Locomotor adaptations and after-effects to resistance during walking in individuals with spinal cord injury.

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ABSTRACT

Muscle activity during the swing phase of walking is influenced by proprioceptive feedback pathways. Previous studies have shown that feedback and anticipatory motor commands contribute to locomotor adaptive strategies to prolonged exposure to a resistance against leg movements during walking. The purpose of this study was to determine whether people with motor-incomplete spinal cord injuries (SCI) modulate flexor muscle activity in response to different levels of resistance in a similar way as uninjured controls. A second purpose was to determine whether people with motor-incomplete SCI have the capacity to form anticipatory motor commands following exposure to resistance. Subjects walked on a treadmill with the Lokomat robotic gait orthosis. The Lokomat applied different levels of a velocity-dependent resistance, normalized to each subject's maximum voluntary contraction of the hip flexors. Each condition consisted of 20 steps against resistance followed by 20 steps without. Electromyography and kinematics of the lower limb were recorded. Although both groups responded to the resistance with an overall increase in rectus femoris activity during swing, the SCI group showed weak modulation of muscle activity to different levels of resistance. Following removal of the resistance, both groups showed after effects, but they were manifested differently. Controls responded to the removal of resistance with a high step while the SCI subjects exhibited increased step length. The size of the after effect was related to the amount of added resistance. In addition, the SCI group showed a negative relationship between the size of the after effect and locomotor function.

Key Words: locomotion, spinal cord injury, adaptation, after-effects
INTRODUCTION

Functional locomotion depends critically on sensory input. During the swing phase of walking, the level of flexor muscle activity is influenced by proprioceptive feedback from muscle receptors (Lam and Pearson 2001). Experiments in cats showed that assisting limb flexion during the swing phase causes a decrease in hip flexor muscle activity while resisting limb flexion enhanced hip flexor muscle activity (Lam and Pearson 2001; McVea et al. 2005). Other studies have indicated that proprioceptive (group I) feedback pathways from the flexor muscles to spinal locomotor centres could contribute to this phenomenon (Lam and Pearson 2002; Quevedo et al. 2000).

In human infants and adults, loading or resisting the leg during the swing phase of walking also enhances flexor muscle activity (Lam et al. 2006; Lam et al. 2003; Noble and Prentice 2006). Such responses occur immediately and are appropriate for overcoming the resistance to maintain safe locomotion (Garrett and Luckwill 1983; Ghori and Luckwill 1989; Lam et al. 2006). For example, the application of a velocity-dependent resistance against hip and knee joint motion has been shown to result in an immediate increase in the activity of the rectus femoris during the swing phase (Lam et al. 2006). This response disappeared once the resistance was removed, consistent with the idea that the adaptive response in the rectus femoris is mediated by feedback mechanisms, possibly via muscle afferent feedback pathways.

Persistent changes in sensory input during walking can also contribute to the formation of anticipatory locomotor commands (Emken and Reinkensmeyer 2005; Fortin et al. 2009; Lam et al. 2006; Lam et al. 2003; Noble and Prentice 2006; Pang et al. 2003). For example, with repeated exposure to forces that resist flexion during the
swing phase of walking, healthy subjects formed anticipatory motor commands in
response to the resistance (Lam et al. 2006; Noble and Prentice 2006). The
development of these anticipatory motor commands was revealed once the disturbance
was removed and after-effects (high-stepping in this example), persisting for several
steps, could be observed (Lam et al. 2006; Noble and Prentice 2006). The observation
of such after-effects is consistent with the concept that modification of central
commands occurred in order to adapt locomotor output to the new task environment.

Many people who have had a motor-incomplete SCI do recover some walking
function, despite varying degrees of neuromuscular and sensory deficits (Burns et al.
1997; Dietz et al. 1998). Harkema et al. (1997) have already demonstrated that
extensor muscle activity during locomotion in people with clinically complete SCI is
responsive to changes in body load and lower limb kinematics. For motor adaptations
requiring the capacity to anticipate the dynamics of the task, there is evidence from
subjects with stroke that suggests that cortical areas are important (Patton et al. 2006;
Takahashi and Reinkensmeyer 2003). Following spinal cord injury, descending motor
input from the cortex could be compromised, depending on the severity and extent of
the lesion. Here we wish to understand the extent to which swing phase motor output
can be modulated by changes in proprioceptive feedback in people with motor-
incomplete SCI. We hypothesized that individuals with motor-incomplete SCI will have
the capacity to modulate flexor muscle activity in response to different levels of
resistance during walking. We further hypothesized that following repeated exposure to
resistance, individuals with motor-incomplete SCI would exhibit after-effects indicative of
the capacity for developing anticipatory locomotor commands, but that the extent to
which after-effects are observed will be related to the degree of locomotor recovery.

METHODS

Subjects
Nine individuals with motor-incomplete SCI were recruited to participate in this
study. Their characteristics are listed in Table 1. All participants were classified as grade
D on the American Spinal Injury Association Impairment Scale (AIS). All were
ambulatory and were able to walk at least 10 meters over ground. None of the subjects
had any other medical condition or cognitive disorder that would have prevented them
from engaging in exercise. In addition, 17 able-bodied individuals (age 22-75 years)
were recruited into two control groups (see below). The UBC Behavioural Research
Ethics Board approved all procedures and all subjects gave their written informed
consent.

