Proprioceptive Modulation of Hip Flexor Activity During the Swing Phase of Locomotion in Decerebrate Cats

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Abstract

Lam, Tania and Keir G. Pearson. Proprioceptive modulation of hip flexor activity during the swing phase of locomotion in decerebrate cats. J Neurophysiol 86: 1321–1332, 2001. This study examined the influence of proprioceptive input from hip flexor muscles on the activity in hip flexors during the swing phase of walking in the decerebrate cat. One hindlimb was partially denervated to remove cutaneous input and afferent input from most other hindlimb muscles. Perturbations to hip movement were applied either by 1) manual resistance or assistance to swing or by 2) resistance to hip flexion using a device that blocked hip flexion but allowed leg extension. Electromyographic recordings were made from the iliopsoas (IP), sartorius, and medial gastrocnemius muscles. When the hip was manually assisted into flexion, there was a reduction in hip flexor burst activity. Conversely, when hip flexion was manually resisted or mechanically blocked during swing, the duration and amplitude of hip flexor activity was increased. We also found some specificity in the role of afferents from individual hip flexor muscles in the modulation of flexor burst activity. If the IP muscle was detached from its insertion, little change in the response to blocking flexion was observed. Specific activation of IP afferent fibers by stretching the muscle also did not greatly affect flexor activity. On the other hand, if conduction in the sartorius nerves was blocked, there was a diminished response to blocking hip flexion. The increase in duration of the flexor bursts still occurred, but this increase was consistently lower than that observed when the sartorius nerves were intact. From these results we propose that during swing, feedback from hip flexor muscle afferents, particularly those from the sartorius muscles, enhances flexor activity. In addition, if we delayed the onset of flexor activity in the contralateral hindlimb, blocking hip flexion often resulted in the prolongation of ipsilateral flexor activity for long periods of time, further revealing the reinforcing effects of flexor afferent feedback on flexor activity. This effect was not seen if conduction in the sartorius nerves was blocked. In conclusion, we have found that hip flexor activity during locomotion can be strongly modulated by modifying proprioceptive feedback from the hip flexor muscles.

INTRODUCTION

The importance of proprioceptive afferent feedback during locomotion is well established (reviewed in Duysens et al. 2000; Rossignol 1996). This has been demonstrated most clearly for the extensor system in a variety of preparations where length- and load-sensitive afferent signals from the legs have been found to be key for the control of extensor activity and the transition to the swing phase (reviewed in Orlovsky et al. 1999). In decerebrate walking cats, it has been found that extensor burst duration is regulated by load-sensitive afferents from extensor muscles (Duysens and Pearson 1980; Whelan et al. 1995) and by stretch-sensitive afferents from flexor muscles (Hiebert et al. 1996). Extensor burst amplitude is controlled largely by feedback from length- and force-sensitive afferents in the extensor muscles (see Pearson 1995 for review).

Investigations on the modulation of the swing phase by muscle afferents have so far been limited. Much of the literature regarding peripheral afferent modulation of the swing phase has focused on the role of cutaneous afferents (reviewed in Rossignol 1996). Few reports have presented evidence that proprioceptive inputs from noncutaneous sources influence the swing phase. Orlovskii and Shik (1965) put forth this concept in their study of the response to elbow braking during treadmill locomotion in dogs. Despite an applied braking force during swing, the forelimb was able to rapidly compensate and resume forward advancement with the speed and inter-joint coordination as during undisturbed stepping. There was a 30-ms delay from the time of braking to the time that a correction was observed. This short delay suggested that the mechanism used to stabilize limb trajectory during swing is realized at relatively low levels of the nervous system (Orlovskii and Shik 1965). Thus they contended that segmental mechanisms exist that serve to ensure a relatively standard swing phase in order for safe paw placement and support to occur. Involvement of flexor muscle afferents during the swing phase is also indicated from results of experiments using decerebrate cat treadmill locomotion and fictive locomotion (Hiebert et al. 1996; McCrea et al. 2000; Perreault et al. 1995; Quevedo et al. 2000). Perreault et al. (1995) have reported that stimulation of afferents from sartorius, tibialis anterior (TA), and semitendinosus muscles at group I strength during flexion can prolong the flexor phase during fictive locomotion, while stimulation at group II strength during flexion consistently terminated flexion and reset the rhythm to extension. Recently, however, McCrea et al. (2000) reported that afferents from iliopsoas and sartorius muscles stimulated at both group I and II strengths can prolong and enhance ongoing flexion. Evidence of a group I disynaptic excitatory pathway to flexor motoneurons has also recently been demonstrated during fictive locomotion (Degtyarenko et al. 1998; Quevedo et al. 2000).
During decerebrate treadmill stepping, stimulation of the nerve to the extensor digitorum longus (EDL) muscle at both group I and II strengths can increase the duration of flexion (Hiebert et al. 1996). TA stimulation at group II strength can also prolong flexor activity (Hiebert et al. 1996). Thus previous results from fictive and decerebrate cat locomotion have demonstrated an influence from flexor muscle afferents onto the locomotor central pattern generator (CPG) and thus indicate a role for these afferents in modulating the swing phase.

