

Human Lumbosacral Spinal Cord Interprets Loading During Stepping

SUSAN J. HARKEMA,³ SEANNA L. HURLEY,³ UDAY K. PATEL,³ PHILIP S. REQUEJO,³
BRUCE H. DOBKIN,^{1,3} AND V. REGGIE EDGERTON^{2,3}

¹Departments of Neurology and ²Physiological Science and ³The Brain Research Institute, University of California, Los Angeles, California 90095

Harkema, Susan J., Seanna L. Hurley, Uday K. Patel, Philip S. Requejo, Bruce H. Dobkin, and V. Reggie Edgerton. Human lumbosacral spinal cord interprets loading during stepping. *J. Neurophysiol.* 77: 797–811, 1997. Studies suggest that the human lumbosacral spinal cord can generate steplike oscillating electromyographic (EMG) patterns, but it remains unclear to what degree these efferent patterns depend on the phasic peripheral sensory information associated with bilateral limb movements and loading. We examined the role of sensory information related to lower-extremity weight bearing in modulating the efferent motor patterns of spinal-cord-injured (SCI) subjects during manually assisted stepping on a treadmill. Four nonambulatory subjects, each with a chronic thoracic spinal cord injury, and two nondisabled subjects were studied. The level of loading, EMG patterns, and kinematics of the lower limbs were studied during manually assisted or unassisted stepping on a treadmill with body weight support. The relationships among lumbosacral motor pool activity [soleus (SOL), medial gastrocnemius (MG), and tibialis anterior (TA)], limb load, muscle-tendon length, and velocity of muscle-tendon length change were examined. The EMG mean amplitude of the SOL, MG, and TA was directly related to the peak load per step on the lower limb during locomotion. The effects on the EMG amplitude were qualitatively similar in subjects with normal, partial, or no detectable supraspinal input. Responses were most consistent in the SOL and MG at load levels of <50% of a subject's body weight. The modulation of the EMG amplitude from the SOL and MG, both across steps and within a step, was more closely associated with limb peak load than muscle-tendon stretch or the velocity of muscle-tendon stretch. Thus stretch reflexes were not the sole source of the phasic EMG activity in flexors and extensors during manually assisted stepping in SCI subjects. The EMG amplitude within a step was highly dependent on the phase of the step cycle regardless of level of load. These data suggest that level of loading on the lower limbs provides cues that enable the human lumbosacral spinal cord to modulate efferent output in a manner that may facilitate the generation of stepping. These data provide a rationale for gait rehabilitation strategies that utilize the level of load-bearing stepping to enhance the locomotor capability of SCI subjects.

INTRODUCTION

In the absence of supraspinal input, the adult cat spinal cord can generate efferent motor patterns that enable independent stepping (Barbeau and Rossignol 1987; Lovely et al. 1986). The oscillating neural circuitry responsible for this task (Grillner and Zangger 1979) is highly dependent on peripheral modalities associated with stepping such as weight bearing and joint position (de Guzman et al. 1991; Forssberg 1979; Grillner and Dubuc 1988; Lovely et al. 1990). The neural pathways in the lumbosacral spinal cord of the cat respond to sensory information specifically associated with locomotion to generate the efferent motor patterns necessary to execute stepping (Edgerton et al. 1996).

Whether similar locomotor neural circuits are present in humans has not been determined, although it now appears that at least some of the circuitry necessary to generate fictive locomotion has been conserved in nonhuman primates (Hultborn et al. 1993).

Spinal-cord-injured (SCI) humans with minimal supraspinal input generated motor patterns that improved their locomotor ability (Barbeau and Blunt 1991; Dietz et al. 1995; Wernig and Müller 1992). SCI subjects with no supraspinal input below their lesions can generate oscillating motor patterns in the lower limbs without the sensations of weight-bearing stepping (Bussel et al. 1988; Rosenfeld et al. 1995) and can generate steplike efferent patterns when partially supported over a moving treadmill belt and manually assisted with stepping (Dietz et al. 1995; Dobkin et al. 1995). These studies suggest that the human lumbosacral spinal cord can generate steplike oscillating electromyographic (EMG) patterns when assisted by the sensory information associated with weight-bearing stepping. It remains unclear, however, to what degree these efferent patterns depend on the phasic peripheral sensory information associated with bilateral limb movements and loading.

The aim of this study was to assess the role of sensory information related to lower-extremity weight bearing in modulating the efferent motor patterns of SCI subjects when provided with body weight support during manually assisted stepping on a treadmill. The effect on motor pool output of varying the load on the lower limbs during stepping was compared among SCI subjects who had minimal or no detectable sensorimotor function below the level of lesion and neurally intact subjects.

METHODS

Subject population

Two nondisabled (ND) and four chronic SCI subjects were studied. SCI subjects were classified with the use of the American Spinal Injury Association (ASIA) impairment scale, which categorizes individuals by their sensorimotor function below the level of lesion and designates a cumulative motor score (Ditunno et al. 1994) (Table 1). In brief, ASIA classifies individuals as follows: A, no sensory or motor function below the lesion including the sacral segments S₄–S₅; B, sensory but not motor function is preserved below the neurological level; and C, motor function is preserved below the neurological level but there is no active movement against gravity. SCI subjects in this study could not voluntarily stand or step over ground. Their injuries were at the thoracic level (Table 1) and a result of traumatic injury (*SCI-A1*, *SCI-A2*, and *SCI-B1*) or ischemia (*SCI-C1*). Sensory evoked potentials were recorded from the popliteal fossa, lumbar spine, and scalp (vertex positive peak) with the use of unilateral and bilateral posterior tibial

TABLE 1. *Clinical characteristics of SCI subjects*

Subject	Age, yr	Weight, kg	Postinjury, yr	Injury Level	ASIA		SEP
					Grade	Motor score	
SCI-A1	39	74	2	T ₇	A	0	Absent
SCI-A2	39	68	11	T ₉	A	0	Absent
SCI-C1	43	96	2	T ₈	C	R = 8, L = 0	Present
SCI-B1	59	68	2	T ₄	B	0	Present

SCI, spinal-cord-injured; ASIA, American Spinal Injury Association; SEP, somatosensory evoked potential; R, right; L, left.

nerve electrical stimulation (Table 1). No response confirmed the absence of detectable supraspinal conductivity to the lower limbs in the ASIA A subjects. Subjects took no medications during these studies. Informed consent was obtained from each subject and the experiments were approved by the University of California at Los Angeles Human Subjects Protection Committee.

Experimental procedures

Soleus (SOL), medial gastrocnemius (MG), and tibialis anterior (TA) EMG activity, ankle and knee joint angles, level of body weight support, and amount of lower limb loading were measured during stepping on a treadmill. All subjects wore a harness connected to an overhead motorized lift that allowed adjustment of lower limb loading. Body weight load (BWL) on the lower extremities of ND subjects ranged from no limb loading (0%) to full loading (100%). Loading of SCI subjects ranged from no limb loading to the maximum loading level at which knee flexion during stance could be avoided (50–80% BWL). The different loads used for each sequence of steps were randomly ordered. SCI subjects were assisted as necessary during stepping. Trainers held each leg distal to the patella to assist with knee extension during stance and distal to the ankle to assist with swing and foot placement. A third person stabilized the pelvis. ND subjects stepped unassisted.

EMG activity was recorded with the use of surface electrodes. Bipolar surface EMG electrodes were placed on the following muscles bilaterally: SOL, distal to the gastrocnemius muscle belly and medial to the Achilles tendon; MG, below the popliteal crease; and TA, below the tibial tuberosity and lateral to the tibial crest. Knee and ankle angles were measured by electrogoniometers; foot contact was detected with the use of 2.5 cm² pressure-sensitive switches (footswitch) placed on the heel, two metatarsals (1st and 5th), and toe; and body weight support was measured by a force transducer attached to the suspension apparatus.

Data acquisition

A 24-channel hard-wired system (Konigsberg Instruments, Pasadena, CA) was used to acquire EMG, electrogoniometer, footswitch, and force transducer signals. EMG data from the SOL, MG, and TA were sampled from 0.1 to 1 kHz and AC coupled into a differential amplifier. The signals were pulse-interval modulated to a fiberoptic transmitter and relayed to decoding electronics. All analog signals were digitally sampled at 1 kHz (National Instruments, Austin, TX).

Limb load was measured for 20 s during each load condition by shoe insert pressure sensors with the use of a computerized measurement device (TEKSCAN, Boston, MA) that acquires plantar forces during stepping. The pressure sensors contained a grid of 960 pressure-sensing cells (0.252 cm each) consisting of two perpendicular layers of conductive ink, each photolithographed with parallel traces on a mylar substrate. The analog signal was digitized at a frequency of 50 Hz by electronics located in a cuff unit placed above the lateral malleolus.

Limb load sensor calibration

All sensors were dynamically calibrated (Carollo et al. 1993) with the use of a force platform (Kistler, Amherst, NY), with ND individuals matched by body weight and shoe size to SCI subjects. Calibrations from ND persons were used for all SCI subjects because they were not capable of over-ground walking. The force signal from the sensors was compared with the signal from a force plate recorded simultaneously during a step for each subject. The peak forces (maximum amplitude during contact) and mean forces (average of the amplitude during contact) from the pressure sensors and the force plate from several steps were compared for each subject. A significant ($P < 0.05$), positive linear relationship was observed for both the peak ($y = 0.98x - 10$, $r = 0.99$) and mean forces ($y = 1.0x - 46$, $r = 0.96$) from the pressure sensors versus the force plate.

During each experiment, the load values determined by the sensors were also compared with body weight support data simultaneously acquired by the force transducer attached to the suspension apparatus. The numerical sum of the loading (sum of the force from the left and right sensors) and unloading (force from the force transducer) closely approximated the subject's body weight, verifying the accuracy of the load measurements during stepping on the treadmill. Mean values from the sum of the force from the sensors from both limbs and force transducer signals during 20 s of stepping at each load condition were compared. This analysis was completed for all loading conditions for each subject. For all subjects, linear regression demonstrated an inverse relationship (slope of -0.92 ± 0.15 , mean \pm SE) between mean unloading and loading and the intercepts closely corresponded to the individual's body weight.

