Modulation of Coordinated Muscle Activity during Imposed Sinusoidal Hip Movements in Human Spinal Cord Injury

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Abstract

Individuals with chronic spinal cord injury (SCI) often demonstrate multijoint reflex activity that is clinically classified as an extensor spasm. These responses are commonly observed in conjunction with an imposed extension movement of the hips, such as movement from a sit to a supine position. Coincidentally, afferent feedback from hip proprioceptors has also been implicated in the control of locomotion in the spinalized cat. Because of this concurrence, we postulated that extensor spasms that are triggered by hip extension might involve activation of organized interneuronal circuits that also have a role in locomotion. If true, imposed oscillations of the hip would be expected to produce activity of the leg musculature in a locomotor pattern. Furthermore, this muscle activity would be entrained to the hip movement.

The right hip joints of 10 individuals with chronic SCI, consisting of both complete (ASIA A) and incomplete (ASIA B,C) injuries, were subjected to ramp and hold (10s) movements at 60°/s and sinusoidal oscillations at 1.2, 1.88, and 2.2 rad/s over ranges from 40° to –15 ° (±5°) using a custom servomotor system. Surface EMG from seven lower extremity muscles and sagittal-plane joint torques were recorded to characterize the response. Ramp and hold perturbations produced coactivation at the hip, knee, and ankle joints, with a long duration (5-10s). Sinusoidal perturbations yielded consistent muscle timing patterns that resulted in alternating flexor and extensor joint torques. EMG and joint torques were commonly entrained to the frequency of movement, with rectus femoris, vastus medialis and soleus activity coinciding with hip extension and medial hamstrings activity occurring during hip flexion. Individual muscle timing patterns were consistent with hip position during normal gait, except for the vastus medialis. These results suggest that reflexes associated with extensor spasms may occur through organized interneuronal pathways, such as spinal centers for locomotion.
Introduction

Extensor spasms in individuals with chronic spinal cord injury (SCI) have been postulated to be the result of excitation of organized spinal interneuronal circuits, such as those associated with spinal locomotor generators (Schmit and Benz 2002). Locomotor pathways receive strong input from hip proprioceptive cues (Grillner and Rossignol 1978; Hiebert et al. 1996; Kriellaars et al. 1994; Lam and Pearson 2001; Pang and Yang 2000, 2001; Van de Crommert et al. 1998), which is consistent with triggering of extensor spasms by imposed extension movements of the hip (Schmit and Benz 2002). This study will demonstrate that imposed sinusoidal hip movements entrain reflex activity in leg muscle groups in a manner that is consistent with locomotion.

Individuals with chronic SCI often have a secondary condition referred to as spasticity. Spasticity is classically defined as a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex (Lance 1980). While this definition may be appropriate for many populations with neural injuries, it does not encompass many of the clinically-observed components of spasticity in SCI. One specific component of SCI spasticity that is poorly described by this definition is the motor behavior often named “extensor spasms”.

Extensor spasms, which result in substantial reflexive muscle activity, are commonly observed in the clinic during movement of SCI patients from a sit to a supine position (Kuhn 1950; Little et al. 1989). The sit to supine movement consists largely of a bilateral hip extension, which has lead to experimental reproduction of spasms using controlled unilateral extension of the hip (Schmit and Benz 2002). The response to this trigger consists of a multijoint reflex and is characterized by hip flexion, knee extension, and ankle extension torques. Since this is a multijoint response, it cannot be attributed to stretch reflexes alone; therefore, extensor spasms must be mediated through polysynaptic pathways involving activation of organized interneuronal circuits located within the isolated spinal cord that are modulated by hip proprioceptive input.
Because of the dependence on hip afferents, it has been postulated that the multijoint extensor reflex response is associated with interneuronal pathways that make up spinal locomotor circuits. Adults with chronic SCI have illustrated evidence for hip afferent modulation of locomotion during partial weight supported treadmill training, specifically associated with enhanced swing following pronounced imposed hip extension at the end of stance (Dietz et al. 2002b; Dietz et al. 1998; Dobkin et al. 1995; Harkema 2001). Locomotion is also modulated by hip proprioceptors in human infants walking on a treadmill, again with swing enhanced by exaggerated hip extension during terminal stance (Pang and Yang 2000). These observations are consistent with animal experiments in which the swing phase of the locomotor cycle is initiated and modulated by the hip afferent input from hip flexor muscles in the spinalized cat (Grillner and Rossignol 1978).

Entrainment of alternating flexor and extensor activity also occurs with the mechanical application of a sinusoidal movement pattern at the hip of the spinalized cat. The entrainment pattern is consistent with the frequency of the sinusoidal oscillation (Andersson and Grillner 1983; Conway et al. 1987; Kriellaars et al. 1994). Previous tests of hip-triggered reflexes in human SCI show a strong prolonged response when the limb is moved into extension and held; however, significant cocontraction is observed, raising questions about whether the characteristic torque response of hip flexion, knee extension, and ankle extension is the result of global muscle activation, with the strongest muscle groups having the dominant torque response (Schmit and Benz 2002).