Experimental Setup
All subjects wore a body weight support harness and were fitted to the Lokomat
driven gait orthosis (Hocoma AG, Volketswil, Switzerland). The level of body weight
support provided to each SCI subject (see Table 1) was adjusted to ensure that there
was appropriate stance phase kinematics during walking, using clinical gait observation
(no knee buckling during stance, hip extension through end-stance) (Behrman and
Harkema 2000; Behrman et al. 2005). Subjects in the first control group (n=8) did not
receive any body weight support. In a second control group (n=9), subjects walked with
the Lokomat while being supported at 0, 20, 40, and 60% of body weight. Subjects were
strapped to the exoskeleton device by leg cuffs around the mid-thigh, upper shank, and
lower shank while a waist belt provided trunk support. In the SCI subjects, passive foot
lifters were used to support the ankle. Control subjects did not use the passive foot
lifters.

The Lokomat was programmed to apply velocity-dependent resistance (viscous
force field) against hip joint movement during walking (Lam et al. 2006). This resistance
(M) can be defined by: \( M = B \times \text{vel}_H \), where \( B \) is the viscous (or damping) coefficient
(N·m·sec/rad) while \( \text{vel}_H \) is the angular velocity (rad/sec) of the hip joint. When \( B \) is set
to zero, no resistance is applied.

Participants were given several minutes to familiarize themselves with walking in
the Lokomat. During this time, no resistance was applied and subjects had to walk of
their own volition. The treadmill speed was adjusted to each subject’s tolerance, with
most SCI subjects able to walk at 1.8 km/h (Table 1). All control subjects walked at a
treadmill speed of 1.8 km/h.

Subjects then underwent testing of their maximum voluntary contraction (MVC)
using isometric strength testing in the Lokomat (Bolliger et al. 2008). Three trials were
performed to calculate an average hip flexor maximum voluntary force. Baseline walking
data with no resistance was then recorded (1 minute). Following this, subjects
underwent separate walking trials during which different levels of resistance were
applied. The resistance was applied unilaterally to the weaker leg of the SCI subjects or
to the right leg of the control subjects. The resistance levels were scaled to each
individual’s hip flexor maximum voluntary contraction (MVC). Five to 7 walking trials with
resistance (targeted at 1, 3, 5, 7, 10, 15, or 20% of MVC) were recorded from each
subject. Subjects in the second control group underwent 2 resistance trials (targeting 10
and 20% of MVC) at each level of BWS (0, 20, 40, and 60%). The presentation order of the different resistance levels was randomized for each subject. Each trial consisted of 20 steps against resistance immediately followed by 20 steps without resistance for a total of 40 steps per trial. The resistance was always turned off during mid-stance during which angular velocity is low compared to the rest of the cycle. Previous research has found that able-bodied subjects will adapt to resistance within about 5 steps (Lam et al. 2006). Hence we felt that walking for 20 steps against resistance would provide adequate time for adaptation to occur while minimizing the possibility of fatigue in the SCI subjects. Subjects were not told when the resistance was present or removed; they were instructed to walk regularly throughout all trials.

**Data Collection**

Surface electromyography (EMG) was used to record activity over the muscle bellies of the rectus femoris (RF), biceps femoris (BF), tibialis anterior (TA) and the medial gastrocnemius (MG) (Delsys Inc., Boston, MA). We used the Lokomat’s position sensors to record hip and knee joint angles and a twin-axis electrogoniometer (Biometrics Ltd., Gwent, UK) to record ankle angle. Ankle angle was only obtained in 5 control and 4 SCI subjects. Force-sensitive resistors (FSRs, Interlink Electronics, Camarillo, CA) placed under the heel of the foot and big toe allowed for detection of foot contact and toe-off times. These signals were sampled at 1000 Hz and stored on a computer for off-line analysis. An active infrared marker (Optotrak, Northern Digital Inc, Waterloo, ON) was also placed over the 5th metatarsal head to record foot trajectory and stride length during walking. These data were collected at 100 Hz and synchronized to the other data with a synchronization pulse. All data were recorded from the weaker leg in the SCI subjects and from the right leg in the control subjects.
Offline processing of the data were performed using custom-written routines in Matlab (Mathworks, Natick MA). The EMG data was rectified and low-pass filtered at 100 Hz using a digital zero-lag 4th-order Butterworth filter. All other signals were low-pass filtered at 6 Hz using a digital zero-lag 4th order Butterworth filter. EMG and kinematic data were divided into individual gait cycles as determined by the FSRs. The step cycle was defined as the period between consecutive foot contacts. For inter-subject comparison, the EMG amplitude for each muscle was normalized to the peak rectified EMG value of that muscle during the averaged baseline steps in each subject. The average EMG amplitude of the RF and TA were calculated over the mid-swing phase of each step (approximately 70-90% of the step cycle in each person). For the BF, average EMG amplitude over the pre-swing phase was used (approximately 50-70% of the step cycle) and for the MG, average EMG amplitude over the stance phase was calculated. These periods of interest were in keeping with previous work (Lam et al. 2006). Kinematic data were quantified by calculating the peak flexion angle of the hip and knee, peak dorsiflexion angle at the ankle, and peak foot trajectory height during the swing phase of each step. The stride length of each step was also computed.