Data analyses

Data from ~800 steps from the six subjects were analyzed. Data reduction was performed with the use of Labview (National Instruments, Austin, TX) customized by our laboratory. The EMG data were rectified and high-pass filtered at 32 Hz. Force signals from the pressure sensors were interpolated from 50 Hz to 1 kHz and synchronized with EMG signals. Onset of EMG bursts was defined as the time when the signal amplitude remained above the threshold (mean of the baseline + 3 SD) for 50 ms. The end of the EMG burst was defined by the time when the signal amplitude remained below the threshold level for 150 ms. Bursts were analyzed independent of where they occurred in the step cycle; that is, the complete EMG burst in each step was quantified regardless of whether it was synchronized with the onset and termination of loading or muscle stretch. Mean EMG amplitude was calculated by dividing the sum of the amplitudes of each burst by the burst duration. For each step, limb peak load was determined by subtracting the minimum force signal from the maximum. Percent BWL was calculated by dividing the limb peak load by body weight. Mean EMG amplitude versus limb peak load was plotted for the SOL, MG, and TA muscles of each subject.

Muscle-tendon length was calculated as percent shank length on the basis of goniometer measurements and the regression equations

derived by Hawkins and Hull (1990). The rate of change of muscle-tendon length (referred to as velocity of muscle-tendon length change) was determined by the derivation of the calculated muscle-tendon length curves. The relationship between mean EMG activity and increases in muscle-tendon length (muscle-tendon stretch) were compared in two ways. First, the mean EMG activity (as described above) was plotted versus the maximum muscle-tendon stretch of each step cycle, independent of whether the EMG activity of the muscle was synchronized with the muscle-tendon stretch. The mean EMG activity was also plotted versus the velocity of muscle-tendon stretch. Second, to assess the immediate response of the EMG activity to muscle-tendon stretch, the mean EMG activity was determined solely during the period of the muscle-tendon stretch incorporating a delay for signal conduction velocity (70 ms). The mean EMG activity during this period was plotted versus muscle-tendon stretch and velocity of muscle-tendon stretch.

Waveforms of EMG activity (low-pass filtered at 5 Hz), muscle-tendon length, velocity of muscle-tendon length change, and limb load were generated from eight consecutive steps per BWL condition to investigate relationships within a step cycle. In loaded conditions, a step cycle was taken from initial foot contact of one limb to the following initial foot contact of the same limb. For trials with no limb loading, step cycles were taken from two consecutive points of maximum dorsiflexion determined by ankle goniometer signals. This ankle position during no loading closely approximated the ankle angle at initial contact during loaded stepping. For step cycle averaging, the eight step cycles were normalized to the mean cycle duration, averaged, and expressed as percent of the step cycle duration.

Statistics

The correlation among EMG mean amplitude and each variable (peak load, muscle-tendon stretch, and velocity of muscle-tendon stretch) were evaluated by regression analysis during a step cycle and during EMG activity. In most cases, quadratic ($y = ax^2 + bx + c + \text{error}$) regressions were reported for each muscle (SOL, MG, and TA) because they provided substantially higher correlation coefficients (thus explaining more of the variability) and lower *P* values than linear ($y = ax + c + \text{error}$) regressions (Tables 3 and 4). When there was a nonsignificant quadratic term and a significant linear term, the linear regression was reported. Additionally, stepwise regression (minimum tolerance for entry into model = 0.010, α to enter = 0.150, α to remove = 0.150) of all variables per muscle was performed.

RESULTS

Motor pool activity during stepping on a treadmill with partial body weight support

EMG bursts of the primary ankle muscles were synchronized with the stepping cadence in all subjects during weight-bearing stepping with body weight support, as shown by representative data from one ND and two SCI subjects (*ND-1*, *SCI-C1*, and *SCI-A2*; Fig. 1). The SOL and MG were active during stance (as displayed by load sensors and footswitches) in all subjects. The MG consistently preceded the onset of loading as well as the initiation of the SOL burst in *SCI-A2*. The TA was active primarily during swing in both *ND-1* and *SCI-C1*, whereas cocontraction with plantarflexors was observed in *SCI-A2*. Cocontraction of dorsiflexors with plantarflexors was also observed in both *SCI-B1* and *SCI-A1* (not shown). Clonic EMG activity (characterized by high amplitude and low frequency) as detected in *SCI-C1* was often seen in the bursts of SCI subjects.

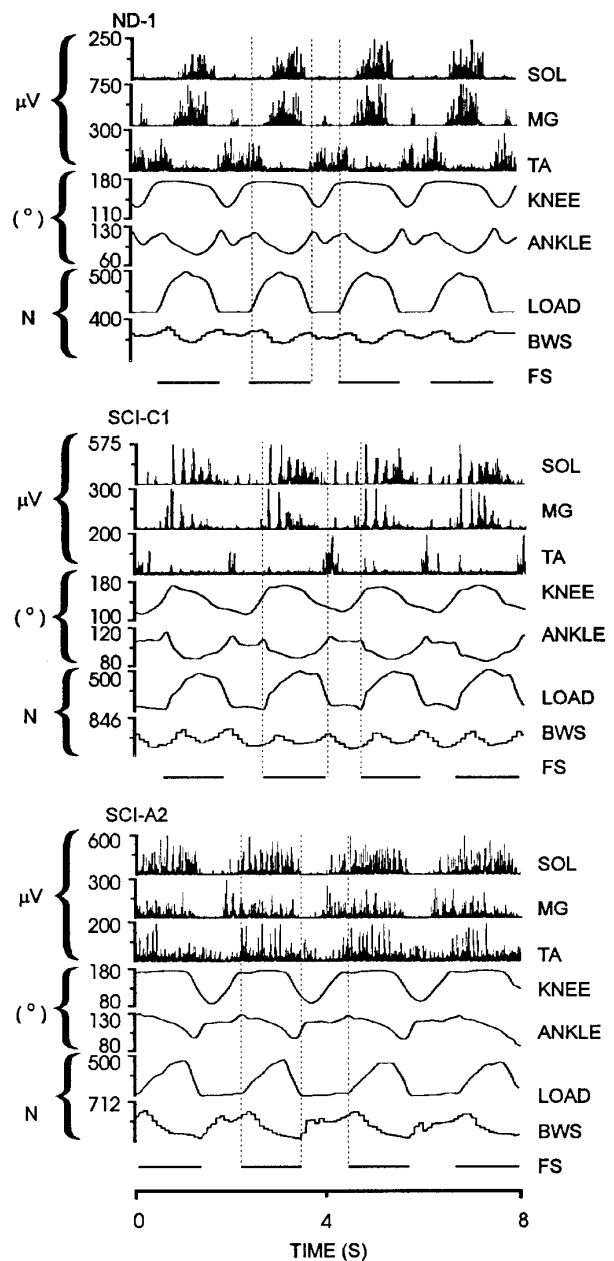


FIG. 1. Representative electromyographic (EMG) activity (μV ; rectified, high-pass filtered at 32 Hz) from soleus (SOL), medial gastrocnemius (MG), and tibialis anterior (TA); knee and ankle angles ($^\circ$); limb load (N); body weight support (BWS, N); and foot contact (FS) during stepping at a treadmill speed of 0.45 m/s. Data from a nondisabled (ND) subject (*ND-1*) and 2 spinal-cord-injured (SCI) subjects (*SCI-C1* and *SCI-A2*) are shown. The ND subject stepped independently and SCI subjects stepped with assistance. Dashed lines: transition between stance and swing phases of a step cycle as detected by the footswitches.

Overall knee and ankle dynamics were similar among SCI subjects that were assisted and the unassisted ND subjects during stepping.

SOL EMG response to limb load

SOL EMG mean amplitudes were modulated by limb peak load in all subjects. The EMG mean amplitudes of each step of each subject for the range of loads studied are shown in Fig. 2. Each point represents the average of the SOL EMG

