Spastic Reflexes Triggered by Ankle Load Release in Human Spinal Cord Injury

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Wu, Ming and Brian D. Schmit. Spastic reflexes triggered by ankle load release in human spinal cord injury. J Neurophysiol 96: 2941–2950, 2006; doi:10.1152/jn.00186.2006. The rapid decrease in firing of load-sensitive group Ib muscle afferents during unloading may be particularly important in triggering the swing phase of gait. However, it still remains unclear whether load-sensitive muscle afferents modulate reflex activity in human spinal cord injury (SCI), as suggested by studies in the cat. The right hip of 12 individuals with chronic SCI was subjected to ramp (60°/s) and hold (10 s) movements over a range from 40° flexion to 0–10° extension using a custom servomotor system. An ankle dorsiflexion load was imposed and released after the hip reached a targeted position using a custom-designed pneumatic motor system. Isometric joint torques of the hip and knee, reaction torque of the ankle, and surface electromyograms (EMGs) from eight muscles of the leg were recorded following the imposed hip movement and ankle load release. Reflexes, characterized by hip flexion torque, knee extension, and coactivation of ankle flexors and extensors, were triggered by ankle load release when the hip was in an extended position. The ankle load release was observed to enhance the reflexes triggered by hip extension itself, suggesting that ankle load afferents play an important role in spastic reflexes in human SCI and that the reflex pathways associated with ankle load afferents have important implications in the spinal reflex regulation of human movement. Such muscle behaviors emphasize the role of ankle load afferents and hip proprioceptors on locomotion. This knowledge may be especially helpful in the treatment of spasms and in identifying rehabilitation strategies for producing functional movements in human SCI.

INTRODUCTION

Limb load is likely to be an important sensory cue for triggering spastic reflex activity in people with chronic spinal cord injury (SCI). Spasticity is classically defined as a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks (Lance 1980) and thus has been associated primarily with stretch reflex excitability. Many of the clinically observed components of spastic reflexes in SCI involve more than stretch reflexes, resulting in complex patterns of reflex activity in muscles throughout the leg that are manifested as flexor or extensor spasms (e.g., Benz et al. 2005). Some of these spastic reflexes may involve spinal networks that are also associated with reflex control of functional movements, such as standing or walking and therefore are likely to involve multiple sensory pathways including limb load afferents. In this study, we postulated that extensor spasms are influenced by load afferents of the foot and ankle, consistent with reflex regulation of locomotion.

Extensor spasms are commonly observed in SCI patients in the clinic, especially during movement from a sit to a supine position (Kuhn 1950; Little et al. 1989), suggesting that hip proprioceptors play an important role in this reflex pathway. Reflex muscle activity strongly resembling extensor spasms has also been experimentally produced using controlled unilateral extension of the hip (Schmit and Benz 2002; Steldt and Schmit 2004) and knee (Wu et al. 2005). The response to imposed hip extension consists of a multijoint reflex characterized by hip flexion, knee extension, and mixed responses at the ankle, although a net ankle plantar flexion is common. Because extensor spasms involve activation of muscles throughout the leg, the response must be mediated through polysynaptic pathways involving activation of interneuronal circuits, such as those associated with the spinal reflex control of locomotion.

The possible involvement of the locomotor reflex pathways in extensor spasms is suggested by the coincidental observation that both locomotion and extensor spasms are influenced by sensory signals from the hip proprioceptors. Spinal locomotor networks in cats receive input from hip proprioceptive cues, originating primarily from stretch of the hip flexors (Grillner and Rossignol 1978; Hiebert et al. 1996; Kriellaars et al. 1994; Lam and Pearson 2001). In human SCI, similar pathways are suggested to be important for eliciting muscle activity in the legs during locomotion (Dietz et al. 2002; Dobkin et al. 1995; Harkema et al. 1997; Van de Crommert et al. 1998). Further, imposed hip extension movements alone trigger multijoint spastic reflexes that have many similarities with the muscle activity produced during pathologic locomotion (Schmit and Benz 2002; Steldt and Schmit 2004). These similarities suggest that walking and extensor spasms might invoke common neural pathways in chronic SCI.