In the current study, we tested whether sinusoidal oscillations of the hip in chronic human SCI would produce entrainment of muscle activity of the leg, similar to studies in spinalized cats. Power spectral analyses of both torque and smoothed EMG responses were conducted to examine entrainment. Resultant vector analysis in a polar coordinate system was used to determine the relative phasing of leg muscle activity during the imposed movements. The sinusoidal responses were contrasted to the response to ramp and hold perturbations, and compared to typical locomotor patterns to identify the similarities and differences with walking. We postulated that similarities to
locomotion would exist due to the excitation of common neural pathways in normal walking and during extensor spasms in chronic SCI.
Materials and Methods

Subjects

Ten subjects with chronic SCI were recruited into this study. Inclusion criteria included a history of SCI (> four mo.) with associated spasticity. Participants (mean age: 30.7 ± 6.94, range: 19-42) included four clinically complete (American Spinal Injury Association (ASIA) classification A) and six clinically incomplete (ASIA B or C) individuals with cervical (six or seven subjects) or thoracic (three or four subjects) SCI (Table 1). At the time of the study, five of the ten subjects were prescribed anti-spastic medications to reduce the intensity and frequency of spasms. Exclusionary criteria included: multiple central nervous system lesion sites or secondary lesions of the cord, the presence of significant complications such as skin breakdown, urinary tract infection, other secondary infections, heterotopic calcification, respiratory failure or other concurrent illness limiting the capacity to conform with study requirements, the inability to give informed consent and significant osteoporosis. Informed consent was obtained and all procedures were conducted in accord with the Helsinki Declaration of 1975 and approved by the Institutional Review Board of Marquette University.

Please insert Table 1 near here

Test Apparatus

A novel apparatus, shown in Figure 1, was constructed for measuring the multijoint torque response to imposed movements at the right hip. This apparatus included a knee-ankle brace with built-in torque transducers aligned with the axes of rotation of the knee and ankle. A footplate included a clamp to be placed on the dorsum of the foot and a strap to secure the heel. The hip-knee and knee-ankle links were adjustable to fit a wide range of leg sizes. The entire leg brace was affixed to a newly constructed velocity controlled servomotor drive system (Kollmorgen MT704A1-R1C1,
Northampton, MA). Hip torque, knee torque and ankle torque were measured using hollow-flanged transducers (Himmelstein, Inc., Hoffman Estates, IL). Position of the hip joint was measured using a potentiometer coupled to the servomotor drive shaft.

*Please insert Figure 1 near here*

Surface EMGs were recorded from the tibialis anterior (TA), soleus (Sol), medial gastrocnemius (MG), vastus medialis (VM), rectus femoris (RF), medial hamstrings (MH), and hip adductors (Add) of the right leg in all subjects. ElectroTrace ET001 (Jason, Huntington Beach, CA) Ag/AgCl snap type diposable hydrogel monitoring electrodes were placed over the belly of each muscle on clean, slightly abraded skin in a bipolar arrangement. The Octopus AMT-8 (Bortec, Calgary, Alberta Canada) EMG system was used for EMG amplification (500–1500x) and filtering (10-500 Hz).

All signals were low-pass filtered (450 Hz), and sampled at 1000 Hz using a data acquisition card (National Instruments, Austin TX) on a personal computer. Custom LabVIEW software (National Instruments) was used for acquiring the data as well as outputting the velocity command signal for the servomotor system.

**Imposed Hip Movements**

Movements were imposed to the right hip of all ten subjects. Each subject was transferred to a tri-section therapy table and placed in a supine position. The center of rotation of the right hip joint was aligned with the axis of rotation of the servomotor system and the brace was adjusted to align the ankle and knee with the appropriate torque transducer. Alignment of the hip was confirmed by a lack of leg translation during imposed flexion and extension of the hip. The pelvis was secured to the table with a strap across the iliac crest to inhibit pelvic rotation. The foot was placed in a footplate and secured using a clamp placed on the dorsum of the foot and the heel. The leg was placed in the
brace with the knee at 10-30° flexion and the ankle at 20-25° plantarflexion, as summarized by Table 2. The initial knee angle was set to 30° flexion. Manual hip extension perturbations were made to elicit an extensor spasm response. If no response occurred, the knee was extended further, which usually helped in eliciting a larger response. After the ankle and knee angles were set, these joints were held isometric for the duration of the test. The contralateral limb was supported in a slightly flexed position at the hip.

Please insert Table 2 near here.

Controlled hip movements were imposed by the Kollmorgen servomotor system. The start position was set at 40° of hip flexion and the end position at 10-20° of hip extension, a position that corresponded to maximum hip extension. As a result, a slightly different hip range of movement was used for each subject. Figure 2 illustrates the motion of the hip relative to the sagittal coordinate system. Two types of imposed of hip movements were made: sinusoidal oscillations at three separate frequencies and ramp stretch and hold movements. During the ramp and hold sequences the motor moved the hip at a constant velocity of 60°/s for three cycles, holding for ten seconds in both flexion and extension. During the sinusoidal oscillations, the hip was moved through ten cycles at frequencies of 1.2 [lowest], 1.88 [medium], 2.2 rad/s [highest] (~0.2, 0.3, and 0.35 Hz) with no pause in extension or flexion. These frequencies roughly correspond to stepping speeds of approximately 0.21, 0.31, and 0.36 m/s, respectively. Table 2 displays the peak velocities as they corresponded to the different subject range of motions. Note that the peak velocity varied between subjects since the frequency was controlled and the hip range of motion was subject-dependent. A timed delay of 3 minutes was allowed between trials. The protocol began with a ramp stretch movement followed by three sinusoidal movements with the 3 frequencies applied in random order. This format was
repeated for a total of 3 ramp stretch movements and 9 sinusoidal movements. At end of the twelve trials, the subject’s leg was moved slowly in extension at 2 deg/s increments to measure the passive torque of the hip. Also, the leg was oscillated from 40º flexion to 20º flexion at frequencies of 3 rad/s and 5 rad/s to identify the inertial properties of the leg.

*Please insert Figure 2 near here.*

**Analysis**

**Ramp Stretch Movements**

Joint torque data were obtained for the hip, knee, and ankle during the 10 s hold periods, with the hip in the maximum extended or flexed position. The gravitational/passive torque offset was removed by subtracting a torque measurement in the end position, when there was no muscle activity (e.g. at the end of the hold period). The signals were then low-pass filtered (LPF, 5 Hz) using a 4th order Butterworth filter (*butterfiltfilt; Matlab command;* The Math Works, Inc., Natick, MA) and plotted against time. The pattern of muscle activity was identified for each subject and related to the measured reflex torque. The duration of the torque response was calculated using a threshold of ±3 Nm for the hip, ±1 Nm for the knee, and ±0.5 Nm for the ankle. These values were used because they eliminated inclusion of signal noise related to resonant vibration during the isometric reading. The resulting values represented the reflex torque at each joint and were used as a measure of the net reflex response at each joint. Rectified EMGs were evaluated to detect the timing of muscle activity during and following imposed hip movements.

