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A Role for Hip Position in Initiating the Swing-to-Stance Transition in Walking Cats

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McVea, D. A., J. M. Donelan, A. Tachibana, and K. G. Pearson. A Role for hip position in initiating the swing-to-stance transition in walking cats. *J Neurophysiol* 94: 3497–3508, 2005. First published August 10, 2005; doi:10.1152/jn.00511.2005. In this investigation, we obtained data that support the hypothesis that afferent signals associated with hip flexion play a role in initiating the swing-to-stance transition of the hind legs in walking cats. Direct evidence came from observations in walking decerebrate cats. Assisting the flexion of the hip joint during swing advanced the onset of activity in ankle extensor muscles, and this advance was strongly correlated with a reduction in the duration of hip flexor muscle activity. The hip angle at the time of onset of the flexion to extension transition was similar during assisted and unassisted steps. Additional evidence for the hypothesis that sensory signals related to hip flexion are important in regulating the swing-to-stance transition came from four normal animals trained to walk in a variety of situations designed to alter the coordination of movements at the hip, knee, and ankle joints during the swing phase. Although there were exceptions in some tasks and preparations, the angle of the hip joint at the time of onset of extensor activity was generally less variable than that of the knee and ankle joints. We also found no clear relationships between the angle of the limb and body axes, or the length of the limb axis, and the time of onset of extensor activity. Finally, there were no indications that the stretching of ankle extensor muscles during swing was a factor in regulating the transition from swing-to-stance.

INTRODUCTION

A major focus of recent research on the neuronal control of walking has been on establishing the functional role of sensory signals in regulating stepping (Dietz and Duysens 2000; Duysens et al. 2000; Lam and Pearson 2002b; Pearson 2003). Sensory feedback is crucial for adapting motor output to injury or growth (Pearson 2000) and ensures that locomotor output is appropriate for the task at hand (Prochazka 1996; Rossignol et al. 1988). One of the most important roles for feedback during walking is to control the timing of the transition from stance to swing. This has been demonstrated in the walking systems of insects (Bässler and Büschges 1998; Pearson and Duysens 1976), cats (Grillner and Rossignol 1978; Pearson 1995; Whelan et al. 1995), and human infants (Pang and Yang 2000). In the hind leg of the cat, the sensory receptors involved in mediating the stance-to-swing transition have not been fully identified, but they include Golgi tendon organs in ankle extensor muscles (Pearson et al. 1998; Whelan et al. 1995) and muscles spindles fom hip flexor muscles (Hiebert et al. 1996). Behavioral observations in spinal cats and human infants also indicate the importance of afferent signals from the hip in controlling the initiation of the swing phase (Grillner and Rossignol 1978; Pang and Yang 2000).

Given the importance of afferent feedback in controlling the transition from stance to swing, it is reasonable to ask whether the transition from swing to stance is similarly controlled. Afferent regulation of this transition would be an effective way to ensure correct placement of the foot during stance despite the different movements required by different locomotor tasks. A crucial aspect of a stable transition to stance is a sufficiently protracted leg, thus making the position of the hip a good candidate for an afferent signal regulating the swing-to-stance transition. Evidence for this role has come from recent observations of the effects of perturbing hip movements in decerebrate walking cats (Lam and Pearson 2001). Assisting hip flexion during the swing phase results in a shortening of flexor burst duration and an increase in duration of the subsequent bursts in extensors. Resisting hip flexion during swing has the opposite effect. In some situations, resisting hip flexion leads to the maintenance of flexor activity for the duration of the resisting perturbation (Lam and Pearson 2001). Another indication for a role of hip afferents in promoting the transition from flexion to extension is that imposed flexion movements of the hip during fictive locomotion in DOPA/nialamide-treated spinal cats shorten flexor burst duration when applied near the end of the flexor bursts (Andersson and Grillner 1981). Recently a similar observation has been made in the foreleg during fictive locomotion in decerebrate cats (Saltiel and Rossignol 2004). Protraction of the shoulder near the end of the burst activity in flexor motoneurons shortens the duration of flexor bursts and promotes an earlier onset of extensor activity. Thus in both hind legs and forelegs, there are good indications that sensory receptors in proximal regions of the legs are involved in controlling the timing from swing to stance.

An important aspect of this transition in the hind legs is the initiation of activity in knee and ankle extensor muscles commencing ~80 ms before ground contact. This prestance extensor activity occurs during the first extension (E1) phase of the locomotor cycle, i.e., during the period when the knee and ankle joints are extending (Engberg and Lundberg 1969). Thus gaining an understanding of the mechanisms regulating the timing of swing-to-stance transition requires knowledge of the neuronal mechanisms responsible for initiating burst activity in the knee and ankle extensor muscles near the end of the swing phase. As mentioned earlier, there is now some evidence from reduced preparations to suggest that afferent signals linked to flexion movement of the hip joint can influence the timing of

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the onset of extensor activity. If this is also true for normal walking animals, then we predicted that some feature(s) of the kinematics of movement at the hip joint should be correlated with the timing of the onset of the E1-associated extensor activity. A similar logic was used in an earlier study reporting a role for hip afferents in regulating the extension to flexion (stance to swing) transition (Grillner and Rossignol 1978). Thus one objective of this investigation was to determine the relationship between joint kinematics and the time of onset of ankle extensor activity in normal walking cats. To dissociate movements at the hip from movements at the knee and ankle joints, we examined animals stepping in a variety of situations: a horizontal treadmill, up and down steps, and stepping over objects.

Another objective of the present investigation was to examine in more detail the influence of imposing flexion movements of the hip on extensor activity in decerebrate walking cats. This influence was examined only qualitatively in the earlier study (Lam and Pearson 2001). Here we determined the relationship between the time of termination of flexor bursts and the time of onset of the subsequent extensor bursts with the aim of establishing the extent to which these two events are linked when hip flexion is assisted. In addition we measured the value of hip joint angle at the time of the transition from flexor to extensor activity with and without assisting hip flexion during the swing phase to assess whether a sensory signal related to hip position could be a factor in the initiation of the swing-to-stance transition. A preliminary description of some of our findings has been published (Pearson et al. 2003).

METHODS

Two sets of experiments were performed in this investigation. The first was designed to examine the influence of assisting hip flexion during walking in decerebrate walking cats, and the second was designed to investigate of the relationship between electromyographic (EMG) activity in ankle extensor and hip flexor muscles and the kinematics of hind leg movement in normal walking cats. All experimental procedures were approved by the Health Sciences Animal Welfare Committee at the University of Alberta.

Assisting hip flexion in decerebrate walking cats

The procedure for examining the effects of assisting hip flexion was similar to that used in an earlier investigation from our laboratory (Lam and Pearson 2001). Briefly, each animal ($n = 2$) was anesthetized with isoflurane, and a tracheal cannula inserted for continued administration of the anesthetic. Blood pressure was monitored via a cannula inserted into one carotid artery, and the other carotid was ligated. One jugular vein was cannulated for the administration of fluids and drugs. The left hind leg was then partially dennervated by cutting the saphenous, sural, superficial peroneal, and distal tibial nerves. This removed cutaneous input from most of the hind leg. Bipolar recording electrodes (Cooner Wire AS632) were then sewn into the iliopsoas (IP) and medial gastrocnemius (MG) muscles of both hind legs. The wires of these electrodes were led under to skin to a multi-terminal connector positioned above the animal's back. Next, the iliac crests were exposed, and a stout wire was threaded through holes drilled in both crests. The two ends of the wire were clamped to each crest. This wire was later clamped to an external frame to support the hindquarters while the animal was walking on a treadmill. Reflective markers (diameter: ~0.5 cm) were placed above the iliac crest, the hip joint, and knee and ankle joints and on the paw and toe of the left leg. These markers were used to determine the kinematics of leg movements using the Peak Motus 8.2 motion-analysis system (Peak Performance Technologies). Triangulation was used to determine the position of the knee joint.