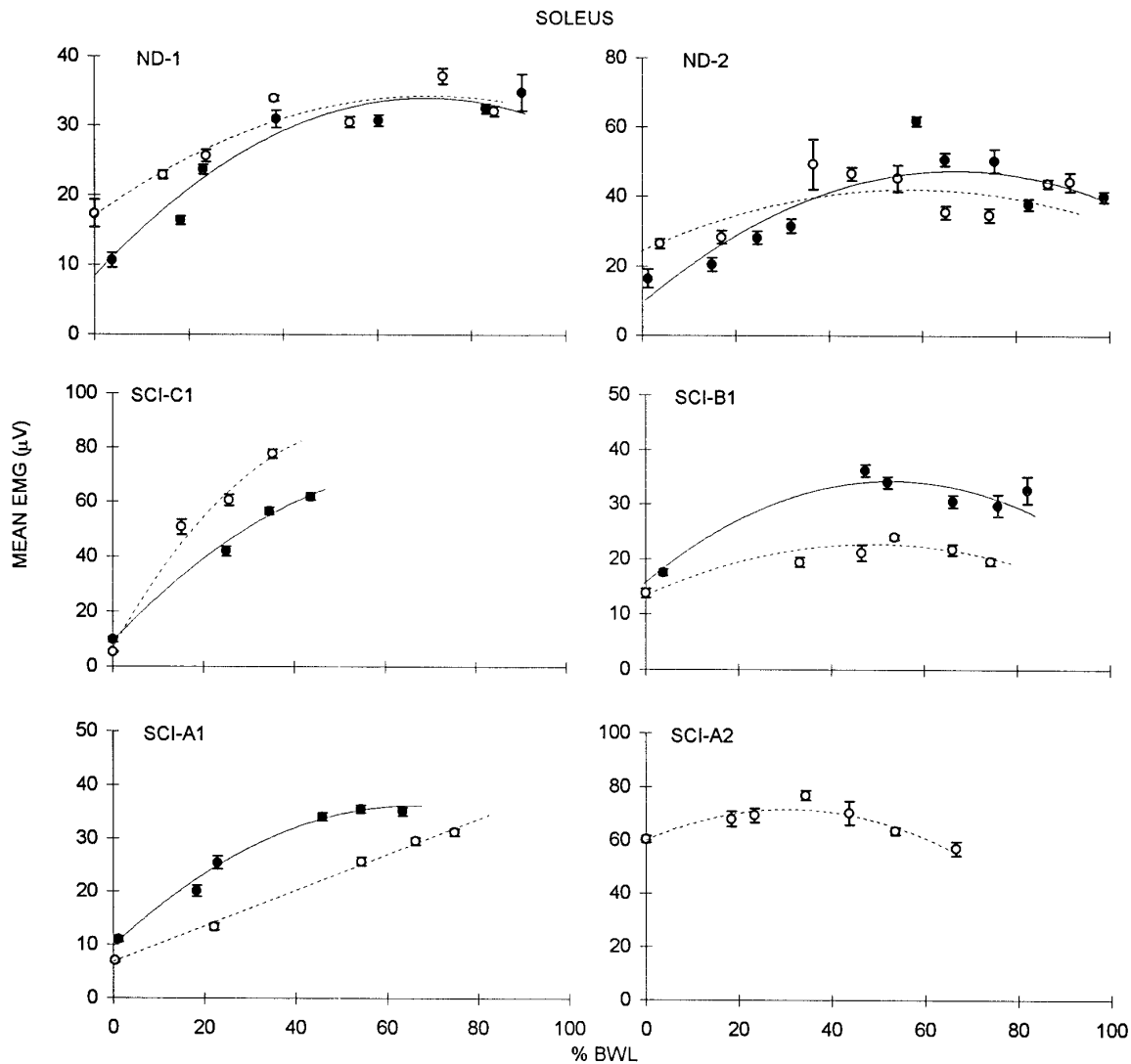


FIG. 2. Averaged SOL EMG mean amplitude per burst (μV ; rectified, high-pass filtered at 32 Hz) vs. limb peak load [% body weight load (BWL)] for each subject. Each point represents the average of SOL EMG amplitudes (means \pm SE) within a 10% interval of the BWL range (i.e., 0–10%, 10–20%, etc.). Data from the right limb (\circ) and left limb (\bullet) of each subject are shown separately. Data were not available for every BWL interval. The regression line was based on individual data points from each step cycle and drawn for each muscle when there was a significant relationship ($P < 0.05$; see Table 2 for correlation ratios).

mean amplitude of 5–30 steps within a 10% BWL range. Regression lines were determined from single steps as plotted in Fig. 3 (Table 2). A significant relationship between SOL EMG mean amplitude and limb peak load was detected in 11 of 11 muscles studied. The responsiveness to loading was most consistent at loads of <40–50% BWL. The magnitude of the effects and the shapes of the regression lines differed among subjects and even between limbs within some subjects (Fig. 2, Table 2). The statistical model of best fit between SOL EMG mean amplitude and limb peak load was curvilinear in 10 of 11 muscles. In one muscle there was no significant curvilinear relationship but a significant linear one (see right leg data for *SCI-A1* in Fig. 2, Table 2). SOL and MG EMG activity was not detected during stepping from the left limb of *SCI-A2* during multiple experiments. These data illustrate that SOL EMG amplitudes were modulated as a function of load in subjects with intact minimal or no detectable supraspinal input to the motor pools studied.

SOL EMG responses to muscle-tendon length changes

The relationships among SOL EMG mean amplitudes, level of load, and changes in muscle-tendon stretch were examined to determine whether limb loading affected the range of joint motion. These altered kinematics could change muscle-tendon stretch during gait, which could modulate EMG activity through reflex pathways. The relationships among SOL EMG mean amplitude, limb peak load, muscle-tendon stretch, and velocity of muscle-tendon stretch per step were examined. Representative data are shown for one subject from each functional class (Fig. 3, *A–C*, *E*, *G–I*, *K*, *M–O*, and *Q*). Statistical analyses of the relationships between SOL EMG mean amplitude and muscle-tendon stretch for one limb of each subject are listed in Table 2. A positive relationship between SOL EMG mean amplitude and limb peak loading was evident for the ND and both SCI subjects (Fig. 3, *A*, *G*, and *M*) shown. Higher limb peak loading resulted in a significant increase in muscle-tendon stretch in

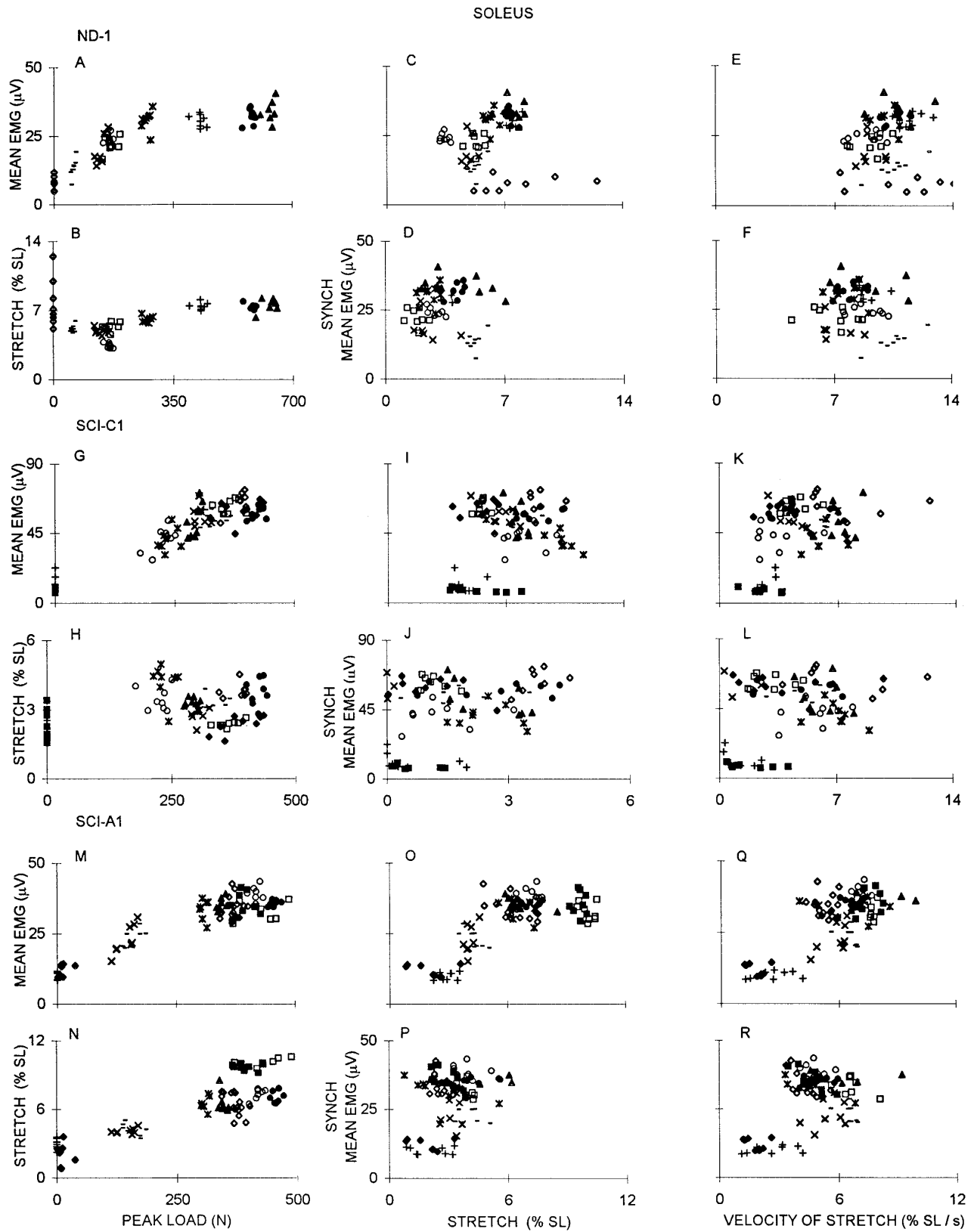


FIG. 3. Relationships among SOL EMG mean amplitude (μV), limb peak load (N), SOL muscle-tendon stretch [% shank length (SL)], and velocity of SOL muscle-tendon stretch [% shank length per s (SL/s)] from *ND-1* (A–F), *SCI-C1* (G–L), and *SCI-A1* (M–R) over a range of loading conditions are shown. Muscle-tendon stretch and velocity of muscle-tendon stretch were measured during an entire step cycle (C, E, I, K, O, and Q) and also during the period synchronized with SOL EMG activity (SYNCH mean EMG; D, F, J, L, P, and R). Each data point represents 1 step and each symbol represents a series of consecutive steps at 1 level of body weight support. There was a significant relationship between SOL muscle-tendon stretch and limb peak load in *ND-1* (B; $r = 0.58$), *SCI-C1* (H; $r = 0.50$), and *SCI-A1* (N; $r = 0.86$).

TABLE 2. Correlation coefficients between mean EMG amplitude and limb load, muscle-tendon stretch, and velocity of muscle-tendon stretch for the soleus

Subject	Leg	During Step Cycle			During EMG Activity	
		Load	Stretch	Velocity of stretch	Stretch	Velocity of stretch
ND-1	L	0.93	0.34	0.36	NS	NS
ND-1	R	0.82				
ND-2	L	0.75	0.44	0.49	0.42	0.33*
ND-2	R	0.57				
SCI-C1	L	0.94	0.49	0.56	0.35*	0.36*
SCI-C1	R	0.94				
SCI-B1	L	0.71	NS	NS	0.27*	NS
SCI-B1	R	0.71				
SCI-A1	L	0.95	0.85	0.78	0.31*	0.62
SCI-A1	R	0.96*				
SCI-A2	R	0.57	0.46	0.36	0.46	0.38

EMG, electromyogram; L, left; R, right; NS, not significant. Correlation coefficients are reported from significant ($P < 0.05$) quadratic regressions unless marked with an asterisk. * Linear regressions provided the only significant results.

