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Ankle Load Modulates Hip Kinetics and EMG During Human Locomotion

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Abstract: The purpose of this research was to examine the role of isolated ankle-foot load in regulating locomotor patterns in humans with and without spinal cord injury (SCI). We used a powered ankle-foot orthosis to unilaterally load the ankle and foot during robotically assisted airstepping. The load perturbation consisted of an applied dorsiflexion torque designed to stimulate physiological load sensors originating from the ankle plantar flexor muscles and pressure receptors on the sole of the foot. We hypothesized that 1) the response to load would be phase specific with enhanced ipsilateral extensor muscle activity and joint torque occurring when unilateral ankle-foot load was provided during the stance phase of walking and 2) that the phasing of subject produced hip moments would be modulated by varying the timing of the applied ankle-foot load within the gait cycle. As expected, both SCI and nondisabled subjects demonstrated a significant increase ($P < 0.05$) in peak hip extension moments (142 and 43% increase, respectively) when given ankle-foot load during the stance phase compared with no ankle-foot load. In SCI subjects, this enhanced hip extension response was accompanied by significant increases ($P < 0.05$) in stance phase gluteus maximus activity (27% increase). In addition, when ankle-foot load was applied either 200 ms earlier or later within the gait cycle, SCI subjects demonstrated significant phase shifts (~100 ms) in hip moment profile ($P < 0.05$; i.e., the onset of hip extension moments occurred earlier when ankle-foot load was applied earlier). This study provides new insights into how individuals with spinal cord injury use sensory feedback from ankle-foot load afferents to regulate hip joint moments and muscle activity during gait.

Introduction

Spinal cord injury (SCI) can significantly impair an individual's ability to walk. However, strong evidence indicates that both incomplete and complete SCI individuals have the capacity to produce locomotor patterns when they receive "appropriate afferent feedback"

(Harkema 2008) such as proprioceptive information related to repetitive, alternating, lower limb loading, and flexion/extension movements consistent with walking. Thus identifying sensory-motor pathways that modulate human locomotor patterns may provide a foundation for developing targeted therapies and neuroprosthetic devices aimed at improving gait in SCI populations. Limb load sensory feedback may be especially effective for enhancing the magnitude and timing of functional muscle activity during walking. The importance of limb load feedback is highlighted by research on the decerebrate cat indicating that the efferent response to load afferents can account for potentially half of the extensor muscle activity occurring during gait (Donelan and Pearson 2004; Hiebert and Pearson 1999). Similarly, the effect of load afferents to modulate muscle recruitment during gait has been observed in humans both with (Dietz et al. 2002; Harkema et al. 1997) and without (Dietz et al. 2002; Mazzaro et al. 2005; Sinkjaer et al. 2000; Stephens and Yang 1999; Yang et al. 1991) SCI and in human infants (Yang et al. 1998a), whose developing nervous system provides a model for studying adult SCI gait. In addition, during infant stepping, the timing of limb loading has been shown to have significant effects on the initiation and duration of stance and swing (Pang and Yang 2000; Yang et al. 1998b). As such, increasing our understanding of the effects of load afferents on SCI locomotor function will likely be valuable for developing effective interventions.

Non-human animal research suggests that the primary limb load feedback regulating locomotion arises from the load-sensitive group Ib afferents of the ankle plantar flexors (Conway et al. 1987; Duysens and Pearson 1980; Guertin et al. 1995; Whelan et al. 1995) and the cutaneous afferents located on the plantar surface of the foot (Duysens and Pearson 1976). Specifically, stimulating the ankle plantar flexor load-sensitive afferents (Pearson et al. 1992) and cutaneous afferents (Duysens and Pearson 1976) during stance excites the limb extensor muscles while inhibiting flexors, effectively prolonging stance and delaying swing initiation. Furthermore, a rapid decrease in afferent firing, as occurs during unloading of the ankle plantar flexors, appears to trigger the stance-to-swing transition (Grillner and Rossignol 1978; Pearson et al. 1992). Similar findings during fictive locomotion suggest that ankle load afferents act directly on spinal locomotor generators (Conway et al. 1987) making this

sensory-motor pathway potentially valuable for targeted SCI gait rehabilitation.

In human SCI, sensory feedback from load afferents of the ankle (i.e., group I muscle afferents) and foot (i.e., cutaneous afferents) are likely to affect gait and volitional movement of the legs. Experimentally, multijoint reflexes consisting of hip flexion and knee extension can be triggered in human SCI subjects following a controlled unilateral hip extension (Schmit and Benz 2002; Steldt and Schmit 2004). This polysynaptic reflex response is believed to be mediated through similar neural pathways as those associated with reflex control of locomotion. Of interest is that this hip flexion reflex response can be enhanced in SCI subjects by removing a dorsiflexor ankle torque immediately following hip extension in a manner analogous to the limb unloading that occurs during the late stance phase of gait (Wu and Schmit 2006). These studies provide evidence that complex responses can be produced by sensory cues typically attributed to reflex regulation of locomotion in human SCI; however, the direct effects on stepping are unknown.

Stimulating limb load afferent pathways during gait yields a phase-specific response. Several studies have demonstrated a positive force feedback loop during the stance phase of gait. For example, excitation of the load-sensitive group I ankle extensor muscle afferents (Grey et al. 2007; Sinkjaer et al. 2000; Yang et al. 1991) or cutaneous receptors of the plantar surface of the foot (Duysens et al. 1990; Yang and Stein 1990) during the stance phase of gait contributes to ongoing ankle extensor muscle activity. In contrast, stimulation of these same sensory pathways during the swing phase of gait will not facilitate extensor activity. In fact, stimulation of cutaneous afferents of the plantar surface of the foot during the swing phase of human walking has been shown to enhance ongoing flexor activity (Duysens et al. 1990; Yang and Stein 1990). This phase-dependent nature of reflex pathways during human walking is exemplified by the amplitude modulation of soleus H-reflexes, which are greatest during stance and inhibited during swing (Capaday and Stein 1986).

The generation and control of hip flexion, extension, and abduction torque is likely an important determinant of walking ability for humans with SCI (Kim et al. 2004). The reliance on hip torque to

power walking relative to knee and ankle torque may be increased in SCI subjects similar to the changes observed in stroke (Nadeau et al. 1999) and elderly populations (DeVita and Hortobagyi 2000). Several studies have shown that in human SCI subjects, modulating load at the ankle joint can trigger and/or enhance hip movements (Schmit et al. 2000, 2002; Wu and Schmit 2006). Similarly, during fictive locomotion, stimulation of the group I ankle extensor afferents during bursting results in an increase in amplitude and duration of ipsilateral ankle, knee, and hip extensor activity (Guertin et al. 1995). Collectively, these data suggest that limb load sensory feedback may modulate hip torque production and thus influence walking ability in SCI populations. However, afferent mediation of hip torque during human SCI locomotion has not been measured.

Thus the purpose of this study was to identify the role of sensory feedback from ankle-foot load afferents (group I muscle afferents of the plantar flexor muscles and pressure-sensitive cutaneous afferents on the sole of the foot) on the amplitude and timing of muscle activity and subject-produced hip joint moments during stepping in human SCI and control subjects. First we examined the effect of ankle-foot load (created by applying a dorsiflexor torque about the ankle joint) on the amplitude and phasing of hip joint moments and lower limb muscle activity when subjects stepped with no ankle-foot load, ankle-foot load during stance, or ankle-foot load during swing. Based on previous studies, we hypothesized that unilateral ankle-foot load applied during the stance phase would enhance ipsilateral extensor hip joint moments and muscle activity. We also hypothesized that the effects of ankle-foot load would be phase specific, manifested as enhanced flexor muscle activity and hip joint moments when ankle-foot load was applied during swing. Second we examined temporal modulations of hip joint moments when an applied ankle-foot load of constant duration was shifted within the gait cycle to occur 200 ms earlier or later than normal stance phase. We postulated that modulations in the timing of the applied ankle-foot load would result in a corresponding phase shift in hip joint moments.