The actual amount of resistance that was applied by the Lokomat during each step of the resistance trials was calculated by multiplying instantaneous hip angular velocity by the $B$ value used in that trial. The resistance was then normalized to each subject’s MVC and expressed as a %MVC. Throughout the manuscript, the amount of added resistance refers to the average maximum amount of resistance experienced during the swing phase of the gait cycle.
The 10-meter walk test (10MWT) (Van Hedel et al. 2005) and the Walking Index for Spinal Cord Injury (WISCI-II) (Dittuno and Dittuno Jr 2001) were used as indicators of over ground locomotor capacity in the SCI subjects. For the 10MWT, subjects were timed while they walked along a 10-meter straight path. Three trials were collected and averaged together to determine gait velocity. The WISCI is a 21-point ordinal scale with higher values associated with better locomotor capacity (decreased dependence on walking aids and/or personal assistance to perform walking).

**Statistical Analysis**

All statistical analyses were conducted with a commercially available software package (SPSS Inc, Chicago, IL). A critical alpha value of 0.05 was used to test the significance of all the statistical tests. All group comparisons throughout the manuscript are between the SCI group and the first control group, unless expressly stated. When warranted after ANOVA testing, all post hoc pair-wise comparisons using the Bonferroni correction were performed. For all correlations analysis, a look-up table was used to determine significant values based on the sample size of each group.

To characterize the changes in the walking pattern (EMG and kinematic variables) associated with resistance, we focused on trials where the actual amount of resistance peaked at 10% of MVC. A group (control, SCI) by condition (baseline, resistance) repeated measures ANOVA was used to characterize the overall effect of resistance on EMG and kinematic variables during the swing phase (for this analysis, all 20 steps against resistance were averaged together). To track changes in the gait pattern over the course of the 20 steps against resistance, a group (control, SCI) by
ANOVA was performed. Pearson’s correlation coefficient (r) was used to determine if there was a relationship between the change in RF EMG activity and the amount of added resistance during the swing phase. Differences in RF modulation to resistance between the SCI and control group was tested by plotting the percent change in RF EMG amplitude vs. the actual amount of added resistance in each subject. The data were fitted with a linear equation and the slope of this line determined. An independent t-test was used to compare slope values in the SCI vs. control subjects.

To determine if the amount of body weight support affected the response to resistance, we plotted the percent change in RF EMG amplitude vs. the actual amount of added resistance in each subject of the second control group. The data were fitted with a linear equation and the slope of this line determined. A repeated measures ANOVA across the different BWS levels (0, 20, 40, and 60% BWS) was performed on these slope values. An independent t-test was also used to compare the slope values from the first control group with the data from the second control at 0% BWS.

To characterize the after-effect responses, we again focused on trials where the actual amount of resistance peaked at 10% of MVC. A group (control, SCI) by time (baseline, 1st step after resistance removed) repeated measures ANOVA was used to compare the change from baseline in hip, knee, or ankle flexion, foot trajectory height, and stride length of the first step taken after the resistance was removed and baseline. To track changes in the gait pattern following the removal of resistance, a group (control, SCI) by time (baseline, 1st step, 10th step, and 20th step after resistance
removed) repeated measures ANOVA was performed. To confirm that after effects were washed out within the 20-step trials, we divided all data into 4 categories based on the amount of resistance that was applied: 0-2%, 2-4%, 4-6%, and >6% MVC. The percent change in stride length (for the SCI group) and foot trajectory height (in controls) was calculated for each of the 20 steps following removal of resistance in each of these categories. A group (0-2%, 2-4%, 4-6%, >6% MVC) by time (baseline, step 1, 2 … 20 after resistance removed) repeated measures ANOVA was performed to determine when the percent change in stride length/foot trajectory height was not significantly different from zero (main effect of time).

To determine if the amount of body weight support affected the size of the after-effects in foot trajectory height, a condition (baseline, 1st step after resistance removed) by BWS (0, 20, 40, and 60% BWS) repeated measures ANOVA was performed on the data collected from the second control group.

Pearson’s r was used to determine if there was a relationship between the amount of added resistance and the size of the after-effect. To determine if there was any relationship between locomotor impairment due to SCI and the size of the after-effects, Spearman’s rank correlation coefficient (ρ) was calculated between the percent change in stride length and over ground gait velocity (as measured by the 10MWT) and the dependence on ambulatory aids for over ground walking (as measured by the WISCI score). The same calculation was done for the percent change in peak hip flexion during swing.

RESULTS
**EMG response to resistance**

Figure 1 illustrates the averaged EMG activity patterns during baseline walking and during steps against resistance for 1 control (left panel) and 1 SCI subject (Subject 3, Table 1) (right panel). An average of 20 steps in both conditions are plotted. The amount of added resistance in these examples was comparable between the two subjects (Fig. 1, bottom plots), with the peak level of resistance occurring during the swing phase. In these examples, the average resistance during the swing phase was 5% of each subject’s MVC, peaking at approximately 10% of MVC. It is evident from these examples that RF EMG activity during swing increased and peak hip flexion during swing decreased during steps taken against resistance. Note that the individual with SCI did not have active ankle dorsiflexor control, as reflected by the lack of activity in the TA.

