Locomotor adaptations and aftereffects to resistance during walking in individuals with spinal cord injury

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Houldin A, Luttin K, Lam T. Locomotor adaptations and aftereffects to resistance during walking in individuals with spinal cord injury. J Neurophysiol 106: 247–258, 2011. First published May 4, 2011; doi:10.1152/jn.00753.2010.—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 aftereffects, but they were manifested differently. Controls responded to the removal of resistance with a high step, whereas the SCI subjects exhibited increased step length. The size of the aftereffect was related to the amount of added resistance. In addition, the SCI group showed a negative relationship between the size of the aftereffect and locomotor function.

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, whereas 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 centers 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. 2003, 2006; 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 (RF) 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 RF 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. 2003, 2006; 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 aftereffects (high-stepping in this example), persisting for several steps, could be observed (Lam et al. 2006; Noble and Prentice 2006). The observation of such aftereffects is consistent with the concept that modification of central commands occurred to adapt locomotor output to the new task environment.

Many people who have had a motor-incomplete spinal cord injury (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 SCI, 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 aftereffects indicative of the capacity for developing anticipatory locomotor commands but that the...
extent to which aftereffects 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 able to walk at least 10 m on 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 yr) were recruited into 2 control groups (see below). The University of British Columbia Behavioural Research Ethics Board approved all procedures, and all subjects gave their written informed consent.

Experimental setup. All subjects wore a body weight support (BWS) harness and were fitted to the Lokomat driven gait orthosis (Hocoma, Volketswil, Switzerland). The level of BWS 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 BWS. 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 midthigh, upper shank, and lower shank, whereas 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: \( M = B \times \omega \), where \( B \) is the viscous (or damping) coefficient (N m s rad\(^{-1}\)) whereas \( \omega \) is the angular velocity (rad s\(^{-1}\)) of the hip joint. When \( B \) is set to 0, 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 0.5 m/s (Table 1). All control subjects walked at a treadmill speed of 0.5 m/s.

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 min). 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 MVC. Five to seven 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 midstance during which angular velocity is low compared with the rest of the cycle. Previous research has found that able-bodied subjects will adapt to resistance within ~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 RF, biceps femoris (BF), tibialis anterior (TA), and the medial gastrocnemius (MG; DelSys, Boston, MA). We used the Lokomat position sensors to record hip and knee joint angles and a twin-axis electrogoniometer (Biometrics, Gwent, United Kingdom) to record ankle angle. Ankle angle was only obtained in five control and four 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 1,000 Hz and stored on a computer for offline analysis. An active infrared marker (Optotrack; Northern Digital, Waterloo, ON, Canada) was also placed over the fifth 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 was performed using custom-written routines in MATLAB (MathWorks, Natick, MA). The EMG data were rectified and low-pass-filtered at 100 Hz using a digital zero-lag fourth-order Butterworth filter. All other signals were low-pass-filtered at 6 Hz using a digital zero-lag fourth-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 intersubject 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 midswing phase of each step (approximately 70–90% of the step cycle in each person). For the BF, average EMG amplitude over the preswing 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

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Subj, subject; M, male; F, female; T, thoracic; C, cervical; Med, medicine; N/A, not applicable; WISCI, Walking Index for Spinal Cord Injury; BWS, body weight support.
calculated 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-m 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 overground locomotor capacity in the SCI subjects. For the 10MWT, subjects were timed while they walked along a 10-m 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 in the SCI subjects. For the 10MWT, subjects were timed (after resistance removed) for the 10 steps against resistance for 1 control (Fig. 1, left) and 1 SCI subject (subject 3; Table 1; Fig. 1, right).

To determine whether the amount of BWS affected the size of the aftereffects 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 2nd control group.

Pearson $r$ was used to determine whether there was a relationship between the amount of added resistance and the size of the aftereffect. To determine whether there was any relationship between locomotor impairment due to SCI and the size of the aftereffects, Spearman rank correlation coefficient ($\rho$) was calculated between the percent change in stride length and overground gait velocity (as measured by the 10MWT) and the dependence on ambulatory aids for overground 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 (Fig. 1, left) and 1 SCI subject (subject 3; Table 1; Fig. 1, right). An average of 20 steps in both conditions are plotted. The amount of added resistance in these examples was comparable between the 2 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 ~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. 2, B–E), and gait kinematics (Fig. 2, F–J) over the 20 steps against resistance that was ~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 with baseline ($P = 0.019$), no significant group effects...
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 with 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 overground 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 nine 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
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 (\( P = 0.21 \)). Post hoc testing showed that controls exhibited greater knee flexion in the first step without resistance compared with 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 with 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 aftereffects 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 aftereffects 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 aftereffects in the SCI group was significantly greater than that in the control group (\( P = 0.02 \)).