After this preparatory procedure, the animal was transferred to a frame mounted above a treadmill. The head was placed in a sterotaxic holder and wire through the iliac crest fixed to a supporting frame. Approximately 2.5-cm-wide surgical tape was wrapped around the thigh of the left hind leg, and a loop of 1.5 mm string was attached to the anterior edge of the tape. This loop was used to manually assist hip flexion during walking sequences. The animal was then decerebrated by transecting the brain stem rostral to the superior colliculus and mammillary bodies and removed from the anesthetic immediately following decerebration. Both animals began to walk spontaneously ~30 min later, although electrical stimulation of the mesencephalic locomotor region (Grillner and Shik 1973; Shik et al. 1966) was used to facilitate walking in one animal.

EMG and kinematic analysis of hind leg movements in intact animals

The second objective of this investigation was to determine which kinematic parameters of hind leg movement correlate with the onset of ankle extensor activity immediately preceding the swing-to-stance transition. This was examined in four intact adult cats walking in a variety of situations: a horizontal treadmill at different speeds (described as "treadmill" in figures), along a series of steps at −25, 0, and 25° angles (described as "down pegs," "level pegs," and "up pegs" in figures), and stepping over an object placed on a horizontal walkway (described as "leading" or "trailing" in figures). Figure 4 shows three of these tasks. Each animal was first trained daily for 1–2 wk to walk consistently in the three situations. Training consisted of inducing animals to participate in the various tasks with food and affection rewards, and lasted for ~1 h/day. Next, bipolar EMG electrodes (Cooner Wire AS632) were implanted into muscles of the right hind leg under general anesthetic (isoflurane) and aseptic conditions. In all animals, EMG electrodes were placed in the knee extensor vastus lateralis (VL), the hip flexor iliopsoas (IP), and the ankle extensors soleus (Sol), lateral gastrocnemius (LG), medial gastrocnemius (MG). In three animals, electrodes were also placed in knee flexor/hip extensor semitendinosus (ST) muscle. The leads from the EMG electrodes were led under the skin to a multi-pin socket fixed with screws and dental acrylic to the animal's skull. While anesthetized, adhesive reflective markers (diameter: 0.5 cm) were placed over the iliac crest, the hip, knee, and ankle joints, and on the end of the paw and the fifth digit of the right hind leg.

Data collection and analysis

In decerebrate cats, data were collected for the duration of time that an animal walked regularly (between 1 and 2 h). In intact cats, data were collected for ~10–20 trials/day for one to two tasks, rotating through the various tasks over the course of 1–2 wk. Because we were seeking kinematic parameters that were related to the onset of stance in different types of movements, we did not screen trials based on speed or range of movement. We did screen trials qualitatively for smoothness of movement.

During regular sequences of walking in the decerebrate animals, and in intact animals walking under different conditions, the EMG signals were recorded on an eight-channel Vetter 4000A PCM recorder. One channel of the recorder was reserved for a signal from the Peak Motus motion-analysis system for later use in synchronizing the EMGs with video data. During all trials video data were recorded, and a time code and a signal for synchronizing EMG with video were added to the video data. The Peak Motus system was later used to track joint movements and calculate the kinematics of movements at the hip, knee, and ankle joints. The length and velocity of MG was calculated from the knee and ankle angles. The length when both knee

and ankle joints were at 90° was taken as 0, and changes from this were calculated using trigonometry assuming that the proximal attachment of MG is located on the femur 0.5 cm from the knee joint and the length of the attachment of MG on the calcanium is 1.5 cm from the ankle joint (Goslow et al. 1973). The velocity of the length changes was calculated by numerically differentiating the calculated length.

After the storage of EMG and video data, the EMGs recorded from decerebrate walking animals were digitized off-line at 1 kHz using the Axotape data-acquisition system (Axon Instruments). The Peak Motus system was used to track the movements of the joints and calculate the kinematics of the hip, knee, and ankle. Custom-written software (Matlab) was used to measure cycle periods, burst durations, the relative timing of the onset of burst activity in different muscles, and the relationship between muscle activity onset and kinematics. Figure 5 shows kinematic data and ankle extensor activity (MG muscle) from a down pegs and a leading trial. The angles measured at the time of onset of MG activity are indicated by the dotted lines.

The EMG data from intact animals was digitized (600 or 1,200 Hz) using Peak Motus. Custom-written software in Matlab was used to rectify and low-pass filter (1st-order Butterworth, 20 Hz) raw EMG signals as well as examine the relationship between kinematics and the initiation of muscle activity.

Statistical analysis

A two-tailed *F*-test, which tests for equality of variance, was used to compare the amount of variance among angles at the time of ankle extensor activation (Woolson and Clarke 2002). Linear regression was used to calculate the correlation coefficients between variables. Student's *t*-test was used to test for changes in cycle periods and burst durations. ANOVA test was used to compare hip angles in assisted and unassisted conditions.

RESULTS

In normal cats walking on a horizontal surface, the onset of stance (i.e., foot contact with supporting surface) in a hind leg is preceded by extension at the knee and ankle joints. These extension movements are associated with activity in the extensors across the knee and ankle joints (Engberg and Lundberg 1969), and this activity is an early neuro-muscular event underlying the transition from swing to stance. The concept we wished to examine in this investigation was whether sensory feedback via afferents arising in the hip region contributes to the initiation of burst activity in the ankle and knee extensor muscles.

Influence of imposed hip flexion during walking in decerebrate cats

A previous study from this laboratory (Lam and Pearson 2001) reported that assisting the flexion movement of the hip during treadmill walking in decerebrate cats shortened the duration of bursts in the hip flexor muscle IP. In that study, the shortening of the duration of hip flexor activity in response to hip flexion was associated with an advanced onset of burst activity in the ankle extensor muscle MG. In the present investigation, our initial goal was to examine the relationship between the time by which MG burst activity was advanced and the time by which IP burst activity was shortened.

Figure 1*A* shows EMG data for a short period of walking during which flexion of the hip was assisted during one step cycle. These data clearly show that assisting hip flexion reduced the duration of the burst in the IP muscle (a2 compared with a1) and advanced the following burst of activity in the MG muscle, which is consistent with previous observations. To quantify these and similar data, we plotted the reduction in the

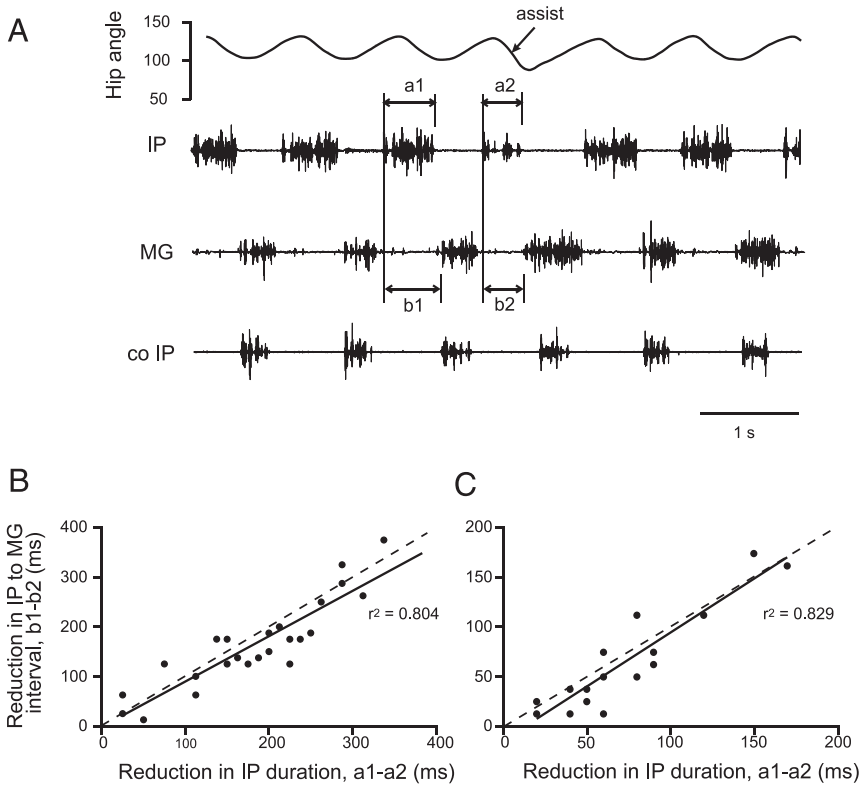

FIG. 1. Assisting hip flexion in decerebrate walking cats reduces the duration of iliopsoas (IP) bursts and advances the onset of activity in medial gastrocnemius (MG). *A*: electromyographic (EMG) recordings showing the parameters measured to quantify the changes in timing of the IP and MG bursts. During this sequence, the flexion movement of the hip during 1 step cycle (labeled "assist") reduced the duration of the IP burst (a2 compared with a1) and advanced the onset of the following MG burst (b2 compared with b1). *B* and *C*. scatter plots for data from 2 animals showing that the advance in the time of onset of the MG bursts (b1–b2) produced by assisting hip flexion is highly correlated with the reduction in IP burst duration (a1–a2). —, best fitting lines to the data; - - -, expected relations if the advance in the onset of MG bursts exactly equaled that reduction of the duration of IP bursts.