The purpose of this study was to determine whether afferent feedback from hip flexor muscles is involved in the modulation of flexor activity in walking decerebrate cats. Using natural stimuli to perturb flexor muscle contractions, we show that modifying the feedback from hip flexor muscles, particularly the sartorius muscles, can have a powerful influence on the amplitude and duration of flexor burst activity. Preliminary results from this study have been published in abstract form (Lamb and Pearson 2000).

METHODS

The data presented in this paper were obtained from experiments on 16 adult cats of both sexes. All procedures were approved by the University of Alberta Health Sciences Animal Welfare Committee.

Preparation

Animals were initially anesthetized with halothane. The trachea was then cannulated for the continuous administration of the anesthetic. One carotid artery was ligated and the other cannulated for monitoring the blood pressure. One jugular vein was cannulated for the administration of drugs. Intramuscular fine-wire recording electrodes (Cooner Wire AS632, Cooner Wire, Chatsworth, CA) were sewn into the following muscles of the left hindlimb: iliopsoas (IP), anterior head of sartorius (SartA), medial head of sartorius (SartM), and medial gastrocnemius (MG). Electrodes were also sewn into the IP and MG muscles on the right side. Electrode leads were threaded under the skin to a connector located externally on the animal’s back. To eliminate cutaneous input and afferent input from other muscles, the following nerves were cut: the two branches of the sural, saphenous, distal tibial, femoral (distal to the sartorius nerve), and either the deep and superficial peroneal (distal to its branch to the tibialis anterior muscle) or the common peroneal nerve. In one animal, the cranial and caudal gluteal nerves were also cut. The hamstring nerves were cut in five animals, and the obturator nerve was cut in four animals.

The nerves to the SartA and SartM muscles were identified and looped off with a thread (n = 8 animals). This enabled us to examine the specific role of the sartorius muscles during swing by blocking conduction in their nerves when the looped thread was tied off and pulled. Interruption of the nerve supply to the sartorius muscles was confirmed by the elimination of sartorius electromyographic (EMG) signals. In a number of animals (n = 15), a hole was drilled into the tibia about 6 cm above the ankle joint and a screw inserted. The screw served as the distal anchor for the blocking device.

After these procedures, the animal was placed over a motorized treadmill and fixed in a stereotaxic frame. The animal was then decerebrated by transecting the brain stem at a 50° angle from the anterior edge of the superior colliculus. The anesthesia was discontinued at this time. Within 1 h, spontaneous stepping usually occurred in response to the moving treadmill belt. The speed of the treadmill was set between 0.3 and 0.4 m/s. Manual stimulation to the perineum was sometimes used to evoke stepping. When spontaneous treadmill locomotion could not be consistently evoked, electrical stimulation of the mesencephalic locomotor region (MLR) was used (n = 8 animals). Typical stimulus parameters were 15 Hz, 0.5-ms pulses, 100–200 μA. Generally, locomotion generated by MLR stimulation shares many characteristics of locomotion in intact animals (Shik et al. 1966). We did not observe any overall qualitative differences between the locomotor pattern of spontaneously and MLR-evoked walking animals. Thus the data were combined and presented as a group.