In the current study, we hypothesized that load afferents of the ankle would also alter or produce extensor spasms. Load afferents from ankle plantar flexors and the skin of the foot have been proven to be important modulators of spinal locomotion in the cat (Conway et al. 1987; Duysens and Pearson 1976, 1980; Guertin et al. 1995; Pearson and Collins 1993; Pearson and Duysens 1976; Pearson et al. 1992; Whelan et al. 1976).
TABLE 1. Subject data

<table>
<thead>
<tr>
<th>Subject</th>
<th>Level of Injury</th>
<th>ASIA Class</th>
<th>Age, yr</th>
<th>Post-Injury Time</th>
<th>Medicaions</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>T10-11</td>
<td>A</td>
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<td>7 yr</td>
<td>baclofen (intrathecal pump)</td>
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<tr>
<td>B</td>
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<td>C</td>
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</tr>
<tr>
<td>C</td>
<td>T2</td>
<td>B</td>
<td>69</td>
<td>2 yr</td>
<td>baclofen (20 mg q.i.d.)</td>
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<tr>
<td>D</td>
<td>C7</td>
<td>B</td>
<td>26</td>
<td>3 yr</td>
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<td>C6-8</td>
<td>A</td>
<td>31</td>
<td>5 yr</td>
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</tr>
<tr>
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<td>T6-7</td>
<td>B</td>
<td>23</td>
<td>5 yr</td>
<td>None</td>
</tr>
<tr>
<td>G</td>
<td>C6</td>
<td>B</td>
<td>43</td>
<td>26 yr</td>
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</tr>
<tr>
<td>H</td>
<td>C6</td>
<td>C</td>
<td>42</td>
<td>15 yr</td>
<td>baclofen (40 mg t.i.d.)</td>
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<tr>
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</tr>
<tr>
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<td>T6</td>
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<tr>
<td>K</td>
<td>C7</td>
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<tr>
<td>L</td>
<td>C6-7</td>
<td>A</td>
<td>48</td>
<td>22 yr</td>
<td>None</td>
</tr>
</tbody>
</table>

1995). For example, in spinalized cats walking on a treadmill, the swing phase will not initiate if the contralateral limb is not in position to bear load (Grillner and Rossignol 1978). In contrast, the rapid decrease in afferent firing during unloading appears to trigger the swing phase of gait in a manner similar to stretch of the hip flexors. Analogous effects of load release have also been observed in human stepping infants (Pang and Yang 2000; Yang et al. 1998). Further, limb loading during body weight-supported treadmill training modulates EMG activity according to the phase of the gait cycle in human SCI (Dietz et al. 2002; Harkema et al. 1997), suggesting an important role for load afferents in walking. If the aforementioned spastic reflexes produced by imposed hip extension activate locomotor pathways in human SCI, load-sensitive afferents may also trigger similar reflexes (i.e., extensor spasms).

In this study, we tested whether release of ankle load modulates spastic reflexes produced by imposed hip extension in 12 individuals with chronic SCI. The reflex responses to ankle load release were characterized using hip torque measurements and electromyograms (EMGs) from eight muscles of the leg. The responses were compared among four different ankle load conditions during imposed hip extension, namely: no ankle load, sustained ankle load, ankle load release timed with imposed hip extension, and ankle load release applied 2 s after imposed hip extension. We postulated that the spastic reflex activity produced by ankle load release would be similar to the muscle activity produced by imposed hip extension, consistent with the excitation of neural pathways common to locomotion.

METHODS

Subjects

Twelve subjects with clinical features described in Table 1 were recruited into this study. Inclusion criteria included chronic SCI (>1 yr) with associated spasticity. All subjects enrolled in the study were >2 yr postinjury. Participants (mean age: 38, range: 23–69) included both complete [American Spinal Cord Injury Association Classification (ASIA) A] and incomplete (ASIA C and D) individuals with SCI. At the time of the study, 4 of 12 subjects were prescribed antispastic medications (baclofen) to reduce the intensity and frequency of spastic reflexes. Exclusionary criteria included multiple CNS lesion sites, urinary tract infection, other secondary infections, heterotopic ossification, respiratory insufficiency, significant osteoporosis, or the inability to give informed consent. Informed consent was obtained, and all procedures were conducted in accord with the Helsinki Declaration of 1975 and approved by the Institutional Review Boards of Northwestern University, Chicago, IL and Marquette University, Milwaukee, WI.