IP burst duration relative to the duration of the immediately preceding IP burst ($a_1 - a_2$ in Fig. 1*A*) versus the reduction in the interval between the onset of IP bursts and the onset of the following MG burst ($b_1 - b_2$ in Fig. 1*A*). Plots for two animals are shown in Fig. 1, *B* and *C*. The reduction in IP duration and the advance in MG burst onset parameters were strongly correlated ($r^2 = 0.80$ and 0.83), and the best fitting lines were close to the line of identity (dotted in Fig. 1, *B* and *C*). It is important to note that the time of the onset of the IP bursts of the assisted cycles relative to the time of onset of the preceding IP bursts was not influenced by the imposed hip flexion (Figs. 3*A* and 4, *B* and *C*), thus eliminating the possibility that the hip flexion simply shortened the IP bursts, but did not influence the time of onset of MG activity. The shortening of the IP bursts and advance of the MG bursts did not significantly influence the timing of stepping in the contralateral leg. This can be seen in the example in Fig. 1 and was quantified by comparing the change in cycle period of the contralateral leg with the change in IP burst duration (not shown). Changes in the contralateral cycle period were insignificant ($P < 0.05$, paired *t*-test) and not correlated to changes in the IP burst duration ($r^2 = 0.015$ and 0.070 for the 2 animals.) There were also no significant changes in the duration of the contralateral IP burst, indicating the absence of any influence on the timing of the contralateral swing-to-stance transition.

In addition to reducing the duration of the IP bursts, the imposed hip flexions also reduced the amplitude of these bursts (Fig. 2). This can be seen in the example shown in Fig. 2*A* and in the comparisons of the averages of the IP bursts with and without imposed hip flexion (Fig. 2, *B* and *C*). The ▨ in the *top traces* in Fig. 2, *B* and *C,* indicate the average effect of assisting hip flexion in two animals. Note also in these figures that the timing of onset of the IP bursts relative to the preceding IP burst was similar during unassisted (*top*) and assisted (*bottom*) flexion of the hip.

The next issue we explored was the relationship between the hip angle at the time of the transition from flexor (IP) to extensor (MG) activity. Figure 3*A* shows records of the hip angle and IP activity for a short walking sequence during which hip flexion was assisted for one cycle. Note the clear reduction in the duration and amplitude of the IP bursts produced by the imposed movement and that the hip angle at the times of the termination of the IP burst was similar during the assisted cycle (indicated by the arrows on the plot of hip angle) and unassisted cycles. The lower ends of the bar graphs in Fig. 3, *B* and *C,* show the averages of the hip angle at the time of termination of the IP bursts for assisted and unassisted cycles in two animals. In one animal (*left*), the hip angle at the time of termination of the IP bursts was not significantly altered when hip flexion was assisted (83° with both unassisted and assisted cycles; $P = 0.133$, 1-way ANOVA), whereas in the second animal, the hip angle at the time of termination of the IP bursts was reduced by ~5° (109° with unassisted cycles and 104° with assisted cycles; $P = 0.0025$, 1-way ANOVA). A noteworthy difference in the stepping behavior of the two animals was that the magnitude and rate of hip flexion was much larger in the first (Fig. 3, *D* and *E*). Furthermore, the assisted movements in the second animal noticeably increased the magnitude of hip flexion, and the change in angular velocity produced by the perturbation was larger in the this animal (compare slopes of flexion movements in Fig. 3, *D* and *E*).

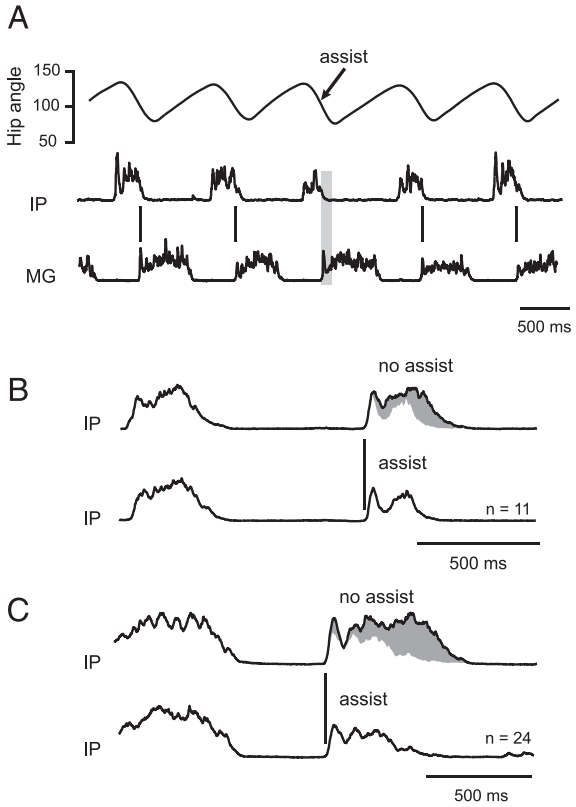

FIG. 2. Assisting hip flexion in decerebrate walking cats reduces the magnitude of IP bursts. *A*: rectified and filtered EMG records from IP and MG muscles during a short walking sequence showing that assisting hip flexion on 1 cycle (labeled assist) reduced the magnitude and duration of the IP. These records were from a different animal than those shown in Fig. 1*A*. Note also the advance in the time of onset of MG activity associated with the reduction in IP burst duration (▨) and the close linkage of the termination of IP bursts with the onset of MG bursts (|). *B* and *C*: averages from 2 animals of the rectified and filtered IP bursts during unassisted (*top*) and assisted (*bottom*) flexions of the hip. ▨, difference between the 2 sets of averages. Note that hip flexion decreased the magnitude and duration of the IP bursts. Note also that assisting hip flexion had no influence on the timing of the onset of IP bursts relative to the preceding IP bursts. |, the reference times for the averages.