Perturbations to swing of the left leg were applied in one of two ways: manual resistance or assistance (Fig. 1A) or blocking hip flexion (Fig. 1B). Resistance was applied manually by the experimenter holding the ankle during the swing phase and applying a force opposing the swinging limb. Assistance was applied in the same way except the limb was pushed forward during swing. During the manual perturbation trials, the experimenter maintained light hold of the ankle to avoid interfering with normal stepping sequences but at the same time prepared to administer a perturbation. Manual perturbations during swing were applied rhythmically and synchronized with the naturally occurring flexor burst activity. The manual perturbations lasted for less than or more than the normal swing duration during assistance and resistance, respectively, as estimated by the experimenter holding the limb. To ensure that excessive force was not used to manually perturb the limb, we videotaped an experimental session.
in one animal with markers on the trunk, hip, and knee joints. The hip joint angle was measured using these markers, and we found that the excursion of the hip joint did not differ greatly between the different conditions, based on data from 20 randomly chosen steps in each condition. During undisturbed stepping, the movement of the hip joint ranged from an extended position of approximately 100° to an extended position of 150° (mean ± SD) to a flexed position of 30° ± 1.9°. During manual assistance, the range was from approximately 100° to approximately 140° ± 5.7°. In this animal, the manual assistance perturbations that were applied were approximately 50–150 ms less than the normal swing duration. The manual resistance perturbations were approximately 100–150 ms more than the normal swing duration.

The use of the blocking device (Fig. 1B) allowed better timing of the resistance to swing. This device allowed leg extension but firmly prevented full flexion at the hip. The device’s position could be adjusted such that hip flexion could be blocked at various times during the swing phase. A force transducer coupled with the device allowed measurement of the time of the onset of the block.

Data analysis

The analog data (raw EMG signals and force transducer signal) were recorded on tape using a Gould 4000A PCM machine. A hard copy of the data were printed out using a Gould TA11 chart recorder from which sequences of regular consistent stepping were identified (usually 10–20 steps). The EMG analog data were full-wave rectified and filtered with a band-pass filter (high pass 10 Hz, low pass 30 Hz). The force signal was filtered at a low-pass frequency of 30 Hz. Each EMG channel and the force signal was then digitized at a sampling rate of 500 Hz and stored on computer using a data acquisition system (Axotape 2.0.2, Axon Instruments). Custom-written computer software programs were used to average and plot the digitized EMG traces, measure burst amplitude, and measure burst duration and cycle period. The amplitude of the EMG signal was calculated as the average amplitude over the whole duration of each burst for each muscle. Cycle duration was defined as the time from the onset of an IP burst to the next IP burst. Average burst durations and flexor burst amplitudes were calculated for undisturbed and perturbed trials from stepping sequences from each animal. Any response in the EMG burst duration or amplitude to the perturbations was expressed as a percentage of the average duration or amplitude of a previous sequence of undisturbed stepping. Descriptions in the text of “burst activity” refer to both amplitude and duration. The onset of blocking hip flexion was measured as the interval between the onset of the IP burst and the onset of force indicated by the force transducer signal. Student’s t-tests were used to determine significant differences, and the level of significance was set at 0.05.

RESULTS

Sensory input from hip flexor muscles influences flexor burst activity

In this study, we present data demonstrating that sensory input from hip flexor muscles during the swing phase can modify the amplitude and duration of burst activity in flexor muscles. The flexor muscles we focused on were the ilioptosar and the two heads of sartorius (Fig. 1C). All effects were obtained from the partially denervated hindlimbs of decerebrate cats during treadmill locomotion. The denervation eliminated cutaneous input as well as afferent input from most other muscles in the hindlimb.

Manual assistance to hip flexion was applied to advance hip flexion during decerebrate walking (n = 7). Data from a representative animal are shown in Fig. 2. Figure 2A shows a sequence of continuous stepping during which manual assistance was applied (denoted by asterisks) followed by undisturbed, control stepping. Once manual assistance was discontinued, flexor burst activity returned to control. In addition, contralateral stepping remained consistent throughout the disturbances and afterward. Averaged flexor bursts are shown in Fig. 2B (averages were taken from the same sequence of steps that is partially shown in the example in Fig. 2A). The major effect was the decrease in flexor burst duration when manual assistance to hip flexion was applied.

