Identification of Flexion Withdrawal Reflex Using Linear Model in Spinal Cord Injury

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The aim of this study was to identify the characteristics of the flexion withdrawal reflex modulated by the hip angle and hip movement in spinal cord injury (SCI). The influence of the hip position and passive movement were tested in 6 subjects with chronic SCI. Each subject placed in a supine position and lower leg was fixed with the knee at 5~45 degree flexion and the ankle at 25~40 degree plantar flexion. A train of 10 stimulus pulses were applied at 200 Hz to the skin of the medial arch to trigger flexion reflexes. From results of the regression analysis, static properties of normalized muscle activation of flexor muscles have the linear relationship with respect to hip angle ($P<0.05$). In order to verify the neural contribution of flexion reflex, we compared the static and dynamic gains of estimated muscle activations with measured EMG of ankle flexor muscle. Form this study, we postulate that the torque and muscle response of flexion withdrawal reflex have linear relationship with hip angle and angular velocity.

Key Words: Flexion Reflex, Neuromuscular Model, Spinal Cord Injury, Electromyogram (EMG)

1. Introduction

The purpose of this simulation study was to identify the characteristics of flexion withdrawal reflex modulated by the hip angle and the hip movement in individuals with spinal cord injury (SCI). The flexion reflex has been studied since Sherrington (1910) first described the withdrawal of a limb from noxious stimuli. The flexion reflex response can provide information about the neural status of the spinal cord (Schmit et al., 2003; Spaich et al., 2004) and be used for producing or assisting the swing phase of gait cycle in spinal cord injured patients (Lee and Johnston, 1976; Granat et al., 1993). For example, flexor spasms are one component of the spastic reflexes that are clinically observed in human SCI, and are associated with an increase in the excitability of the flexion reflex pathways (Shahani and Young, 1971). This increase in flexion reflex excitability can even result in flexion reflex responses to other types of stimuli including imposed movements of the ankle (Schmit et al., 2000; 2002). These movement-triggered reflex responses are likely to originate from muscle afferents, since intramuscular stimulation also triggers flexor reflexes (Hornby et al., 2004). These observations that imposed movements of the leg produce flexion reflexes indicate that convergent afferent inputs from skin and muscle contribute substantially to flexion reflexes

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in human SCI, and ultimately to the clinical manifestation of flexor spasms. Since Lee and Johnston proposed electrical induction of the flexion reflex during the ambulation of hemiplegic patients, the flexion reflex response have been implemented into the simple gait pattern in the FES ambulation program of complete and incomplete SCI patients.

Convergence of multiple afferent systems onto flexion reflex pathways in the spinal cord suggests that flexor spasms in SCI might be modulated by proprioceptive feedback from the leg. This knowledge could have implications in the management of spasticity in SCI. In the cat, Lundberg (Lundberg, 1979) demonstrated that the afferents comprising the FRA (high threshold muscle afferents, joint afferents and cutaneous afferents) converge onto common interneuronal pathways. For example, convergent input to neurons in flexion reflex pathways from nociceptors and group II muscle afferents has been demonstrated by stretch activation (Kirkwood et al., 1987) or electrical stimulation (Steffens and Schomburg, 1993) of muscle afferents during cutaneous triggers of the flexor reflex in cats.

In the present study the effects of hip posture and movement on joint torque and the activation level of the flexor muscles during flexion reflexes were analyzed with linear model and compared to the effects on ankle muscle activation. First, joint torque response at the hip and ankle were identified using linear model. Second, flexor muscle activation levels associated with hip angle and angular velocity were also estimated from the measured torque response using estimates of the muscle moment arms to calculate muscle force and a Hill-based muscle model to account for length and velocity changes in the muscles. In order to verify the neural contribution of flexion reflex, we compared the static and dynamic gains of estimated muscle activations with measured EMG of ankle flexor muscle. Since flexion reflexes are used to aid in swing during therapeutic interventions aimed at restoring locomotion (Field-Fote, 2000; Graupe, 2002), it is important to consider the effects of the hip angle and the hip movement on the reflex response to electrocutaneous stimuli.

2. Methods

2.1 Experimental setup

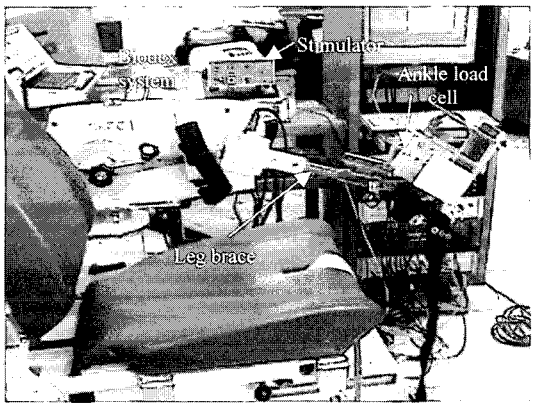
Six subjects were recruited into this study through the inpatient and outpatient physical therapy clinics of the Rehabilitation Institute of Chicago. Inclusion criteria included SCI and a history of spasms reported by the subject and/or physical therapist. All participants had complete SCI (American Spinal Injury Association (ASIA) Scale A) at levels ranging from C5 to T6, as summarized in Table 1. Exclusionary criteria included multiple CNS 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 the inability to give informed consent. Subject consent was obtained and all procedures were conducted in accordance with the Helsinki Declaration of 1975 and approved by the Institutional Review Boards of Northwestern University, Chicago, Illinois, USA.