Test apparatus

The experimental apparatus for this study was an instrumented hip/ankle actuator modified from a device used in a previous study (Steldt and Schmit 2004). Briefly, the apparatus, shown in Fig. 1, was constructed for measuring the multijoint torque response to imposed hip movement and ankle load release. This apparatus included a static knee brace connected to ankle and hip actuators. The foot was placed in a footplate with a clamp placed on the dorsum of the foot and a strap to secure the heel. The hip-knee and knee-ankle links were adjustable to fit a wide range of leg sizes. The knee/ankle portion of the leg brace was affixed to a servomotor drive system (MT 704A1-R1C1, Kollmorgen, Northampton, MA) as described previously (Steldt and Schmit 2004). A newly constructed ankle load actuator, which included a rotary pneumatic motor (HI-ROTOR PRN 50D, Kuroda, Kanagawa, Japan), a solenoid valve (PIV73B4S NC, Metal-Work, Brescia, Italy) and an air-compressor (Jun-Air, Nørresundby, Denmark), was affixed to the knee-ankle brace to impose a load to the ankle joint. Hip, knee, and ankle torques were measured using hollow-flanged transducers (Himmelstein, Hoffman Estates, IL). Position of the hip and ankle joints were measured using potentiometers coupled to the servomotor and pneumatic motor drive shafts, respectively.

Surface EMGs were recorded from the tibialis anterior (TA), soleus (SOL), medial gastrocnemius (MG), vastus medialis (VM), rectus femoris (RF), medial hamstrings (MH), lateral hamstrings (LH), and hip adductors (ADD) of the right leg in all subjects. Ag-AgCl 2.5-cm
square pregelled electrodes (Vermed, Bellows Falls, VT) were applied with 2.5-cm center-to-center spacing over the respective muscle bellies on lightly abraded skin. Active preamplifiers with shielded leads were attached to the electrodes and connected to an Octopus AMT-8 EMG unit (Bortec, Calgary, Alberta, Canada). All channels were amplified (total gain = 1,000) low-pass filtered (450 Hz) and sampled (1,000 Hz) using a data-acquisition card (National Instruments, Austin TX) on a personal computer. Custom LabVIEW software (National Instruments) was used for acquiring the data as well as outputting the velocity command signal to the servomotor system and load command signal to the valve for the pneumatic motor system.

**Imposed hip movement and ankle load**

Movements were imposed on the right leg of all 12 subjects. Each subject was transferred to a tri-section therapy table and placed in a supine position (table horizontal) with a pillow under her/his head. The center of rotation of the right hip joint was aligned with the axis of rotation of the servomotor system, the ankle joint was aligned with the axis of pneumatic motor, and the brace was adjusted to align the knee with the appropriate torque transducer. Alignment of the hip and ankle joints was confirmed by an absence of leg translation during manually imposed flexion and extension of the joint, assessed visually. The pelvis was secured to the table with a strap across the iliac crest to limit pelvic rotation. The leg was placed in the brace with the knee at the appropriate torque transducer. The start position was set at 40° of hip flexion and (slightly plantarflexed).

**Analysis**

Two types of joint torque data were obtained for each movement trial: the reflex joint torques during the stretch and the reflex joint torques during the 10-s hold period with the hip in the maximum extended posture. The torque measurements recorded immediately at the start and end of the movements were excluded from the analysis due to large inertial artifacts. During the hold periods, the gravitational–passive torque offset was removed by subtracting a torque measurement in the end position, when there was no muscle activity (i.e., at the end of hold period). To calculate the reflexive hip torque during the stretch movement, the gravitational torque and passive resistance were removed.

The mean torque signal was calculated during each pause and the resulting signal contained only the passive and gravitational torque (verified by an absence of detectable EMG signal in any of the recorded muscles). The passive resistance of the joint was then calculated by fitting a third order polynomial \((polyfit/polyval; Matlab command, The Math Works, Natick, MA)\) to the mean torque versus hip angle data according to Eq.1

\[
\tau_{\text{passive+gravity}} = (a_1\theta + a_2\theta^2 + a_3\theta^3 + a_4)
\] (1)

The reflex joint torques were then calculated for each test trial using Eq.2

\[
\tau_{\text{active}} = \tau_{\text{measured}} - \tau_{\text{passive+gravity}}
\] (2)

The resulting reflex torque signals were low-pass filtered using a fourth-order Butterworth filter \((\text{butterfiltfilt; Matlab command, The Math Works})\) with a cutoff frequency at 10 Hz.

The peak hip torque was the primary measurement used to compare the reflex response across conditions because the ankle and knee torques were mechanically influenced by the ankle load (i.e., knee torques were affected by stretch of biarticular muscles). The peak torque of the hip joint was identified for each movement trial in each load condition. Then for all 12 subjects in whom reflex responses were conducted for comparison. A timed delay of 3 min was allowed between trials. Each set of conditions was repeated three times, with the four conditions randomized for each set of trials. At the end of the 12 test trials, the subject’s leg was moved slowly into extension at 2° increments to measure the combined gravitational and passive torque of the hip. A 5-s pause was instituted at each increment to allow any minor spastic reflex activity to subside.