SCI-A1 ($r = 0.86$; Fig. 3N) and also SCI-C1 and ND-1, but with relatively low correlation ($r = 0.50$ and 0.58 , respectively; Fig. 3, H and B). In five of the six subjects there was a significant curvilinear relationship between SOL EMG mean amplitude and SOL muscle-tendon stretch and between SOL EMG mean amplitude and velocity of muscle-tendon stretch during the step cycle. The changes in SOL EMG mean amplitudes in all subjects were correlated more closely with peak load per step (Fig. 3, A, G, and M) than to changes in muscle-tendon stretch (Fig. 3, C, I, and O) or velocity of muscle-tendon stretch (Fig. 3, E, K, and Q; Table 2) per step.

If the EMG responses were attributable directly to an immediate response to muscle stretch alone, as in a stretch reflex, then the EMG burst would have to be confined to the period of the step cycle corresponding to when the muscles are being stretched. To address this issue, the relationships among EMG mean amplitude, muscle-tendon stretch (Fig. 3, D, J, and P), and velocity of muscle-tendon stretch (Fig. 3, F, L, and R) that occurred only during lengthening of the muscle and the time period of EMG activity (synchronous mean EMG) were examined. The correlation between SOL EMG mean amplitude and muscle-tendon stretch during EMG activity was lower than the correlation between the SOL EMG mean amplitude and limb peak load in each subject, and lower than the correlation between SOL EMG mean amplitude and muscle-tendon length changes during the entire step cycle in ND-1, ND-2, SCI-C1, and SCI-A1.

Further, multivariate, forward stepwise regression analysis selected limb peak load as the parameter primarily responsible for the variability in SOL EMG mean amplitude in all subjects except SCI-A2 (velocity of muscle-tendon stretch during the step cycle was selected first). For this subject, limb peak load became the primary variable at loads of $<50\%$ BWL. Overall, the muscle-tendon stretch contributed modestly to EMG amplitude, but could have been the primary modulating factor in only one subject.

The significant relationships between the level of load per

step with SOL EMG amplitude, as previously demonstrated, could have occurred as a result of a response to the integrated afferent signals accumulated throughout a complete step cycle or as an immediate response to afferent stimuli. To address these issues the relationships among SOL EMG amplitude, limb load, muscle-tendon length, and velocity of muscle-tendon length change were examined. Representative data from two consecutive steps at a single body weight support condition, but when different limb peak load levels occurred inadvertently, are shown for ND-1 and SCI-A1 (Fig. 4). Although differences in the detail of the relationships among EMG amplitude, limb load, muscle-tendon length, and velocity of muscle-tendon length change were observed between ND-1 and SCI-A1, the general patterns of coordination were similar.

The highest load occurred during the second step, whereas the greatest stretch occurred in the first step in both ND-1 (Fig. 4, A-C) and SCI-A1 (Fig. 4, E-G), further illustrating that the effect of load on SOL EMG amplitude was not simply a function of muscle-tendon stretch. These data demonstrate that SOL EMG amplitude increased in response to an increase in limb peak load between the two consecutive steps, whereas a greater increase in length occurred during the step at the lower load. Further, the EMG amplitude was inversely related to the velocity of muscle-tendon length change (positive velocity in Fig. 4, D and H), indicating that the velocity of muscle-tendon stretch within these steps is not a principal factor in regulating the EMG amplitude. Thus muscle-tendon length within a step did not have an important modulatory effect on SOL EMG amplitude for the entire step cycle.

In both subjects, the peak SOL EMG activity and peak load were closely synchronized (Fig. 4, B and F). The relationship between EMG amplitude and limb load during stance was similar in both subjects and was highly phase dependent. The response of the SOL EMG activity to load was dependent on the step cycle phase, because the EMG amplitude was modulated differently, at the same absolute load level, during loading and unloading phases of stance. A highly phase-dependent EMG amplitude modulation was also evident when plotted with muscle-tendon length (Fig. 4, C and G) or velocity of muscle-tendon length change (Fig. 4, D and H). In both subjects there was a direct relationship between SOL EMG activity and muscle-tendon length during the loading phase of stance. Yet at the same muscle-tendon length SOL EMG activity was significantly higher during the steps taken at the higher limb peak load compared with the lower limb load condition. The SOL EMG amplitude was not directly related to muscle-tendon velocity (Fig. 4, D and H).

MG EMG responses to limb load

As in the SOL, MG motor pool mean amplitude was modulated by limb peak load during manually assisted and independent stepping (Fig. 5). A significant relationship between MG EMG mean amplitudes and limb peak load was observed in both ND subjects and three of the four SCI subjects studied (SCI-C1, SCI-A1, and SCI-A2; Fig. 5, Table 3). The statistical model of best fit was curvilinear in five muscles (ND-1, SCI-A1, and SCI-A2) and linear in four muscles (ND-2 and SCI-C1). A decrease in MG EMG activ-

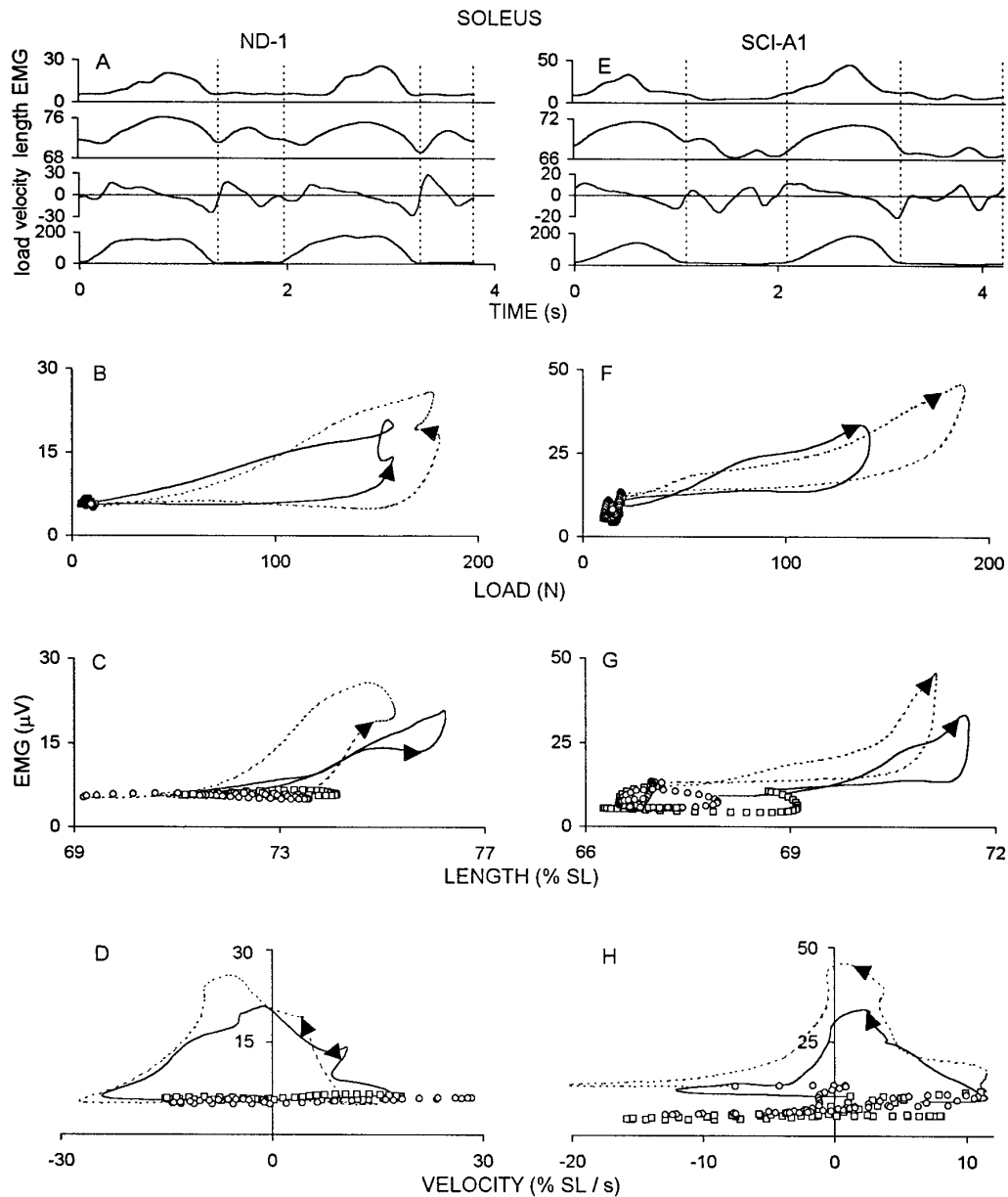


FIG. 4. SOL EMG amplitude (μV ; rectified, high-pass filtered at 32 Hz, low-pass filtered at 5 Hz), limb load (N), SOL muscle-tendon length (% SL), and velocity of SOL muscle-tendon length change (% SL/s) from 2 consecutive steps with inadvertently different limb loads from *ND-1* (A–D) and *SCI-A1* (E–H). All data are shown relative to the phase of the step cycle (s) in A and E. Vertical dashed lines: transitions between stance and swing phases. SOL EMG amplitude vs. limb load (B and F), SOL EMG amplitude vs. muscle-tendon length (C and G), and SOL EMG amplitude vs. velocity of muscle-tendon length change (D and H) are illustrated. The stance phases of the steps are represented by a dashed line (high load) and solid line (low load). The swing phases of the steps are represented by open symbols (circles, high load; squares, low load). Arrows indicate the direction of the step cycle and are located at the time point that represents 50% of the stance phase.

ity was observed at the higher limb peak loads studied in the left limb of *SCI-A1*.