The test apparatus consisted of a leg brace with torque transducers aligned with the axes of rotation of the ankle, as shown in Figure 1. The foot was placed in a footplate and secured using clamps

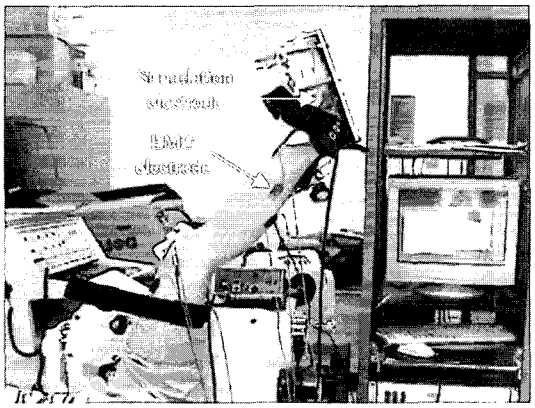
Table 1 Clinical features of subjects

Subject	Level of Injury	ASIA Impairment Scale	Age (years)	Time post injury	Knee flexion angle (deg)	Ankle plantar flexion (deg)
A	C5	A	33	1 y	45	25
B	C6	A	33	1 y	5	35
C	T3	A	46	5 y	20	40
D	T6	A	18	1 y	10	30
E	C5	A	24	5 y	40	35
F	T4	A	37	2 y	22	35

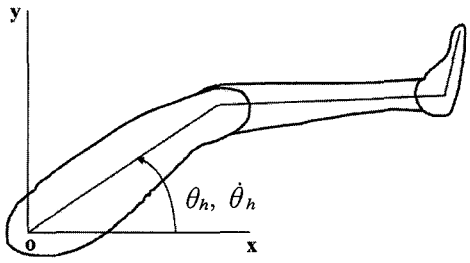
placed on the dorsum of the foot and on the heel. The hip-knee and knee-ankle links were adjustable to fit a wide range of leg sizes. The entire leg brace was affixed to the motor of a Biodex Rehabilitation Testing System 2 (Biodex Medical Systems Inc., Shirley, NY), with the axis of the motor aligned with the axis of rotation of the hip. Hip torque, position, and velocity signals were recorded from the transducers on the Biodex. In addition, ankle and knee torques were recorded from reaction torque meters (S. Himmelstein and Co., Hoffman Estates, IL) incorporated into the leg brace. All signals were low-pass filtered (450 Hz) and sampled at 1,000 Hz using a data acquisition card (National Instruments, Austin, TX) on a personal computer. Custom LabVIEW software (National Instruments) was used for controlling data acquisition, timing of imposed movements and electrical stimulation.



(a) Test apparatus



(b) Flexed hip position (hip angle=70 deg)



(c) Coordinate system of hip posture

Fig. 1 The experimental apparatus for testing flexor reflexes is shown. Joint torques were measured at ankle using transducers incorporated into the instrumented leg brace, and at the hip using the torque transducer of the Biodex system. The motor of the Biodex was used for imposing constant velocity movements to the leg. Stimuli were applied to the skin of the foot in the region of the medial arch

Surface electromyograms (EMGs) were recorded from the tibialis anterior. Active Delsys electrodes (model DE2.1, Delsys Inc., Boston, MA) were applied to lightly abraded, degreased skin over the respective muscle belly. The signal was amplified (X 1,000) and filtered (20-450 Hz; Bagnoli-8, Delsys Inc.). The signals were sampled at 1,000 Hz using the same computer system used for acquiring the position and torque data.

Flexion reflexes were elicited by electrical stimulation through bipolar surface electrodes (Blue Sensor, Medicotest, Rugmarken, DK) placed 1 cm apart at the foot. Stimulating electrodes were placed on the medial arch of the foot and electrical stimulation was triggered using custom-made software and delivered through a constant current stimulator (Model DS-7A, Digitimer Stimulator, Hertfordshire, UK). The electrical stimulus train consisted of a 50 mA, 200-Hz pulse train composed of 10 monophasic pulses (each pulse of 1-ms duration).