Methods

Subjects

Sixteen SCI [37 ± 9.8 (SD) yr, 76.2 ± 14.9 kg, 3 female; Table 1] and 10 nondisabled (ND; 28 ± 3.8 yr, 67 ± 9 kg, 4 female) subjects gave written informed consent and participated in the study. The Northwestern University Institutional Review Board approved the experimental protocol. SCI subjects were all >1 yr post injury and had a spinal cord lesion occurring between C₁ and T₁₀ due to nonprogressive etiology. Two of the SCI subjects were classified as clinically complete [American Spinal Injury Association Impairment Scale (ASIA) A] (Ditunno et al. 1994). The remainder of the SCI subjects had incomplete SCIs (12 ASIA C, 2 ASIA D). Subjects were excluded from the study if they had any of the following: concurrent severe medical illness, history of peripheral nerve injury in the lower legs, history of traumatic head injury, history of cardiovascular or pulmonary complications, history of metabolic (endocrine, hepatic) or renal dysfunction, and inability to tolerate 30 min of standing without orthostasis. Subjects did not alter their medications for this study. Five of the SCI subjects were prescribed antispasticity medications (baclofen) to reduce the intensity and frequency of spasms.

TABLE 1. Subject profile

Subject	Sex	Age	Body Weight, kg	ASIA Level	SCI Level	Post Injury, yr	Ambulatory	Medications	Experiment 1	Experiment 2
SCI-1	M	40	99.8	C	C ₅	7	Household	None	x	—
SCI-2	M	49	89.3	C	C ₃₋₇	7	Yes	Baclofen 110 mg/day; Dantrium 200 mg/day; Topamax	x	—
SCI-3	F	40	54.4	C	C ₅	3	No	Neurontin 600 mg t.i.d.	x	—
SCI-4	M	34	77.1	C	C ₅	2	No	Baclofen 5 mg t.i.d.; Flomax	x	—
SCI-5	F	38	79.8	C	C ₄₋₅	2	Yes	Baclofen 20 mg 4x/day; Zanaflex 2mg 2x/day	x	x
SCI-6	M	24	63.5	C	T ₇₋₁₀	7	Household	4-AP; Ditropan 10 mg 2x/day	x	—

Subject	Sex	Age	Body Weight, kg	ASIA Level	SCI Level	Post Injury, yr	Ambulatory	Medications	Experiment 1	Experiment 2
SCI-7	M	28	77.1	C	C ₇	6	No	Baclofen Pump; Sanctura 20 mg; Lorazepam 0.5 mg; Hydrocodone 5/500 mg	x	—
SCI-8	M	44	67.5	C	C ₅₋₇	11	Yes	None	x	—
SCI-9	M	51	77.1	D	C ₄₋₅	14	Yes	None	x	—
SCI-10	M	22	72.6	A	T ₆	4	No	Baclofen 30 mg 2x/day; Ditropan; Zanaflex	x	—
SCI-11	M	35	72.6	A	C ₇	4	No	None	x	—
SCI-12	M	38	77.1	C	C ₅₋₆	24	Yes	None	—	x
SCI-13	M	36	77.3	C	C ₄	3	Yes	Coumadin	—	x
SCI-14	F	29	43.1	D	C ₁₋₂	4	Yes	None	—	x
SCI-15	M	30	99.8	C	C ₅	6	Household	None	—	x
SCI-16	M	58	90.7	C	T ₇	34	Yes	None	—	x

Profile of spinal cord injury (SCI) subjects participating in this study. An "X" in the final two columns indicates participation in a specific experiment(s).

Equipment

We constructed a 4.3-kg ankle-foot loading device to provide a controllable and overt sensory stimulation to the limb load receptors of the foot and ankle (i.e., group I ankle plantar flexor muscle afferents) during stepping (Fig. 1). The device consisted of an ankle-foot orthosis that used a low-friction ball-bearing joint to allow free sagittal plane rotation about the ankle joint. The orthosis was rigidly attached to the distal end of a commercially available robotic gait orthosis (Lokomat; Hocoma, Zurich, Switzerland) used to assist walking (described in the following text). When pressurized, a pneumatic cylinder created a dorsiflexor torque about the ankle joint. The dorsiflexor torque was adjusted for each subject to ~0.5 Nm/kg, approximately half the peak torque experienced during normal walking (Eng and Winter 1995). A solenoid valve, regulating air flow to the pneumatic cylinder, was controlled by a laptop computer equipped with an analog input-output card (National Instruments, Austin, TX) running custom LabVIEW software (National Instruments). Note that when the solenoid valve was opened to release air pressure during stepping, the rapid expulsion of air from the pneumatic cylinder created a brief auditory

hiss that was detectable by the subjects. The act of pressuring the cylinder did not provide auditory cues detectable over the background noise of the Lokomat hip and knee actuators.

Ankle-Foot Loading Device

FIG. 1. A unilateral powered orthosis attached to the distal end of a Lokomat was used to mechanically stimulate the load receptors of the ankle and foot during stepping movements. A hinge joint (A) allowed sagittal plane rotation at the ankle joint. When pressurized, a pneumatic cylinder (B) created a dorsiflexor torque that was measured by a load cell (C) placed in series with the pneumatic cylinder.

The Lokomat was used to provide assistance to subjects during stepping and to prescribe a consistent kinematic gait pattern. Four DC motors, one aligned at each hip and knee joint, moved the legs in the sagittal plane. Subjects also wore an overhead harness attached to a pulley/counterweight support system that was adjusted to support between 0 and 100% of the subjects' body weight. There were several benefits of using a Lokomat. First, the device provided consistent and measurable levels of assistance. Variability in assistance could alter sensory feedback (cutaneous at the robotic/subject interface and proprioceptive if gait kinematics change) and obscure results. Second, we calculated joint torques during stepping by instrumenting the Lokomat with load cells (JR3, Woodland, CA) at each subject/robot interface of the lower limbs (Hidler 2004). Finally, we used the Lokomat motors and scaffolding to support and carry the ankle-foot loading device. By attaching the device to the Lokomat, subjects did not have to adjust their stepping patterns to accommodate for the added mass.

Measurements

We recorded bilateral lower limb joint kinematics, kinetics, and electromyographic (EMG) activity from subjects during stepping. Hip and knee joint kinematics were measured using the joint sensors of the Lokomat. Sagittal plane motion about the ankle joint was measured with a potentiometer rigidly attached to the "ankle" joint of the ankle-foot loading device. The subjects' thigh and shank kinetics were measured directly from the 6-df load cells attached to the leg attachment cuffs of the Lokomat. During treadmill stepping trials, an ADAL3D-F/COP/Mz split belt treadmill with embedded force plates (HEFGroupe, Andrézieux Bouthéon, France) measured ground reaction forces. EMG signals were recorded from seven major muscles of the legs (Motion Lab Systems, Baton Rouge, LA). Two experiments were conducted for this study, each using slightly different EMG recordings. For *experiment 1*, active surface EMG electrodes were secured to the skin over the bellies of the following muscles bilaterally (soleus, medial gastrocnemius, tibialis anterior, vastus lateralis, vastus medialis, rectus femoris, and medial hamstrings). During *experiment 2*, the EMG setup was altered to record from gluteus maximus instead of vastus lateralis. EMG signals were amplified ($\times 10,000$) and low-pass filtered

(500 Hz). All analog signals were sampled at 1,000 Hz using a data-acquisition card (National Instruments) on a PC running custom Matlab software (The Mathworks, Natick MA).

Protocol

Two separate experiments were performed in this study. All ND subjects and 11 SCI subjects participated in the first experiment. Six incomplete SCI subjects participated in the second experiment. Only one SCI subject participated in both experiments. This subject performed the two experimental sessions on separate days.

All treadmill and airstepping trials were performed at 0.55 m/s. We selected a relatively slow walking speed because it increased the step cycle duration and allowed for sizable variations in the timing of the applied limb loading.

During the first experiment, subjects initially stepped on the treadmill with Lokomat assistance without wearing the ankle-foot loading device and the minimum bodyweight support needed to maintain an upright trunk posture (Behrman and Harkema 2000). We recorded data from 2 min of treadmill stepping. The right-side ground reaction force and hip position data were immediately analyzed and used to calculate the timing of normal stance and swing phases relative to hip position. In the subsequent portion of the experiment, we used real-time hip position data to trigger the applied dorsiflexor torque from the ankle-foot loading device.