MG EMG responses to muscle-tendon length changes

The relationships among MG EMG amplitude, limb peak loading, muscle-tendon stretch, and velocity of muscle-tendon stretch were examined to determine whether joint mechanics affecting muscle-tendon stretch could be providing the sensory cues that regulate motor pool modulation. All data points used to derive the correlations in Table 3 were

plotted for one subject from each functional group in Fig. 6 as described previously for the SOL. The higher loads were associated with greater muscle-tendon stretch in all three subjects (*ND-1*, $r = 0.71$; *SCI-C1*, $r = 0.69$; *SCI-A1*, $r = 0.87$; Fig. 6, B, H, and N). However, in several cases the relationships between MG EMG mean amplitude and selected variables associated with muscle-tendon stretch were not significant (Table 3, see *ND-1*, *ND-2*, *SCI-B1*, and *SCI-A2*). Further, significant relationships between EMG mean amplitude and muscle-tendon stretch that occurred during a step cycle or length changes that were synchronized with

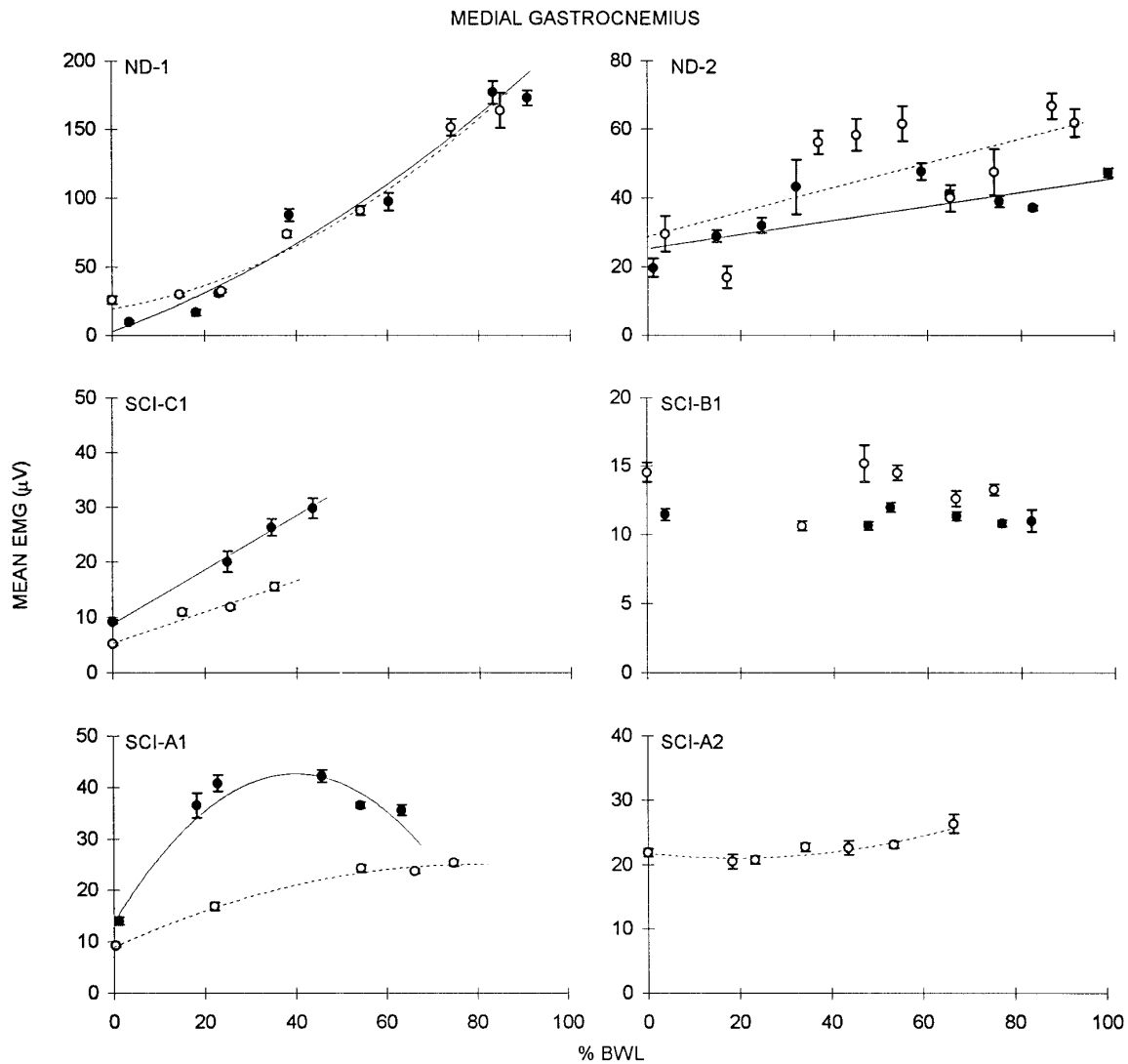


FIG. 5. Averaged MG EMG mean amplitude per burst (μV) vs. limb peak load (% BWL) are presented for each subject. Each point represents the average of MG EMG amplitude (mean \pm SE) within a 10% interval of the BWL range. Data from the right limb (\circ) and left limb (\bullet) of each subject are shown separately. Data were not available for every BWL interval. The regression line was based on individual data points from each step cycle and drawn for each muscle when there was a significant relationship ($P < 0.05$; see Table 3 for correlation ratios).

MG EMG activity were not as highly correlated as those observed between EMG mean amplitude and limb peak load (Table 3). In *ND-1* and *SCI-C1*, the shape of the relationships between EMG amplitude and limb peak load (Fig. 6, *A* and *G*) were not similar to those between MG EMG mean amplitude and muscle-tendon stretch during a step (Fig. 6, *C*, *E*, *I*, and *K*) or synchronized with MG EMG activity (Fig. 6, *D*, *F*, *J*, and *L*). A multivariate, forward stepwise regression model incorporating all of these variables selected limb peak load as the parameter primarily responsible for the variability in MG EMG mean amplitude in both of these subjects as well as *ND-2*.

MG EMG mean amplitude did not respond to limb peak load or muscle-tendon stretch in a graded manner in *SCI-A1* (Fig. 6, *M* and *O*). Yet, higher MG EMG amplitude occurred at higher limb peak loads (Fig. 6*M*), increased muscle-tendon stretch (Fig. 6, *O* and *P*), and higher velocities muscle-tendon stretch (Fig. 6, *Q* and *R*) in this subject. Multivariate regression analysis selected velocity of muscle-

tendon stretch during the step cycle as the primary parameter, limb peak load as the secondary parameter, and velocity of muscle-tendon length change during EMG activity as the tertiary parameter responsible for the variability in MG EMG mean amplitude in *SCI-A1*. These data suggest that muscle-tendon stretch during EMG activity may play a more dominant role in MG EMG amplitude modulation in *SCI-A1* than in the other subjects studied, and a more prominent role than in the modulation of the SOL EMG amplitude of *SCI-A1*. In subject *SCI-A2*, however, muscle-tendon velocity did not significantly affect the MG EMG amplitude, but a significant, although low, correlation was detected with muscle-tendon stretch during the step cycle and during EMG activity (Table 3).

To address whether MG EMG activity was generated by instantaneous afferent stimuli or an integration of afferent information, the relationships among MG EMG amplitude, limb load, muscle-tendon length, and muscle-tendon velocity were examined within a step cycle. The overall shape of the

TABLE 3. Correlation coefficients between mean EMG amplitude and limb load, muscle-tendon stretch, and velocity of muscle-tendon stretch for the medial gastrocnemius

Subject	Leg	During Step Cycle			During EMG Activity	
		Load	Stretch	Velocity of stretch	Stretch	Velocity of stretch
ND-1	L	0.97	0.71*	NS	0.53*	NS
ND-1	R	0.95				
ND-2	L	0.68*	NS	0.54	0.37*	0.25
ND-2	R	0.43*				
SCI-C1	L	0.71*	0.50	0.17	0.45	0.31
SCI-C1	R	0.87*				
SCI-B1	L	NS	NS	NS	NS	NS
SCI-B1	R	NS				
SCI-A1	L	0.90	0.81	0.80	0.76	0.78
SCI-A1	R	0.93				
SCI-A2	R	0.46	0.35*	NS	0.34*	NS

For abbreviations, see Table 2. Correlation coefficients are reported from significant ($P < 0.05$) quadratic regressions unless marked with an asterisk. * Linear regressions provided the only significant results.

curves of the relationship between MG EMG amplitude and limb load differed between *ND-1* and *SCI-A1*, in contrast to the similarities observed between the two subjects for the SOL. Yet, in both subjects, higher MG EMG amplitude occurred in the step with higher limb loading. In both steps from *ND-1*, the highest MG efferent motor pool amplitude occurred during limb peak load (Fig. 7, A and B), in contrast to that observed in *SCI-A1* (Fig. 7, E and F). Even though there was a reduced sensitivity of MG EMG amplitude to limb peak load in *SCI-A1* at higher limb loads (see Fig. 6M) in two consecutive steps with varying load, a higher MG EMG peak amplitude corresponded to a higher limb peak load for its respective step (Fig. 7, E and F). In *ND-1*, during the step with the greater muscle-tendon stretch (lower limb load), the peak EMG amplitude was lower (Fig. 7C). In *SCI-A1* the muscle-tendon lengths and velocities were similar in both steps, yet the step with the higher limb peak load attained a higher EMG peak amplitude (Fig. 7, G and H). In *SCI-A1* the onset of MG activity occurred before limb loading (Fig. 7, E and F) and corresponded to increases in muscle-tendon length (Fig. 7G). At the onset of limb load the EMG amplitude declined and then began increasing again, reaching a peak at the greatest muscle-tendon length in both steps. In the second step, however, muscle-tendon length continued to increase while the EMG activity decreased. The EMG amplitude declined during stance while muscle-tendon length was maintained at the greatest muscle stretch and while limb load was still increasing. These results indicate a phase-dependent response that did not seem to relate directly to either instantaneous limb load or muscle-tendon length changes.