Figure 2 illustrates the average changes in cycle duration (Fig. 2A), EMG amplitude (Fig. 2B-E), and gait kinematics (Fig. 2F-J) over the 20 steps against resistance that was about 10% of MVC during the swing phase in control and SCI subjects. The overall changes in gait patterns (averaged across the 20 steps against resistance) are also plotted on the same graphs. ANOVA results yielded significant changes between baseline and resistance in RF EMG amplitude and peak hip flexion angle.

The RF showed an overall significant main effect of condition (baseline vs. resistance) \( p = 0.019 \), but no main effects of group \( p = 0.72 \) and no interaction effects \( p = 0.48 \) (Fig. 2B). The *group by time* (baseline, 1st step, 10th step, and last step against resistance) repeated measures ANOVA on RF amplitude showed a
significant main effect of time (p = 0.03) but no main effects for group (p = 0.71) and no interaction effects (p = 0.84) (Fig. 2B).

At the hip, there was an overall significant decrease in peak flexion angle across the steps taken against resistance compared to baseline (p = 0.019), no significant group effects (p = 0.38) and a significant interaction effect (p < 0.001) (Fig. 2F). Post hoc testing showed that the control group showed a significant decrease in peak hip flexion across all steps against resistance (p < 0.001).

The group by time analysis on peak hip flexion showed significant main effects for time (p = 0.01), no significant main effects of group (p = 0.89), and a significant interaction effect (p < 0.001) (Fig. 2F). Post hoc testing showed that in the controls, there were significant decreases in peak hip flexion angle in the 1st (p = 0.003), 10th (p = 0.003), and last step against resistance (p = 0.002) compared to baseline. There were no differences in peak hip flexion angle among the 1st, 10th, and last step against resistance in the SCI group (p > 0.05).

**Modulation of muscle activity in response to different levels of resistance**

Figure 3A illustrates the relationship between the amount of added resistance and the change in EMG amplitude (averaged across all steps at a given level of resistance). Although both the control and SCI subjects showed increases in RF EMG, only the control subjects showed a significant relationship between the amount of added resistance and the change in RF EMG amplitude (r = 0.71, p < 0.05, Fig. 3A). There was no significant relationship between the amount of added resistance and the change in RF EMG amplitude in the SCI subjects (r = 0.29, p > 0.05, Fig. 3A). The average slope of the line of best fit between the percent change in RF EMG amplitude and the
amount of added resistance was 3.11 (SD 2.77) in the controls and 0.12 (SD 0.64) for the SCI subjects. There was a significant difference in these slope values between the two groups (p = 0.01). In two subjects, the minimum speed possible on our treadmill was more than twice their over ground walking speed. They were able to complete the experimental protocol but we wished to ensure that the relatively fast treadmill speed did not affect the EMG modulation pattern. Even with their data removed, the average slope in the SCI subjects remained low at 0.24 (SD 0.61) and was significantly different from the controls (p = 0.02).

To verify that differences in the modulation of RF activity between the control and SCI groups were not due to the different amounts of BWS required by the SCI group, we conducted a second control experiment in 9 able-bodied control subjects who walked under different levels of BWS (see Methods). Figure 3B illustrates the change in RF EMG amplitude as a function of the amount of added resistance for different levels of BWS (0, 20%, 40%, and 60% of body weight). The average slope of the line of best fit between the percent change in RF EMG amplitude and the amount of added resistance at 0% BWS was 3.60 (SD 5.76), at 20% BWS was 5.09 (SD 7.98), at 40% BWS was 2.03 (SD 2.00), and at 60% BWS was 2.51 (SD 2.00). The magnitude of the slopes of the lines of best fit through each of the data sets were not significantly different from each other (main effect of BWS: p = 0.547). There was also no significant difference between the data from the first control group and that of the second control group at 0% BWS (p = 0.740).
After-effects following removal of resistance

During each resistance trial, the resistance was removed without advance notice to the subjects. Figure 4 illustrates the hip, knee, ankle and foot kinematics after the resistance was removed in 1 control subject (Fig. 4A) and 1 SCI subject (Fig. 4B). The data are from the first step without resistance following 20 steps against swing phase resistance that was about 10% of each subject’s MVC (same subjects and trial used in Fig. 1). An after-effect can be discerned in both examples, as illustrated by the increased hip and/or knee flexion in the first step without resistance. However, while the control subject exhibited a high stepping after-effect (increased foot trajectory height) in the first step after resistance, the end-point manifestation of the after-effect in the SCI subject was a longer stride length (Fig. 4, bottom panels).