Next the subjects performed a series of airstepping trials. For these trials, the ankle-foot loading device was attached unilaterally to the right leg of the Lokomat and donned by the subjects. On the left "nonperturbed limb," no assistive, supportive, or restraining device of any kind was worn on the foot and ankle joint. Subjects were given 100% bodyweight support and elevated ~25 cm above the treadmill surface. In this position, we recorded 20 s of data while the subjects performed airstepping during five different unilateral ankle-foot loading conditions. The conditions were as follows; NO LOAD (ankle-foot device disengaged), STANCE (dorsiflexor torque applied during normal stance phase), EARLY (dorsiflexor torque equal in amplitude and duration to STANCE applied 200 ms earlier in the gait cycle than

normal stance phase), LATE (dorsiflexor torque equal in amplitude and duration to STANCE applied 200 ms later in the gait cycle than normal stance phase), and SWING (dorsiflexor torque applied during normal swing phase). Load was applied for a duration of 1.16 s during the STANCE, EARLY, and LATE conditions. During the SWING condition, load was applied for 0.7 s. During the NO LOAD condition and the "off" periods of the remaining conditions, the subject's right (perturbed) foot remained strapped to the ankle-foot loading device but was allowed free rotation in the sagittal plane (flexion-extension). For each condition, subjects were asked to either relax completely, allowing the robotic devices to move their lower limbs (passive), or they were asked to do their best to volitionally move their limbs with the timing of the Lokomat (active). The purpose of collecting passive trials was twofold. First, the passive NO LOAD condition was used to calculate the active hip moments as described in detail in the following text. Second, examining differences in passive and active trials during the three loading conditions (NO LOAD, STANCE, and SWING) was valuable for assessing the effect of volitional drive on ankle-foot load mediated reflex modulations. The order of the conditions was quasi-randomized with the passive and active trials always back to back (although the order of passive and active trials was varied) for each condition. Subjects were given rest periods as needed between conditions with a minimum rest period of 1 min occurring every two trials. We recorded data from at least two trials of every condition. For each trial, subjects stepped for ~30 s with the designated ankle-foot load before data recording.

A major finding from *experiment 1* was that SCI subjects had significant increases in hip extension torque when load was applied to the ankle-foot during the STANCE condition. In this experiment, the medial hamstrings were the only measured hip extensor muscle group. Post experiment analysis of the hamstrings activity was not sufficient to explain the observed changes in hip torque. Therefore we conducted a second experiment to specifically investigate changes in EMG activity of a uniarticular hip extensor, gluteus maximus, in response to ankle-foot load during stepping in incomplete SCI subjects. During *experiment 2*, we repeated the protocol used during the first experiment, but only the passive and active NO LOAD and STANCE conditions were performed.

Kinetic calculations

All kinematic and kinetic data were smoothed using a fourth-order Butterworth low-pass filter (cut-off frequency: 7 Hz) with zero lag. We calculated the applied ankle dorsiflexor torque created by the ankle-foot loading device by multiplying force measured from a tension-compression load cell (Transducer Techniques, Temecula, CA) attached in series with the pneumatic cylinder by the moment arm of the cylinder about the ankle joint. We also calculated the subject's active contribution to sagittal plane hip joint torques during airstepping using experimental methods previously described in detail (Hidler 2004; Hidler and Neckel 2006). Three-dimensional forces recorded at the load cell attached in series between the Lokomat thigh cuff and the Lokomat upper leg were multiplied by their respective moment arm distance about the hip joint and then summed with the load cell torque measurements to get total hip joint torque during stepping. For each subject, we created an average representative total hip joint torque profile over a complete gait cycle using 8–20 steps recorded during the passive NO LOAD airstepping condition. We assumed that during the passive NO LOAD condition, total hip joint torque was created entirely by passive tissue (i.e., muscle and tendon) and nontissue components (i.e., limb inertia and gravity) and that these components were consistent between steps because the Lokomat constrained kinematic trajectories. Thus we estimated the subjects' active hip moments (i.e., muscular contribution) by subtracting the subject's representative passive NO LOAD joint torque profile from the total hip joint torque calculated for each individual gait cycle recorded during all airstepping conditions (Hidler 2004). This method allowed us to look at variability in hip joint torque between individual steps even for the passive NO LOAD condition. Throughout the remainder of this paper when we refer to hip moments, we are referring only to the active component.

We separated each gait cycle into extensor and flexor hip moment regions and created corresponding hip moment-angle plots. Calculating the area of the positive and negative regions of the moment-angle plots yielded positive and negative work, respectively. We calculated total hip extensor and flexor work by summing the absolute value of the positive and negative work performed. Calculating total hip extensor and flexor work (in addition to peak

moments) was important because it provided a quantifiable performance measure of both the direction and magnitude of the hip moment that subjects produced over the course of the entire gait cycle.

Analysis

To examine changes in hip kinetics, we found the average peak flexion and extension moments and total work performed by the hip flexor and extensor muscles during the gait cycle. We ran a repeated-measures ANOVA ($\alpha = 0.05$) to look for differences in these four hip kinetic measurements between the passive NO LOAD, active NO LOAD, passive STANCE, active STANCE, passive SWING, and active SWING conditions. When appropriate, a Newman-Keuls multiple comparisons test with a family-wise error rate of $\alpha = 0.05$ was used to check for differences between the active NO LOAD condition (which we selected as our baseline performance measure) and the other five conditions. Ankle torque was not compared between conditions because applied ankle loads directly influence the ankle torque. Similarly, knee torque was not compared because biarticular ankle-knee muscles could mechanically transmit the applied ankle torque to the knee.

To examine changes in EMG amplitude, normalized root mean square (RMS) EMG values were calculated for each subject and condition during both the stance (1.16 s) and swing (0.7 s) phases of the gait cycle. RMS values were calculated from high-pass filtered (cutoff frequency: 20 Hz) and rectified EMG data. RMS EMG values were normalized to the average RMS EMG value occurring during the active NO LOAD condition. We ran two repeated-measures ANOVAs to look for differences in EMG amplitude during the stance and swing phases of the following six conditions: passive NO LOAD, active NO LOAD, passive STANCE, active STANCE, passive SWING, and active SWING. Again the significance level was set at $\alpha = 0.05$, and a Newman-Keuls multiple comparisons test with a family-wise error rate of $\alpha = 0.05$ was used to check for differences between the active NO LOAD condition and the other five conditions when appropriate.

Finally, we examined the effect of modulating the timing of ankle-foot load on hip moment patterns. Specifically, we performed a cross-correlation to calculate the phase shift between the subjects' hip

moment and hip joint angle during the active EARLY, active STANCE, and active LATE conditions. The cross-correlation is a method of detecting common periodicities between two signals and the phase shift indicates the magnitude of time difference between the two series (Li and Caldwell 1999). The phase shift between hip moment and hip angle was calculated from time series data recorded during 8–16 continuous steps for each subject and condition. Because the Lokomat held hip kinematics constant across conditions, phase shift changes indicated a temporal modulation of the entire hip moment relative to the gait cycle. A normalized measure was calculated for each subject by finding the difference in phase shift between the STANCE condition and both the EARLY and LATE conditions. This procedure was also used to verify the temporal changes in the applied ankle torque within the gait cycle. We ran a repeated-measures ANOVA to look for relative differences in hip joint torque phasing among the active STANCE, active EARLY, and active LATE conditions. We set the significance level at $\alpha = 0.05$ and used a Newman-Keuls multiple comparison test with a family-wise error rate of $\alpha = 0.05$ to check for differences where appropriate.

Results

Response to NO LOAD, STANCE, and SWING

Hip Kinetics.

ND subjects had significant differences in peak hip extension moment (ANOVA, $P < 0.0001$), total hip extension work (ANOVA, $P < 0.0001$), peak hip flexion moment (ANOVA, $P < 0.0001$), and total hip flexion work (ANOVA, $P < 0.0001$) in the perturbed limb among the NO LOAD, STANCE, and SWING conditions (Figs. 2 B and and3;3; Table 2). Post hoc testing demonstrated that ND subjects increased the amplitude of all four hip kinetic variables during the active NO LOAD condition compared with the passive NO LOAD condition (Newman-Keuls, $P < 0.05$; Fig. 3, Table 2). When load was applied to the foot during the active STANCE condition, ND subjects significantly increased ipsilateral peak hip extension moment [-0.593 ± 0.380 (SD) Nm/kg] by 43% compared with the active NO LOAD condition (-0.415 ± 0.237 Nm/kg; Newman-Keuls, $P < 0.05$; Figs. 2B and

and 3; Table 2). This increased extensor moment occurred during the stance phase (Fig. 2B). In addition, during the *passive* STANCE condition, peak hip extension moment (-0.315 ± 0.153 Nm/kg), and total hip extension work (0.155 ± 0.115 J/kg) also increased to levels that were not significantly different from the *active* NO LOAD condition (Newman-Keuls, $P > 0.05$; Table 2). During both the passive and active SWING conditions, ND subjects did not significantly change any ipsilateral hip kinetic variable when compared with the active NO LOAD condition (Newman-Keuls, $P > 0.05$; Fig. 3; Table 2).

