stretch reflex hyperexcitability (Nielsen et al. 2007), and we suspect it may also partly underlie increased central activation during lengthening contractions.

Varying levels of HPAD between SCI and control subjects may also contribute to different lengthening Hmax amplitudes. The HPAD phenomenon refers to a reduction in transmitter release from previously activated Ia fibers (Curtis and Eccles 1960) and may also decrease H-reflex amplitudes during passive muscle lengthening in healthy adults (Crone and Nielsen 1989; Hultborn et al. 1996). As data suggest HPAD is reduced in animal models of SCI (Hultborn et al. 1996) as well as spastic patients (Aymard et al. 2000; Nielsen et al. 1995), including those with SCI (Nielsen et al. 1993; Schindler-Ivens and Shields 2000), this may also partially explain differences in H-reflex modulation between subject groups. Consistent with Schindler-Ivens and Shields, we think it is most likely that changes in both presynaptic inhibition and HPAD result in alterations to Ia transmission following chronic SCI.
Contribution of Spinal Reflex Excitability to Increased Central Activation

Voluntary efforts of 75% MVC potentiated H-reflexes in both subject groups. The Hmax/Mmax of healthy adults during 75% MVC contractions were similar to those reported for MVCs in two previous studies (Duclay et al. 2011; Duclay and Martin 2005). Potentiation from passive to active states has previously been attributed to increased excitability of the motor pool due to descending drive (Burke et al. 1989; Schieppati 1987). The primary difference between groups was the greater potentiation of lengthening H-reflexes relative to isometric and shortening H-reflexes demonstrated by SCI subjects. Controls continued to demonstrate a relative depression of lengthening Hmax compared with isometric and shortening Hmax, while SCI subjects generated lengthening Hmax/Mmax values that were no different than isometric or shortening. As SCI subjects demonstrated reduced passive lengthening Hmax values compared with passive shortening Hmax, this suggests there was a selective disinhibition/facilitation of Ia transmission during lengthening contractions.

Interestingly, although there were significant correlations between spinal reflex excitability during passive muscle lengthening and measures of increased central activation during lengthening MVCs (CAR value and SOL EMG), we did not observe any correlation between active Hmax amplitudes and lengthening CAR values. Close examination of the active H-reflex data revealed the lack of correlation was due to a clear ceiling effect, with three of the SCI subjects producing Hmax amplitudes that were over 90% of Mmax during lengthening and isometric contractions. Therefore, it is likely these subjects reached the limits of Ia excitation of motoneurons.

Notably, the predominant absence of significant correlations between clinical measures of spastic motor behaviors and objective electrophysiological measures of spinal reflex excitability has been previously reported (Crone et al. 1994; Kim et al. 2015; Nielsen et al. 2007) and may be partly due to the difficulty of discriminating passive and active properties of muscle stiffness using clinical exams (Lorentzen et al. 2010).

Influence of Sensory Inputs on Motor Commands Post-SCI

The potential for specific sensory inputs in combination with volitional descending commands to facilitate motor output following SCI has previously been demonstrated. Zijdewind and colleagues (2012) demonstrated in SCI subjects that adding various concurrent inputs (e.g., vibration, stimulation, contralateral contraction) during isometric MVCs of thenar muscles could increase force, maximal motor unit firing rates, and surface EMG activity above what could be achieved during isometric MVC alone. Whether this is strictly due to increased excitation or removal of inhibition within spinal circuits, or some combination of both is unclear. Regardless, there appears to be a reserve of neural drive to muscle post-SCI that can be accessed through various means. Our data suggest that, in SCI subjects, imposed muscle lengthening may be another form of afferent input that accesses this reserve when combined with descending drive. Based on principles of experience-dependent plasticity, the activated synapses will likely strengthen with chronic use (Kleim and Jones 2008). Hence, these data support development of future rehabilitation protocols incorporating high-intensity lengthening contractions. The benefits of such a protocol are likely to be further augmented by the positive structural adaptations and improvements in neural activation of muscle which seem to be maximized by lengthening contractions over other types of contractions (Enoka 1996).

Change in Ia transmission to motoneurons is likely one factor among many that may contribute to differential activation patterns. Different sensory pathways (e.g., Ib, II, cutaneous) as well as supraspinal changes post-SCI may further contribute to the control strategies utilized by the nervous system after injury. For example, recent studies have suggested a disinhibition/facilitation of lower limb motor cortical representations resulting from homonymous afferent stimulation (Roy et al. 2010), as well as reduced intracortical inhibition compared with healthy adults during a fatigue protocol (Nardone et al. 2013). Future investigation of motor cortex excitability and intracortical inhibition during dynamic contractions will provide more detailed information on supraspinal changes post-SCI that may affect activation strategies.

Conclusion

In conclusion, subjects with incomplete SCI demonstrate greater central activation during lengthening MVCs than during isometric or shortening MVCs, as well as increased efficacy of Ia transmission during muscle lengthening compared with controls. Passive lengthening Hmax values were positively correlated with increases in central activation during lengthening MVCs. Importantly, Ia excitation of motoneurons was facilitated by volitional activation in SCI, especially during lengthening contractions. Combined, the present study presents compelling evidence that patterns of muscle activation are altered following SCI, and that enhanced efficacy of Ia-α motoneuron transmission contributes to greater central activation during lengthening contractions.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

Author contributions: H.E.K., D.M.C., and T.G.H. conception and design of research; H.E.K. performed experiments; H.E.K. analyzed data; H.E.K., D.M.C., and T.G.H. interpreted results of experiments; H.E.K. prepared figures; H.E.K. drafted manuscript; H.E.K., D.M.C., and T.G.H. edited and revised manuscript; H.E.K., D.M.C., and T.G.H. approved final version of manuscript.

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