**Sinusoidal Movements**

Torque data for the hip, knee, and ankle were acquired during the entire movement cycle. In order to calculate the reflexive hip torque, the gravitational torque, passive resistance, and inertial torque were removed. The effects of gravity, passive joint resistance and inertia were each calculated
using a separate set of hip perturbations. Once determined, the net reflex response was calculated by subtracting the inertia, gravity and passive torques from the torque measured during the oscillations.

Passive resistance and gravitational torque of the leg were determined by moving the leg throughout the entire range of motion at 2°/s pausing for 2-10 sec for a total 20 increments throughout the range of motion. The mean torque signal was calculated during each pause, and thus the resulting signal contained only the passive and gravitational torque. Gravitational torque alone was calculated from the mean torque measurements collected in the middle of the range of motion, during which time the passive resistance was negligible. A cosine function, multiplied by a scalar, ($K_{leg}$) was fit to the torque data using a least squares regression (backslash operator; Matlab command), resulting in an estimate of the gravitational torque (Equation 1). The passive resistance of the hip joint was then calculated by fitting a 3rd order polynomial (polyfit/polyval; Matlab command) to the mean torque data, with the gravitational torque subtracted (Equation 2).

$$
\tau_{gravity} = K_{leg} \cos(\theta_{hip})
$$

(Equation 1)

$$
\tau_{passive} = (a_3 \theta_{hip}^3 + a_2 \theta_{hip}^2 + a_1 \theta_{hip} + a_0)
$$

(Equation 2)

The inertial properties of the leg were estimated from hip torque data obtained during separate oscillations of the leg from 40° flexion to 20° flexion at 3 rad/s and 5 rad/s. By keeping the hip in a flexed position, no muscle activity was elicited and no passive resistance was encountered; therefore, the inertial constant, $I_{leg}$, could be calculated by correcting for gravity only (Equations 3a, 3b, 3c). $I_{leg}$ was determined through least squares regression (backslash operator; Matlab command).
From the gravitational constant $K_{\text{leg}}$, the inertial constant $I_{\text{leg}}$, and the 3rd order polynomial coefficients associated with the passive resistance, the active torque was calculated for each trial using Equation 4.

\[
\tau_{\text{inertia}} = \tau_{\text{measured}} - \tau_{\text{gravity}}
\]

\[
I_{\text{leg}} \ddot{\theta}_{\text{hip}} = \tau_{\text{measured}} - K_{\text{leg}} \cos(\theta_{\text{hip}}) \quad (3b)
\]

\[
I_{\text{leg}} \ddot{\theta}_{\text{hip}} = \frac{\tau_{\text{measured}} - K_{\text{leg}} \cos(\theta_{\text{hip}})}{\ddot{\theta}_{\text{hip}}} \quad (3c)
\]

The active torques at the knee and the ankle were calculated by correcting only for gravity and inertial artifacts of the foot-shank and foot, respectively. The passive resistance of the knee due to biarticular muscles of the hip and knee were assumed to be negligible (<2 Nm), a result supported by subjective observations of knee torque during slow trials into hip extension. The resulting torque measurements represented the reflex response at each joint. These biomechanical measurements of the responses were absolute measures of the leg output, which could be compared across subjects to determine the relative size of the response. The disadvantage of the torque measurements is that they could not account for muscle coactivation at the joints, which required EMG measurements for interpretation.

For analysis, smoothed rectified EMG signals were calculated. Surface EMG’s were rectified and enveloped using a 4 Hz, low-pass, 8th order Butterworth digital filter (butter/filtfilt; Matlab command). EMG signals were then summed over the last five cycles, normalized to the
maximum and plotted in a polar coordinate system, where 180° represents full extension of the leg. The last five cycles were used in this analysis to eliminate the effects of transient modulations in the total responses, which were occasionally observed in the first five cycles.

**Frequency Analysis**

In order to determine whether oscillation of the hip produced reflex responses that were entrained to the movement, the frequency content of the reflex response was identified. Both smoothed rectified EMG and torque signals were examined for spectral content using a Discrete Fourier Transform (DFT) method that squares the magnitude of the DFT within Matlab *(periodogram; Matlab command)*. All data was zero-padded to length of $2^{17}$ (131072). A rectangular window was used to obtain the best resolution. The maximum spectral peak was obtained from the power spectral density estimate (PSD). The maximum spectral peaks were then examined and compared with its corresponding time series signal in order to determine whether the peak occurred at the frequency of the movement. Similar peak frequency content was used to indicate entrainment of the response to the movement.

**Phase Analysis**

Muscle timing patterns during each sinusoidal cycle were examined using circular statistics according to methods outlined by Batschelet (Batschelet 1981). Phase analysis of EMG signals was only pursued if the EMG signals demonstrated sufficient muscle activity. Amplified EMG noise levels typically did not exceed 5mV; therefore, a threshold of 10mV for enveloped and rectified EMG was utilized as the criterion for including individual trials in the analysis. Similarly, a threshold of ±5 Nm for hip extension/flexion, ±4 Nm for knee extension/flexion, and ±1 Nm for ankle extension/flexion were implemented for inclusion of reflex torque signals. This threshold was set so as not to include noise associated with the sinusoidal torque calculation of active muscle torque.
Phase analysis consisted of using rectified and smoothed EMG signals that were normalized from 0 to 360, where 180 represents full extension of the hip. The signals were then plotted in a polar coordinate system. For each movement cycle, the Cartesian coordinates of the resultant vector for each muscle were calculated using equations 5a and 5b. From these x, y coordinates the polar angle was found using equation 5c. The vector length, r, was then normalized to a unit vector size in a conversion back to x, y coordinates. After normalization, the mean polar angles and vector length, r, were calculated (Equations 5c and 5d) across trials of similar frequency if the criteria for a minimum response (see above) were met. The mean polar angles and vector lengths were then used for a phase analysis. Similar analysis was performed on joint torque signals from sinusoidal trials. The torque signals were first half-wave rectified in both flexion and extension, then analyzed to find the mean polar angle of the resultant vector using the aforementioned procedure.