Relationships between leg kinematics and the onset of the ankle extensor activity in normal walking cats

Based on our observations in decerebrate walking cats, as well as results from previous studies (Lam and Pearson 2001, 2002a), we formed the hypothesis that signals related to hip position during swing contribute to initiating the transition from swing to stance. The issue we next explored was whether this is also true in normal walking cats. Initially we attempted to assist hip flexion in cats walking normally on a treadmill to establish whether this perturbation advanced the onset of extensor activity. However, this strategy failed because walking was consistently disrupted in an unpredictable manner thus preventing a clear assessment of the responses to the imposed flexion movements. As an alternative we examined a variety of kinematic parameters of hind leg movement when animals walked in different situations (Figs. 4 and 5) and looked for parameters that were most closely correlated with the onset of

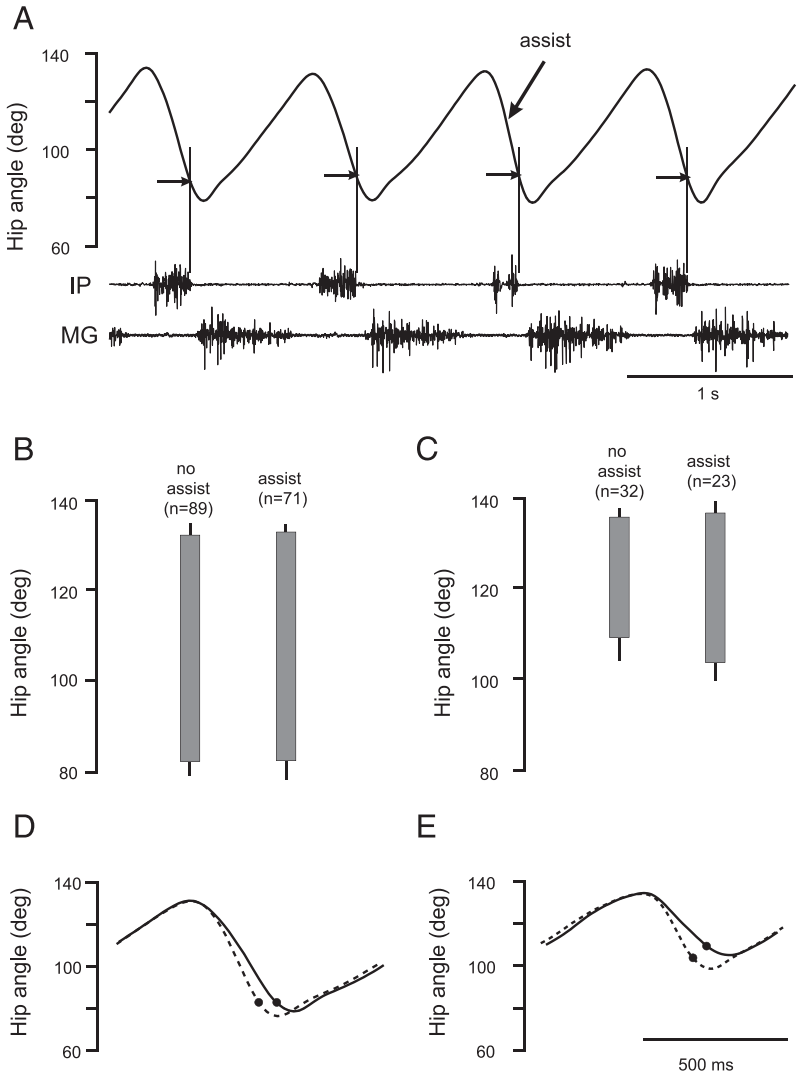

FIG. 3. Hip angle at the time of the transition from flexor (IP) to extensor (MG) activity is similar during assisted and unassisted cycles in decerebrate walking cats. *A*: short sequence of stepping showing a plot of the hip angle (*top*) and EMG records from IP and MG (*bottom*) during 3 unassisted and 1 assisted (labeled assist) cycles. The horizontal arrows indicate the hip angle at the time of termination of the IP bursts. *B* and *C*: bar graphs for 2 animals showing the range of the hip flexion from the time of swing onset (*top*) to the time of termination of IP bursts (*bottom*). Short vertical lines indicate SDs. *D* and *E*: superimposed plots of hip angle during 1 unassisted (solid lines) and 1 assisted (dotted lines) cycle illustrating the influence of assisting hip flexion on hip movement in the 2 animals yielding the data in *B* and *C,* respectively. The solid dots indicate the times the IP bursts terminated in these trials.

extensor activity during the swing phase. We predicted that one of these parameters would be the hip angle.

Consistent with this prediction was our finding that the angle at the hip at the time of MG burst onset remained relatively constant in all the tasks in three of the four animals (Fig. 6). In the fourth animal, the hip angle at the onset of MG activity was similar in five of the six tasks. In contrast to the relative constancy of hip angle at the onset of MG activity, the knee and ankle angles at the same instant varied considerably depending on the task. The highest variation was seen for the ankle, which had a range of 50–60°, twice that of the hip. This is apparent in Fig. 5, which shows two example trials for one cat. The hip angle at the time of MG onset is similar for all four steps, whereas the knee and ankle angles vary by ~40°. This suggested that the position of the hip was an important signal for the initiation of MG activity. However, it should be emphasized that this does not indicate that the position of the hip was the sole afferent signal related to this initiation. The differences between the angles of the hip at the time of MG onset during different tasks suggests that other signals, in addition to signals from the hip, are probably involved in terminating swing. The pattern of variation in the knee and ankle angles at the time of onset of MG activity for the different tasks was consistent in the four animals. For example, this ankle angle was always smallest when the leg was leading over an object and largest when walking on the treadmill (Fig. 6).

We also examined the angular velocity of the hip joint and found that it varied up to fivefold from trial to trial at the time of the swing-to-stance transition even within the same task (data not shown). Furthermore, the maximum angular velocity of the hip occurred ≤300 ms before the onset of extensor activity.

Although our observations on joint angles indirectly support the hypothesis that afferent signals related to hip position play a role in initiating extensor activity, another possibility is that a signal derived from a combination of information from sensory receptors distributed throughout the leg is a critical factor regulating the onset of extensor activity. For example, this signal could provide information about global variables such as the position of the paw relative to the body or to the ground. The former has been termed "limb axis" by Bosco and Poppele (2001). To examine whether these variables could provide a reliable signal for initiating the swing-to-stance transition, we calculated the length and angle of the limb axis at the time of extensor activation (we define limb axis as the

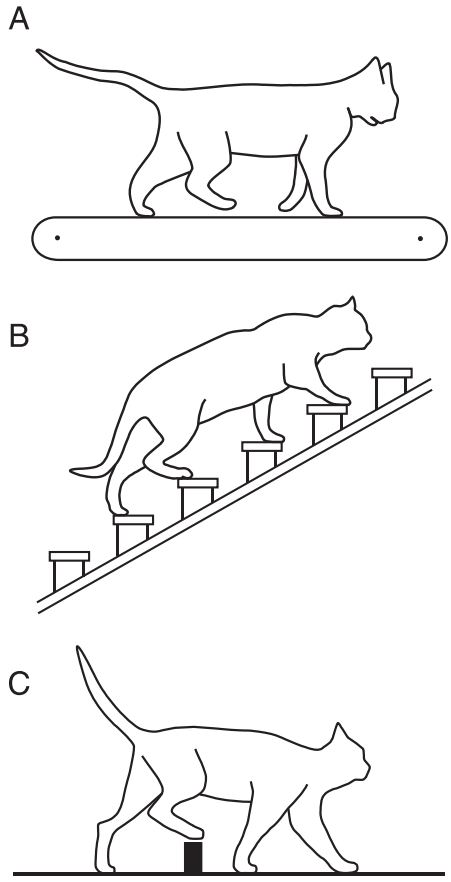

FIG. 4. Locomotor tasks used to examine the kinematics of leg movement during the swing phase in intact walking cats. *A*: walking on a horizontal treadmill. *B*: walking on pegs. The slope of the pegs was either up (as shown), horizontal, or down. *C*: stepping over blocks. The experimental leg could either lead (as shown) or trail.

angle between the toe, the hip, and a vector from the hip, forward and parallel to the walking surface) as well as the distance and direction of the toe from where it eventually contacted the ground for the different tasks.

Figure 7 compares the variability of the limb axis angle to the variability of the hip, knee, and ankle angles at the time of the onset of MG activity for all tasks. In all four animals, the angle of the limb axis was more variable that the hip angle but less variable than the knee or ankle angles. We also examined the variability of these angles at the time of onset of MG activity within tasks (data not shown). The variability of all angles was lower for any individual task than that for all tasks pooled. Importantly, the difference between variability of the hip angle for individual tasks and for all tasks pooled was generally much less than the difference between the variability of individual and pooled tasks for other angles. This is consistent with our hypothesis that signals related to the hip angle contribute more to regulating the swing-to-stance transition the signals related to the other three angles. Examination of the *length* of the limb at the time of onset of MG activity revealed a large variability between tasks and major differences in the profiles of the limb axis length for the different tasks (Fig. 8). The limb axis length ranged from ~15 to 20 cm at the time of the onset of MG activity, and during steps down pegs, for example, the limb axis length generally started quite short and became longer, but during leading steps over obstacles, the limb axis started long and shortened dramatically through the swing phase. We also examined the position of the toe relative to where it eventually touched the ground at the time of onset of MG activity. Again, there was no consistent pattern in the distance or the angle between the toe and the point where the toe eventually touched ground and the time MG became active. This can be seen in Fig. 6 in which the distance from the toe at the time of MG burst onset to the position of ground contact varied depending on the task.