Tests of flexion reflexes were conducted during isometric conditions and during imposed movements of the hip. Each subject was transferred to the seat of the Biodex, secured with a lap belt and a pair of belts crossing the chest and placed in a supine position by reclining the seat. The leg was placed in the brace with the knee at 5~45 degrees flexion and the ankle at 25~40 degrees plantar flexion. The hip was aligned with the axis of the

Biodex motor and the brace was adjusted to align the ankle and knee with the torque meters. Controlled hip movements were imposed by the Biodex system. Stimuli were applied with the hip held isometrically in three positions: flexed (70 deg), extended (10 deg) and in the midposition (40 deg). Additionally, the response was measured to stimuli applied at midposition during 30 deg/s constant velocity flexion and extension movements (Figure 1), initiated from the fully flexed or extended posture. A control movement was provided (no stimulation) to account for inertia, gravitational and hip triggered reflex torques at all three joints. Eight trials of each isometric and movement condition were tested, in random order.

2.2 Identification of flexion reflex using Hill-based muscle model

To investigate the influence of the hip position and hip movement on the flexion reflex, we assumed that the joint torque and neural response of flexion reflex were a linear function of hip angle and angular velocity. Muscle activation levels of flexor muscles were estimated using measured torque data.

Active muscle force (F_m) generated by flexion reflex triggered by foot stimulation was modeled with Hill-based contractile elements consisting of three factors describing the length-tension, the force-velocity property, and activation of muscles (Hill, 1938; Zajac, 1989).

$$F_m = aF_m^o f_l(\tilde{l}_m) f_v(\tilde{v}_m) \tag{1a}$$

$$\tilde{l}_m = \frac{l_m}{l_m^o},\ \tilde{v}_m = \frac{v_m}{v_m^o} \tag{1b}$$

where a is activation of muscle fiber, F_m^o is peak isometric muscle force, l_m^o is optimal muscle fiber length and v_m^o is maximum shortening velocity. The length-tension (f_l) and force-velocity (f_v) characteristics of each muscle-tendon were derived by Hill based muscle model. Muscle parameters were determined from several literatures (Brand et al., 1986; Delp et al., 1990; Friederich and Brand, 1990).

Eq. (2) shows the relation between tendon (F_t) and muscle force (F_m) with pennation angle (α).

$$F_t = F_m \cos \alpha \tag{2}$$

Muscle moment of hip, knee and ankle joint were calculated from tendon force multiple by muscle moment arm (r_m).

$$\tau = F_t r_m \tag{3}$$

Flexor muscles having important effects on flexion withdrawal reflex of lower limb were considered in order to investigate the neural response of reflex. These muscles are iliopsoas for hip flexion and tibialis anterior for ankle dorsiflexion. In this study, the tibialis anterior can be used to validate the model and the iliopsoas cannot be measured, thus it must be estimated from the model.

In order to identify static and dynamic gains of flexion reflex, we modeled the activation level of each muscle as combination of linear model of hip angle and angular velocity when constant stimulation input was applied at the medial arch of the foot. In this study, we did not consider the influence of stimulation intensity. However, further study about scaling factor associated with stimulation intensity will be useful to identify the effect of stimulation intensity in the flexion reflex.

$$a = B_0 + B_1\theta_h + B_2\dot{\theta}_h \tag{4}$$

where B_1, B_2 are static and dynamic gains of flexion reflex, respectively. Under isometric condition, activation level of each muscle was simplified to Eq. (5). We postulated that the hip proprioceptors, including the hip joint afferents and hip muscle afferents converged with the cutaneous afferents from the noxious foot stimulation onto common spinal neuronal pathways to result in a flexion withdrawal reflex that was modulated by hip joint angle.

$$a = B_0 + B_1\theta_h \tag{5}$$

First, we examined the ankle joint where we assumed that the ankle torque could be attributed to activation of one major flexor muscle, the tibialis anterior, which remained the same length during changes in hip angle. The ankle joint torque was given by Eq. (6) as follows:

$$\tau_{ankle}=K_{TA}(B_0^{TA}+B_1^{TA}\theta_h) \quad (6)$$

where $K_{TA}=r_{TA}F_{TA}^{o}f_l(\tilde{l}_{AA})\cos\alpha_{TA}$, subscript : TA (tibialis anterior). The constant, K_{TA}, was calculated using published values for the muscle properties (Delp et al., 1990). Note that the muscle activation, a, was incorporated into the B coefficients. We determined the estimated muscle activation level using peak measured ankle torque data. The static gain of the hip angle, B_1^{TA}, was then calculated for each subject using a linear regression of the isometric data at the three tested hip angles and the slope was tested for significance ($\alpha=0.05$).

A similar analysis was then applied to the TA EMG data in order to validate the analysis. The EMG data were band pass filtered between 10-250 Hz (4th order Butterworth) and rectified. The resulting signals were smoothed (4th order Butterworth low pass filter at 5 Hz) and the amplitude during the 2s following the stimulus was calculated. A linear regression of the TA areas was then used to calculate the static gain for each subject. These gains were then compared to the B values obtained from the torque analysis.