In order to determine whether there was a significant phasing in EMG and torque signals, Raleigh’s test for one-sidedness was performed (α=0.05). All data sets that showed a significant trend were then plotted in polar coordinates, where the vector length, r, was used to determine significance (Batschelet 1981).

\[
\bar{x} = \frac{1}{n}(\cos(\varphi_1) + \cos(\varphi_2) + ... + \cos(\varphi_n)) \quad (5a)
\]
\[
\bar{y} = \frac{1}{n}(\sin(\varphi_1) + \sin(\varphi_2) + ... + \sin(\varphi_n)) \quad (5b)
\]
\[
\bar{\varphi} = \arctan\left(\frac{\bar{y}}{\bar{x}}\right) \quad (5c)
\]
\[
r = \sqrt{\bar{x}^2 + \bar{y}^2} \quad (5d)
\]

**Amplitude Analysis**

The peak hip flexion, knee flexion, ankle flexion, hip extension, knee extension, and ankle extension torques were calculated for each movement trial, in each subject to identify the effect of movement frequency on the amplitude of the response. The mean of the three 1.2 rad/s, 1.88 rad/s, and 2.2 rad/s trials were calculated for each subject. A two-factor ANOVA was used compare the
effect of movement frequency and subject on the mean torque for each joint in order to determine whether the magnitude of the reflex response was velocity dependent.
Results

Ramp and Hold Movement Responses

In this study, EMG and reflex torque responses were measured during both ramp-hold and sinusoidal hip movements in order to illustrate that sensory input from the hip joint is important in modulating the timing and duration of leg muscle activity in chronic human SCI (n=10). Typical EMG and torque responses for both movement types are shown in Figure 3. Note that EMG responses occasionally exhibited bursting activity, consistent with clonus (Beres-Jones et al 2003). In the EMG analysis, the signals were rectified and low pass filtered at 4Hz, effectively filtering out this clonic response.

Please insert Figure 3 near here.

Ramp and hold extension of the hip produced net hip flexion torque in all 10 subjects. Knee extension torque was generated in 5 of 10 subjects (Subjects 4, 5, 6, 7, & 10), while the other 5 subjects showed no knee torque response. Ankle torques had more variability than hip and knee responses: 4 subjects had ankle extension torque (Subjects 1, 3, 6, & 9), 2 produced ankle flexion torque (Subjects 4 & 5), 2 exhibited alternating flexor and extensor torque (Subjects 7 & 10, e.g. Figure 3) and 2 had no response. In addition, reflex responses to ramp and hold flexion movements occurred in 6 of the 10 subjects (Subjects 1, 2, 5, 6, 7, & 10). The typical torque response to ramp and hold hip flexion was hip extension (Subjects 1, 2, 5, 6, 7, & 10), knee flexion (Subjects 5, 6, 7 & 10), and variable ankle responses (pure extension (Subjects 7 & 10), both flexion and extension (Subject 6)).

Typically, the duration of the reflex response to hip extension was significantly reduced when a flexion was immediately imposed to the hip. In Figure 3 it can be observed that both the EMG and
torque responses were prolonged when holding the leg in extension. In fact, 7 of the 10 ten subjects with hip flexion responses produced a hip flexion torque of greater than 3 Nm for longer than 5 seconds. In addition, 4 out of 5 subjects with knee extension responses held knee extension torques above 3 Nm for over 4 seconds. Ankle extension responses were held above 1 Nm for more than 3 seconds in 3 of 6 subjects that exhibited ankle extensor torques. In 4 subjects, hip flexion moments were held above 3 Nm for the entire ten seconds that the limb was held in extension. These prolonged responses ceased when the limb was moved into flexion. This cessation of hip flexion and knee extension torques upon flexion of the limb were consistent with the results seen with the sinusoidal perturbations, in which the duration of both the EMG and torque response were equal to the period of the imposed sinusoidal oscillation (see next section).

**Torque and EMG during Sinusoidal Oscillations**

The duration of the torque and EMG responses during sinusoidal oscillations was dependent on the frequency of the movement. Typically, the EMG and torque responses were entrained to the frequency of the oscillation. These observations were quantified by identifying the frequencies of the peaks of the power spectrum of each signal.

In general, the maximum spectral peak for the torque response occurred at the frequency of the movement in all ten subjects; however, there were exceptions for individual trials in Subjects 1, 4, 5, 6, 7, 9 and 10. Figure 4 illustrates the power spectrum for hip, knee, and ankle torque responses during a single trial in Subject 7. Peak A corresponds to the frequency of the movement. In Figure 4 this peak was maximal in hip and knee torque responses. Peak A was the maximum spectral peak in 88 of 90 hip, 90 of 90 knee, and 59 of 90 ankle movement trials exhibiting entrainment. The number 90 represents the total number of trials across all subjects and frequencies (3 frequencies x 3 trials x 10 subjects). Peak B (Figure 4) corresponds to twice the frequency of the movement. This peak was occasionally the largest (ankle responses (4 of 90), hip (2 of 90) and knee (0 of 90)). Finally, peak C
corresponds to a lower frequency component. This phenomena was observed at the ankle joint in 27 of 90 trials. Figure 4 shows that this low frequency response corresponded to a large amplitude, long duration ankle plantar flexion (extension) that increased during the first 3 cycles. It was also observed that the high frequency peak (peak B) was larger than the power at the oscillation frequency (peak A) in a majority of the cases where peak C was the maximum. The torque vs. hip angle plots in Figure 4 illustrate that in cases where a large, high frequency peak (peak B) was observed, the ankle created an extension moment upon both flexion and extension of the hip, accounting for the location of the spectral peak at double the oscillation frequency.