Another sensory signal that might be used to initiate activity in the ankle and knee extensors is from the extensor muscles themselves. To examine this possibility in more detail, we calculated the profiles of length and velocity during the swing phase from the kinematics of knee and ankle movements (see METHODS) in an attempt to identify a consistent relationship between the time of onset of MG activity and the length and lengthening velocity of the MG muscle. The first observation was that MG length at the time of MG burst onset varied considerably from task to task (Fig. 9). Assuming that gamma drive to muscle spindles is similar from task to task, this observation makes it unlikely that signals related only to muscle length are involved in initiating the onset of MG bursts. Another observation was the absence of any consistent relationship between the latency from the time of peak lengthening velocity to MG burst onset and the maximum lengthening velocity (Fig. 10). We did observe a stronger inverse relationship when animals were walking on the treadmill at different speeds. However, the slope of this relationship was steep and the latencies at the slower walking speeds were quite large (~300 ms).

A final observation of relevance to the issue of the mechanism(s) regulating the onset of extensor activity was that there was a qualitative difference in timing and duration of the IP bursts in intact animals compared with decerebrate walking animals. In decerebate animals, the termination of IP activity was always tightly coupled to the onset of MG activity (Fig. 1), whereas is intact animals, this was not always the case depending on the task (Fig. 11). In some situations, IP was observed to be active during the E1 phase, and thus active at the same time as the E1-related activity in MG. When this occurred there was very often a clear distinction between F- and E1-related activity in IP as shown in Fig. 11, *B* and *C*, with the termination of the F component of the IP bursts closely linked to the onset of MG activity (Fig. 11, - - -). The relevance of these observations to our understanding of the swing-to-stance transition is considered in the DISCUSSION.

DISCUSSION

Recent studies in the cat have indicated that the timing of the swing-to-stance transitions in the hind legs and forelegs are regulated to some extent by proprioceptive signals from the hip and shoulder regions, respectively (Lam and Pearson 2001, 2002a; Saltiel and Rossignol 2004). A priori, sensory feedback related to hip position might be particularly important in regulating swing-to-stance transitions in the hind legs under a variety of conditions for two reasons. First, during the swing phase of walking, the position of the hip is most reflective of the protraction of the leg, whereas the positions of the knee and ankle joints are more reflective of the height of the foot relative to the ground. Second, the movement of the hip during loco-

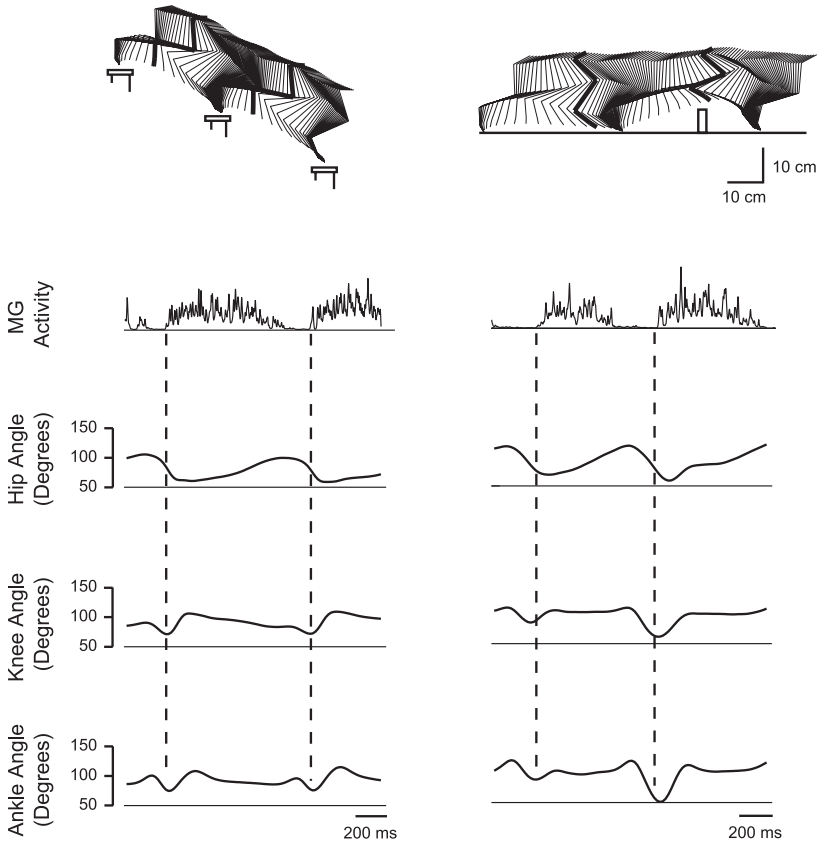

FIG. 5. Example data from a ′down pegs“ and a ”leading” trial. *Top*: stick figures of the hind leg at a rate of 60 Hz. Bold traces show the position of the leg at the time of onset of MG bursts. *Bottom*: rectified and filtered MG EMGs and angles of the hip, knee, and ankle. Dotted lines indicate the time of MG bursts. Note that the angle of the hip was relatively constant at the time of MG activation despite the variation in the interval between the onset of MG bursts and ground contact (as seen by the position of the bold traces in the *top* relative to the position at ground contact). Also note the variation in the knee and ankle angles at the time of onset of MG activity.

motion is biphasic, passing through any one position only once during the swing phase. In contrast, the joints of the knee and ankle both extend and flex during the swing phase and thus may pass through the same position twice during the swing phase. The findings of the present investigation provide additional evidence that afferent signals arising from receptors in the hip region are an important component of the mechanism that regulates the swing-to-stance transition.

Although the position of the hip is a good candidate for an afferent signal regulating swing-to-stance transitions, other signals are also involved. For example, our laboratory has reported inhibitory coupling between, first, the systems that generate flexor activity of the two hindlegs (Lam and Pearson 2001), and secondly, the systems that generate flexor activity of ipsilateral fore and hind legs (McVea et al. 2005). Furthermore, studies of fictive locomotion have shown that a range of afferents, such as group I afferents from ankle and knee extensors as well as group II afferents from hip and knee flexors, have an effect on the duration of flexor activity (McCrea 2001).

Hip flexion influences swing-stance transition in decerebrate walking cats

Our conclusion that hip afferents have a role in regulating the timing of the swing-to-stance transition is strongly supported from our observations in decerebrate walking cats. First, assisting flexion movements of the hip joint shortened the duration of bursts in the IP muscle (hip flexor) and promoted an earlier onset of activity in the MG muscle (ankle extensor) that closely matched the shortening of hip flexor activity (Fig. 1). Because the onset of burst activity in ankle and knee extensors shortly before ground contact is the first neuromuscular event in the swing-to-stance transition, this observation demonstrates directly that signals related to hip position could be involved in initiating this transition. Assuming that the basic network for the timing of activity in the central pattern generator (CPG) is mutual inhibition between flexor and extensor half-centers (Lundberg 1980), the simplest explanation for the changes in timing of the IP and MG bursts is that sensory signals generated during hip flexion act to terminate activity in the flexor half-center and release the extensor half-center from inhibition. Alternatively, changes in timing of the IP and MG bursts could be produced by reflex modification of interneuronal networks located between the CPG and motoneurons or even by direct reflex actions on motoneurons. However, we believe these alternative possibilities are less likely because of the strong correlation between the time of termination of the IP bursts and the time of onset of the MG bursts (Fig. 1, *B* and *C*). Moreover, no studies on nonwalking preparations have reported any reflex action from hip muscle afferents on ankle extensor motoneurons. On the other hand, the group Ia afferents from hip extensors are known to form inhibitory connections onto IP motoneurons (Eccles and Lundberg 1958), so it is quite conceivable that increased activity in these afferents partially explains the reduction in the magnitude of the IP bursts we observed when hip flexion was assisted (Fig. 2).