In order to determine an estimate of the static gain for the population, a regression analysis was conducted on the normalized EMG and torque-derived activation levels. The activation gain derived from the torque recordings for the entire population was calculated for the ankle. The activation values for each subject were normalized by the mean activation obtained in the three positions for each subject. A linear regression analysis was then run on the population data to determine an estimate of the effect of hip posture on the ankle muscle activation during the flexor reflex. Similarly, the EMG areas were normalized by the mean response and a linear regression was conducted on the population data.

Hip joint response is more complicate than ankle joint because length of hip flexor muscle is also changed according to hip angle. We determined the estimated muscle activation level using peak measured hip torque data. Hip joint torques are given by Eq. (7).

$$\tau_{hip}=K_{IP}(B_0^{IP}+B_1^{IP}\theta_h) \quad (7)$$

where $K_{IP}=r_{IP}F_{IP}^{o}f_l(\tilde{l}_{IP})\cos\alpha_{IP}$, subscript : IP (iliopsoas).

The dynamic response was measured using electrical stimuli applied at the midposition of the hip (hip angle=40 deg) with constant angular velocity (30 deg/s), initiated from the full flexed (hip angle=70 deg) or extended (hip angle=10 deg) posture. A control movement was provided (no stimulation) to account for inertia, gravitational and hip triggered reflex torques at all three joints. The neural contribution was distinguished from the biomechanical contribution by accounting for the length and velocity properties of the hip flexors. Since the stimuli were all applied at the same hip angle, the dynamic gain (B_2 in Eq. 4) was then estimated using a simplified equation (Eq. (8)). In addition, the dynamic gain was estimated separately for the hip flexion and hip extension perturbations, since both movements appeared to produce an increase in the flexor reflex response.

$$a=B_{0d}+B_2\dot{\theta}_h \quad (8)$$

For the ankle, the joint torque was assumed to be determined as shown in Eq. (9). We then neglected the force-velocity property of tibialis anterior because the ankle joint was fixed. Since the stimuli were all applied at the same position, the position effect was accounted for by the B_{0d} constant. In addition, the tibialis anterior length was not altered by hip position, making K_{TA} a simple constant. In order to account for intersubject variability a normalized ankle torque data was used, normalizing the torque by the mean peak ankle torque during the static posture.

$$\tau_{ankle}=K_{TA}(B_{0d}^{TA}+B_2^{TA}\dot{\theta}_h) \quad (9)$$

A similar procedure was used to estimate the dynamic gain at the hip; however, since the length and velocity of the iliopsoas muscle were changed during hip movement, these effects on hip torque needed to be accounted for. In this test, the iliopsoas muscle's stretch and shortening velocity were equal but different direction (muscle shortening velocity is 0.022 m/s) during angular velocity of hip joint (30 deg/s). The hip joint torque during the hip movement was given from

Eq. (10). Net peak torque of hip joint was calculated by subtracting torque with stimulation from without stimulation at the mid-position of hip (hip angle=40 deg) during different movement. Again, the peak hip flexion torque used for the calculation of dynamic gain was normalized by the mean hip torque produced during the isometric test.

$$\tau_{hip}=K_{IP}^{Mov,i}(B_{0d}^{IP}+B_2^{IP}\dot{\theta}_h) \qquad (10)$$

where $K_{IP}^{Mov,i}=r_{IP}^{i}F_{IP}^{o}f_l(\tilde{l}_{IP}^{i})f_v(\tilde{v}_{IP}^{i})\cos\alpha_{IP}^{i}$, i= during hip flexion, and during hip extension.

3. Results

We triggered the flexion reflex with high stimulation intensity (50 mA) and the reflex latency has a long lasting latency (80-120 ms). In this study, we proposed a linear model of reflex response associated with hip angle and angular velocity in order to identify reflex characteristics in flexion reflex. To minimize the influence of subjects and the stimulation intensity, the reflex responses are normalized by the mean reflex response obtained in the three positions for each subject.

3.1 Identification of static flexion reflex under isometric conditions

Figure 2 shows that scatter plots of reflex torque amplitude with respect to hip joint angle for the hip, ankle joints, and regression lines for individual subjects. Reflex torques are normalized by the mean response during isometric conditions. The hip and ankle torque response triggered by foot stimulation are linearly modulated by hip angle.

Figure 3 shows that scatter plot of estimated muscle activation, measured EMG with respect to hip angle. Activation levels of tibialis anterior and illiopsoas were calculated from measured peak ankle and hip torque. The results of the linear regression analysis of normalized activation levels for individual subjects are shown in Table 2. Neural response of flexor muscles triggered by the noxious stimulation is significantly influenced by hip angle ($P<0.05$).