Spectral analysis of smoothed rectified EMG responses was generally consistent with the joint torque, usually producing peak spectral peaks at the frequency of movement. Maximum spectral peaks were found at the frequency of the movement in at least one muscle for 8 of 10 subjects, for all three movement frequencies. Out of a total of 90 trials, the RF had a spectral peak at the frequency of the movement for 38 trials, the MH 57 trials, the VM 25 trials, the Sol 30 trials, the Add 13 trials, and the MG and TA 9 trials each. Maximum power at frequencies corresponding peak C were most common in the MG (27 of 90) and TA (26 of 90).
EMG and Torque Phase Analysis in Sinusoidal Oscillations

Muscle activity was synchronized with movement in cases where significant EMG signal was recorded. Surface EMG recordings demonstrated unambiguous muscle activity in 8 of 10 subjects (smooth rectified activity > 10mV). For example, EMG from the Sol, VM, RF, and MH for five hip movement cycles is illustrated in Figure 5. The last five cycles were used for additional analysis because there were some cycle-to-cycle changes in the reflex torque response over the first five cycles. These changes are demonstrated in hip, knee, and ankle torque responses in Figure 4a,d,g. The EMG responses were rectified, smoothed and plotted on a polar plot, as shown in Figure 6A.

Please insert Figures 5 and 6 near here.

The most common pattern of muscle activity was comprised of RF, VM and Sol activity corresponding to hip extension and MH activity corresponding to hip flexion, although occasional exceptions were noted. Figure 6A is an example of the most commonly observed response pattern, in which the activity in the RF, VM, and Sol began immediately after the leg began movement into extension. In fact, a significant phasing of the RF activity with hip extension was observed in 6 of 8 subjects, the VM activity in 5 of 8 subjects, and the Sol activity in 4 of 8 subjects, according to Raleigh’s test for one-sidedness (p<0.05). Conversely, the MH began to fire after the limb moved into flexion and continued until activity in the RF, VM, and Sol was initiated.

There were two exceptions to the muscle activity pattern shown in Figure 6A. Figure 6B is an example of one exception, from Subject 10. In this case the VM and MH were in phase with each other during the flexion phase of the cycle. In addition, VM and RF activity had peaks just before peak extension occurred. The RF also had a peak just after full extension. The RF activity began to subside after the MH began to fire. Both Subject 6 and 10 showed this response; however, the
response was only observed occasionally (Subject 6:7 of 9 trials, Subject 10:5 of 9 trials). The VM was out of phase with the MH in the remainder of the trials.

The polar angles of the mean resultant vector for the EMG signals, which were used to determine the overall phasing each muscle, also demonstrated a phasing of the RF, VM and Sol activity with hip extension and MH phasing with hip flexion. Figure 7 demonstrates the angular locations of the resultant vectors, for the 2.2rad/s, 1.88rad/s, and 1.2rad/s movement frequencies, for trials that met Raleigh’s test criteria (p<0.05) (see Methods for other exclusion criteria). A mean resultant vector phase angle of 180 corresponds to muscle timing in phase with hip extension movements. The phase plots illustrate that for the majority of trials, the RF, VM, and Sol are in phase with the hip extension movement. The majority of MH activity is in phase with the hip flexion movements with only a slight phase lead. As Figure 7 illustrates TA, MG, and Add muscle activity did illustrate patterns that were consistent across subjects. The number of subjects that yielded significant patterns for one-sidedness was especially small in the MG and TA.

_A similar resultant vector analysis, which was performed on the half-wave rectified torque signals, suggested that hip flexion torques and knee extension torques were in phase with the hip extension movements. Figure 8 shows the locations of the resultant vectors for unambiguous responses that satisfied Raleigh’s test criteria for one-sidedness (p<0.05) during the 2.2rad/s, 1.88rad/s, and 1.2rad/s movement frequency trials. The knee extension and hip flexion torque signals were in phase with hip extension movements. Conversely, knee flexion and hip extension torques were in phase with hip flexion movements. Ankle torque phasing patterns were less conclusive. Ankle extension torques only occurred with a significant phasing in two subjects._

Please insert Figure 7 near here.
occurring during hip extension while ankle flexion torques occurred with significant phasing (hip flexion) in three subjects. These results are consistent with the spectral analysis in which the ankle often produced extension in response to both hip flexion and hip extension. This would be expected to produce negligible phasing of the ankle torque in the vector analysis since Raleigh’s test for one-sidedness would not detect for this bimodal pattern.

Please insert Figure 8 near here.

**Frequency/Velocity Dependence**

There was a large disparity in the magnitude of the joint torque responses across subjects. Figure 9 shows the mean peak torque and standard deviations for hip, knee, and ankle torques for each frequency of the oscillation for all ten subjects. Positive torque represents extension and negative values correspond to flexion responses. From Figure 9, subjects 4, 5, 6, 7, 9, and 10 yielded to largest amplitude responses regardless of joint or direction.

Please insert Figure 9 near here.