**TABLE 2. Subject test parameters indicating range of motion and static knee angle**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Static Knee Angle</th>
<th>Ankle Plantar Flexion Angle</th>
<th>Hip Flexion Angle</th>
<th>Hip Extension Angle</th>
<th>Hip Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>15</td>
<td>30</td>
<td>40</td>
<td>-10</td>
<td>50</td>
</tr>
<tr>
<td>B</td>
<td>40</td>
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<tr>
<td>C</td>
<td>30</td>
<td>20</td>
<td>40</td>
<td>-10</td>
<td>50</td>
</tr>
<tr>
<td>D</td>
<td>15</td>
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<tr>
<td>F</td>
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<td>-10</td>
<td>50</td>
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<tr>
<td>G</td>
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<td>55</td>
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<td>0</td>
<td>40</td>
</tr>
<tr>
<td>H</td>
<td>30</td>
<td>10</td>
<td>40</td>
<td>-10</td>
<td>50</td>
</tr>
<tr>
<td>I</td>
<td>40</td>
<td>20</td>
<td>40</td>
<td>0</td>
<td>40</td>
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<tr>
<td>J</td>
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<td>L</td>
<td>25</td>
<td>30</td>
<td>40</td>
<td>-10</td>
<td>50</td>
</tr>
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</table>
signals, across all four load conditions, were normalized to the mean of the area values for each muscle to account for the differences in the EMG magnitudes across subjects and muscle groups. A one-way ANOVA was used to statistically compare the EMG activities across the ankle load conditions ($\alpha = 0.05$).

Preliminary analysis suggested that the ankle EMG responses to imposed hip movements were dependent on whether the ankle loading perturbation produced lasting clonus in the ankle. Subjects were separated into two groups based on the occurrence of clonus to test whether ongoing clonic muscle activity had a significant effect on the reflex response to hip extension and ankle load release. The response to ankle loading was defined as clonic if the EMG signals of the SOL or MG contained at least three consecutive bursts of EMG activity, with silent periods between them. A paired $t$-test was used to compare the EMG activities of the TA, MG, and SOL across two load conditions, sustained load and no load, for subjects with clonus and without clonus, respectively. Significance was tested at $\alpha = 0.05$.

**RESULTS**

Torque and EMG reflex responses to imposed ramp-and-hold movement of the hip, combined with four different ankle load conditions, suggested that the spastic reflexes triggered by hip extension were modulated by ankle load release. The effects of ankle load condition were consistent across all (11 of 12) chronic SCI subjects with reflex responses, always resulting in an increase in response to load release. Representative torque responses to sustained ankle load during hip movement and to load release combined with the hip extension movement are shown for subject D in Fig. 2A, where time $= 0$ s corresponds to the end of movement into hip extension. Typically, hip flexion torque increased following the ankle load release at the end of hip movement, reaching its peak at $\sim 1.2$ s in this case, and then slowly declined to zero during the hold period. Following the ankle load release, the extension torque response of the knee increased and reached a peak at approximately the same time as the peak hip flexion torque. Sustained ankle load following the hip extension resulted in a similar torque response pattern, e.g., hip flexion and knee extension, however, the magnitudes of the responses were smaller.

The EMG data were consistent with the torque data, with similar patterns of activity across all load conditions and larger magnitude EMG signals when load release was applied at the end of the hip extension movement. For example, the surface EMG responses to hip extension movement combined with ankle load release and sustained load are shown in Fig. 2B for subject D. During the hold period, the muscle activities of the knee and ankle flexors, represented by the LH and TA, were activated with the ankle load release. Muscle activity of the RF (a hip flexor/knee extensor) was also observed following the load release, coinciding with the measured hip flexion and knee extension torques. For the sustained ankle load condition, the RF was also active following imposed hip extension but with a smaller magnitude. No tonic muscle activities of knee and ankle flexors, represented by LH and TA, were observed following the hip extension movement. The ankle extensors, represented by SOL, were often activated by the stretch associated with ankle dorsiflexion during the initial loading, which complicated interpretation of the SOL activation at the end of the hip extension movement. The SOL muscle activity generally increased following load release, although a long-lasting SOL activity associated with the initial stretch was also observed in the sustained load condition.