An analysis of the kinematics of the hip joint at the time of extensor activation and flexor burst termination showed the hip angle could be similar in both control and assisted step cycles (Fig. 3). In one animal, the hip angles at the time of the termination of the IP bursts were virtually identical in the two

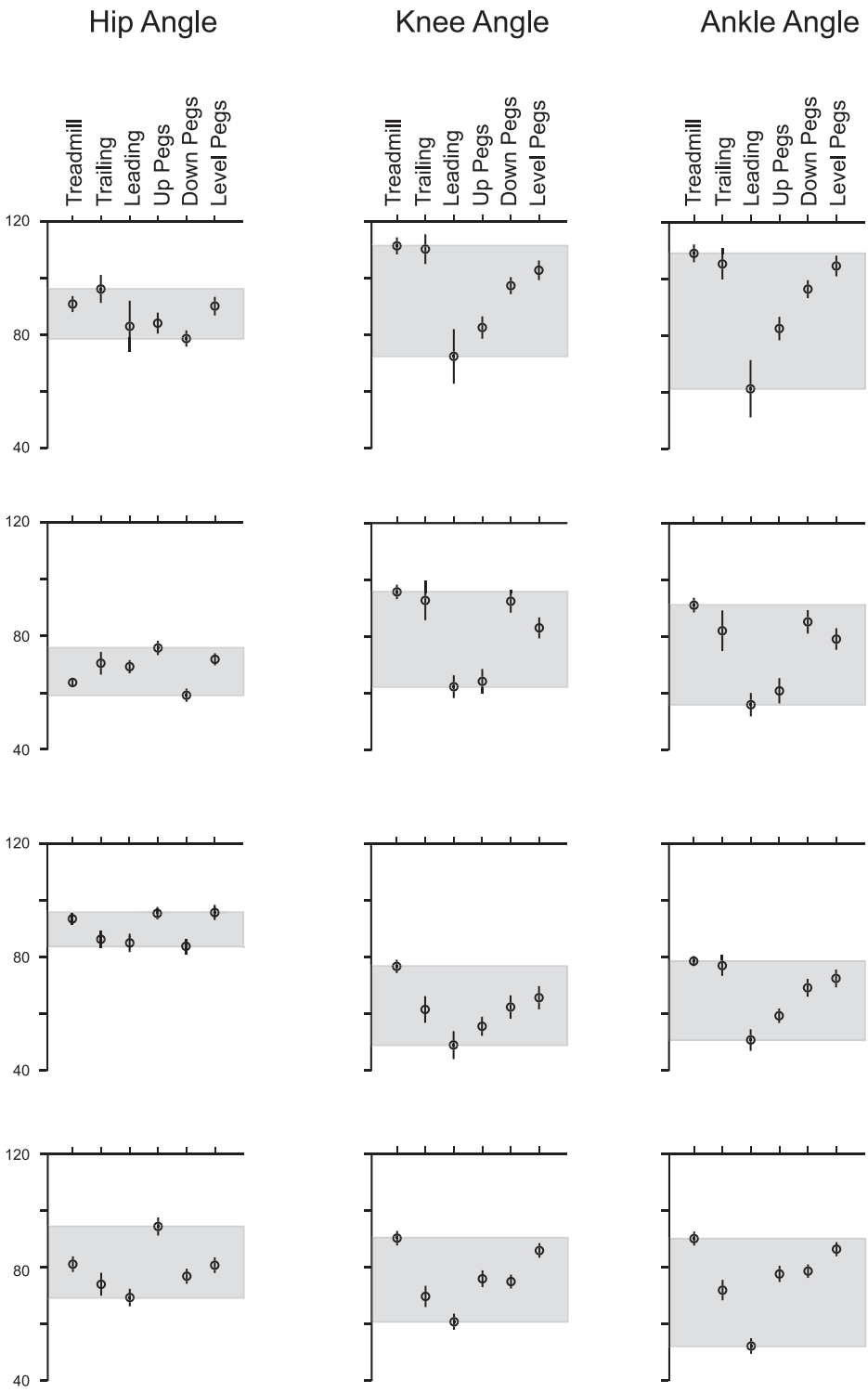

FIG. 6. Hip angle is the least variable joint angle at the onset of extensor activity during the swing phase. The 3 main columns show the angles of the hip (*left*), knee (*middle*), and ankle (*right*) at the time of onset of MG activity for the 4 experimental animals (each row represents 1 animal). For each joint, the data for the different tasks are shown in 6 adjacent sub-columns. Circles represent means and bars represent 95% confidence intervals for each task. Shaded area represents the range of the means of all 6 tasks. This range is smallest for the hip for all animals. See Fig. 9 for the number of trials for each condition.

situations (Fig. 3*B*), whereas in the second animal it was decreased by ~5° during assisted trials (Fig. 3*C*). We have no explanation for this difference, but it may be due to differences in the strength of hip flexion movements in the two animals (compare Fig. 3, *D* and *E*). In the first animal, these movements were larger in magnitude and closer to those in normal walking animals. If this animal reflects the situation during normal walking, then the fact that hip angle at the time of the termination of IP activity remained constant suggests that a sensory signal related to hip position is involved in initiating the transition from flexor to extensor activity.

Hip position is part of a multi-modal signal controlling stance

Following our results from decerebrate walking animals, we predicted that the position of the hip could be an important signal controlling the swing-to-stance transition in intact walk-

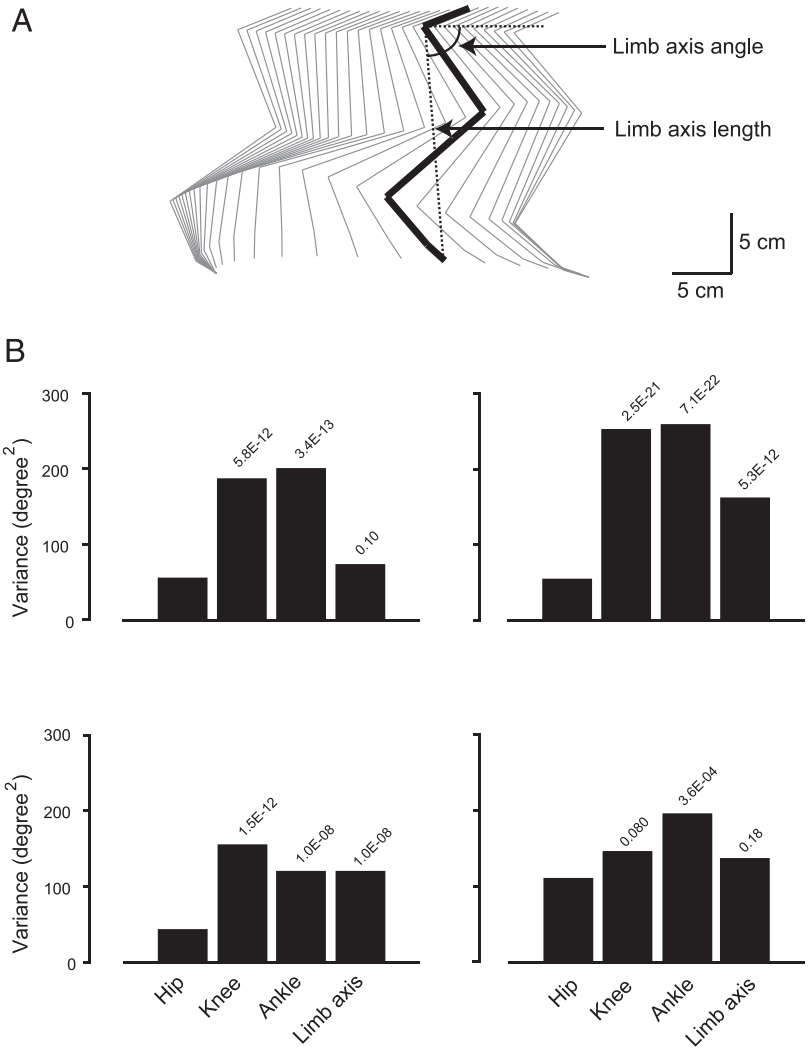

FIG. 7. Hip angle at the time of the onset of extensor activity is less variable than other joint angles or limb axis angle. *A*: stick figure defining limb axis angle and length (see Fig. 8 for data on limb axis length). *B*: variance of joint and limb axis angles at MG activation across all conditions. Each graph shows data from 1 animal. Number of trials for each animal is the sum of trials shown in Fig. 9. *F* statistic above columns for knee, ankle, and limb axis angle is result of 2-tailed *F*-test testing for equality of variance between hip angle and that angle.