When manual resistance was applied to prevent hip flexion during decerebrate walking (n = 7), a significant increase in flexor burst activity was observed. A representative example of the response to manual resistance to hip flexion from a single cat is illustrated in Fig. 3 (same animal as in Fig. 2). The response to manual resistance was opposite of that observed during manual assistance. Figure 3A shows a sequence of undisturbed stepping followed by a bout of stepping during which manual resistance was applied. The increase in activity in the flexor bursts was apparent as soon as the manual resistance was applied (denoted by asterisks). In Fig. 3B, averaged flexor bursts are illustrated (averages were taken from the same sequence of steps that is partially shown in the example in Fig. 3A). In this example, there was an increase in duration in all muscles and an increase in IP burst amplitude during manual resistance.

In Fig. 4A, the average decrease in the duration of the flexor bursts in response to manual assistance for each of the seven animals is illustrated. To eliminate the possibility that reciprocal inhibition from the hip extensor muscles could account for the effects seen during manual assistance, the hamstring muscles were denervated in two cats (JE and M6, indicated by asterisk). In one cat (MI, indicated by double asterisks), both
the hamstrings and the gluteal nerves were cut. No difference in the response to manual assistance to hip flexion was seen between the animals with the hip extensors denervated and those with them intact (*P* > 0.10). Figure 4B illustrates the average increase in the duration of the flexor bursts in response to manual resistance.

An obvious limitation to the manual perturbations is the inability to precisely regulate the timing and amplitude of the resisting or assisting movement. To address this issue, we devised a mechanical blocking device (illustrated in Fig. 1B). This allowed us to block hip flexion at different times during the swing phase. Data from 13 animals were collected while blocking hip flexion in this way.

Similar to the effects of manual resistance, blocking hip flexion resulted in an increase in duration and amplitude of the flexor bursts. An example of the response to the mechanical blocking device is shown in Fig. 5A. In Fig. 5A, the response to blocking hip flexion is illustrated in rectified and filtered EMG records of adjacent stepping sequences, during which flexion was blocked in the first sequence, followed by a sequence of undisturbed stepping. The increase in the duration of the flexor bursts as well as an accompanying decrease in MG burst duration is apparent. Figure 5B illustrates averaged flexor bursts from the same stepping sequence shown in Fig. 5A. During this sequence, the onset of the force occurred 190 ms after the onset of the IP burst (denoted by black arrows). There was a short burst of activity, presumably a short-latency reflex response, occurring after the onset of the block. Following this, there was a general increase in flexor burst duration and amplitude (shaded area). There was also a general trend for a relationship between the onset time of blocking flexion and the degree of increase in flexor burst duration (Fig. 5C). In other words, the largest increase in flexor burst duration tended to occur when blocking of the limb occurred earlier in the flexor phase of locomotion. The duration of the MG bursts also tended to be lower when hip flexion was blocked compared with undisturbed stepping. The increase in flexor burst amplitude was not related to the time of onset of the block (Fig. 5D).

Each point in the scatterplots in Fig. 5, C and D, represents the mean change in flexor burst duration or amplitude (compared with control) of a sequence of stepping during which hip flexion was blocked at a given time.

**Afferents from the sartorius muscles are particularly important for flexor burst modulation**

To differentiate the roles of the hip flexor muscles, we performed a series of experiments where either the IP muscle was detached from its insertion or conduction was blocked in the nerves to the SartA and SartM muscles. Unfortunately, due to technical constraints, the IP muscle had to be detached from its insertion before treadmill locomotion could be elicited. Thus in these experiments, the animal could not serve as its own control for examining the effect of IP tenotomy.

In five experiments, we detached the IP tendon from its distal attachment, effectively leaving the SartA and SartM muscles as the major hip flexor muscles. In these animals, we observed an increase in flexor burst duration and amplitude...
when hip flexion was blocked despite the inability of the IP muscle to provide any sensory input. Figure 6A illustrates averaged filtered and rectified EMG records comparing blocked with undisturbed stepping in a cat with IP tenotomy. In this example, blocking of flexion occurred 132 ms after the onset of the IP burst (denoted by black arrows). In Fig. 6, B and C, we present grouped data from three animals with IP tenotomy (□) compared with six animals without IP tenotomy (■), where hip flexion was blocked between 50 and 150 ms after the onset of the IP burst. Even with the IP detached, the response to blocking flexion appeared largely unaffected. The percent changes in flexor and MG burst duration in animals with IP tenotomy was not statistically significant from those seen in cats with the IP intact (P > 0.10; Fig. 6B). Similarly, the percent increases in flexor burst amplitudes when the IP was detached were also comparable with those seen in cats with the IP intact (P > 0.10; Fig. 6C).