Although the EMG and torque phasing showed no dependence on the frequency (speed) of the movement as visualized from Figures 7 and 8, the amplitudes of the torque responses were significantly affected by the movement frequency. The mean peak hip flexion, knee flexion, ankle flexion, hip extension, knee extension, and ankle extension torques for each movement frequency were determined for each subject. A two factor ANOVA using subject and frequency as test factors indicated that subject had a significant effect on the torque in every joint (p<0.001). Significant effects of frequency were observed for hip extension (p<0.10), knee flexion (p<0.10) and hip flexion (p<0.01). Interaction effects were negligible (p>0.10). These population effects are summarized in
Figure 10. The amplitude of the EMG responses also appeared to depend on the frequency of hip movement in many cases; however, due to a low number of qualified trials, this effect was not tested statistically.

Please insert Figure 10 near here.

Discussion

The majority of subjects in this study yielded organized EMG responses to imposed sinusoidal movements of the hip. This was evidenced by significant ($p<0.05$) phasing of EMG patterns of activity in the RF (6 subjects), VM (5 subjects), MH (6 subjects), and Sol (4 subjects). In addition, torque responses consistently alternated between flexion and extension at the hip and knee, with durations that were similar to with the period of the sinusoidal oscillation. The EMG burst durations were also dependent on the period of the oscillation as illustrated by similar activation patterns across frequencies (Figure 7). The phasing of the EMG activity was dependent on the position of the limb in all measured lower extremity musculature, illustrating an organized response that depended on afferent input from the hip.

The results from imposed hip oscillations contrasted with the responses to ramp and hold perturbations of the hip. Ramp and hold movements produced reflex activity similar to previously reported results (Schmit and Benz 2002), consisting largely of hip flexion, knee extension and ankle extension torques in response to hip extension and predominant co-activation of the ankle and knee musculature, as reflected in the EMG data. These results were similar to patient descriptions of extensor spasms. Conversely, the organization of the muscle activity patterns during hip oscillation appeared to depend upon continued movement of the hip.

Role of Interneurons in the Extensor Spasm Response
Spastic reflex behaviors, which have traditionally been attributed to velocity-dependent homonymous stretch reflexes (Ashworth 1964; Lance 1980), include hyperexcitable multijoint reflexes in chronic human SCI. The traditional definition of spasticity as a hyperexcitable stretch reflex has proven to be adequate when describing spasticity in many types of central nervous system disorders such as stroke (Schmit 2001), cerebral palsy (Engsberg et al. 2000), and multiple sclerosis (Sinkjaer et al. 1993). In contrast, SCI individuals also demonstrate other spastic reflexes, such as flexor spasms which have been associated with an increased flexor reflex response to skin stimuli (Dimitrejevic and Nathan 1968; Shahani and Young 1971) or movement of the ankle (Schmit et al. 2000). Similarly, coactivation of muscles across several joints has been observed in response to a number of stimuli (Beres-Jones et al. 2003; Dimitrejevic and Nathan 1967a, 1967b) including imposed hip extension movements (Schmit and Benz 2002). The results from the current study are consistent with multijoint reflex activation playing a role in spasticity. Clearly movement at the hip produces reflex activity of the ankle and knee musculature that is not stretched by the movement (e.g. Figure 3). These observations suggest that spastic reflexes in human SCI involve more than homonymous stretch reflex pathways, implicating interneuronal pathways that have the potential to coordinate the reflex response.

The increased excitability of reflex pathways involving spinal interneurons raises the question of whether single joint movements can trigger organized multijoint responses that have a functional correlate. Similar to previous studies, the ramp and hold stretch responses in the current study illustrate that single joint movements can trigger multijoint responses, including coactivation at the ankle and knee. In addition, peak joint torque was found to increase with increases in speed during hip oscillations (Figure 10), suggesting that the response may be mediated by spindle afferents. In particular, this mechanism may account for activity of the hamstrings, which are stretched (and activated) during hip flexion. In addition to these results, however, we observed that
the EMGs and joint torques became entrained to oscillatory movements (Figures 4-8), with different muscles demonstrating activity in separate phases of the oscillation. In addition, the entrainment occurred in muscle groups that do not cross the hip joint. The entrainment of the activity alone suggests that the reflex produces more than coactivation at the joints, raising the question of whether the interneuronal pathways that are activated in this reflex correspond to some motor function. Locomotor pathways are a potential neural substrate of these reflex responses to hip oscillation.

*Are these Interneuronal Pathways Associated with the Spinal Centers for Locomotion?*

The initiation of the organized responses observed in this current study is likely to be due to hip afferent feedback to the spinal cord as evidenced by the entrainment of the muscle activity (Figures 4, 5, and 7). Hip afferents have also been shown to have important roles in the modulation of locomotion. The spinalized cat model has revealed that the swing phase of locomotion is initiated by extension of the hip (Grillner and Rossignol 1978). In addition, transitions from stance to swing in the human infant are modulated by hip joint afferents (Pang and Yang 2000, 2001). Our results show that during an imposed extension of the hip, the hip produces a flexion moment (Figure 8), which is a muscle pattern consistent with the initiation of swing.

Fictive locomotion is influenced by hip afferents, in a similar manner. Imposed sinusoidal oscillation to the hip joint during fictive locomotion of the immobilized spinal (Andersson and Grillner 1981, 1983; Conway et al. 1987) and decerebrate cat (Kriellaars et al. 1994) entrain alternating flexor and extensor muscle activity to the frequency of the movement. The torque and EMG responses to imposed hip oscillations in the current study were also entrained to the frequency of the movement, as demonstrated by dominant power in the EMG and torque signals at the frequency of the movement (Figure 4), suggesting a neural mechanism similar to the one implicated during fictive locomotion in the cat. In contrast, the duration of the ramp and hold responses lasted
several seconds and cocontraction of the hip, knee, and ankle musculature was more prominent than cocontraction during sinusoidal responses.