ing cats. By examining the kinematics of leg movements in a variety of situations (Figs. 4 and 5), we found the hip angle at the time of onset of activity in ankle extensor muscles was relatively constant compared with knee and ankle angles (Fig. 6). Although indirect, we interpret this observation as evidence to support the hypothesis. It is important to note that we have not concluded that the position of the hip is the sole signal that triggers a transition from swing to stance. In fact, our data show this to be unlikely for three reasons. First the hip angle at which MG activity begins varies somewhat from task to task. This suggests the strength of the feedback from hip-related afferents is either modulated by descending, task-dependent connections or that other afferent signals are involved. Second, the hip angles at which MG became active varied from cat to cat. Third, *cat 4* had a more varied hip position at the time of MG onset than the other cats. These points suggest that, although the position of the hip is an important part of a multi-modal afferent signal that triggers the transition to stance, the relative contribution of hip position to this signal likely varies from cat to cat and from task to task.

Accepting that multi-modal sensory signals are important in regulating the swing-to-stance transition, with signals from the hip being especially important, we need to consider which receptors give rise to these signals. Receptors in the hip joint capsule are unlikely to be involved because inactivation of these receptors has no effect on entrainment of the fictive locomotor rhythm in decerebrate cats (Kriellaars et al. 1994). More likely possibilities are stretch-sensitive receptors in hip extensor muscles and/or muscle spindles and Golgi tendon organs in hip flexor muscles (Lam and Pearson 2002a; Perrault et al. 1995). As for receptors in other regions of the leg, we know that electrical stimulation of group I afferents arising from spindles and Golgi tendon organs in ankle extensor muscles can reset the fictive rhythm to extension (Conway et al. 1987; Guertin et al. 1995) and thus have the potential for regulating the swing-to-stance transition. However, if we assume that gamma drive is similar during the different tasks examined, we can use the length and velocity of the MG muscle as an approximate indication of from group Ia afferent activity at the time of the swing-to-stance transition. The large variation in the length of MG at the time of activation (Fig. 9) and the weak relationship between the interval from the time of maximum MG velocity to MG activation and the maximum MG velocity (Fig. 10), indicate that signals from receptors in the ankle extensors are unlikely to have a significant role in controlling the swing-to-stance transition in intact walking cats.

Another possible signal regulating the timing of the swing-to-stance transition is one derived from a combination of

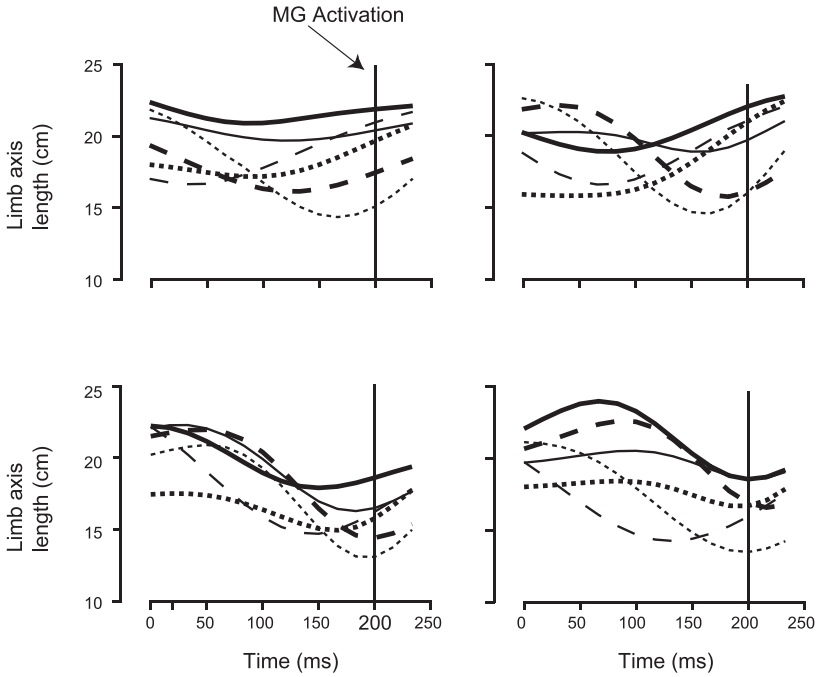

FIG. 8. Limb axis length at the time of MG activation varies with locomotor task. Average limb axis length throughout 1 step for different tasks. Each trace is the average of between 4 and 15 trials, time-locked on MG activation (shown as vertical line at 200 ms). The pattern of limb axis length through steps differs from task to task as does the length at the time on MG activation. Thick solid, treadmill; thick dashed, up pegs; thick dotted, down pegs; thin solid, level pegs; thin dashed, trailing over object; thin dotted, leading over object.

information from sensory receptors distributed throughout the leg to indicate global variables such as the position and angle of the paw relative to the hip (Bosco and Poppele 2001). However, we found that the distance between the toe and the body at the time of ankle extensor activation varied widely depending on the task (Fig. 8) and that the orientation of the toe relative to the body was less consistent than the angle of the hip (Fig. 7). We cannot definitively say that the endpoint of the limb is not a factor in activating extensors in late swing, but if it is, then the computation of the endpoint would necessarily require information about the position of all the joints of the leg. Our data, particularly those from decerebrate animals, show that the position of the hip would be an important component in this computation.

Integration of hip position signals into the CPG

An important question raised by our results is how sensory signals related to the hip position might be integrated into the CPG to influence transitions from flexor to extensor half-centers. This requires an understanding of the functional organization of the CPG. At issue is whether or not the onset of

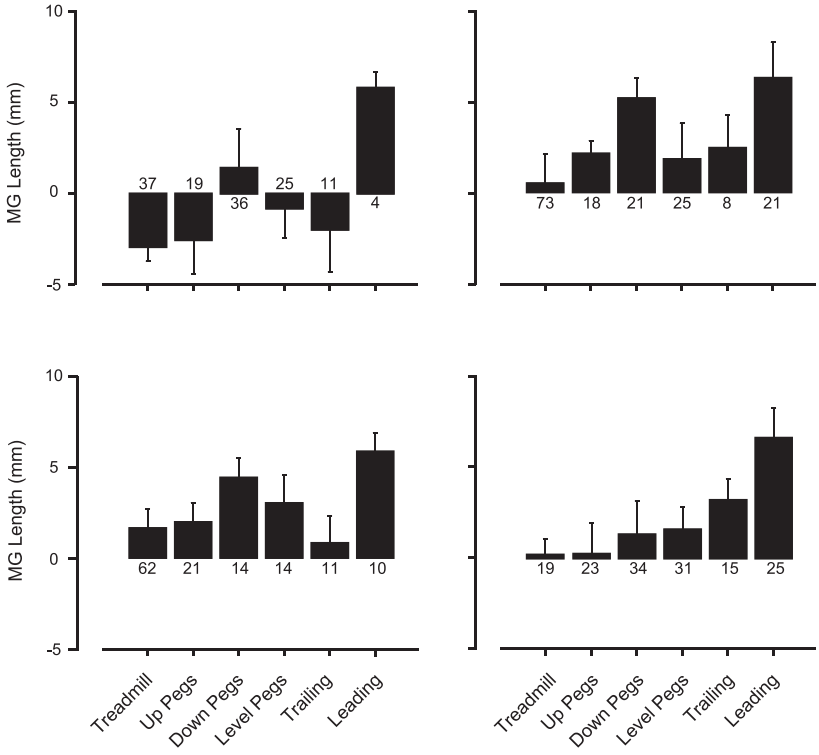

FIG. 9. The length of the MG muscle at the time of MG activation varies from task to task. Muscle length was calculated relative to length when knee and ankle angles are 90° (see METHODS). Each set of graphs shows the average length of MG at the time it became active during different tasks for one animal. Error bars represent one SD, and the numbers indicate the number of trials. Panels correspond to those in Figs. 6 and 7, thus these numbers also indicate the number of trials for Figs. 6 and 7.