Figure 5 illustrates the average change in foot trajectory height, stride length, peak hip flexion, peak knee flexion, and peak ankle dorsiflexion during the swing phase of the first step following removal of resistance that was about 10% of MVC during the swing phase. Both the control and SCI group showed after-effects in peak hip flexion during swing upon removal of the resistance. The average increase in peak hip flexion was 4.7° (SD 2.8°) in the controls and 4.9° (SD 2.1°) in the SCI subjects (Fig. 5A). These corresponded to a 19.4% (SD 16.6%) and a 26.9% (SD 25.7%) increase in peak hip flexion in the control and SCI subjects, respectively. There was a significant main effect of condition (baseline vs. 1st step after resistance removed) (p < 0.001). There were no significant group effects (p = 0.08) or interaction effects (p = 0.49).

At the knee, there was a significant main effect of group (p = 0.003) and a significant interaction effect (p = 0.03) but no significant main effect of condition (p =
Post hoc testing showed that controls exhibited greater knee flexion in the first step without resistance compared to baseline (average increase: 6.2° (SD 3.9°), p = 0.02, Fig. 5B). No significant changes were noted in the ankle.

For peak foot trajectory height, there were significant main effects for condition (p = 0.02), group (p < 0.001) and interaction effects (p = 0.02). Post hoc testing showed that there was a significant increase in peak foot trajectory height in the control subjects (average increase: 13.1 mm (SD 8.6 mm), p = 0.02), but not in the SCI subjects (Fig. 5D). For stride length, there were significant main effects for condition (p = 0.01) and significant interaction effects (p = 0.003), but no significant main effects for group (p = 0.35). Post hoc testing showed that stride length was significantly greater in the first step following resistance removal compared to baseline in the SCI subjects (average increase: 51.5 mm (SD 19.9 mm), p < 0.01, Fig. 5E), but not in the controls.

Since both the control and SCI group showed robust after-effects at the hip, we analyzed the percent change in peak hip flexion of the first step taken after every resistance trial. The average size of the hip after-effects in the control subjects was 11.1% (SD 15.1) and that in the SCI group was 20.5% (SD 23.7%) above baseline. The size of the hip after-effects in the SCI group was significantly greater than that in the control group (p = 0.02).

Data from the second control group are presented in Figure 6 and show that the amount of BWS did not affect the size of after-effects. Peak foot trajectory height during baseline walking and the first step following removal of resistance are plotted for each level of BWS. There was no main effect of BWS (p = 0.08) and no significant interaction
effects ($p = 0.84$). However, there was a significant main effect of walking condition (baseline vs. 1st step after resistance removed, $p < 0.001$).

Figure 7 illustrates the average changes in swing phase RF EMG amplitude (Fig. 7A), peak hip flexion (Fig. 7B), peak knee flexion (Fig. 7C), stride length (Fig. 7D), and peak foot trajectory height (Fig. 7E) over the 20 steps of de-adaptation following removal of resistance that was at 10% of MVC during the swing phase.

In the RF, there was a significant main effect of time ($p = 0.006$) but no group ($p = 0.57$) or interaction effects ($p = 0.46$) (Fig. 7A). Peak hip flexion during swing also showed a significant main effect for time ($p < 0.001$) but not for group ($p = 0.10$) and no interaction effects ($p = 0.07$) (Fig. 7B). For the knee, there was a significant main effect for group ($p = 0.01$) but no main effects for time ($p = 0.45$) and no interaction effects ($p = 0.36$) (Fig. 7C). For stride length, there was a significant interaction effect ($p = 0.01$) but no main effects of time ($p = 0.07$) or group ($p = 0.32$) (Fig. 7D). Post hoc multiple comparison testing showed that in the SCI group, there was a significant difference between baseline and the first step after resistance was removed ($p < 0.001$) (Fig. 7D).

Finally, for foot trajectory height, there were significant main effects for time ($p = 0.01$), group ($p < 0.001$), and a significant interaction effect ($p = 0.01$) (Fig. 7E). Post hoc multiple comparison testing showed significant difference between baseline and the first step after resistance was removed ($p = 0.02$) (Fig. 7E).

The percent change in stride length in the SCI subjects (Fig. 7F) and peak foot trajectory height in the control subjects (Fig. 7G) are plotted for each step taken following removal of different levels of resistance. Although there was a trend for larger after-effects following higher levels of resistance, both of these parameters returned to
within 5% of their baseline values by the mid-point of the de-adaptation trials. For the percent change in stride length in the SCI subjects, there was a main effect of time (p < 0.001). Post hoc multiple comparison tests showed that by the 7th step after the resistance was removed, the percent change in stride length was not significantly different from zero. For the percent change in foot trajectory height in the control subjects, there was a main effect of time (p < 0.001). Post hoc tests showed that by the 5th step after the resistance was removed, the percent change in foot trajectory height was not significantly different from zero.

Figure 8 illustrates the relationship between the size of the after-effect in the first step without the resistance and the amount of resistance that had been added. In the control subjects, there was a positive relationship between foot trajectory height in the first step following removal of the resistance and the amount of added resistance (r = 0.32, p > 0.05). In the SCI subjects, there was a positive relationship between stride length in the first step following removal of the resistance and the amount of added resistance (r = 0.48, p > 0.05).