Hip afferent input has also been shown to be one factor that is important in the modulation of alternating flexor and extensor rhythms in adult humans with chronic SCI. Involuntary locomotor-like activity was found in a single incomplete SCI subject (Calancie et al. 1994). This activity was induced when moving from a sit to supine position, with the knees fully extended. This activity suggests that the spinal center for locomotion is modulated by hip afferent input. Other studies have provided anecdotal evidence of alternating flexor and extensor activity after sit to supine movements (Kuhn 1950). These observations are consistent with modulation of locomotor rhythms through hip afferent input during body weight supported treadmill training of patients with chronic SCI (Dobkin et al. 1995) as well as with the observed modulation of muscle activity with hip oscillation in the current study.

Many studies have been conducted that demonstrate that reflex pathways associated with locomotion are recruited using a treadmill, partial body weight support, and the assistance of physical therapists moving the legs in a gait pattern (Dietz et al. 1995; Dietz et al. 2002a; Dietz et al. 1998; Dobkin et al. 1995; Harkema et al. 1997). During these activities, it has been noted that extension of the limb produces an involuntary hip flexion that initiates swing (Dobkin et al. 1995). These results are consistent with the hip torque produced during this experiment; however, the knee extension torque observed with imposed hip extension was inconsistent with swing, rather, it is typically more closely associated late stance phase. Figure 11 outlines the EMG activity differences between non-SCI gait (Perry 1992), SCI gait (Dobkin et al. 1995, Pepin et al. 2003), and the unilateral sinusoidal oscillations in this study. There are clear similarities in the EMG patterns for the MH, Sol, and RF. The quadriceps (VM/ VL) activity in all cases was, however, much more prolonged and in most cases completely out of phase with what is seen in non-SCI gait. A possible reason for this difference in activity pattern is the absence of other afferent input to the spinal locomotor centers.
Role of Other Afferent Input to the Involved Interneuronal Pathways

The spontaneous alternating flexion and extension movements triggered by sit to supine movements discussed earlier (Calancie et al. 1994) were manifested only if the hip extension occurred while the knees were extended. In this current study, reflex responses would not occur if the knees were flexed beyond a critical angle. For example, responses typically occurred if the knee was flexed to about 30°; however, in some cases the knee had to be extended further in order to elicit a response. In one case the knee was extended to 10° flexion in order to get a response. This is evidence that knee proprioceptors may play a role in the modulation of this activity. The knee proprioceptors have not been shown to play a large role in modulation of locomotor rhythms in the spinalized cat preparation; however, knee proprioceptors may be more important in human gait because of the differences in bipedal and quadrupedal locomotion and the need for an enhanced support mechanism at this joint. In human SCI, locomotor activity can be produced with “hip-only” walking, with the knees held in an extended posture (Dietz 2002b), suggesting that knee proprioceptors might not play a critical role in regulating locomotion. It is unknown whether hip afferents influence locomotor activity with the knees held in a more flexed posture.

Unlike knee proprioceptors, limb load afferents have proven to be an important modulator in the spinal centers for locomotion. Spinalized cat preparations have illustrated that swing phase will not initiate if the contralateral limb is not in position to bear load (Grillner and Rossignol 1978). Also, stepping infant studies have illustrated that the load on the ipsilateral limb must be decreased to initiate swing phase (Pang and Yang 2000). Chronic spinal cats also illustrate the ability to correct for loss of ground support in the ipsilateral limb during treadmill walking. The cat achieves this correction by flexing the ipsilateral limb upon stepping into a hole in the treadmill. The contralateral
leg extends the knee and hip to help support the body until the ipsilateral limb is ready for weight support (Hiebert and Gorassini 1994). It was concluded that the lack of input from load sensitive afferents in the ipsilateral limb had a major contributing role in this response.

Similar evidence linking ankle load afferents to locomotor modulation has been found in the chronic human SCI. Limb loading during body weight supported treadmill training modulates EMG activity of ankle musculature according to the phases of the gait cycle (Dietz et al. 2002b; Harkema et al. 1997). The unilateral hip extension movements applied in this study did not incorporate an ankle-loading paradigm. We postulate that an absence of the ankle afferent input into the organized interneuronal pathways may cause the difference in the phasing of VM activity in the current study (Figure 11). For example, in the spinalized cat, removal of contralateral limb support, similar to treadmill walking where the contralateral limb steps into a hole, inhibits swing phase in the ipsilateral limb and even produces knee extensor activity (Hiebert and Gorassini 1994). The loading of the ankle at the appropriate time in the sinusoidal cycle would likely affect the phasing of the ankle and quadriceps musculature and needs to be examined further.

*Effects of Intersubject Variability*

Previous studies have also postulated that the level of the spinal cord lesion has an effect on the response (Dietz et al. 1999). This may be due to, in part, to involvement of the cervical spinal regions in the locomotor generating circuits (Dietz 2002a). No conclusions could be drawn about level of injury in the current study because consistent responses were seen in Subject 5 (T6 ASIA A) and Subject 1 (T6 ASIA A) and small responses were found in Subject 3 (C7- T1 ASIA B) and Subject 8 (C4-5 ASIA C). Similarly, no conclusions about whether the location of the lesion or the nature of injury (incomplete vs. complete) had an effect on the response could be made because of the varying amounts of anti-spasm medications that each subject was taking at the time of the study. No significant differences in the magnitude and phase of the torques or EMGs were observed when
comparing subjects on spasticity medications to those without medications (p>0.10), although the sample size was small (n = 4, 5) for this comparison. Despite the lack of significant differences, the subjects with the largest and most consistent responses were not prescribed anti-spasm medications (Subjects 6, 7 and 10), however, Subject 9 yielded a consistent reflex response and this subject was taking baclofen. The effects of spasticity medications on the magnitude and phasing of muscle activity responses to hip oscillations is an important topic for future studies since the response to hip oscillation may be indicative of the clinical incidence of extensor spasms and may impact locomotor training.