TA EMG responses to limb load

There was a significant relationship between mean TA EMG amplitude and limb peak load in 10 of the 11 muscles studied (Fig. 8, Table 4) but the correlation coefficients were lower than in the plantarflexors. The statistical model of best fit was linear in four muscles, curvilinear in six. A

higher level of TA EMG activity was evident in ND subjects than in SCI subjects during no limb loading. Despite the low levels of activity in the TA of SCI subjects, EMG mean amplitude did respond to limb peak load. Subjects *SCI-C1*, *SCI-B1*, and *SCI-A1* showed the highest correlations (Fig. 8 and Table 4, right leg data). Subject *SCI-B1* demonstrated some load dependence from ~50 to 80% BWL.

TA EMG responses to muscle-tendon length changes

We examined the relationships among TA EMG amplitude, limb load, muscle-tendon length, and velocity of muscle-tendon length change within a step. Representative data averaged from eight step cycles are shown for *ND-1* (Fig. 9A) and *SCI-A2* (Fig. 9B). In both subjects, higher TA EMG amplitude was detected with increased limb peak loading. However, unlike the plantarflexors, minimal modulation of TA EMG amplitude was synchronized to load or muscle-tendon stretch in the subjects studied. In ND subjects the EMG activity primarily occurred during the swing phase, although, especially at the higher limb loading conditions, observable TA EMG activity was present during stance. A significant correlation between TA EMG mean amplitude and muscle-tendon stretch and velocity of muscle-tendon stretch during a step cycle was observed in *ND-1* and *ND-2* (Table 4). However, a significant correlation was observed between TA EMG mean amplitude and muscle-tendon stretch and velocity of muscle-tendon stretch during EMG activity in *ND-2* but not in *ND-1*. These measurements were taken only during periods when the TA EMG activity was observed and the TA was lengthening. This represents only a portion of the total EMG activity per step cycle.

In three of the SCI subjects (*SCI-A1*, *SCI-A2*, and *SCI-B1*), TA EMG activity occurred during stance, which corresponded primarily to the period of shortening of the TA muscle, as exemplified by *SCI-A2* (Fig. 9B). In *SCI-B1* and *SCI-A1*, a significant correlation was evident between TA EMG mean amplitude and muscle-tendon length during the step cycle (Table 4). Only *SCI-C1* had TA EMG activity during swing (in SCI subjects significant TA lengthening is observed during swing). There was a significant correlation between muscle-tendon velocity during the step cycle in *SCI-A1* that was not observed in the other three SCI subjects. No significant relationships were found between TA EMG mean amplitude and muscle-tendon stretch during EMG activity in *SCI-C1* or *SCI-B1*. A significant relationship was observed in *SCI-A1* and *SCI-A2*, again noting that these measurements represent only a small portion of the total TA EMG activity.

DISCUSSION

These data demonstrate that the level of loading through the limbs during cyclic activity can provide important information that facilitates the generation of steplike efferent patterns by the human lumbosacral spinal cord. Although a modulation of the EMG amplitude and coordination of the EMG bursts in lower limb muscles by load of completely spinalized cats (de Guzman et al. 1991; Edgerton et al. 1991) and decerebrate cats (Conway et al. 1987; Duysens and Pearson 1980) has been described previously, Dietz et al. (1995) reported that in SCI humans with no supraspinal

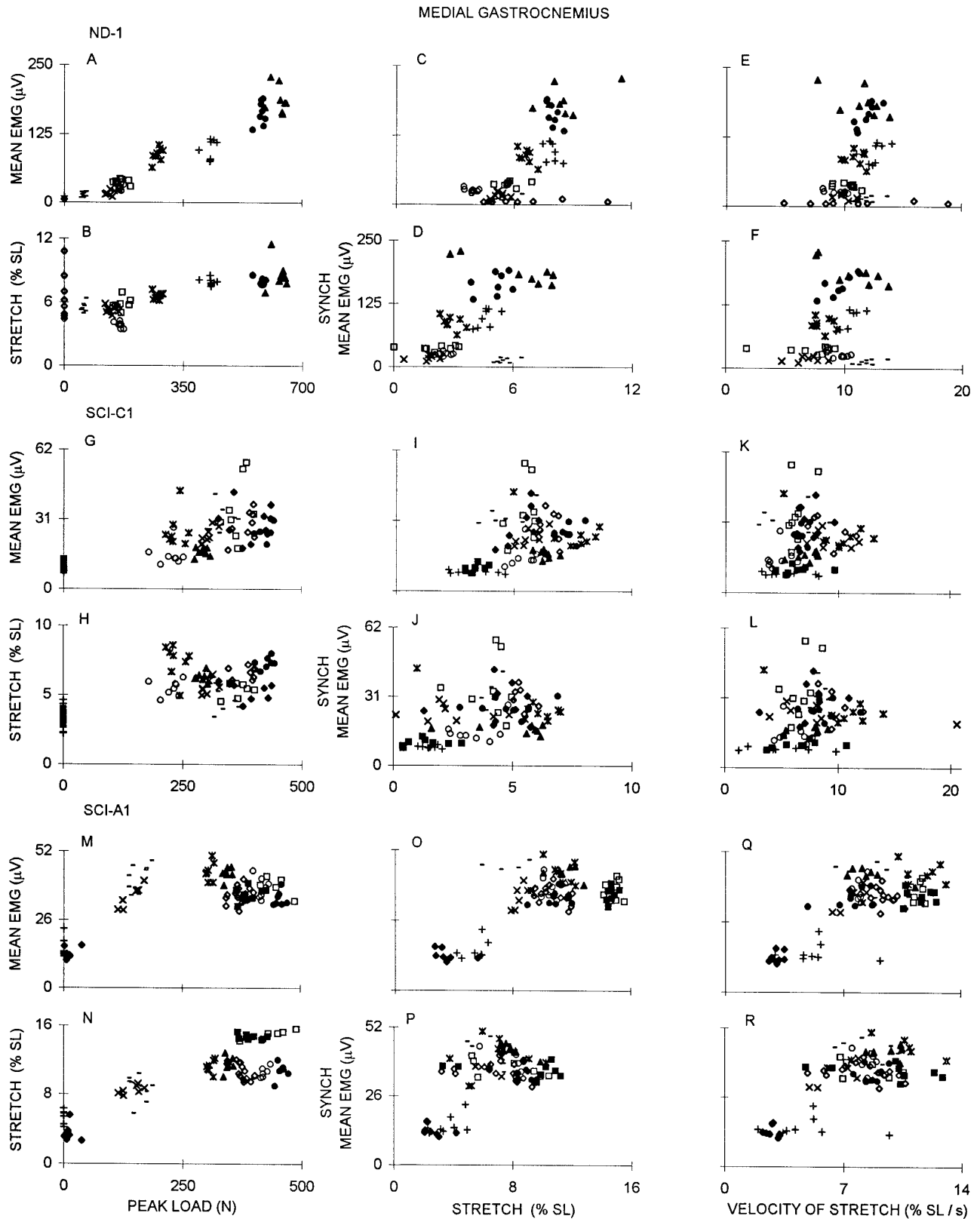


FIG. 6. Relationships among MG EMG mean amplitude (μV), limb peak load (N), MG muscle-tendon stretch (% SL), and velocity of MG muscle-tendon stretch (% SL/s) from ND-1 (A-F), SCI-C1 (G-L), and SCI-A1 (M-R) over a range of loading conditions. Muscle-tendon stretch and velocity of muscle-tendon stretch were measured during an entire step cycle (C, E, I, K, O, and Q) and also during the period synchronized with MG EMG activity (SYNCH mean EMG; D, F, J, L, P, and R). Each data point represents 1 step and each symbol represents a series of consecutive steps at 1 level of body weight support. There was a significant relationship between MG muscle-tendon stretch and limb peak load in ND-1 (B; $r = 0.71$), SCI-C1 (H; $r = 0.69$), and SCI-A1 (N; $r = 0.87$).