To further investigate the possible role of muscle afferents from the IP muscle, the tendon of the muscle was detached with a part of its bony insertion intact in two animals. The muscle was then tied to a muscle puller (Hiebert et al. 1996). The IP muscle afferents were activated by a stretch via the muscle puller of 5–9 mm, 50-ms rise time, and 300–500 ms duration. Figure 7A illustrates an example of a bout of stepping during which the IP was stretched during flexion (5 mm, 500 ms duration). Averaged data, taken from the same animal whose locomotor activity is partially shown in Fig. 7A, is shown in Fig. 7B (n = 100 steps). The average increase in IP burst duration was 7.3 ± 16.6%. SartA burst duration increased by 6.4 ± 16.0%, and SartM burst duration increased by 7.4 ± 14.8%. The average increase in IP burst amplitude was 6.1 ± 18.2%. SartA burst amplitude changed by −2.7 ± 14.4%, and SartM burst amplitude increased by 3.2 ± 12.6%.

The response to stretch of the IP muscle was variable and subtle with a large range of responses and small changes in duration or amplitude, consistent with the finding that IP tenotomy had no effect on the response to blocking hip flexion (Fig. 6).
Since IP tenotomy had little effect on the response to blocking flexion and the response to stretch of the isolated IP muscle was negligible, we turned our attention to examining the role of proprioceptive feedback from the sartorius muscles during the swing phase. In one experiment, the SartA and SartM muscles were cut near their distal attachments, leaving the IP as the sole major hip flexor. In this animal, we noticed that the response to blocking forward swing was diminished compared to those observed in other cats whose SartA and SartM muscles were intact. In subsequent experiments (n = 8), we looped a thread around the SartA and SartM nerves. Following a period of baseline stepping, we tied off and pulled on this thread to block conduction in the nerves. We determined that conduction was successfully blocked by the loss of sartorius EMG activity. In five animals, we were able to record responses to blocking hip flexion both before and after conduction block in the SartA and SartM nerves. Figure 8A illustrates an example of a sequence of stepping during which flexion was blocked in one of these animals. Note also the decrease in the level of activity in the IP muscle after conduction block in the sartorius nerves (Fig. 8A). Figure 8B1 shows the characteristic response to blocking flexion in the IP muscle before conduction in the sartorius nerves was blocked (shaded area). Averaged IP EMG records from the same cat are shown in Fig. 8B2 after conduction in the sartorius nerves was blocked. As illustrated, the response to blocking the limb during flexion was diminished (shaded area). Figure 8C shows data from five animals demonstrating the general diminished response to blocking. Each point in the scatterplot represents the average IP burst duration and amplitude from a stepping sequence (n = 5 animals, mean ± SE in parentheses). There was a significantly smaller overall response in IP burst duration and amplitude to blocking swing after conduction block in the sartorius nerves (P < 0.01).

Influence of contralateral limb activity on ipsilateral flexor burst activity

Although we were able to obtain a prolongation of the hip flexor burst when the swinging limb was blocked, we sought to find out what was limiting this prolongation. We reasoned that inhibitory coupling between the flexor generating system in the two limbs would limit the extent to which the flexor bursts could be prolonged (Lundberg 1981). If this is true, then flexor activity in the two hindlimbs should not be observed at the
same time. We quantified this idea by comparing the duration of the ipsilateral IP burst with the interval between the onset of ipsilateral IP activity and the onset of contralateral IP activity. These values are indicated as $Y$ and $X$, respectively, in Fig. 9A. If the flexor half-centers are mutually inhibitory, then the values of $X$ and $Y$ should be correlated. $Y$ should not be much greater than $X$, although the reverse is possible: the duration of the ipsilateral IP burst ($Y$) can be much shorter than the interval to the onset to the next contralateral IP burst ($X$). To test this idea, we attempted to lengthen the interval $X$ by delaying the onset of the burst in the contralateral hip flexors by briefly stopping the treadmill during midstance of the contralateral limb (Fig. 9A). When this was done, the duration of the ipsilateral IP burst was prolonged for the duration of the delay to the onset of the contralateral flexor burst in 21% of the trials (Fig. 9B, Undisturbed, ■). In the remainder of the trials, we observed either a cessation of the locomotor rhythm or a continuation of ipsilateral stepping with no change to the duration of the IP bursts (Fig. 9B, Undisturbed, □).