Conclusions

This study has illustrated that hip afferent input can modulate the timing of lower extremity muscles in a predictable fashion, but hip proprioceptors alone cannot create consistent locomotor rhythms. The reflex response manifested from sinusoidal oscillations of the hip in subjects with chronic SCI is likely due to organized pathways located within the lumbosacral region of the spinal cord. We believe that these pathways are associated with the spinal centers for locomotion because of the organized timing of flexor and extensor muscles. The alternating flexor and extensor activity was not entirely consistent with locomotor patterns because of the ill-timed uniarticular knee extensor activity, which may be due, at least in part, from the absence of other afferent feedback such as limb load afferents. Locomotor training is becoming a very important tool in the rehabilitation protocol in human SCI. Proper afferent input must be present during this training in order to obtain appropriate muscle activation synergies.

Acknowledgements
This project was funded by NIH grant NS40901 and a research grant from the Whitaker Foundation.


**Figure/Table Legends:**

**Table 1:** Subject information indicating age, injury level, ASIA class, year of injury, and medications the subjects were prescribed at the time of the study.

**Figure 1:** Kollmorgen servomotor system with leg brace attachment. The servomotor drive system actuates the leg from the hip joint while the knee and ankle joints are held isometric by the leg brace. Reaction torque transducers at the hip, knee, and ankle measure the joint torque response to the imposed movement.

**Table 2:** Subject test parameters indicating range of motion, isometric knee and ankle angles, and peak sinusoidal movement velocities.

**Figure 2:** Relative motion of the limb during imposed hip movements. The leg is oscillated from 40º of hip flexion to 15º (±5º) of hip extension.

**Figure 3:** Sample data for subject 10 are shown. The top plots indicate the hip movement trajectories. The subsequent traces are typical EMG responses for sinusoidal (left) and ramp/hold (right) hip movements. The bottom plots are the torque produced at the hip, knee, and ankle joints for the respective movements. Extension torque is positive and flexion torque is negative.

**Figure 4:** Figures 4a-c represent the hip torque response (Nm) vs. time (sec), the frequency response of the hip torque time series, and the hip torque (Nm) vs. hip angle ( ) relationships for Subject 7, respectively. Figures 4d-f, and 4g-i represent the same relationships for knee and ankle torque responses, respectively. Peak A corresponds to the frequency of the imposed hip movement. Peak B corresponds to frequency components twice the frequency of the movement. Peak C represents a frequency component associated with low frequency modulation of torque amplitude.

**Figure 5:** EMG (V) vs. time (sec) and associated hip motion profile for 5 cycles of a single 2.2 rad/s frequency trial in subject 7. The MH activity is in phase with imposed hip flexion movements. The Sol, RF, and VM show activity during imposed hip extension movements.

**Figure 6:** Figures 6A and 6B display normalized (maximum) smoothed and rectified Sol, RF, VM, and MH plotted on a polar plane (r=1) over last 5 cycles, where 180 corresponds to maximum hip extension. 6A: Representative 1.88rad/s frequency trial from Subject 7. Sol, VM, RF begin activation upon hip extension and cease upon flexion. MH activates upon hip flexion. 6B: Representative 1.88rad/s frequency trial from Subject 10. Subject 10 illustrated similar activity to Subject 7 in Sol and MH. VM had activity components in phase with MH.

**Figure 7:** Significant muscle phasing patterns (Raleigh test, α<0.05) for all EMG activity during 1.2 rad/s (dark-gray), 1.88 rad/s (light gray), and 2.2 rad/s (black) movements is shown. A phase angle of 0 deg corresponds to hip flexion. An angle of 180 deg corresponds to maximum hip extension. The vectors on the polar plots indicate timing of muscle activity during the movement (direction) and consistency of phasing (radius, r=1 is maximum). The number of subjects, n, illustrating these patterns are listed next to their respective muscle plots.

**Figure 8:** Significant torque response phasing patterns (Raleigh test, α<0.05) for all unambiguous responses during 1.2 rad/s (dark-gray), 1.88 rad/s (light gray), and 2.2 rad/s (black) movements. The vectors on the polar plots indicate timing of the responses during the movement (direction) and
vector strength estimate (radius, r= 1/ max). The number of subjects, n, illustrating these patterns are listed next to their respective plots.

**Figure 9:** Mean peak torque for similar frequency trials (n=30) and standard deviations across the hip, knee, and ankle joints. Flexion responses are negative, and extension responses are positive for 1.2 rad/s (white), 1.88 rad/s (dark gray), and 2.2 rad/s (light gray) frequencies. The figure reveals that Subjects 4, 5, 6, 7, 9, and 10 yielded the largest amplitude responses, regardless of joint.

**Figure 10:** Mean peak reflex torques 2.2 rad/s, 1.88 rad/s, and 1.2 rad/s movement frequencies are shown for each joint torque. Frequency has a significant effect on hip extension, hip flexion and knee flexion torque responses. The trend was similar in all joint torques. (* p<0.10) (** p<0.01)

**Figure 11:** Estimated comparison of muscle activity patterns in various lower extremity muscles during non-SCI (light gray) (Perry 1992), SCI gait (black) (Dobkin et al. 1995, Pepin et al. 2003), and the imposed unilateral sinusoidal pattern in this current study (gray). The x-axis represents a percentage of the gait/sinusoidal cycle. The vertical dotted line marks the transition from stance to swing. The bars represent the timing patterns for representative data sets of the different paradigms. The gray-hatched bar indicates the VM pattern observed in Subjects 6 and 10.
### Table 1

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### Table 2

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Figure 1
Figure 2
Figure 3
Figure 6
Figure 7
Figure 8

- Hip Flexion
- Knee Flexion
- Ankle Flexion
- Hip Extension
- Knee Extension
- Ankle Extension

- 2.2 rad/sec
- 1.88 rad/sec
- 1.2 rad/sec
Figure 9
Figure 10
Figure 11