Statistically, the reflexive hip flexion torque was increased following ankle load release compared with the no load and sustained load conditions. The mean intrasubject SD for load and load release were 26 and 18%, respectively (normalized by the mean peak hip torque for each subject). The mean peak
values across 11 of the 12 subjects for these three ankle load conditions are shown in Fig. 3A (the peak torque from 1 subject was excluded because the data point was classified as an outlier, defined as a magnitude that was >3 SD above the population mean). The one-way ANOVA (ankle load condition) indicated that the reflex responses at the hip joint were significantly affected by the ankle load (P < 0.05). Post hoc pairs comparisons indicated that the value of peak torque was significantly greater with load release than for the other two load conditions (Tukey test, P < 0.05). The mean peak torque was 22.5 Nm for the load release condition, compared with 13.7 and 16.2 Nm for no load and sustained load, respectively.

The results of the statistical analyses of the EMG data of the thigh muscles were similar to the hip torque results, indicating a significantly larger response in the load release condition compared with the sustained load and no load conditions. An ANOVA analysis of the integrated EMG signals of the RF, MH and ADD in 11 and VM in 12 of the 12 subjects indicated that muscle activities were significantly affected by load condition (P < 0.05). The RF, MH, and ADD data from one subject were excluded because the data were classified as outliers (the magnitudes were >4 SD above the mean). Post hoc test pairs comparisons indicated that the integrated EMG signals from RF, MH, ADD, and VM were significantly greater with ankle load release than for other load conditions (Tukey test, P < 0.05). The EMG signals were significantly different between sustained load and load release for RF and VM, between no load and load release for MH, and between no load and load release, sustained load and load release for ADD. The magnitudes of the differences are summarized in Fig. 3B.

The results from the ankle muscles differed from those of the thigh muscles. Based on the one-way ANOVA, the integrated EMG signals from the MG, SOL, and TA in 11 of 12 subjects demonstrated that the magnitude of the ankle muscle activities were also significantly affected by load condition (ANOVA, P < 0.05); however, the post hoc pairs comparisons indicated that the response was greater with sustained ankle load than for the other load conditions (Tukey test, P < 0.05). Specifically, the EMG signals from TA and MG were significantly greater for both sustained load and load release when compared with no load. The EMG of the SOL was significantly greater for the sustained load than no load. The mean values of the integrated EMG signals are summarized in Fig. 3B for the ankle muscles.

The larger ankle muscle responses during the sustained load condition appeared to be associated with clonic muscle activity at the ankle. In some cases, the initial loading of the ankle, which preceded the imposed hip extension, appeared to trigger lasting clonus in the ankle. Specifically, the initial ankle loading perturbation triggered clonus in 6 of the 12 subjects. Figure 4 demonstrates the typical muscle activities of TA, SOL, and

FIG. 4. Typical EMG responses to imposed hip extension movement and 2 ankle load conditions, no load and sustained load, are shown for subject G. EMG activities with cyclical bursts in the SOL, MG, and TA were elicited following loading of the ankle. Co-contraction of the ankle extensors and flexors was commonly observed with ankle load. EMG activities without cyclical bursts in the MG were also elicited following the hip extension with no ankle load.

FIG. 3. A: peak hip flexion torque responses for 12 subjects are indicated at 3 ankle load conditions, i.e., no load, sustained load, and load release. The data shown include the mean ± SD for 12 subjects and 3 trials. The positive direction of the torque was hip flexion. B: relative change in integrated EMG activity of 8 muscles: RF, vastus medialis (VM), medial hamstrings (MH), LH, adductors (ADD), TA, medical gastrocnemius (MG) and SOL for 12 subjects and 3 ankle load conditions are shown. Data were normalized to the mean integrated EMG activity obtained at all load conditions. Data represent the mean ± and SDs of 12 subjects during the hold period of hip extension. * significant difference (P < 0.05).
MG with and without ankle load following the hip extension movement for subject G, where time = 2 s corresponds to the end of the movement into hip extension. Following the ankle loading, the TA (an ankle dorsiflexor), and the plantarflexors (MG and SOL) were activated, with EMG activity lasting for >9 s for this subject. For the condition without ankle load, very little muscle activity was observed in the TA and SOL, and the MG activity was reduced, although activity was still evident, following imposed hip extension.