Data from the 2nd control group are presented in Fig. 6 and show that the amount of BWS did not affect the size of aftereffects. Peak foot trajectory height during baseline walking and the 1st 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 deadaptation 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 \((P = 0.01)\) and group \((P < 0.001)\) and a significant interaction effect \((P = 0.01; \text{Fig. 7E})\). Post hoc multiple comparison testing showed a significant difference between baseline and the first step after resistance was removed \((P = 0.02; \text{Fig. 7E})\).

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 aftereffects following higher levels of resistance, both of these parameters returned to within 5% of their baseline values by the midpoint of the deadaptation 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 seventh step after the resistance was removed, the percent change in stride length was not significantly different from 0. 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 fifth step after the resistance was removed, the percent change in foot trajectory height was not significantly different from 0.

Figure 8 illustrates the relationship between the size of the aftereffect 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 aftereffects. For the aftereffects in stride length, there was a negative relationship to the 10MWT \((\rho = -0.55, P > 0.05; \text{Fig. 9A})\) and the WISCI \((\rho = -0.44, P > 0.05; \text{Fig. 9B})\), indicating that larger aftereffects were associated with poorer locomotor capacity. Similarly, for the aftereffects in peak hip flexion, there was a negative relationship to the 10MWT \((\rho = -0.31, P > 0.05; \text{Fig. 9C})\) and the WISCI \((\rho = -0.27, P > 0.05; \text{Fig. 9D})\).

DISCUSSION

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 with 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. Aftereffects following removal of the resistance were manifested in the controls as increased peak foot trajectory height, whereas in the SCI group they were manifested as increased stride length. In addition, the size of the aftereffects 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 with
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; Ghorı́ 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 (Ghorı́ 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 with 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 (Bonnard et al. 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.

Aftereffects following removal of resistance. Subjects in both groups showed aftereffects immediately following the removal of the resistance. Both groups showed aftereffects in peak hip flexion, and the magnitude of these aftereffects were significantly higher in the SCI group compared with controls. However, there was an interesting difference in the way the aftereffect was manifested between the two groups. In the controls, the increase in foot trajectory height during the aftereffects 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 aftereffects can be considered as clues to the strategies used 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 aftereffects 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 aftereffects suggest that the control subjects employed a multijoint 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 aftereffect manifested by changes in stride length. Indeed, positive work by the hip flexors during swing is a contributing factor influencing stride length (Winter 1992).

Aftereffects and neurological injury. We observed a trend that the size of the aftereffects was negatively correlated to locomotor capacity (e.g., faster overground walking speeds), suggesting that in individuals who have recovered better locomotor function, the capacity to generate 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 aftereffects following a period of sustained perturbation during stepping (Hodgson et al. 1994; Lou and Bloedel 1988). Significant aftereffects during reaching movements have also been reported in stroke subjects following adaptation to a novel force field, although the size of the aftereffects was smaller compared with control subjects (Patton et al. 2006). In addition, the size of the aftereffects are only weakly correlated to functional improvement poststroke, if at all (Patton et al. 2006; Takahashi and Reinkensmeyer 2003). In contrast, when we compared aftereffects at the hip, we found that the SCI group showed larger aftereffects than the controls. However, the controls certainly showed more robust aftereffects in terms of changes in overall lower limb kinematic patterns (hip and knee aftereffects), whereas the aftereffects in the SCI group were relegated to one joint parameter (hip only). Thus the magnitude of the aftereffects 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 aftereffect in only one kinematic parameter. If we use this framework to evaluate the magnitude of the aftereffects 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 cer-

![Fig. 9. Relationship between size of aftereffect and locomotor capacity. The relationships between the size of the stride length aftereffect vs. the 10-m walk test (10MWT; A) and the Walking Index for Spinal Cord Injury (WISCI) score (B) and between the size of the hip aftereffect vs. the 10MWT (C) and WISCI score (D) are shown. Each point represents data from 1 trial in each SCI subject. Each data set was fit with a linear equation (black lines).](http://jn.physiology.org/2011/106/7/256)
ebellar 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 spino-cerebellar 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 with controls. However, SCI are very diverse in their specific injury patterns, resulting in lesions along different ascending and descending tracts to and from the brain, brain stem, 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 overground locomotor capacity tended to have smaller aftereffects following the removal of the resistance, in terms of hip flexion and stride length. They also did not show strong aftereffects in any of the other kinematic parameters. Thus improvements in overground 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 reestablish such adaptive locomotor control mechanisms based on specific characterization of injury along the spinal cord.

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DISCLOSURES

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REFERENCES