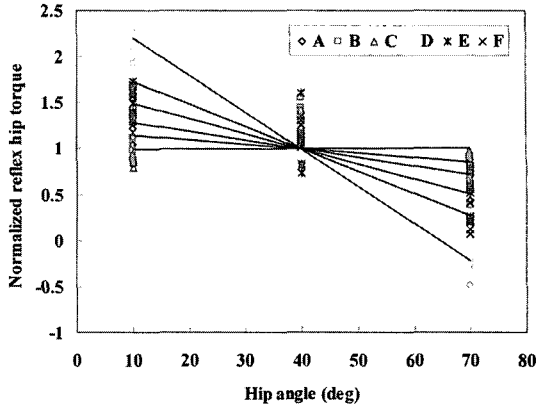


Fig. 2 Scatter plots of reflex torque amplitude vs. hip joint angle for the hip and ankle joints and regression lines for individual subjects. Reflex torques are normalized by the mean reflex response during isometric conditions

The mean static gains of the joint torque, muscle activation levels and measured EMG are shown in Figure 4. Mean static gains of hip and ankle torque are −0.0158, −0.0048, respectively. Mean static gains of the tibialis anterior and iliopsoas were −0.0048 and −0.0175, respectively. Static gains of measured EMG in tibialis anterior were similar with the estimated activation level.

3.2 Identification of dynamic flexion reflex during passive hip movement

We identified the dynamic flexion reflex triggered by the foot stimulation during different hip movement. We defined counterclockwise angular

velocity as positive direction. The flexion reflex response during hip movement was generally larger than the isometric response. The isometric flexor reflex response was compared to the response during hip flexion and extension, with the stimulus applied to the skin of the foot, at the identical hip angle.

Mean reflex torque response for individual subjects with respect to hip angular velocity are shown in Figure 5. Excitations of the spinal mo-

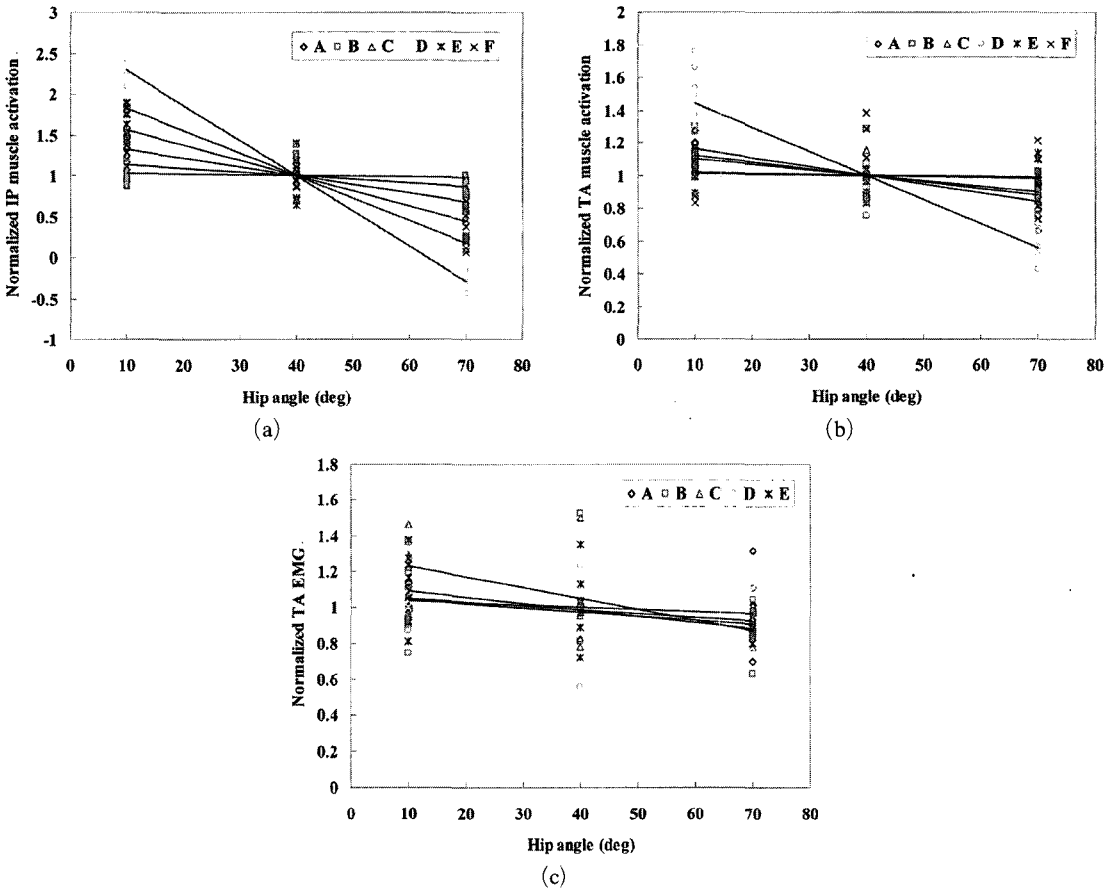


Fig. 3 Scatter plots of estimated flexor muscle activation vs. hip joint angle for the flexor muscles and regression lines for individual subjects. Reflex muscle activations are normalized by the mean reflex response during isometric conditions