Locomotor recovery in the SCI subjects was measured by the 10MWT and the WISCI. Unfortunately, 10MWT data were missing for one subject (subject 8). Figure 9 illustrates the relationship between locomotor recovery and the size of the after-effects. For the after-effects in stride length, there was a negative relationship to the 10MWT ($\rho = -0.55$, $p > 0.05$, Fig. 9A) and the WISCI ($\rho = -0.44$, $p > 0.05$, Fig. 9B), indicating that larger after-effects were associated with poorer locomotor capacity. Similarly, for the after-effects in peak hip flexion, there was a negative relationship to the 10MWT ($\rho = -0.31$, $p > 0.05$, Fig. 9C) and the WISCI ($\rho = -0.27$, $p > 0.05$, Fig. 9D).
In this study, we evaluated changes in lower limb muscle activity and kinematic patterns in response to different levels of resistance against leg movements in people with motor incomplete SCI compared to controls. Although both groups showed an increase in swing phase RF EMG activity with resistance, the SCI subjects tended to show weak modulation of RF muscle activity in response to the different levels of resistance. In contrast, control subjects showed a strong relationship between the change in RF activity and amount of added resistance. After-effects following removal of the resistance were manifested in the controls as increased peak foot trajectory height while in the SCI group, they were manifested as increased stride length. In addition, the size of the after-effects in the SCI group tended to be negatively related to the degree of locomotor recovery.

**Flexor muscle response during the swing phase**

Both subject groups showed an increase in RF activity compared to baseline in the presence of resistance. As the resistance was applied against the hip joint, the increase in RF activation during swing is unsurprising given its role as a hip flexor and is also consistent with our previous results (Lam et al. 2006). Changes in transmission through proprioceptive feedback pathways from load- or length-sensitive muscle afferents could have contributed to this response to the resistance (Lam and Pearson 2001). This possibility is consistent with observations in human adults where rapid mechanical obstructions against the swinging limb during walking result in rapid EMG responses in ongoing flexor muscle activity (Dietz et al. 2004; Ghori and Luckwill 1989). The response latencies to such obstructions were thought to correspond to the involvement of polysynaptic spinal (Dietz et al. 2004) or supraspinal pathways (Ghori
and Luckwill 1989). With respect to the potential contribution of supraspinal pathways to
the adaptation of RF during swing phase disturbances, Bonnard et al. (2002) showed
that motor evoked potentials in the RF during a constrained walking situation are larger
compared to unconstrained walking. Locomotor adaptations to the constrained walking
condition consisted of increased EMG activity in proximal leg muscles. Thus, RF activity
during locomotion could be modulated by changes in corticospinal transmission during
walking tasks requiring adaptations in motor output of proximal leg muscles (Bonnard et
al. 2002), although it cannot be established which components of the descending
pathways are involved (2002; Capaday et al. 1999).

Although there was an overall increase in RF activity in response to the
resistance, only the control group demonstrated a strong relationship between the
change in RF muscle activity and the amount of added resistance. This relationship did
not occur in the SCI group. It appears that the grading of muscle activity in response to
incremental increases in applied resistance is not present in the SCI subjects, at least
across the range of resistance levels we used here. One possibility is that there could
be impaired rate modulation during ramped increases of voluntary force, although this
has only been shown in motor units of the small hand muscles in individuals with motor-
incomplete SCI (Zijdewind and Thomas 2003). We also cannot discount the fact that we
were unable to access important muscles around the hip, such as the iliopsoas, and
therefore may have missed possible modulation of their responses to the different levels
of hip resistance.
After-effects following removal of resistance

Subjects in both groups showed after-effects immediately following the removal of the resistance. Both groups showed after-effects in peak hip flexion and the magnitude of these after-effects were significantly higher in the SCI group compared to controls. However, there was an interesting difference in the way the after-effect was manifested between the two groups. In the controls, the increase in foot trajectory height during the after-effects was accompanied by an increase in both hip and knee flexion during swing. In contrast, the SCI group exhibited a longer stride length following removal of the resistance that was accompanied only by an increase in hip flexion during swing. We surmise that this reflects differences in the locomotor strategies employed to adapt to the hip resistance.

The features of the motor patterns that are revealed by after-effects can be considered as clues to the strategies utilized by the locomotor system to adapt to the perturbation. For instance, locomotor adaptations to different types of force fields during swing are accompanied by specific changes in lower limb EMG and kinematic patterns (Blanchette and Bouyer 2009; Fortin et al. 2009; Lam et al. 2006; Noble and Prentice 2006). Once the perturbation was removed, corresponding after-effects were observed and accompanied, at least for a few steps, by the persistent change in EMG activation (Blanchette and Bouyer 2009; Fortin et al. 2009; Lam et al. 2006). In the present study, the features of the after-effects suggest that the control subjects employed a multi-joint strategy to adapt to the hip resistance. The combined increase in hip and knee joint flexion could account for the overall increase in foot trajectory height during swing.

Although foot clearance height is usually considered to be primarily influenced by ankle dorsiflexion (note that there was no change in ankle angle in our results), it is also very
sensitive to small changes in knee angle (Winter 1992). The SCI subjects, in contrast, appeared to employ a more local strategy relegated to the hip, which translated to an after-effect manifested by changes in stride length. Indeed, positive work by the hip flexors during swing is a contributing factor influencing stride length (Winter 1992).