Individuals demonstrating clonus in response to the initial ankle load had a larger ankle muscle response to imposed hip extension for the sustained load condition compared with no load. To test this observation, the subjects were grouped into two categories, depending on whether clonus was observed during the initial load application. A paired $t$-test indicated that the difference in muscle activities of TA, MG, and SOL for the sustained load and no load conditions was significantly larger for the clonus group than the nonclonus group ($P < 0.05$ for all 3 muscles). For the clonus group, the mean values of EMG signals increased 185% for TA, 195% for MG, and 374% for SOL for sustained load compared with no load. For the nonclonus group, no significant difference was observed for the muscle activities of TA, MG, and SOL for the sustained and no load conditions, although the trend of a larger response with sustained ankle load was still be observed for the MG and SOL ($P = 0.91$ for TA; $P = 0.15$ for MG, and $P = 0.48$ for SOL). These data are summarized in Fig. 5.

The timing of the load release also appeared to effect the reflex response. Figure 6 demonstrates the typical torque responses and EMGs of subject N for load release at the end of hip extension movement and after a delay of 2 s, where time = 0 s corresponds to the end of hip extension. Typically, following the load release, the magnitude of the hip flexion torque increased gradually and reached its peak value at ~2 s after load release (1.8 s in this case; Fig. 6A). For the delayed load release condition, the hip flexion torque reached its peak values at ~4 s after hip extension (3.5 s in this subject). The time delay between the two torque peaks (~2 s) was equivalent to the time interval between the two load release conditions.

The corresponding surface EMG responses to load release and delayed release for subject N are shown in Fig. 6B. The timing of load release affected the timing of the burst of muscle activities following the load release. The pattern of the reflex muscle activities in response to load release and delayed release were similar. However, the onset of the muscle activity following the delayed load release was postponed with a time...
interval approximately equivalent to the time difference between the load releases (i.e., 2 s). Specifically, the mean time interval between peak hip flexion torque for load release and delayed release was 1.7 ± 0.8 (SD) s.

To determine whether there was a significant difference in the pattern of muscle activity between the reflex response to ankle load release timed with hip extension and the response triggered by load release 2 s later, the EMG signals were compared. The integrated EMG areas of the eight muscles (Fig. 7), i.e., RF, VM, MH, LH, ADD, TA, MG, and SOL, following the ankle load release and delayed load release were calculated for a duration of 2 s following the ankle load release in each case. Paired t-test demonstrated no significant difference between the two load conditions for seven muscles ($P = 0.2$ for RF, $P = 0.1$ for VM, $P = 0.9$ for MH, $P = 0.6$ for LH, $P = 0.5$ for ADD, $P = 0.1$ for TA and $P = 0.3$ for MG). One muscle, the SOL was significantly larger for the load release (no delay) test ($P < 0.05$).

**Discussion**

Ankle load release with the hip in an extended position produced a reflex pattern that resembled the response to an imposed hip extension movement alone, even when the load release was delayed by two seconds after the end of the imposed hip extension. Further, when load release was timed with hip extension, the magnitude of the reflex was consistently enhanced compared with hip extension alone in all 11/12 subjects with chronic SCI. These observations suggest that proprioceptive feedback from hip flexors converges with sensory inputs from ankle and foot load afferents onto a common spinal network. Interestingly, maintained load of the ankle did not suppress the response to reflexes imposed by hip movement; however, an ongoing clonus reaction at the ankle associated with the initial loading may also have impacted the reflex response to imposed hip extension in six of the test subjects. In total, the results of this study suggest that ankle load afferents play an important role in spastic reflexes in human SCI and that the reflex pathways associated with ankle load afferents have important implications in the spinal reflex regulation of human movement.

**Possible role of locomotor networks**

One explanation for the multijoint spastic reflexes triggered by both hip proprioceptors and ankle load afferents is that the reflex activates a spinal reflex network normally designated for control of standing or walking. Ankle load release with the hip in extension produced a response composed of hip flexion, knee extension, and coactivation of ankle extensor and flexor muscles, similar to the reflex response to imposed hip extension (see also Schmit and Benz 2002; Steldt and Schmit 2004). As has been noted previously (Schmit and Benz 2002), this pattern of joint torques somewhat resembles the pattern associated with the late stance phase of gait. Although the observations are circumstantial, the position of the ipsilateral leg (approximating late stance), the net joint torques produced by the reflex (similar to late stance) and the sensitivity to hip proprioceptors and ankle/foot load afferents are all consistent with the activation of a locomotor reflex. These similarities appear despite the fact that the subjects were not walking in the current study, the afferent feedback from the contralateral leg was not similar to gait, and the current study was conducted with subjects in a supine posture.