FIG. 2. Mean gait cycle data from the perturbed limb of 8 incomplete spinal cord injury (SCI, A) and 10 nondisabled (B) subjects. Hip kinematics, which were constrained by the Lokomat, did not change between conditions. Most notably when a dorsiflexor torque was applied to the ankle during STANCE, subjects increased hip extension moments. For display purposes, 1 incomplete SCI subject was excluded from the figure because their hip torque was ~ 180 out of phase of the other subjects. However, their data were included in all statistical tests.

FIG. 3. Mean $\pm$ SE of peak moment and total joint work generated by the perturbed limb during the gait cycle. Data are from all incomplete SCI and nondisabled (ND) subjects. All values were normalized to bodyweight. *, significantly different from the active NO LOAD condition.

TABLE 2. Peak hip moments and total work

				NO LOAD Active	NO LOAD Passive	STANCE Active	STANCE Passive	SWING Active	SWING Passive
Incomplete SCI									
Peak flexion moment	6.51	8.5	0.000164	0.232 $\pm$ 0.284	0.075 $\pm$ 0.091	0.313 $\pm$ 0.367	0.251 $\pm$ 0.204	0.408 $\pm$ 0.368	0.368 $\pm$ 0.315
Peak extension moment	10.13	8.5	0.000003	-0.374 $\pm$ 0.220	-0.073 $\pm$ 0.082	-0.904 $\pm$ 0.477	-0.515 $\pm$ 0.229	-0.728 $\pm$ 0.500	-0.530 $\pm$ 0.273
Total flexion work	5.68	8.5	0.000477	0.070 $\pm$ 0.091	0.029 $\pm$ 0.036	0.095 $\pm$ 0.154	0.048 $\pm$ 0.069	0.150 $\pm$ 0.119	0.148 $\pm$ 0.135
Total extension work	12.01	8.5	0.000000	0.202 $\pm$ 0.121	0.029 $\pm$ 0.036	0.542 $\pm$ 0.287	0.340 $\pm$ 0.206	0.327 $\pm$ 0.292	0.168 $\pm$ 0.153
ND									
Peak flexion moment	15.19	9.5	0.000000	0.371 $\pm$ 0.114	0.068 $\pm$ 0.026	0.429 $\pm$ 0.139	0.318 $\pm$ 0.158	0.481 $\pm$ 0.171	0.297 $\pm$ 0.113
Peak extension moment	13.24	9.5	0.000000	-0.415 $\pm$ 0.237	-0.067 $\pm$ 0.041	-0.593 $\pm$ 0.380	-0.315 $\pm$ 0.153	-0.532 $\pm$ 0.280	-0.293 $\pm$ 0.133
Total flexion work	8.50	9.5	0.000010	0.176 $\pm$ 0.074	0.019 $\pm$ 0.010	0.149 $\pm$ 0.109	0.097 $\pm$ 0.093	0.218 $\pm$ 0.156	0.116 $\pm$ 0.069
Total extension work	6.87	9.5	0.000077	0.198 $\pm$ 0.206	0.018 $\pm$ 0.011	0.309 $\pm$ 0.275	0.155 $\pm$ 0.115	0.254 $\pm$ 0.235	0.112 $\pm$ 0.088
Complete SCI									
Peak flexion moment	—	—	—	0.015 $\pm$ 0.007	0.017 $\pm$ 0.005	0.214 $\pm$ 0.030	0.197 $\pm$ 0.054	0.392 $\pm$ 0.225	0.393 $\pm$ 0.225
Peak extension moment	—	—	—	-0.026 $\pm$ 0.004	-0.019 $\pm$ 0.005	-0.221 $\pm$ 0.105	-0.216 $\pm$ 0.112	-0.131 $\pm$ 0.046	-0.127 $\pm$ 0.039
Total flexion work	—	—	—	0.006 $\pm$ 0.003	0.007 $\pm$ 0.002	0.069 $\pm$ 0.001	0.069 $\pm$ 0.001	0.122 $\pm$ 0.038	0.101 $\pm$ 0.067

	F	df	P	NO LOAD Active	NO LOAD Passive	STANCE Active	STANCE Passive	SWING Active	SWING Passive
Total				0.013 ±	0.007 ±	0.103 ±	0.095 ±	0.063 ±	0.063 ±
extension work				0.007	0.002	0.078	0.088	0.056	0.056

Values are means ± SD. ND, nondisabled. Subject produced peak hip moments (Nm/kg) and total hip work (J/kg) for the loaded (ipsilateral) limb. *F* ratio, *df* and *P* values are given for each individual repeated-measure ANOVA that was run comparing the six conditions. Highlighted values are significantly different from the NO LOAD active condition.

The incomplete SCI subjects also had significant differences in peak hip extension moment (ANOVA, $P < 0.0001$), total hip extension work (ANOVA, $P < 0.0001$), peak hip flexion moment (ANOVA, $P < 0.0001$), and total hip flexion work (ANOVA, $P < 0.0001$) of the perturbed limb among the NO LOAD, STANCE, and SWING conditions (Figs. 2A and 3; Table 2). Post hoc testing demonstrated that the incomplete SCI subjects increased the amplitude of peak hip flexion and extension moments during the active NO LOAD condition compared with the passive NO LOAD condition (Newman-Keuls, $P < 0.05$; Table 2). When load was applied to the foot during the active STANCE condition, the incomplete SCI subjects significantly increased their ipsilateral peak hip extension moment (-0.904 ± 0.477 Nm/kg) by 142% and total hip extension work (0.542 ± 0.287 J/kg) by 168% compared with the active NO LOAD condition (-0.374 ± 0.220 Nm/kg and 0.202 ± 0.121 J/kg; Newman-Keuls, $P < 0.05$; Figs. 2A and 3; Table 2). During the *passive* STANCE condition, the incomplete SCI subjects increased both peak hip extension moment (-0.515 ± 0.229 Nm/kg) and total hip extension work (0.340 ± 0.206 J/kg) enough that they were not significantly different from the *active* NO LOAD condition (Newman-Keuls, $P > 0.05$; Table 2). When load was applied to the foot during the passive SWING condition, the incomplete SCI subjects significantly increased their ipsilateral total hip flexion work (0.148 ± 0.135 J/kg) compared with the active no load condition (0.070 ± 0.091 J/kg; Newman-Keuls, $P < 0.05$; Figs. 3; Table 2). The incomplete SCI subjects increased hip extension moments during the stance phase when load was applied during stance and increased hip flexion moments during the swing phase when load was applied during swing (Fig. 2A).

Ankle-foot loading also affected the ipsilateral hip moments of the complete SCI subjects. Subjects increased their mean peak hip

extension moment from -0.026 ± 0.004 Nm/kg during the NO LOAD condition to -0.221 ± 0.105 Nm/kg when load was applied to the ankle-foot during the STANCE condition (Fig. 4; Table 2). In addition, the complete SCI subjects had sizable changes in the peak flexion hip moment when load was applied to the ankle-foot during the SWING condition (0.393 ± 0.225 Nm/kg) compared with the NO LOAD condition (0.015 ± 0.007 Nm/kg) (Fig. 4; Table 2). The complete SCI subjects demonstrated no observable differences in hip moments between the active and passive trials during any of the three conditions.

FIG. 4. Mean gait cycle data from the perturbed limb of 2 complete SCI subjects. Each set of 3 graphs shows data from a single subject averaged from 8 to 16 steps. A: data recorded from complete SCI-10. B: data recorded from complete SCI-11.