The combination of blocking hip flexion and delaying the onset of contralateral swing resulted in prolongation of ipsilateral IP burst activity in 82% of the trials (Fig. 9B, Block Flexion, ■ and □). In 57% of these trials (■), the duration of the IP burst was extended for the duration of the delay to the onset of the contralateral flexor burst. In the remaining 43% of these trials (□), the duration of the IP burst was, on average, prolonged by 63% (and at least by 12%) compared with the preceding IP burst but terminated at least 10 ms before the resumption of contralateral flexor activity. In the remaining 18% of the trials, we observed either continuation of alternating bursts between ipsilateral flexors and extensors or cessation of ipsilateral flexor activity (□).

If conduction in the sartorius nerves was blocked, the dominant response was either a cessation of locomotor rhythm or a continuation of ipsilateral stepping with no change in the duration of the IP bursts compared with the preceding step (Fig. 9B, Block Flexion and Sartorius Nerve Block). Prolongation of ipsilateral IP bursts was rarely observed (8% of the trials; □). Even when prolongation was observed, it never lasted for the duration of the delay to the onset of the contralateral flexor burst.

The example shown in Fig. 9A represents the upper limit of the relationship between ipsilateral IP burst duration ($Y$) and the interval between the onset of the ipsilateral IP and the subsequent contralateral IP ($X$). If the flexor half centers on both sides inhibit each other, then the greatest extension of ipsilateral flexor activity should coincide with the duration of time that the onset of contralateral flexor activity is delayed (exemplified in Fig. 9A). Indeed, we found that for all animals tested, the relationship between $X$ and $Y$ was strongly correlated. In Fig. 9C, the values $X$ and $Y$ from steps taken from 1 cat ($n = 489$ steps) are plotted. Data points that are located at the top right of the plot are those measured from the trials where the onset of contralateral swing was briefly inhibited and hip flexion was blocked. Data points that are clustered close to the bottom left of the plot (where $X$ is $<2000$ ms) are those measured from sequences of undisturbed stepping and those during which hip flexion was blocked. The relationship between $X$ and $Y$ was significantly correlated ($r = 0.99$, $P < 0.05$).

**DISCUSSION**

The purpose of this investigation was to determine whether modification of afferent feedback from hip flexor muscles influences the burst activity in these muscles during the swing
phase of stepping in decerebrate walking cats. Modifying afferent feedback with natural perturbations to the contracting muscles was found to strongly influence the duration and amplitude of bursts in these muscles. Assisting hip flexion movements decreased flexor burst activity (Fig. 2), while resisting hip flexion enhanced flexor burst activity (Figs. 3 and 5). These effects occur in the absence of sensory input from the skin and other leg muscles, and they depend largely on modification of feedback from the sartorius muscles (Fig. 8). Furthermore, sensory feedback from the sartorius muscle was capable of maintaining flexor burst activity for long periods when stepping in the contralateral leg was inhibited at the same time that ipsilateral hip flexion was resisted (Fig. 9).

**Modulation of flexor activity by flexor muscle afferents during the swing phase**

On the basis of previous findings and the results from this investigation, a scheme for explaining the role of afferent feedback during the swing phase of walking is shown in Fig. 10. This scheme bears a close resemblance to the pathways by which extensor group I muscle afferent input can enhance extensor activity during the stance phase (reviewed in Pearson 1995). Assuming that the CPG for each leg consists of flexor and extensor half-centers that mutually inhibit each other (Lundberg 1981), input from group I afferents from hip flexor muscles could enhance activity in flexor motoneurons via monosynaptic and disynaptic pathways (Degtyarenko et al. 1998; Lundberg 1981; Quevedo et al. 2000) and via the locomotor CPG (McCrea et al. 2000; Perreault et al. 1995).