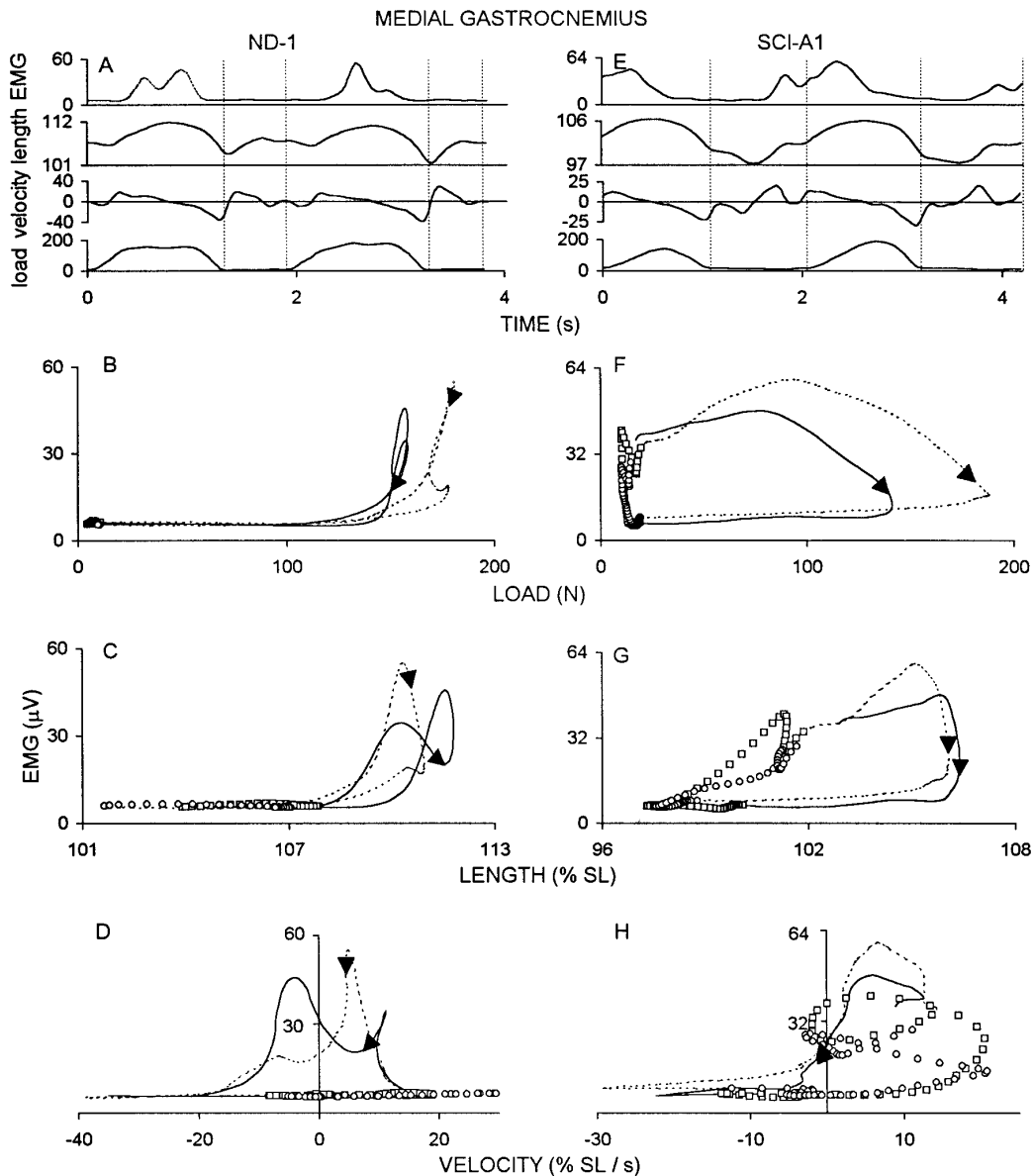


FIG. 7. MG EMG amplitude (μV), limb load (N), muscle-tendon length (% SL), and velocity of muscle-tendon length change (% SL/s) from 2 consecutive steps with inadvertently different limb peak loads from ND-1 (A–D) and SCI-A1 (E–H). All data are shown relative to the phase of the step cycle in A and E. Vertical dashed lines: transitions between stance and swing phases. MG EMG amplitude vs. limb load (B and F), EMG amplitude vs. muscle-tendon length (C and G), and MG EMG amplitude vs. velocity of muscle-tendon length change (D and H) are illustrated. The stance phases of the steps are represented by a dashed line (high load) and solid line (low load). The swing phases of the steps are represented by open symbols (circles, high load; squares, low load). Arrows indicate the direction of the step cycle and are located at the time point that represents 50% of the stance phase.

input “there was little correlation between the amplitude of EMG activity and unloading of the body weight.” The present data demonstrate, however, that the level of loading does provide cues that modulate the motor pool activity in a similar manner in subjects with either full, partial, or no supraspinal control of the motor pools that innervate the lower limb muscles.

Sensory modalities that contribute to weight-bearing stepping

The load-imposed modulation of EMG amplitude illustrates one of many possible kinetic and kinematic signals

concurrent with stepping that shape the output of the motor pools controlling the lower limbs. A more parsimonious interpretation has been that the motor patterns seen in clinically complete SCI patients reflect only the rhythmic muscle stretches induced by the mechanical events associated with stepping (Rossignol and Barbeau 1995; Stewart et al. 1991). The present study shows that, although sensory information associated with muscle-tendon stretch and velocity of muscle-tendon stretch during a complete step cycle could have had some effect on the efferent motor patterns of the lower limbs, the EMG amplitudes were clearly less coupled to length factors than limb peak load per step. Higher correlations were not consistently observed when comparing these

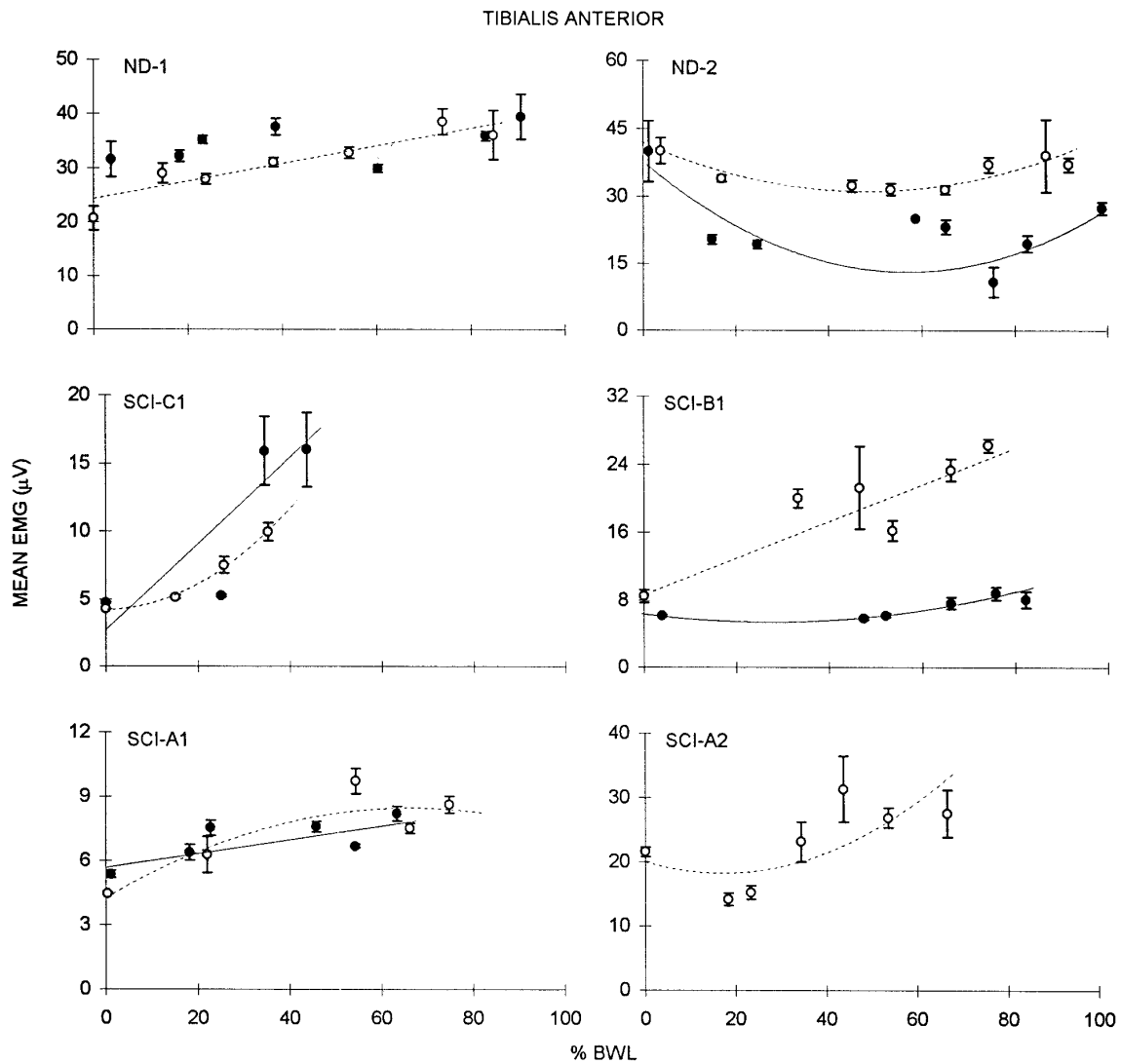


FIG. 8. Averaged TA EMG mean amplitude per burst (μV) vs. limb peak load (% BWL) for each subject. Each point represents the average TA EMG amplitudes (means \pm SE) within a 10% interval of the BWL range. Data from the right limb (\circ) and left limb (\bullet) of each subject are shown separately. Data were not available for every BWL interval. The regression line was based on individual data points from each step cycle and was drawn for each muscle when there was a significant relationship ($P < 0.05$; see Table 4 for correlation ratios).

kinetic variables only during the period when an EMG burst occurred. Although the present data are consistent with the possibility that muscle-tendon stretch could contribute to the total ensemble of proprioceptive and cutaneous information, it is evident that this single mechanical event does not play the dominant role in EMG amplitude modulation during manually assisted stepping on a treadmill with body weight support.

Sensory modalities other than limb peak loading and muscle-tendon length could play a role in the modulation of lower limb motor pools during stepping in humans. For example, temporal and spatial distributions of the loads on the soles of the feet may be important in the regulation of motor pool activity during locomotion (Aniss et al. 1992; Fung and Barbeau 1994; Yang and Stein 1990). Further, limb unloading has been shown to be an important signal for the termination of stance and the initiation of swing in cat locomotion (Conway et al. 1987; Duysens and Pearson 1980; Pearson and Duysens 1976). The present data empha-

size that there are other afferent systems that are not closely associated with limb loading that can markedly affect the output of the motor pools. For example, a series of steps at the same loading condition resulted in varying levels of EMG activity in the muscles studied. Hip joint position (Andersson and Grillner 1981; Andersson et al. 1978b; Grillner and Rossignol 1978), contralateral limb information (Berger et al. 1984; Burke et al. 1991), and cutaneous stimulation (Abraham et al. 1985; Andersson et al. 1978a; Duysens 1977) could have a significant effect on EMG amplitudes. It is feasible that all of these sources of sensory information contribute to defining the kinetic and kinematic events associated with stepping in ND as well as SCI subjects (Wernig and Müller 1992).