Table 2 Results of the linear regression analysis of normalized activation levels in the flexor muscles

Subject	Tibialis anterior				Illiopsoas			
	B_0	B_1	r_2	P-value	B_0	B_1	r_2	P-value
A	1.2176	−0.0054	0.8041	<0.05	1.4234	−0.0106	0.8502	<0.05
B	1.1644	−0.0041	0.6411	<0.05	1.1701	−0.0043	0.6082	<0.05
C	1.1347	−0.0034	0.7146	<0.05	1.0381	−0.0010	0.0552	>0.05
D	1.5926	−0.0148	0.8745	<0.05	2.7174	−0.0429	0.9867	<0.05
E	1.0202	−0.0005	0.0194	>0.05	1.7408	−0.0185	0.8302	<0.05
F	1.0178	−0.0004	0.0040	>0.05	2.1071	−0.0277	0.9839	<0.05
Mean	1.1912	−0.0048	0.5096		1.6995	−0.0175	0.7191	
S.D	0.2121	0.0053	0.3937		0.6321	0.0158	0.3534	

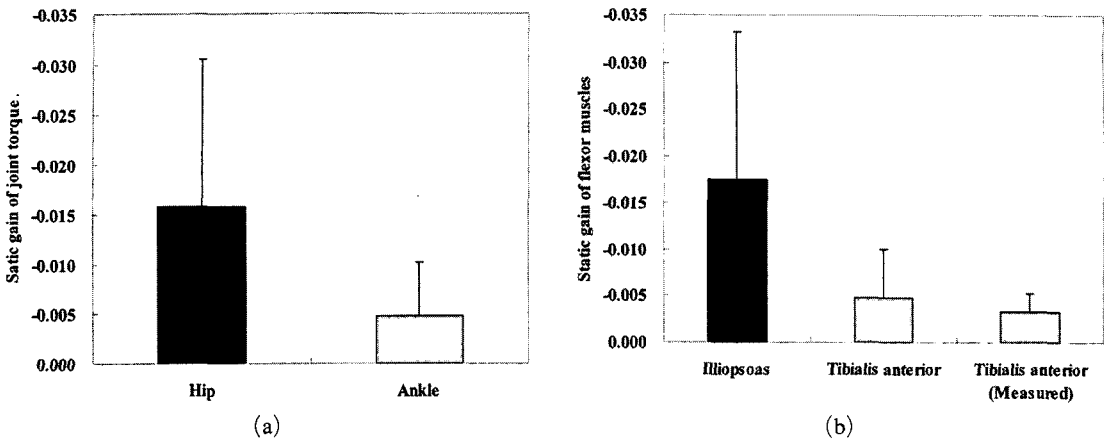


Fig. 4 Mean static gains of joint torque and activation levels of flexor muscle. Reflex response are normalized by mean response from isometric conditions

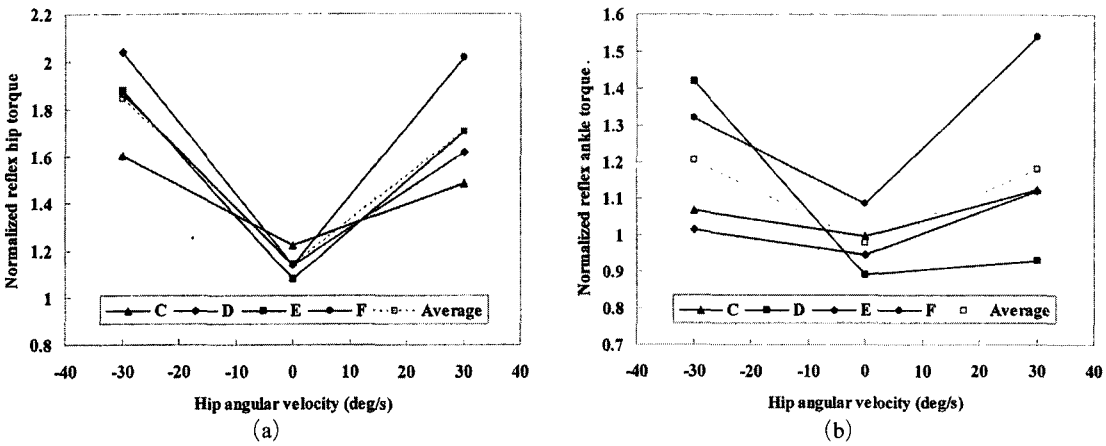


Fig. 5 Mean reflex torque response vs. hip angular velocity at the hip and ankle joints. Reflex torque are normalized by mean response from isometric conditions

torneurons are enhanced by hip movement so that torque responses during hip movement are bigger than those during isometric condition.