In the nonperturbed limb, both the ND and incomplete SCI subjects had significant differences in peak hip extension moment (ANOVA, $P < 0.0001$) and total hip extension work (ANOVA, $P < 0.0001$) between conditions. However, post hoc testing revealed significant differences in these two hip extensor kinetic variables only between active and passive trials (Newman-Keuls, $P < 0.05$). For both extensor variables, the ND group had significantly greater responses

for the active NO LOAD condition (-0.832 ± 0.375 Nm/kg, 0.391 ± 0.217 J/kg) compared with all three passive conditions [NO LOAD (-0.046 ± 0.027 Nm/kg, 0.015 ± 0.008 J/kg), STANCE (-0.352 ± 0.0302 Nm/kg, 0.213 ± 0.188 J/kg), and SWING (-0.325 ± 0.203 Nm/kg, 0.145 ± 0.089 J/kg)]. For the incomplete SCI subjects, the peak hip extension moment and total hip extension work were both significantly greater during the active NO LOAD condition (-0.543 ± 0.371 Nm/kg, 0.248 ± 0.202 J/kg) than the passive NO LOAD condition (-0.122 ± 0.162 Nm/kg, 0.036 ± 0.043 J/kg). In addition, only the ND subjects had significant differences in peak hip flexion moment (ANOVA, $P < 0.0001$) and total hip flexion work (ANOVA, $P = 0.0027$) between the conditions. Post hoc testing indicated that differences were only significant between passive (passive NO LOAD, 0.046 ± 0.026 Nm/kg, 0.015 ± 0.008 J/kg) and active (active NO LOAD, 0.284 ± 0.172 Nm/kg, 0.106 ± 0.078 J/kg) conditions (Newman-Keuls, $P < 0.05$). The complete SCI subjects had no observable differences in hip kinetics in the nonperturbed limb between any of the stepping conditions.

EMG.

In general, the muscles directly loaded by the ankle-foot device (i.e., soleus and medial gastrocnemius) had the greatest changes in EMG amplitude between conditions. When load was applied to the ankle-foot during the active STANCE condition, the ND subjects significantly increased ipsilateral RMS EMG amplitude of the medial gastrocnemius by 250% (ANOVA, $P < 0.0001$; Newman-Keuls, $P < 0.05$) during the stance phase of the gait cycle compared with the EMG amplitudes during the active NO LOAD condition (Figs. 5B and 6A; Table 3). Similarly, the incomplete SCI subjects significantly increased ipsilateral RMS EMG amplitude of the soleus (ANOVA, $P < 0.0001$) by 179% and the medial gastrocnemius (ANOVA, $P = 0.0009$) by 81% during the stance phase of the gait cycle compared with the active NO LOAD condition (Newman-Keuls, $P < 0.05$; Figs. 5A and 6A; Table 3). When load was applied during the active STANCE condition, the incomplete SCI subjects also had significant increases in ipsilateral RMS EMG amplitude of the tibialis anterior (73% increase) during the stance phase (ANOVA, $P = 0.004$; Newman-Keuls, $P < 0.05$; Figs. 5A and 6A; Table 3).

Incomplete SCI subjects also demonstrated a significant 27% increase in gluteus maximus RMS EMG during the stance phase when load was applied during the active STANCE condition compared with the active NO LOAD condition (ANOVA, $P = 0.0004$; Newman-Keuls, $P < 0.05$; Fig. 7; Table 3).

FIG. 5. Mean rectified low-pass filtered electromyographic (EMG) data from all incomplete SCI (A) and all ND (B) subjects. EMG data were normalized to peak amplitude during the active NO LOAD condition. During the STANCE condition, ankle-foot load was applied before the point indicated by the vertical dashed line and after the dashed line during the SWING condition.

FIG. 6. Mean $\pm$ SE of the root mean square (RMS) EMG, corresponding to either the stance (A) or swing (B) phase of the gait cycle. Data were normalized to the active NO LOAD condition. *, significantly different from the active NO LOAD condition.

FIG. 7. Representative EMG, hip moment and hip joint angle from a single incomplete SCI subject, *SCI-13*, during the active NO LOAD and STANCE conditions. Gray bars during the STANCE condition indicate timing of applied ankle-foot load. EMG data were normalized to the active NO LOAD condition. When load was applied during stance, this subject demonstrated a phase appropriate increase in both gluteus maximus activity and hip flexion and extension muscle moment.

TABLE 3. Stance phase EMG

	<i>F</i>	<i>df</i>	<i>P</i>	NO LOAD Active	NO LOAD Passive	STANCE Active	STANCE Passive	SWING Active	SWING Passive
Incomplete SCI									
Soleus	12.41	8.5	0.000000	1.000 ± 0.000	0.750 ± 0.278	2.791 ± 1.022	1.919 ± 1.018	1.492 ± 0.608	1.185 ± 0.818
Medial gastrocnemius	5.17	8.5	0.000944	1.000 ± 0.000	0.728 ± 0.411	1.814 ± 0.809	1.205 ± 0.534	1.889 ± 1.177	1.631 ± 1.233
Tibialis anterior	4.14	8.5	0.004028	1.000 ± 0.000	0.546 ± 0.314	1.734 ± 0.995	0.974 ± 0.347	1.082 ± 0.324	0.816 ± 0.919
Vastus medialis	7.86	8.5	0.000031	1.000 ± 0.000	0.483 ± 0.355	1.057 ± 0.261	0.579 ± 0.469	1.422 ± 0.384	0.803 ± 0.591
Vastus Lateralis	4.74	8.5	0.001705	1.000 ± 0.000	0.467 ± 0.277	1.500 ± 1.198	0.824 ± 1.140	1.653 ± 0.823	0.904 ± 0.679

		F	df	P	NO LOAD Active	NO LOAD Passive	STANCE Active	STANCE Passive	SWING Active	SWING Passive
Rectus femoris		4.88	8.5	0.001409	1.000 ± 0.000	0.822 ± 0.458	1.254 ± 0.298	0.886 ± 0.311	1.300 ± 0.537	1.039 ± 0.557
	Medial hemstrings	0.82	8.5	0.544955	1.000 ± 0.000	1.195 ± 1.123	1.273 ± 0.631	1.039 ± 0.626	1.295 ± 0.468	0.982 ± 1.425
	Gluteus maximus	11.16	5.3	0.000417	1.000 ± 0.000	0.661 ± 0.222	1.266 ± 0.431	0.708 ± 0.245	—	—
ND										
Soleus		4.69	9.5	0.001577	1.000 ± 0.000	0.549 ± 0.349	2.230 ± 1.420	1.054 ± 0.693	2.831 ± 1.975	1.839 ± 2.475
	Medial gastrocnemius	13.39	9.5	0.000000	1.000 ± 0.000	0.359 ± 0.423	3.503 ± 1.762	0.782 ± 0.673	1.938 ± 1.226	0.491 ± 0.577
	Tibialis anterior	10.57	9.5	0.000001	1.000 ± 0.000	0.188 ± 0.133	0.934 ± 0.435	0.322 ± 0.287	1.222 ± 1.091	0.288 ± 0.216
Vastus medialis		6.71	9.5	0.000095	1.000 ± 0.000	0.326 ± 0.185	1.725 ± 1.534	0.707 ± 0.615	1.652 ± 0.780	0.657 ± 0.813
	Vastus lateralis	10.80	9.5	0.000001	1.000 ± 0.000	0.475 ± 0.266	1.152 ± 0.653	0.607 ± 0.377	1.570 ± 0.797	0.497 ± 0.259
	Rectus femoris	1.63	9.5	0.171437	1.000 ± 0.000	0.746 ± 0.241	1.430 ± 1.013	1.329 ± 1.122	1.518 ± 1.345	1.428 ± 1.255
Medial hamstrings		6.38	9.5	0.000148	1.000 ± 0.000	0.200 ± 0.187	1.163 ± 0.618	0.308 ± 0.233	1.373 ± 1.386	0.330 ± 0.458
Complete SCI										
Soleus		—	—	—	1.000 ± 0.000	1.031 ± 0.044	1.645 ± 1.505	1.315 ± 1.038	1.484 ± 0.347	1.421 ± 0.258
	Medial gastrocnemius	—	—	—	1.000 ± 0.000	1.007 ± 0.010	1.950 ± 0.544	1.669 ± 0.147	1.828 ± 0.854	1.835 ± 0.844
	Tibialis anterior	—	—	—	1.000 ± 0.000	0.928 ± 0.101	2.194 ± 1.787	2.818 ± 2.669	0.808 ± 0.031	0.805 ± 0.036
Vastus medialis		—	—	—	1.000 ± 0.000	1.018 ± 0.025	0.585 ± 0.147	0.586 ± 0.150	1.035 ± 0.071	1.047 ± 0.089
	Vastus lateralis	—	—	—	1.000 ± 0.000	0.969 ± 0.044	0.637 ± 0.122	0.634 ± 0.126	0.953 ± 0.180	0.967 ± 0.199
	Rectus femoris	—	—	—	1.000 ± 0.000	0.995 ± 0.07	2.622 ± 1.771	2.609 ± 1.752	1.521 ± 0.363	1.471 ± 0.292
Medial hamstrings		—	—	—	1.000 ± 0.000	1.005 ± 0.007	1.056 ± 0.703	1.018 ± 0.649	1.686 ± 0.746	1.645 ± 0.804

Values are means ± SD. Root mean square (RMS) electromyographic (EMG) data for the loaded (ipsilateral) limb occurring during the stance phase of the step cycle. Values are normalized to the no load active condition. *F* ratio, *df* and *P* values are given for each individual repeated-measure ANOVA that was run comparing the six conditions. Highlighted values are significantly different from the NO LOAD active condition.