**After-effects and neurological injury**

We observed a trend that the size of the after-effects was negatively correlated to locomotor capacity (e.g. faster over ground walking speeds), suggesting that in individuals who have recovered better locomotor function, the capacity to generate anticipatory locomotor commands (as manifested by after-effects) is diminished. However, our measures of walking capacity can only function as gross indicators of neural impairment. We cannot discern any changes along specific neural pathways or compensatory mechanisms underlying our subjects’ walking capacity.

Previous findings from human and animal studies suggest that cortical areas are not necessary for the ability to form anticipatory motor commands in response to force perturbations (Hodgson et al. 1994; Lou and Bloedel 1988; Patton et al. 2006). Spinalized or decerebrate animal preparations show after-effects following a period of sustained perturbation during stepping (Hodgson et al. 1994; Lou and Bloedel 1988). Significant after-effects during reaching movements have also been reported in stroke subjects following adaptation to a novel force field, although the size of the after-effects was smaller compared to control subjects (Patton et al. 2006). In addition, the size of the after-effects are only weakly correlated to functional improvement post-stroke, if at all (Patton et al. 2006; Takahashi and Reinkensmeyer 2003). In contrast, when we compared after-effects at the hip, we found that the SCI group showed larger after-
effects than the controls. However, the controls certainly showed more robust after-effects in terms of changes in overall lower limb kinematic patterns (hip and knee after-effects) while the after-effects in the SCI group were relegated to one joint parameter (hip only). Thus, the magnitude of the after-effects during walking tasks should take into account the synergistic changes across multiple joints of the lower limb, and not only a simple assessment of the size of the after-effect in only one kinematic parameter. If we use this framework to evaluate the magnitude of the after-effects we observed here, our results are consistent with those previously observed in stroke (Patton et al. 2006).

We also cannot discount the probable contribution of cerebellar structures and pathways to the locomotor adaptations observed here. Cerebellar structures have been shown to be responsive to treadmill-induced perturbations in decerebrate cats (Yanagihara and Kondo 1996; Yanagihara and Udo 1994). During locomotion, inactivation of nitric oxide pathways in the cerebellum was shown to prevent the ability of decerebrate cats to adapt to a split-belt treadmill (Yanagihara and Kondo 1996). Humans with cerebellar lesions also show deficits in the adaptation to speed perturbations during walking (Rand et al. 1998), split-belt treadmill perturbations (Morton and Bastian 2006) and prism adaptation during walking (Morton and Bastian 2004). It is possible that some of the participants we tested here had a SCI that resulted in injury along spinocerebellar pathways. Injury along these pathways, which provide important proprioceptive feedback to the cerebellum during locomotion (Arshavsky et al. 1972; Bosco et al. 2006), could have contributed to the altered adaptation patterns to the resistance, compared to controls. However, SCI are very diverse in their specific injury patterns resulting in lesions along different ascending and descending tracts to and from...
brain, brainstem and cerebellar structures. Future studies on adaptive motor strategies in SCI would benefit from improved assessments of injury in specific pathways.

Our results suggest that better locomotor recovery (as measured by functional outcomes) is not necessarily associated with a ‘normalization’ of locomotor control. Recovery of locomotor function following SCI has been shown to correspond to high-frequency coherence between antagonist thigh muscles (Norton and Gorassini 2006). However, the fact that such coherence is not observed in control subjects suggests that even though there is recovery of locomotor function, the recovery pathways may not necessarily involve the same as those contributing to walking in uninjured individuals (Norton and Gorassini 2006). We found that those individuals with better over ground locomotor capacity tended to have smaller after-effects following the removal of the resistance, in terms of hip flexion and stride length. They also did not show strong after-effects in any of the other kinematic parameters. Thus, improvements in over ground locomotor function are not necessarily accompanied by the capacity for short-term development of anticipatory locomotor commands. Future studies should investigate the extent to which deficits in adaptive locomotor control affect functional mobility in everyday environments and how rehabilitation strategies could be targeted to re-establish such adaptive locomotor control mechanisms, based on specific characterization of injury along the spinal cord.
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**FIGURE LEGENDS**

*Figure 1. Electromyographic (EMG) response to resistance during treadmill walking.*

Averaged EMG and joint kinematic patterns in (A) a control and (B) SCI subject during baseline (thin black lines) and during steps against resistance (thick black lines). The average maximum amount of resistance during the swing phase was about 10% of MVC in both subjects. All EMG data from each subject were normalized to the peak averaged EMG of each muscle during baseline walking. In the plots of joint kinematics and the added resistance, positive values indicate flexion. All data were normalized in time to 100% of the step cycle.

*Figure 2. Changes in temporal, EMG, and kinematic gait parameters during steps against resistance.*

Step cycle duration (A), EMG amplitude (B-E), peak hip, knee, and ankle flexion during swing (F-H), peak foot trajectory height (I) and stride length (J) averaged across baseline and the 1st, 10th, last, and all steps against resistance in the control (grey diamonds) and SCI (black circles) subjects. The average amount of added resistance during the swing phase was 10% of MVC in both groups. All EMG data were normalized to the peak averaged EMG of each muscle during baseline walking (see Methods). Error bars represent standard deviation. Grey and black asterisks indicate significant differences compared to baseline in the control and SCI groups, respectively.