Figure 6 shows that mean reflex muscle activation levels, measured EMG with respect to hip angular velocity. Dynamic gains of activation levels in tibialis anterior and iliopsoas were calculated from measured ankle and hip peak torque during hip movement. The slopes of normalized activation levels for four subjects during hip movement are shown in Table 3. Mean dynamic gains of tibialis anterior and iliopsoas normalized by mean response were 0.0067, 0.0207 during hip flexion and −0.0075, −0.0107 during hip extension, respectively.

Figure 7 shows the mean dynamic gains of joint torque and muscle activation. Mean dynamic gains of torque response at the hip and ankle joint were 0.0186, 0.0067 during hip flexion and −0.0234, −0.0075 during hip extension. The comparison of dynamic gains between measured EMG and estimated activation level of tibialis anterior are also shown in Figure 7(b).

4. Discussion

We identified the characteristics of flexion withdrawal reflex modulated with changes of hip angle and angular velocity in human spinal cord injury. Static and dynamic gains of the joint tor-

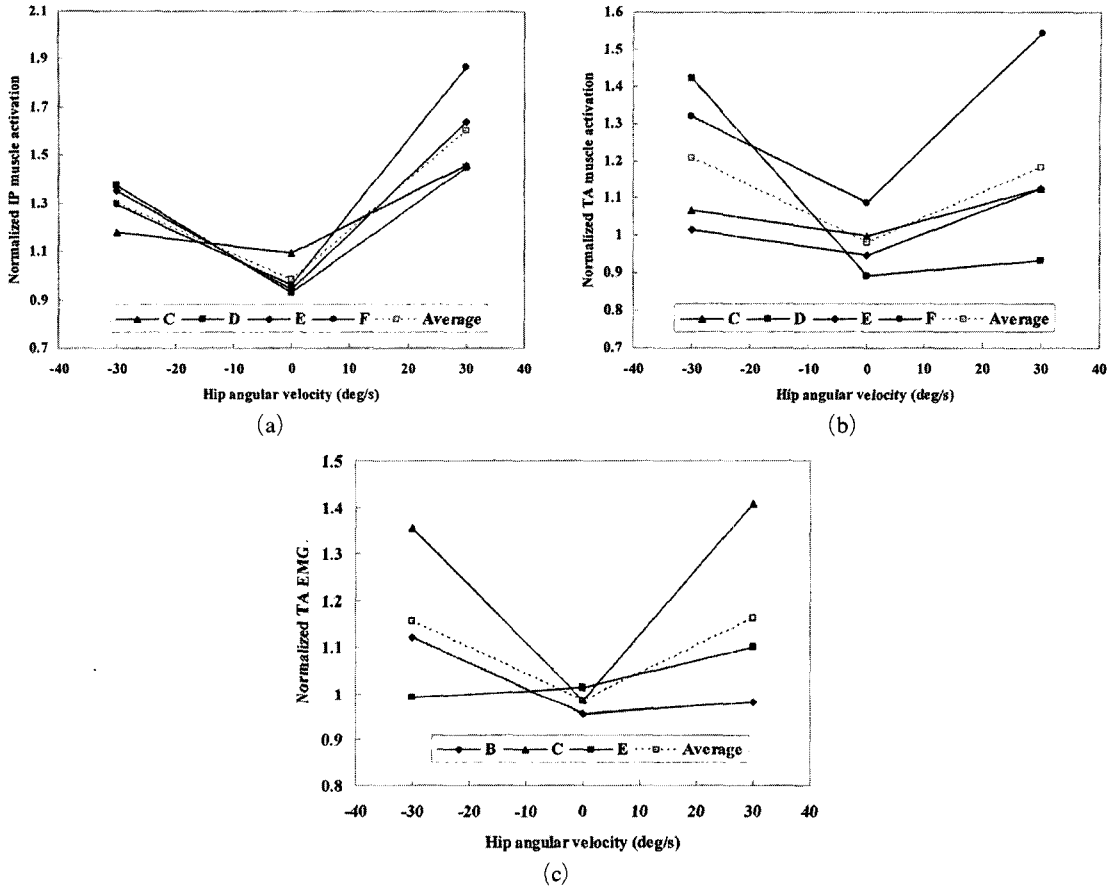


Fig. 6 Mean reflex muscle activation levels for flexor muscles vs. hip angular velocity. Estimated muscle activations are normalized by mean response from isometric conditions

Table 3 Dynamic gains of normalized activation levels in the flexor muscles during the hip movement

Subject	During hip flexion		During hip extension	
	Tibialis anterior	Illiopsoas	Tibialis anterior	Illiopsoas
C	0.0042	0.0122	−0.0023	−0.0029
D	0.0013	0.0172	−0.0176	−0.0148
E	0.0059	0.0232	−0.0022	−0.0137
F	0.0152	0.0302	−0.0078	−0.0111
Mean	0.0067	0.0207	−0.0075	−0.0107
S.D	0.0060	0.0078	0.0072	0.0054

que and estimated muscle activation in the flexion reflex induced by the hip perturbation were examined with linear regression analysis. Position-sensitive afferents, possibly involving muscle afferents and joint afferents from hip perturbation enhanced flexor reflex excitability when the hip was placed in the extended position (hip angle= 10 deg). Flexor reflexes were enhanced during movement in either direction, in comparison to isometric tests at the same joint position. The imposed hip movements are likely to activate group Ia muscle afferents, suggesting unique functional