When load was applied to the ankle-foot during the active SWING condition, ND subjects had significant increases in ipsilateral soleus (ANOVA, *P* < 0.0001; 194% increase) and medial gastrocnemius (ANOVA, *P* = 0.0036; 252% increase) during the swing phase compared with the active NO LOAD condition (Newman-Keuls, *P* < 0.05; Figs. 5B and 6B; 6B; Table 4). In addition, during the

active SWING condition ND subjects significantly increased ipsilateral soleus (ANOVA, $P = 0.0015$; 183% increase), medial gastrocnemius (ANOVA, $P < 0.0001$; 94% increase), vastus medialis (ANOVA, $P < 0.0001$; 65% increase), and vastus lateralis (ANOVA, $P < 0.0001$; 57% increase) during the stance phase compared with the active NO LOAD condition (Newman-Keuls, $P < 0.05$; Figs. 5B and and6A;6A; Table 3). The incomplete SCI subjects also had significant increases in ipsilateral soleus (ANOVA, $P = 0.0002$; 308% increase) during the swing phase compared with the active NO LOAD condition (Newman-Keuls, $P < 0.05$; Figs. 5A and and6B,6B, Table 4). During the stance phase, the incomplete SCI subjects had significant increases in ipsilateral medial gastrocnemius (ANOVA, $P = 0.0009$; 89% increase) when load was applied during the active SWING condition compared with the active NO LOAD condition (Newman-Keuls, $P < 0.05$; Fig. 5A and and6A,6A, Table 3).

TABLE 4. Swing phase EMG

	<i>F</i>	<i>df</i>	<i>P</i>	NO LOAD Active	NO LOAD Passive	STANCE Active	STANCE Passive	SWING Active	SWING Passive
Incomplete SCI									
Soleus	6.22	8.5	0.000234	1.000 ± 0.000	0.832 ± 0.202	1.939 ± 1.451	1.599 ± 1.043	4.081 ± 3.140	3.813 ± 2.524
Medial gastrocnemius	3.13	8.5	0.017626	1.000 ± 0.000	0.774 ± 0.368	1.249 ± 0.231	1.280 ± 0.444	2.732 ± 2.737	2.462 ± 2.264
Tibialis anterior	0.80	8.5	0.55879	1.000 ± 0.000	0.627 ± 0.337	1.594 ± 0.963	1.575 ± 2.178	1.464 ± 0.927	1.016 ± 0.682
Vastus medialis	3.07	8.5	0.019371	1.000 ± 0.000	0.765 ± 0.430	1.211 ± 0.383	0.900 ± 0.488	1.387 ± 0.636	1.189 ± 0.723
Vastus lateralis	2.13	8.5	0.082063	1.000 ± 0.000	0.701 ± 0.472	1.578 ± 0.776	1.055 ± 0.841	1.203 ± 0.664	1.186 ± 0.790
Rectus femoris	1.11	8.5	0.371182	1.000 ± 0.000	0.772 ± 0.302	1.113 ± 0.319	0.971 ± 0.593	1.098 ± 0.222	0.954 ± 0.387
Medial hamstrings	0.67	8.5	0.645492	1.000 ± 0.000	1.264 ± 1.017	1.115 ± 0.337	1.053 ± 0.472	1.131 ± 0.347	1.069 ± 0.389
Gluteus maximus	11.35	5.3	0.000383	1.000 ± 0.000	0.660 ± 0.206	1.126 ± 0.256	0.738 ± 0.241	—	—
ND									
Soleus	12.62	9.5	0.000000	1.000 ± 0.000	0.498 ± 0.223	1.358 ± 1.150	0.819 ± 0.622	2.947 ± 0.940	2.354 ± 1.439
Medial gastrocnemius	4.21	9.5	0.003627	1.000 ± 0.000	0.442 ± 0.283	1.465 ± 0.594	0.696 ± 0.463	3.527 ± 3.845	1.067 ± 0.708
Tibialis anterior	8.84	9.5	0.000007	1.000 ± 0.000	0.157 ± 0.149	1.630 ± 0.609	0.380 ± 0.308	1.309 ± 0.827	0.767 ± 1.201
Vastus medialis	3.80	9.5	0.005899	1.000 ± 0.000	0.250 ± 0.131	1.060 ± 0.568	0.276 ± 0.137	2.246 ± 2.392	1.322 ± 2.370
Vastus lateralis	14.81	9.5	0.000000	1.000 ± 0.000	0.340 ± 0.225	0.868 ± 0.316	0.395 ± 0.272	1.105 ± 0.557	0.417 ± 0.321

	F	df	P	NO LOAD Active	NO LOAD Passive	STANCE Active	STANCE Passive	SWING Active	SWING Passive
Rectus femoris	1.60	9.5	0.179948	1.000 ± 0.000	0.614 ± 0.443	0.949 ± 0.436	0.844 ± 0.758	1.718 ± 2.335	1.974 ± 3.045
Medial hamstrings	8.75	9.5	0.000007	1.000 ± 0.000	0.328 ± 0.245	1.220 ± 0.517	0.364 ± 0.172	1.204 ± 0.642	0.651 ± 0.703
Complete SCI									
Soleus	—	—	—	1.000 ± 0.000	0.967 ± 0.047	3.237 ± 2.567	3.052 ± 2.304	4.833 ± 3.875	4.773 ± 3.790
Medial gastrocnemius	—	—	—	1.000 ± 0.000	0.984 ± 0.023	2.228 ± 0.999	2.092 ± 0.808	2.586 ± 2.285	2.590 ± 2.291
Tibialis anterior	—	—	—	1.000 ± 0.000	1.057 ± 0.080	1.680 ± 1.070	2.356 ± 2.026	4.531 ± 2.650	4.497 ± 2.698
Vastus medialis	—	—	—	1.000 ± 0.000	1.242 ± 0.342	1.159 ± 0.300	1.135 ± 0.334	5.122 ± 4.431	3.952 ± 2.777
Vastus lateralis	—	—	—	1.000 ± 0.000	0.916 ± 0.119	0.995 ± 0.310	0.956 ± 0.365	1.288 ± 0.203	1.179 ± 0.048
Rectus femoris	—	—	—	1.000 ± 0.000	0.997 ± 0.005	1.069 ± 0.041	1.076 ± 0.031	1.043 ± 0.306	1.047 ± 0.300
Medial hamstrings	—	—	—	1.000 ± 0.000	0.880 ± 0.170	1.592 ± 0.664	1.531 ± 0.577	1.458 ± 1.086	1.512 ± 1.163

Values are means ± SD. RMS EMG data for the loaded (ipsilateral) limb occurring during the swing phase of the step cycle. Values are normalized to the NO LOAD active condition. *F* ratio, *df* and *P* values are given for each individual repeated-measure ANOVA that was run comparing the six conditions. Highlighted values are significantly different from the NO LOAD active condition.

In the nonperturbed limb, ANOVAs revealed significant differences in muscle activity between conditions for the ND subjects (ANOVA, *P* < 0.05). Post hoc testing revealed a significant increase in RMS EMG of the soleus (52% increase) and medial gastrocnemius (36% increase) during the stance phase when load was applied to the contralateral limb during the active STANCE condition compared with the active NO LOAD condition (Newman-Keuls, *P* < 0.05). All other differences in RMS EMG activity of the nonperturbed limb occurred between active and passive trials and did not vary by load condition. In the incomplete SCI subjects ANOVA's revealed significant differences in muscle activity between conditions in the nonperturbed limb (ANOVA, *P* < 0.05). However, post hoc testing showed that all differences in RMS EMG activity of the nonperturbed limb occurred only between active and passive trials and did not vary by load condition.