The monosynaptic excitatory pathway from flexor group I afferents to flexor motoneurons is represented as pathway 1 in Fig. 10. In addition to homonymous monosynaptic connections, heteronymous connections exist between flexor group I afferents and flexor motoneurons. For example, the excitatory connection from Ia sartorius afferents to IP motoneurons (Eccles and Lundberg 1958) was proposed by Lundberg (1981).
to influence IP activity during the swing phase of walking. Very recently, Quevedo et al. (2000) demonstrated that mono-synaptic excitation of TA motoneurons could be elicited via heteronymous as well as homonymous flexor group I afferent connections during fictive locomotion in decerebrate cats.

Disynaptic excitatory pathways from flexor group I afferents to flexor motoneurons are represented as pathway 2 in Fig. 10. Evidence for these pathways comes from recent studies of fictive locomotion in decerebrate cats (Degtyarenko et al. 1998; Quevedo et al. 2000). At rest there is little disynaptic excitation of flexor motoneurons from flexor group I afferents (Quevedo et al. 2000). However, during the flexion phase of locomotor activity, excitatory transmission is powerfully enhanced in homonymous and heteronymous group I disynaptic pathways to flexor motoneurons of the hip, knee, ankle, and bifunctional muscles, including sartorius (Quevedo et al. 2000). Degtyarenko et al. (1998) also provide evidence for excitatory disynaptic reflex pathways from EDL and TA group I afferents to their respective motoneurons during the flexion phase of fictive locomotion in cats.

Evidence for group I afferent input from flexor muscles to the locomotor CPG comes from the work of Perreault et al. (1995) and McCrea et al. (2000). They reported that stimulation of group I afferents from the iliopsoas and sartorius muscles enhances and prolongs flexor activity with a concomitant increase in step cycle duration during fictive locomotion in decerebrate cats. The direct pathway from group I muscle afferents to flexor motoneurons via the flexor half-center is indicated by pathway 3 in Fig. 10.

On the basis of these previous findings, the scheme illus-
trated in Fig. 10 can account for our results as follows. Blocking (resisting) hip flexion causes the hip flexors to shorten more slowly than normal, thus resulting in increased spindle and Golgi tendon organ activation from these muscles. While activity patterns in group Ib afferents from flexor muscles during swing has not been reported to date, spindle afferents from the sartorius muscles have been shown to be active during the swing phase in walking cats (Loeb and Hoffer 1985; Loeb et al. 1985). With the slower shortening of the flexor muscles, the expected reduction in group I activity near the end of swing would not occur, and excitatory drive onto the flexor motoneurons would be increased via one or possibly all of the pathways indicated by previous studies (Degtyarenko et al. 1998; Lundberg 1981; Perreault et al. 1995; Quevedo et al. 2000). Enhanced activation in the pathways onto the flexor half-center would lead to prolonged burst activity. The opposite effects would occur when hip flexion is assisted. In this situation, the hip flexors would shorten and unload more rapidly, thus prematurely reducing the excitatory group I input to the flexor motoneurons and resulting in an abbreviation of activity in the flexor half-center and therefore reduce flexor burst duration.

The extent to which afferents other than the group I afferents might be involved in producing the effects we have observed is difficult to assess. For example, there is no clear consensus on the action of group II flexor muscle afferents on flexor activity during swing. During fictive locomotion, McCrea et al. (2000) reported that stimulation of afferents from the iliopsoas and sartorius muscles at group II strengths can enhance flexor activity, whereas Perreault et al. (1995) reported that stimulation of group II afferents in the sartorius nerve resets the locomotor rhythm to extension. In walking decerebrate cats, stimulation of the EDL nerve at group II strengths produces an increase in the duration of the IP burst while stimulation of the TA nerve (at group II strengths) produced either no effect or an excitatory effect on flexor burst duration (Hiebert et al. 1996). Another source of excitatory input to the flexor burst generating system is the nonspindle group II afferents and smaller afferents. These afferents form a major component of the “flexor-reflex afferents” (FRA) (Burke 1999), so if they were activated with the contracting hip flexors they may have contributed to facilitating flexor burst activity when hip flexion was resisted or blocked. Another possible source of afferent input is from the hip joint. However, the effects of blocking hip flexion were significantly weakened following conduction block in the sartorius nerve (Fig. 9B). This procedure would not have influenced hip joint afferents. Thus we believe that hip joint afferents do not contribute substantial afferent input to hip flexor muscle activity during swing. It is conceivable, however, that some of the residual effect of blocking hip flexion, in the absence of feedback from sartorius muscles, is due to input from hip-joint afferents.