*Figure 3. Modulation of RF EMG activity to different levels of resistance*

(A) Relationship between the amount of added resistance and the percent change in RF activity during swing in controls without BWS (grey diamond symbols) and SCI subjects with BWS as needed (black circle symbols). Asterisk represents a significant correlation. (B) Relationship between the amount of added resistance and
the percent change in RF activity in the second control group at four different levels of BWS. Grey and black solid and dashed lines represent the average linear fit of the data for each level of BWS.

**Figure 4. Change in lower limb kinematics in the first step after removal of resistance.**

Average hip, knee, and ankle angle and foot trajectory during baseline stepping (thin black lines) and the first step following removal of resistance (thick black lines) for (A) a control and (B) SCI subject. In the plots of joint kinematics, positive values indicate flexion. The amount of resistance was 10% of MVC. in both subjects. All data were normalized in time to 100% of the step cycle.

**Figure 5. Quantification of after-effects following removal of resistance.**

Comparison of peak hip flexion, peak knee flexion, and peak ankle dorsiflexion angle during the swing phase, as well as stride length and peak foot trajectory height between baseline steps and the first step following removal of resistance, averaged across subjects in each group. The amount of resistance was 10% of MVC. Error bars represent standard deviation. Asterisks represent significant differences between baseline and the first step following removal of resistance.

**Figure 6. Effect of BWS on after-effects following removal of resistance**

Comparison of the peak foot trajectory height during baseline stepping (grey bars) and the first step taken after resistance was removed (black bars) in subjects in the second control group who walked at different levels of body weight support. Error bars represent standard deviation.
Figure 7. Changes in EMG and kinematic gait parameters in steps following removal of resistance

RF EMG amplitude (A), peak hip flexion (B) and peak knee flexion (C) during swing, stride length (D), and peak foot trajectory height during swing (E) averaged across baseline and the 1st, 10th, and 20th step following removal of the resistance in the control (grey diamonds) and SCI (black circles) subjects. The average amount of added resistance during the swing phase was 10% of MVC in both groups. Error bars represent standard deviation. Grey and black asterisks represent significant differences from baseline in the control and SCI subjects, respectively. (F) Step-by-step changes in stride length (% change from baseline) in the SCI subjects and (G) peak foot trajectory height (% change from baseline) in the control subjects during the 20 steps after resistance was removed. Data were grouped according to level of resistance that was added. Dashed vertical line indicates the number of steps after which the percent changes were not significantly different from zero.

Figure 8. Relationship between size of after-effect and amount of added resistance.

Scatterplots comparing the amount of added resistance and (A) percent change in foot trajectory height and (B) percent change in stride length in the control (grey diamonds) and SCI subjects (black circles). Each data set was fit with a linear equation (grey and black lines).

Figure 9. Relationship between size of after-effect and locomotor capacity

The relationship between the size of the stride length after-effect vs. the 10MWT (A) and the WISCI score (B) and between the size of the hip after-effect vs. the 10MWT (C) and WISCI score (D). Each point represents data from one trial in each SCI subject. Each data set was fit with a linear equation (black lines).
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*AIS, American Spinal Injury Association Impairment Scale

**WISCI: Walking Index for Spinal Cord Injury
A. control

B. SCI

- baseline
- resistance

RF

BF

TA

MG

hip angle (degrees)

knee angle (degrees)

ankle angle (degrees)

added resistance (% MVC)

foot trajectory

% step cycle

% step cycle

50 mm

300 mm
A. 

$r = 0.71^*$

% change in RF EMG amplitude

B. 

% change in RF EMG amplitude

added resistance during swing (% MVC)

- $r = 0.29$

- $0\%$ BWS
- $20\%$ BWS
- $40\%$ BWS
- $60\%$ BWS

controls

SCI
A. control

B. SCI

- Hip angle (deg)
- Knee angle (deg)
- Ankle angle (deg)
- Foot trajectory (mm)

baseline
resistance off, step 1

% step cycle
A. Peak hip flexion (deg)

B. Peak knee flexion (deg)

C. Peak ankle dorsiflexion (deg)

D. Foot trajectory height (mm)

E. Step length (mm)

Controls

SCI

* Indicates significance
foot trajectory height (mm)

level of body weight support

baseline
resistance removed, 1st step
A. RF amplitude (normalized units)

B. Peak hip flexion (deg)

C. Peak knee flexion (deg)

D. Stride length (mm)

E. Peak foot trajectory height (mm)

F. % change in stride length (SCI subjects)

G. % change in foot trajectory height (control subjects)
A. Controls SCI

% change in peak foot trajectory height

peak added resistance during swing (% MVC)

r = 0.32

r = 0.003

B. Controls SCI

% change in stride length

peak added resistance during swing (% MVC)

r = 0.48

r = 0.004
A. % change in stride length vs. 10MWT (gait velocity, m/s)

B. WISCI score vs. 10MWT (gait velocity, m/s)

C. % change in peak hip flexion vs. 10MWT (gait velocity, m/s)

D. WISCI score vs. 10MWT (gait velocity, m/s)