The complete SCI subjects had no observable differences in EMG activity between the active and passive conditions. When ankle-foot load was applied during both the STANCE and SWING conditions

the complete subjects increased soleus, medial gastrocnemius, and tibialis anterior activity during the period of application (Fig. 8). For example, tibialis anterior RMS EMG activity increased an average of 119% during the stance phase of the STANCE condition and 353% during the swing phase of the SWING condition. Of note, during the STANCE condition, the complete subjects increased RMS EMG activity of rectus femurs by 162% during the stance phase and increased medial hamstrings activity by 69% during the swing phase.

FIG. 8. Representative EMG, hip moment, and hip joint angle from a single complete SCI subject (SCI-10) during the active NO LOAD and STANCE conditions. Gray bars during the STANCE condition indicate timing of applied ankle-foot load. EMG data were normalized to the active NO LOAD condition. When load was applied during STANCE, this subject demonstrated an increase in both soleus and tibialis anterior activity during the time of applied load. While both flexion and extension hip moments increased, their direction was out of phase of the direction of joint movement.

Temporal response to STANCE, EARLY, and LATE

The phasing of ankle-foot loading within the gait cycle affected the relative phasing of ipsilateral hip moments in SCI but not ND subjects. During the active EARLY condition, the applied ankle torque profile occurred 182 ± 21 ms (7.9% of the gait cycle) and 188 ± 7 ms (8.2% of the gait cycle) earlier in the gait cycle than during the active STANCE condition for the ND and incomplete SCI subjects, respectively (Fig. 9). As well, during the active EARLY condition, incomplete SCI ipsilateral hip moment profiles occurred 108 ± 118 ms (4.7% of the gait cycle) earlier in the gait cycle when compared with the active STANCE condition. (ANOVA, $P = 0.0037$; Newman-Keuls, $P < 0.05$). Conversely, during the active LATE condition, applied ankle torque occurred 148 ± 37 ms (6.5% of the gait cycle) later in the gait cycle for ND and 186 ± 55 ms (8.1% of the gait cycle) later for the incomplete SCI subjects than during the active STANCE condition (Fig. 9). In incomplete SCI subjects the phasing of their ipsilateral hip moment profile occurred 95 ± 56 ms (4.1% of the gait cycle) later in the gait cycle (Newman-Keuls, $P < 0.05$) during the active LATE condition compared with active STANCE. The ND subjects showed trends to shift hip moment profiles in the direction of the temporal variations in applied ankle torque, however none of these changes were significant (ANOVA, $P = 0.1658$; Fig. 9). In the contralateral (nonperturbed) limb, incomplete SCI subjects had no significant changes in hip moment phasing among the three ankle-foot loading conditions (ANOVA, $P = 0.9263$). In contrast, ND subjects significantly changed the phasing of their contralateral hip moment to occur 94 ± 112 ms earlier in the gait cycle during the active EARLY condition compared with active STANCE (ANOVA, $P = 0.0032$; Newman-Keuls, $P < 0.05$).

FIG. 9. Top 3 figures, representative hip moment and applied ankle torque profiles from incomplete SCI subject SCI-2 (A), a ND subject (B), and complete SCI subject SCI-11 (C). Each figure shows mean data from 8 to 16 during the active EARLY, STANCE, and LATE conditions. D, bottom 2 figures: the calculated phase shift in applied ankle torque and hip moment profile within the gait cycle when compared with the active STANCE condition. Data shown are group means $\pm$ SE. Negative values indicate the profile is shifted earlier in the gait cycle. *, significantly different from the STANCE condition.

The step-to-step temporal variability in hip moments among the EARLY, STANCE, and LATE conditions for the complete SCI subjects was large, making generalizations difficult for this group. However, qualitatively it appeared that one complete SCI subject shifted the phasing of hip flexion-to-extension to correspond with the onset of ankle-foot loading (Fig. 9). The other complete SCI subject showed no observable shifts in the hip moment phasing between conditions.

Discussion

Response to Ankle-foot load applied during STANCE or SWING

We observed a complex multijoint response to ankle-foot load feedback during gait. Specifically, both SCI and ND subjects

substantially increased hip extension torque during the stance phase of the gait cycle when they received concurrent ankle-foot load. This finding was consistent with previous animal research reporting that 20–50% of the extensor muscle activity occurring during locomotion is regulated by ankle muscle load afferents (Donelan and Pearson 2004; Hiebert and Pearson 1999). In addition, we observed an enhancement of extensor muscle activity in muscles that were directly loaded by the applied ankle-foot torque (soleus and gastrocnemius) as well as in muscles that were not perturbed (gluteus maximus). Because these changes in muscle activation occurred throughout the leg, this finding suggests that the efferent response to ankle-foot load is mediated by interneuronal pathways. This potential mechanism agrees with reduced animal locomotion models, indicating that during the extension phase of gait, ankle extensor group I afferents act directly on spinal level central pattern generators to excite extensor motoneurons (Conway et al. 1987; Gossard et al. 1994). Our observation that complete SCI subjects increased hip extensor torque in response to ankle-foot load further supports the possibility that in humans, ankle-foot load afferents act directly on spinal level neural circuitry to modulate locomotor patterns.

SCI subjects increased hip flexion torque when ankle-foot load was applied during the swing phase. Similar responses have been observed in seated SCI subjects, who exhibit a hip flexion response to both electrical (Hornby et al. 2004) and mechanical (Schmit et al. 2002) stimulation of the ankle plantar flexor afferents. This hip flexion reaction is a reversal of the extension response we observed when subjects received stance phase ankle-foot load, suggesting that during human locomotion, the effect of ankle-foot load is phase specific. This modulation is consistent with previous observations of phase-dependent reflex reversals occurring in the lower limb muscles during human walking (Duysens et al. 1990, 1992; Yang and Stein 1990). In addition, our results imply that the observed flexion response is spinally mediated (indicated by a sizable reaction in complete SCI subjects) and modulated by descending input (indicated by no significant changes in ND subjects).

Both ND and SCI subjects demonstrated select increases in muscle activation that occurred out of phase with the timing of ankle-foot loading. For example, the ND subjects had significant increases in

soleus, gastrocnemius, vastus medialis, and vastus lateralis EMG activity during the stance phase of gait when load was applied only during the swing phase. It is of interest that this out-of-phase muscle activity occurred during stance when load was applied during swing phase but not during swing when load was applied during stance phase. Other research has shown that neurologically intact humans will initially respond to novel locomotor perturbations by increasing lower limb muscle activity throughout the gait cycle (Gordon and Ferris 2007). This locomotor strategy likely increases joint impedance and ultimately walking stability (Duan et al. 1997; van Soest et al. 2003). In the current study, it is possible that the peculiarity of applying ankle-foot load during swing resulted in subjects adapting a locomotor pattern that emphasized gait stability.

Response to temporal modulations in ankle-foot load

Studies on human infants (Pang and Yang 2000; Yang et al. 1998b) and cats (Guertin et al. 1995; Pearson et al. 1992; Whelan et al. 1995) have shown that modulating the timing of limb load during stepping will influence the onset and duration of stance and swing phases. Similarly, in the current study, SCI subjects shifted the timing of hip moment profiles within the gait cycle corresponding to temporal modulations of applied ankle-foot load. When ankle-foot load was applied earlier in the gait cycle, SCI subjects shifted the relative timing of their hip moment patterns to occur earlier within the gait cycle. Conversely, SCI subjects shifted their hip moment patterns later in the gait cycle when the timing of ankle-foot load was applied later than normal. These phase shifts in hip moment profiles are analogous to the changes in stance-to-swing and swing-to-stance timing observed in previous studies. Of importance is that the temporal changes in hip moment were not phase locked to the temporal changes in applied ankle-foot loading. For example, in SCI subjects, when the applied ankle-foot load occurred later in the gait cycle, the transition of hip moment (from extension-to-flexion) in late stance tended to precede the removal of ankle-foot load. In this case, delaying ankle-foot load removal delayed but did not prevent the transition of hip moments from extension to flexion. This finding suggests that other factors, such as hip position feedback (Grillner and Rossignol 1978) and feed forward control (Lam et al. 2006), likely play a role in modulating the

timing of hip torque production. In contrast to SCI subjects, ND subjects demonstrated no significant temporal response to small shifts in stance load timing, indicating that descending input can potentially override the locomotor response to ankle-foot load afferents.