The usual criterion for establishing whether a specific input
has an effect on the CPG is examining whether or not the locomotor rhythm has been reset by the disturbance in question (Hultborn et al. 1998). Given the nature of treadmill stepping and the type of perturbations used, we cannot use this criterion to test that the perturbations we used influence the locomotor CPG. However, two lines of evidence are consistent with the proposal that our proprioceptive disturbances to hip flexor activity (i.e., assisting and resisting/blocking flexion) are accessing the CPG. First, when we blocked hip flexion we observed prolongation of flexor activity accompanied by a decrease in the subsequent ipsilateral extensor burst duration (Figs. 5 and 6). Step cycle duration was not significantly affected by the perturbation; however, effects on cycle duration would not be obvious because the speed of the treadmill entrains the locomotor rhythm via the stepping legs. Second, the duration of the flexor bursts could be greatly enhanced by the addition of proprioceptive feedback, and this enhancement is further reinforced when stepping in the contralateral leg is stopped. We propose that flexor burst activity, although enhanced, is not maintained for a long period when flexion is resisted or blocked because the bursts are terminated by inhibitory input from the contralateral flexor burst generator (Lundberg 1981) (pathway 4 in Fig. 10). Indeed, flexor activity from both sides strongly alternate with each other (Fig. 9C). When the onset of the contralateral flexor burst was delayed, prolongation of ipsilateral flexor activity occurred for seconds could easily occur when hip flexion was blocked (Fig. 9, A and B, middle histogram). Without this additional proprioceptive input, prolongation of ipsilateral flexor activity occurred only 21% of the time (Fig. 9B, left histogram).

Feedback from the sartorius muscles is important for enhancing flexor burst activity

We have been able to show a difference in the importance of afferent feedback from the IP and sartorius muscles during the swing phase. When the IP muscle was detached from its insertion, we saw little change in the response to blocking swing. This was consistent with the small effects produced by stretching the IP muscle (Fig. 7). Given these results, we conclude that afferent feedback from the IP muscle has a small role in the modulation of flexor burst activity. Conversely, we found that when conduction in the sartorius nerves was interrupted, the enhancement in hip flexor activity when hip flexion was blocked was not present (Fig. 8). Furthermore, activity in the IP muscle was reduced even during undisturbed stepping after conduction block in the sartorius nerves (Fig. 8A). The heterogeneity among individual muscles’ roles in the modulation of flexor burst activity extends previous findings from decerebrate walking cats. The IP, TA, and EDL muscles have been previously reported to yield different effects on flexor burst activity when their afferents have been activated (Hiebert et al. 1996). Furthermore, given the different anatomical features of the IP and sartorius muscles (see Fig. 1C), it is not surprising that they would each have a unique contribution to the modulation of flexor burst activity.

Functional relevance

The scheme proposed in Fig. 10 could provide for appropriate pathways by which effective flexor activity can be produced despite perturbations to limb motion. For example, this may provide a mechanism for appropriate modifications to the locomotor pattern when animals walk along inclined surfaces during which there is an increase in flexor burst activity during swing (Carlson-Kuhta et al. 1998). Because the leg has to be lifted more against gravity, there is a tendency to reduce the rate of shortening, thus increasing spindle and tendon organ activity above that generated when walking along a horizontal surface. These enhanced afferent signals would then enhance flexor burst activity to accommodate the increased loading of the flexor muscles via the pathways illustrated in Fig. 10. Consistent with this proposal are our unpublished observations that adding a load to the hindlimb of a conscious walking cat (by strapping a weight around the shank) enhances flexor burst activity analogous to that observed in this study.

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