An alternative explanation for the multijoint spastic reflexes triggered in this study is that the hip flexor/knee extensor activity is generated by stretch reflex activation of the biarticular RF due to the imposed hip extension. The load release would presumably enhance the RF stretch reflexes triggered by hip extension. However, the similarity of the spastic reflexes between the load release and delayed release suggests that hip position feedback may be more important than hip velocity feedback, which would be critical to an RF stretch reflex. In addition, and the burst of muscle activity of the SOL, which was shortened following load release, suggests that the load release produced a multijoint response rather than a modulation of a stretch reflex. Overall, these results suggest that stretch reflexes may not be the primary contribution to the multijoint spastic reflexes associated with hip extension and ankle load release, implicating interneuronal pathways that have the potential to be involved in reflex control of functional movements.

The feedback from load receptors at the ankle/foot has been shown to be an important sensory cue for locomotion in the cat. Specially, load-sensitive group Ib muscle afferents from the ankle extensors and cutaneous afferents from the plantar surface of the foot act to inhibit the generation of ipsilateral flexor burst activity and to promote the generation of extensor activity during the stance phase of walking in the cat (Conway et al. 1987; Duysens and Pearson 1976, 1980; Guertin et al. 1995; Pearson and Collins 1993; Prochazka et al. 1997a,b; Whelen et al. 1995). In contrast, the results from the current study did not indicate that sustained ankle load prolonged extensor muscle activity in the leg during a hip-triggered reflex. The response of the ankle plantar flexors was clearly higher during sustained loading; however, the knee extensor activity was slightly smaller (Fig. 3), and ongoing clonus from the initial load.
appeared to be at least partly responsible for the enhanced ankle activity. It is possible that the modest load (18 Nm) of the sustained load stimulus was not effective in enhancing the activity of the extensor muscles.

Another important effect of ankle load afferents on locomotion is that a rapid decrease inafferent firing during unloading appears to trigger the swing phase of gait (Grillner and Rossignol 1978; Pearson and Duysens 1976; Pearson et al. 1992). In the current study, load release provided a strong cue for triggering a reflex response; however, the ensuing response did not strongly resemble swing. Although a strong hip flexion was produced, and activity of the ankle and knee flexors was observed, the dominant response at the knee and ankle was still an extension torque. These observations, however, are consistent with recent flexor reflex experimental results, which show that individuals with SCI have significantly smaller knee flexion torque, or often exhibit a net knee extension torque following noxious stimulation at the medial arch (Deutsch et al. 2005). Reorganization of the flexion reflex pathways within the spinal cord circuitry, or nonuniform muscle atrophy throughout the leg after SCI may account for this altered pattern of joint torques. Spastic knee extension and ankle plantar flexion are commonly problematic during the swing phase of gait in human SCI (Fung and Barbeau 1989), and the reflexive knee and ankle torques observed in the present study may reflect these same locomotor problems.

The effects of load afferents on gait have also been implicated in people with SCI and in human infants. A body load applied during the stance phase of stepping prolongs the stance phase and delays the swing phase of gait in infants (Pang and Yang 2000; Yang et al. 1998). In individuals with SCI, an essential contribution of load related input to the generation of locomotor activity has been recognized. Several studies have shown that physiological locomotor-like leg movement alone (100% body unloading) generated by the application of a driven gait orthosis (Dietz et al. 2002) or by manually assistance (Ferris et al. 2004; Harkema et al. 1997) are not sufficient to generate leg muscle activation in subjects with complete paraplegia. However, leg movements combined with loading of the leg lead to appropriate leg muscle activation. Locomotor training of incomplete paraplegic patients indicates that loading is crucial to the generation of a locomotor pattern (Dietz et al. 1995; Harkema et al. 1997). Therefore the sensitivity of multijoint reflexes to load release in the current study is consistent with activation of a locomotor reflex. To test this hypothesis, however, load release stimuli would need to be applied during locomotion. Artificial stimulation mimicking load release might even have a therapeutic effect on gait through this reflex pathway.