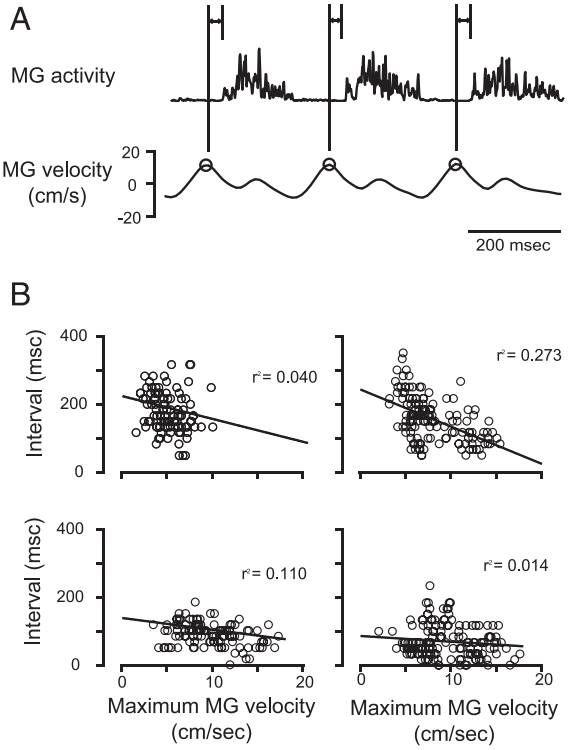

FIG. 10. Maximum lengthing velocity of MG is not correlated to the time from maximum lengthening velocity to the time of MG activation. *A*: 3 step cycles showing the measured parameters. ○, maximum lengthening velocity in MG during the 1st half of the swing phase, and the ↔ shows intervals from maximum velocity to MG activation. *B*: relationships between maximum lengthening velocity in MG and interval from maximum velocity to MG activation for the 4 experimental animals. Data from all 6 tasks are combined in each plot. Results of linear regression are also shown. Note the maximum intervals are large, and the relationship between the 2 parameters is weak.

activity in the knee and ankle extensors is linked to an overall switch from flexor to extensor activity in muscles throughout the leg. Lundberg (1980) has argued that it is not. This conclusion was based on the fact that the EMG recordings of Engberg and Lundberg (1969) showed that IP activity lasted well into the E1 phase and overlapped the early activity in ankle extensors. Lundberg concluded that "the E1 burst of activity in extensors has an origin extraneous to that of the half-centers," and proposed that the half-centers switch from flexor to extensor activity at the time of the termination of IP activity, i.e., at a time close to the time of ground contact. However, another interpretation of the same data, and one consistent with observations we have made in this investigation, is that the overall switch from flexor to extensor activity occurs at the time of onset of extensor activity and that the E1-associated activity in IP is generated by the extensor half-center. Evidence supporting this interpretation is that the termination of IP bursts was normally associated with the onset of MG activity at or near the F-E1 transition when animals walked on the treadmill (Fig. 11*A*) and, in situations in which activity in IP did occur during the E1 phase, that this activity was often distinctly segregated from the preceding flexion associated activity (Fig. 11, *B* and *C*). If the extensor-associated activity in IP is produced via an excitatory pathway linking the extensor half-center to the IP motoneurons, then we must assume that transmission in this pathway is task-dependent to allow for the variable occurrence of IP activity during E1 phase (Fig. 11). This scheme accounts for the strong linkage between the termination of IP bursts on the onset of MG bursts that occurs in decerebrate walking animals (Fig. 3) as a complete closing of the pathway from the extensor half-center to IP motoneurons due to loss of supraspinal facilitation of the pathway. If this scheme is accepted, then we propose that signals generated by flexion of the hip during swing inhibit the interneuronal networks generating the F phase of flexion (flexor half-center) and help promote the switching from the flexor to extensor half-center. This transition is associated with the onset of activity of knee and ankle extensors, and extensor related IP activity may or may not continue depending on the state of the putative connection from the extensor half-center to the IP motoneurons described in the preceding text.

Summary

In this study, we have shown that the position of the hip is an important part of the signal that initiates the swing-to-stance

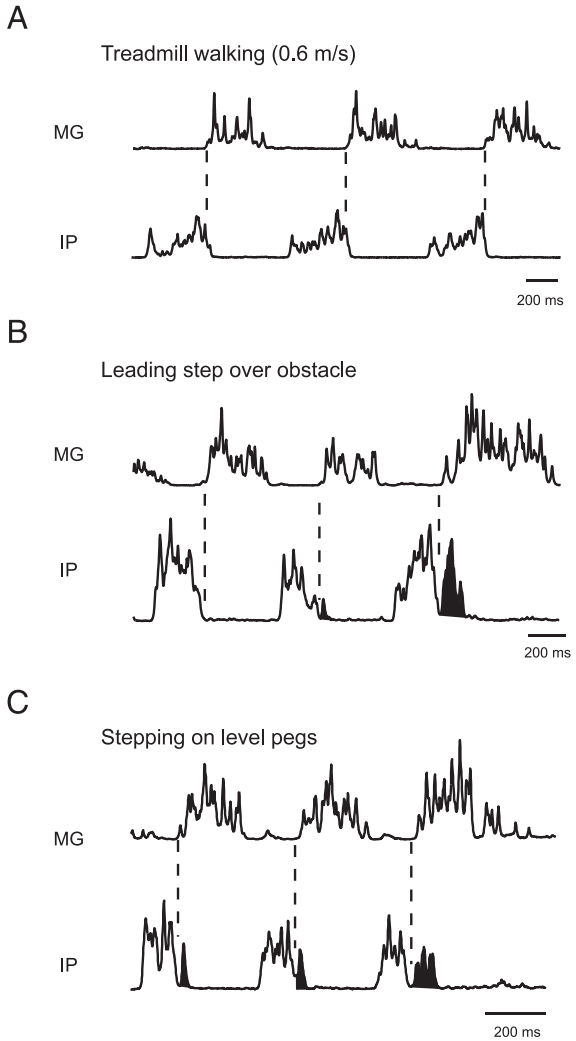

FIG. 11. Activity of IP in intact animals can overlap the onset of activity in MG. Records show rectified and filtered EMGs from IP an MG during short sequences of walking during three tasks. *A*: walking on treadmill. *B*: leading over an object (3rd step is over object). *C*: walking on pegs. Note that there was no overlap of the IP and MG bursts during treadmill walking, but clearly discrete bursts of activity in IP (■) overlapped MG activity when the animal stepped over the object and when it walked on the pegs. These bursts began close to the time of the onset of MG activity (- - -).

transition in the hindlegs of the walking cat. This expands our understanding of the role of sensory feedback in regulating phase transitions during walking and complements our knowledge of the role of hip position in regulating the stance-to-swing transition (Grillner and Rossignol 1978; Hiebert et al. 1996). In this role, the position of the hip is one part of a multi-modal signal that initiates swing (Pearson 2003), and we suggest that the hip has a similar role as part of a distributed signal initiating stance.

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