In human infants, unloading of the plantar flexor muscles as occurs at the end of stance is a trigger for hip flexion (Pang and Yang 2000; Yang et al. 1998b). As such, we expected an enhanced hip flexion torque when ankle-foot load was released at the end of the stance phase. However, unlike the increase in extensor hip moments we observed when ankle-foot load was applied during the stance phase, removal of this load did not result in an increase in hip flexion moments compared with the no load condition. It is possible that we did not see an enhancement in hip flexion moment because the major afferent signal driving hip flexion magnitude is not limb load release. In SCI subjects, limb load release may only modulate temporal aspects of hip flexion torque. As mentioned earlier, stimulation of the load-sensitive afferents of the ankle extensor muscles has been shown to inhibit the onset of hip flexion (Guertin et al. 1995; Pearson et al. 1992; Whelan et al. 1995), which could explain why we saw a modulation in the timing of hip moments occurring with temporal shifts of ankle-foot load but not an enhancement in hip flexion torque with load release. Stretch of the hip flexors as occurs at the end of stance may be the major afferent signal driving hip flexion (Grillner and Rossignol 1978). In this experiment, hip kinematics were rigidly controlled by the Lokomat, and as a result, hip stretch at the end of stance was consistent between all stepping conditions. It is possible that during unconstrained stepping, the enhancement of subject-produced hip extension torque with ankle-foot loading could result in actual kinematic changes (i.e., increase hip extension at the end of stance), which in turn might produce greater hip flexor muscle stretch and ultimately increased hip flexion torque production.

Contralateral response to ankle-foot load

In the current study, we applied ankle-foot load unilaterally. Investigating locomotor modulations in the contralateral limb may provide insight into the regulatory role of afferent feedback on interlimb coordination. Previous studies examining stepping patterns in

humans with complete SCI (Ferris et al. 2004; Kawashima et al. 2005) and human infants (Pang and Yang 2001; Yang et al. 1998b) have shown that movement of one limb can influence contralateral limb stepping patterns. For example, during infant stepping when swing is prolonged in one limb the contralateral leg will prolong stance (Yang et al. 1998b). These studies suggest that spinal neuronal networks use bilateral afferent signals to regulate muscle activity during gait. Thus we might anticipate that subjects in the current study would modulate locomotor activity in both ipsi- and contralateral limbs to unilateral ankle-foot load. However, this was not always the case. SCI subjects demonstrated no significant changes in the contralateral limb between loading conditions. Our results were consistent with past work showing that when complete SCI subjects performed bilateral stepping with unilateral limb loading (similar to the stance condition in the current study) little to no EMG activity was observed in the nonloaded limb (Ferris et al. 2004). However, in contrast to the SCI subjects, the ND subjects in the current study demonstrated significant changes in the contralateral limb. ND subjects increased contralateral ankle extensor EMG activity during the stance phase when the ipsilateral limb was loaded during stance. Thus the increase in the contralateral limb extensor EMG activity was both timed appropriately to the gait cycle (during stance) and occurred out of phase with the timing of load application of the contralateral limb. In addition, when unilateral limb load occurred earlier in the gait cycle than normal, ND subjects made a corresponding shift in the timing of their contralateral hip moment profile. Dietz et al. proposed that interlimb coordination depends on supraspinal input, based on their findings that ND subjects, but not complete SCI, modulate EMG activity in a nonmoving limb when the contralateral limb performs stepping (Dietz et al. 2002). However, evidence from human infant (Yang et al. 1998b) and spinalized cat (Hiebert et al. 1994) stepping studies suggest that the spinal cord has the ability to modulate contralateral limb locomotor activity in response to changes in limb load. Thus it is possible that other factors such as slow walking speed or the amount of previous locomotor training (Ferris et al. 2004) may have limited the effects of interlimb modulations observed in SCI subjects in this study.

Volitional influence on feedback modulation of gait

Understanding how volitional drive influences sensory feedback modulation during gait may be valuable for developing successful rehabilitation strategies for individuals with incomplete SCI who possess some descending control. It has been shown that with operant conditioning, rats with partial SCI can learn to use descending drive to regulate soleus H-reflex gains in a manner that can influence and possibly improve locomotor performance (Chen et al. 2005, 2006). Similar operant conditioning protocols have been used in human SCI subjects to downregulate hyperactive spinal stretch reflexes in the upper limb (Segal and Wolf 1994) and suggest that such training may be valuable for improving motor performance. Furthermore, in humans with incomplete SCI, corticospinal tract function is highly correlated with walking ability (Thomas and Gorassini 2005), suggesting that descending drive may be a factor in determining locomotor function. In the current study, we examined the efferent response to ankle-foot loading when subjects performed stepping movements both passively and actively. Differences in the efferent response between active and passive conditions may give an indication of the influence of volitional drive on load related reflex modulations during gait. During the no load condition, incomplete SCI subjects produced greater peak flexion and extension hip moments when they actively stepped compared with the passive stepping, indicating that subjects could use volitional drive to modulate locomotor patterns. Interestingly, in incomplete SCI subjects there were no differences in hip moments produced between the active no load and the passive stance condition. This finding suggests that in incomplete SCI subjects both volitional drive and afferent feedback can contribute substantially to the underlying locomotor patterns. Further, the combined effect of active stepping while receiving ankle-foot load during the stance condition resulted in greater hip extension moments than when stepping with either component (volitional drive or limb load feedback) alone. This finding indicates that incomplete SCI subjects can use descending drive to modulate their efferent response to sensory feedback and supports current gait rehabilitation methods stressing active patient participation (Behrman et al. 2006).

Study limitations

One potential shortcoming of this study is that the method we used to calculate hip moments may underestimate the actual active (i.e., muscle) component of the moment. We made an estimate of external joint moments by creating an average profile of hip torque during the passive no load condition and then subtracted this value from the total hip torque produced during the other conditions, providing an estimate of active joint moment. Errors could occur if the limb dynamics were not consistent from step to step or if subjects were not completely passive during the baseline conditions. Errors resulting from changes in limb dynamics were likely small because limb kinematics were controlled. However, we did observe low-level rhythmic EMG activity during the passive no load condition in some SCI and ND subjects and, as a result, potentially underestimated the true active moment in some subjects. Although this method may have underestimated the actual moment, it still provided a reasonable means for investigating the relative changes in hip moments between different ankle-foot loading conditions.

Controlling inter- and intrasubject sensory feedback variability during stepping is very challenging. In this study, we sought to examine changes in locomotor patterns with isolated changes in ankle-foot load. We used a Lokomat to prescribe a set kinematic trajectory at the hip and knee joints because of the known importance of limb kinematic feedback in regulating locomotor patterns in human SCI subjects (Beres-Jones and Harkema 2004; Ferris et al. 2004; Kawashima et al. 2005). However, controlling stepping kinematics necessitated applying a torque to the subjects' limbs any time their movement differed from that of the Lokomat. For example, if a subject was able to match the Lokomat movements perfectly during the no load condition, an increase in hip extension torque, as was regularly observed during the STANCE condition, would result in the subject accelerating their thigh against the Lokomat thigh cuff. To maintain kinematics, a braking force would be applied to the subject's thigh at the Lokomat interface. The forces applied to maintain kinematics activated skin afferents in a pattern that was not consistent between conditions (as evidenced by differences in subjects' hip moments) and may have influenced the locomotor performance. While the effect of

cutaneous stimulation along the surface of the thigh and shank has not been clearly established during human walking, it has been suggested to influence locomotor patterns in human SCI populations (Behrman and Harkema 2000). In incomplete SCI subjects, using the Lokomat to resist swing phase movements has been shown to enhance flexor muscle activity (Lam et al. 2008). As well, seated SCI subjects demonstrate an invariant flexion response to cutaneous stimulation regardless of stimulus location on the lower limb (Schmit et al. 2003). If the cutaneous stimulation of the thigh and shank during walking do indeed create a flexion response in spinal cord injury subjects, this may have potentially decreased the magnitude of the major finding of the current study, that SCI subjects increase hip extension torque when the limb is loaded during stance phase.

Conclusions

Our results indicate that humans use ankle-foot load afferents to modulate the amplitude and timing of locomotor patterns in a phase-dependent manner. Specifically, both ND and SCI subjects substantially increased hip extension moments during stance when the ankle-foot was loaded during stance. This work provides significant new information about how the human nervous system uses ankle-foot load afferents to regulate hip activity during gait. Moments produced at the hip during walking have not been well investigated in human SCI. The findings from this study suggest that sensory modulation of hip moments may be a major determinant of walking ability in SCI individuals. This new information on the regulatory role of ankle-foot load afferents and hip moment modulation during walking may be valuable for improving gait rehabilitation.

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Notes

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