The differences in body position, limb movement, and locomotor spinal “state” for the experimental conditions in the current study compared with locomotion might account for the differences in the reflexes triggered by hip extension and ankle load release. For example, reflexes generated in a lying subject may differ from those during locomotion because of differences in descending vestibular drive. Descending vestibular influence appears to affect H reflex responses triggered with the body in an upright versus supine position in subjects with incomplete SCI (Knikou and Rymer 2003). In addition, load afferents of the contralateral leg, which provide critical feedback for the stance-swing transition (Prochazka 1996) may also modulate the spastic reflex response to hip extension and ankle load release. This condition was not well controlled in the current study. Finally, locomotion involves much greater activation of spinal networks, which cycle from “stance” to “swing” states. This underlying modulation of the excitability of spinal networks might be expected to alter the reflex responses to ankle load release, compared with the relatively static, supine posture used in the current study. Further investigation is needed to elucidate the influence of these factors on the spastic reflexes.

**Ankle clonus**

The clonus triggered by the imposed ankle load only appeared to affect the ankle component of the hip-triggered reflex response. Clonus may result from the recurrent activation of stretch reflexes (Hagbarth et al. 1975; Hidler and Rymer 1999), from the action of a central oscillator (Dimitrijevic et al. 1980; Walsh 1976), or from both (Beres-Jones et al. 2003). Because clonus at the ankle only affected the ankle response to imposed hip movement, we concluded that the clonic effects were localized. As a result, any central oscillators that might underlie the ankle clonus were likely to be limited to the ankle muscles, and not more complex oscillatory networks. The restriction of clonus to the ankle might also have been the result of securing the hip and knee joints in a static position, whereas the ankle was theoretically able to move against the load.

Another interesting aspect of the clonus response was the simultaneous activation of the tibialis anterior. Activation of the tibialis anterior in synchrony with the ankle plantar flexors during clonus has also been observed by others (Beres-Jones et al. 2003; Hidler and Rymer 2000) and appears to be a common feature of clonus. The simultaneous activation of the tibialis anterior may be associated with a reciprocal facilitation during stretch of the plantar flexors, as has been reported during tendon taps of the Achilles tendon (Xia and Rymer 2005). Again, activation of this reflex pathway did not appear to extend to the hip triggered reflex because the thigh muscles were relatively unaffected by the clonus in the ankle.

**Role of interneuronal excitability in SCI spasticity**

The results of this study have implications in the assessment of spastic reflexes in human SCI. Specifically, the results emphasize that spastic reflexes in chronic SCI can include multijoint responses to targeted stimuli, strongly suggesting the involvement of interneuronal pathways. Spastic reflex behaviors, have traditionally been attributed to velocity-dependent homonymous stretch reflexes (Ashworth 1964; Lance 1980); however, individuals with SCI also show other spastic reflexes, such as flexor spasms, which have been associated with an increased flexor reflex response to skin stimuli (Dimitrijevic and Nathan 1968; Shahani and Young 1971) or can even be produced by movement of the ankle or knee (Schmit et al. 2000; Wu et al. 2006). Extensor spasms, which also involve multijoint reflex responses, are common and can be triggered by imposed hip or knee extension movements (Schmit and Benz 2002; Wu et al. 2005). As a result of these observations, a clinical scale, the Spinal Cord Assessment Tool for Spastic Reflexes (SCATS), has been developed to quantify each com-
ponent (clonus, flexor spasms, and extensor spasms) of spastic reflexes in SCI (Benz et al. 2005). In particular, the extensor spasm portion of the scale involves simultaneous extension of the knee and hip to trigger the response. Our current results suggest that care must be taken to avoid loading or releasing load from the ankle so that the test may be more consistent.

Spasticity medication may have influenced the magnitude of the reflex response for some of the subjects in the study. Four of the 12 subjects were taking baclofen to manage spasticity. No conclusions regarding the effects of spasticity medications, level of injury, time since injury, severity of injury (ASIA) on the magnitude, and pattern of muscle activity response to load release could be made due to the limited sample size. The four subjects taking baclofen had no noticeable difference in the pattern or magnitude of the response. This topic is important for future studies since the response to load release may be indicative of the clinical incidence of extensor spasms and may impact locomotor training.

Conclusions

Spastic reflexes were triggered by ankle load release with the hip in extension in 12 volunteers with chronic SCI. The ensuing reflex responses resembled the responses to hip extension movement alone with an enhanced magnitude of muscle activities that are consistent with descriptions of extensor spasms. The reflex response manifested from ankle load release in subjects with chronic SCI is likely due to an organized network located within the lumbosacral region of the spinal cord. These networks may involve portions of the spinal cord centers for reflex control of locomotion because of the similarity of these spastic reflexes to the muscle activity of late stance phase of gait and the modulation by afferents from hip position and ankle/foot extensor load.

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References