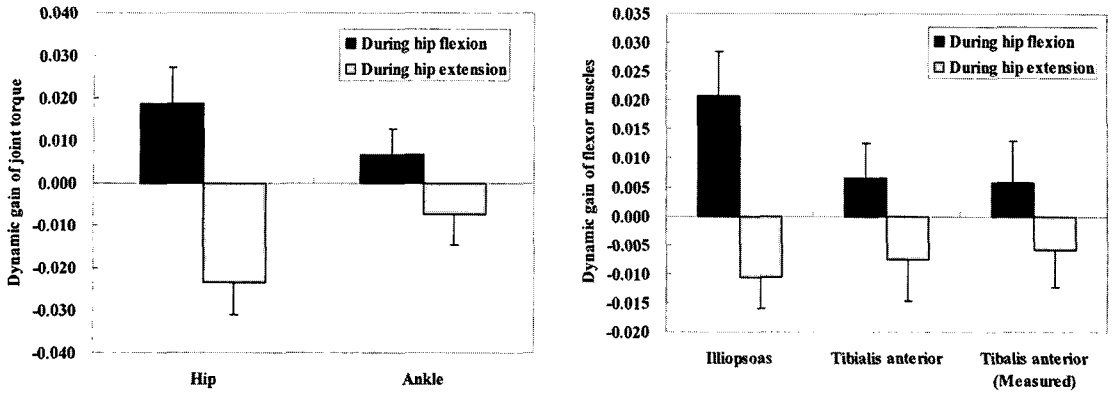


Fig. 7 Mean dynamic gains of joint torque and activation levels of flexor muscle. Reflex response are normalized by mean response from isometric conditions

roles for static and dynamic spindle afferents in spinal reflexes. These observations suggest that hip afferents play a crucial role in spasticity in SCI and is used to aid in swing during therapeutic interventions aimed at restoring locomotion

4.1 Static flexion reflex under isometric conditions

Our results demonstrating an increased flexion reflex of the hip and ankle in an extended hip posture are generally consistent with some observations. One functional explanation for the effects of hip flexor afferents on flexion reflexes arises from observations of the modulation of locomotion by hip flexor afferents in the cat ; in particular, hip flexor afferents play an important role in initiating swing. The potential effects of hip afferents on swing initiation were first reported in acute spinal cats walking on a treadmill (Grillner and Rossignol, 1978). In this preparation, manual manipulations of the limb initiate swing during an imposed extension of the hip, regardless of the state of the contralateral leg. While flexor muscle afferents of many muscles of the leg play a role in modulating swing (Hiebert et al., 1996), the influence of the afferents of the sartorius (a hip flexor/knee flexor) may be particularly potent (Lam and Pearson, 2002). Alternative mechanism needs to be considered is that joint afferents enhance the excitability of flexion reflex pathway (Baxendale and Ferrell, 1980). They observed that flexion reflexes were most easily obtained when the knee joint was extended in decerebrated cats.

4.2 Dynamic flexion reflex during passive hip movement

An imposed movement of the hip, in either direction, appeared to provide an excitatory drive to the flexor reflex pathways in the current experiments. This excitatory drive might be associated with group Ia afferent feedback, activated by the stretch of the hip muscles during the imposed movement. Unlike the isometric conditions, in which the effects were associated exclusively with stretch of the hip flexors, the modulation of the response due to movement did not appear to depend on the movement direction. In particular, ankle flexors increased their response during both flexion and extension movements. These observations suggest that movement of the hip produces a nonspecific excitation of the flexor reflexes, and possibly a general excitation of all spinal neurons. The increased flexor reflex response during passive movement may involve reflex feedback from muscle group Ia afferents. In neurologically-intact individuals, passive cycling movement of the limb has an excitatory effect on TA reflex responses to tibial nerve stimulation (Brooke et al., 1999), which is then suppressed during active pedaling or walking (Zehr et al., 2001). An increased response to cutaneous stimuli is also pro-

duced by imposed movements of the ankle (alone) in SCI (Dimitrijevic and Nathan, 1968), similar to the effects of hip movements in the current study. Generally, the modulation of the electrocutaneous reflex response during cyclical movements has been associated with the locomotor pattern generator (Brown and Kukulka, 1993; Duysens and Van de Crommert, 1998) rather than with reflex modulation, implicating a different role for dynamic afferent feedback on electrocutaneous reflexes during movement. Our results suggest that movement itself enhances flexor reflexes, probably through an intraspinal interaction between dynamic muscle spindle afferents (group Ia) and skin afferents.

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