Phase dependence of EMG modulation

The present results demonstrate a very consistent phase-specific modulation of the efferent output of muscles in re-

TABLE 4. Correlation coefficients between mean EMG amplitude and limb load, muscle-tendon stretch, and velocity of muscle-tendon stretch for the tibialis anterior

Subject	Leg	During Step Cycle			During EMG Activity	
		Load	Stretch	Velocity of stretch	Stretch	Velocity of stretch
ND-1	L	NS	0.35	0.29*	NS	NS
ND-1	R	0.57*				
ND-2	L	0.59	0.60	0.47	0.63*	0.72
ND-2	R	0.43				
SCI-C1	L	0.40*	NS	NS	NS	NS
SCI-C1	R	0.65				
SCI-B1	L	0.57	0.76	NS	NS	NS
SCI-B1	R	0.70*				
SCI-A1	L	0.58*	0.52	0.34*	0.49	0.45
SCI-A1	R	0.65				
SCI-A2	R	0.52	NS	NS	0.52	0.58

For abbreviations, see Table 2. Correlation coefficients are reported from significant ($P < 0.05$) quadratic regressions unless marked with an asterisk. * Linear regressions provided the only significant results.

sponse to loading and the kinematics of a complete step cycle. A phase-dependent modulation of plantarflexor motor pool activity occurred when the limbs were allowed to support weight. Within a step, the relationships among SOL EMG activity, level of load, and muscle-tendon length were remarkably similar in all subjects. A strong phase dependence was indicated by the observations that SOL EMG amplitudes differed substantially throughout the step cycle at the same load, muscle-tendon length, and velocity. This phase-specific modulation was also observed consistently in the MG, whereas phase dependence was not obvious in the TA.

Many studies provide evidence for phase dependence of reflex modulation in humans during walking (Burke et al. 1991; Capaday and Stein 1986; Crenna and Frigo 1987; Duysens et al. 1990, 1992; Yang et al. 1991a,b). Further,

phase-dependent modulations of reflexes in mammals during cyclic motor output in response to cutaneous stimulation (Abraham et al. 1985; Duysens 1977), changes in hip position (Andersson and Grillner 1981; Andersson et al. 1978b), and loading (Conway et al. 1987; Duysens and Pearson 1980) have been reported for a range of experimental conditions in cats. The present data do not add to our understanding of the relative contribution of each afferent system at each point in the step cycle to locomotion in humans. It seems likely, however, that these neural pathways form an ensemble of temporally appropriate inputs that shape the patterns of motor pool output of the lower limbs.

Sensory information interpreted by the human lumbosacral spinal cord

The present results are a clear demonstration that load- and phase-dependent responses of the extensor and flexor motor pools of the lower limbs can be attributed to the human lumbosacral spinal cord, i.e., present in subjects with no detectable supraspinal influence available to the lower limb motor pools. Further, although responses of EMG activity to loading levels in humans with intact supraspinal input have been observed during standing (Horstmann and Dietz 1990) and stepping on a treadmill (Dietz et al. 1995; Finch et al. 1991), there has been no previous report of phase-dependent sensitivity to loading in humans. In the current study, responses of EMG activity to limb peak loading were evident in subjects independent of the amount of supraspinal input available to the lumbosacral motor pools and whether or not manual assistance for stepping was provided, suggesting a general mechanism within the spinal cord for interpreting load during locomotion in humans.

The presence of neural networks in the lumbar spinal cord that can generate motor patterns tightly linked to stepping and levels of loading might be expected given observations from other species (Grillner 1981). Many studies have suggested that the neural circuits of the human locomotor system (Bussel et al. 1988, 1989; Calancie et al. 1994; Crenna and

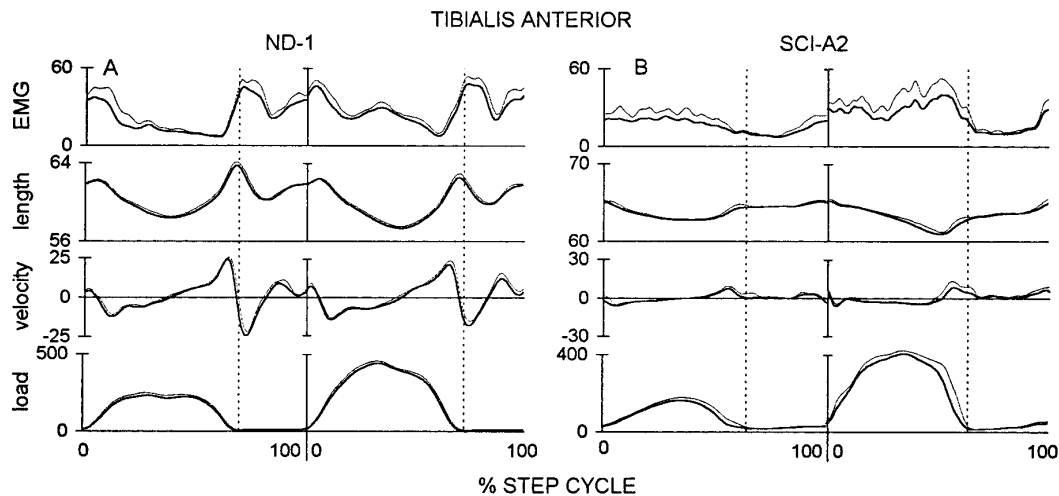


FIG. 9. TA EMG amplitude (μV , rectified, high-pass filtered at 32 Hz, low-pass filtered at 5 Hz), limb load (N), muscle-tendon length (% SL), and velocity of muscle-tendon length change (% SL/s) during a high-load and a low-load condition from ND-1 (A) and SCI-A2 (B) are shown. Consecutive step cycles were averaged by triggering from the point at which stance was initiated (increase in force measured from the pressure sensors). The average (heavy line) and average + SD (light line) for both load conditions relative to the phase of the step cycle (% STEP CYCLE) are shown for each subject in A and B. Vertical dashed lines: transitions between the stance and swing phase.

Frigo 1987; Duysens et al. 1992; Roby-Brami and Bussel 1987) are organized in a manner similar to that in the cat (Gossard and Hultborn 1991). The present study provides additional evidence that the human lumbosacral spinal cord may have maintained at least some of the same neural components that respond to limb load as in cats (Pearson 1995).

Muscle specificity of load modulation

Within a step the limb peak load occurred approximately at the EMG peak amplitude in the SOL in both subjects studied, but was only observed in the MG of the ND subject. SOL activation usually occurred after the initiation of stance in all subjects. Differential regulation of SOL and MG activation during walking has been reported previously (Duysens et al. 1991). In the present study the SOL response to load and muscle-tendon length change was similar in the SCI and ND subjects. The MG motor pools were active before the onset of limb load in SCI but not in ND subjects. Muscle-tendon stretch may play a more important role in initiation of the MG EMG amplitude in the SCI subjects than in ND subjects.

The significance of the coupling of TA EMG amplitude with loading on the same limb is not clear. There was no synchronization between TA EMG peak amplitude and limb peak load within the step cycle. In ND and some SCI subjects, TA activity occurred primarily during the swing phase when there is no or minimal loading of the ipsilateral limb. In most SCI subjects, on the other hand, the TA EMG activity occurred during stance of the ipsilateral limb in a similar manner to that observed in cats after spinal transection (de Guzman et al. 1991). These results indicate that there may be a common source for activation of the plantarflexors and dorsiflexors during stepping in SCI subjects. Further study is required to determine the significance and cause of the response of TA EMG amplitude to load.

Amplitude modulation of the TA could also be due to higher loading on the contralateral limb (Conway et al. 1987; Duysens and Pearson 1980). Load may not only facilitate ipsilateral extension but it may simultaneously facilitate contralateral flexion. Simultaneous activation of MG and contralateral TA has been observed during gait after a perturbation in humans (Berger et al. 1984). Regardless, the enhanced flexor activity has two important implications. First, it seems to facilitate flexion ipsilaterally, even though the timing may not be normal in an SCI subject. Second, it shows that the spinal cord interprets the afferent signals regarding load in an integrative manner such that the response to load is not immediate or reflexive.

Implications of present data

Because the lumbosacral motor pools appear to respond to multiple sensory modalities simultaneously and the type and magnitude of the response is dependent on the phase of the step cycle, it follows that the ensemble of sensory inflow from all sources and modalities concurs with the actual kinetic, kinematic, and cutaneous events appropriate for a specific instant in a step cycle. This interpretation is consistent with observations in chronic spinal cats that the specific manner of loading and the control of other sources of sensory inflow are crucial to the success of weight-bearing stepping

(Barbeau and Rossignol 1987; Edgerton et al. 1991; Lovely et al. 1990). Further, the necessity of the careful control of this sensory information when training a spinalized cat to step indicates an ability of the sensory motor pathways of the lumbosacral spinal cord to "perceive" and respond to the details of step related events (Edgerton et al. 1996). Our present understanding of the human lumbosacral spinal cord is not nearly as extensive as it is for other mammals. The present data do suggest that some of the fundamental properties are at least qualitatively similar in humans and cats.

Although these studies provide extensive analyses of hundreds of steps, the data represent a limited number of subjects. These data do not provide sufficient information to predict the robustness of the EMG response to load in a given muscle for a large population of subjects. These results, however, do show that load can affect the EMG output of plantarflexors and, to a lesser extent, dorsiflexors in subjects independent of the level of deficiency of supraspinal input to the lumbosacral spinal cord.

These data suggest that the human spinal cord, given appropriate sensory inputs, can modulate motor pool output that may facilitate locomotion, providing a fundamental rationale for partial weight-bearing step training as a means of enhancing the mobility of SCI subjects (Barbeau and Blunt 1991; Dietz et al. 1995; Wernig et al. 1995). The efficacy of this rehabilitative approach not only depends on the capacity of the spinal cord to interpret ongoing sensory inflow from the limbs during stepping, but it also assumes some use-dependent plasticity in the sensorimotor pathways that generate stepping. Further studies identifying the effective sensory modalities, the neural pathways involved, and level of adaptability and motor learning available to the human spinal cord are essential for understanding the potential of step training on a treadmill with body weight support for the rehabilitation of individuals with spinal cord injury.

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Address for reprint requests: V. R. Edgerton, 2322 Life Sciences, Dept. of Physiological Science, UCLA, Los Angeles, CA 90095-1527.

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