Contribution of Sensory Feedback to the Generation of Extensor Activity During Walking in the Decerebrate Cat

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Hiebert, Gordon W. and Keir G. Pearson. Contribution of sensory feedback to the generation of extensor activity during walking in the decerebrate cat. J. Neurophysiol. 81: 758–770, 1999. In this investigation we have estimated the afferent contribution to the generation of activity in the knee and ankle extensor muscles during walking in decerebrate cats by loading and unloading extensor muscles, and by unilateral deafferentation of a hind leg. The total contribution of afferent feedback to extensor burst generation was estimated by allowing one hind leg to step into a hole in the treadmill belt on which the animal was walking. In the absence of ground support the level of activity in knee and ankle extensor muscles was reduced to ~70% of normal. Activity in the ankle extensors could be restored during the “foot-in-hole” trials by selectively resisting extension at the ankle. Thus feedback from proprioceptors in the ankle extensor muscles probably makes a large contribution to burst generation in these muscles during weight-bearing steps. Similarly, feedback from proprioceptors in knee extensor muscles probably makes a large contribution to burst generation in these muscles because unloading and loading these muscles, by lifting and dropping the hindquarters, strongly reduced and increased, respectively, the level of activity in the knee extensors. This conclusion was supported by the finding that partial deafferentation of one hind leg by transection of the L4–L6 dorsal roots reduced the level of activity in the knee extensors by ~50%, but did not noticeably influence the activity in ankle extensor muscles. However, extending the deafferentation to include the L7–S2 dorsal roots decreased the ankle extensor activity. We conclude that afferent feedback contributes to more than one-half of the input to knee and ankle extensor motoneurons during the stance phase of walking in decerebrate cats. The continuous contribution of afferent feedback to the generation of extensor activity could function to automatically adjust the intensity of activity to meet external demands.

INTRODUCTION

The function of peripheral afferents, in particular those arising from muscle receptors, has been an important issue in the physiology of locomotion since the early work of Sherrington (1910). In recent years, a primary focus has been on the role of afferent feedback in controlling the timing of the transition from stance to swing. There is evidence that activation of large muscle afferents, arising from muscle spindles and tendon organs in extensor muscles, is capable of regulating the duration of extensor activity associated with stance (Conway et al. 1987; Guertin et al. 1995; Hiebert et al. 1995; Whelan et al. 1995). Also, proprioceptive input from the muscle spindle afferents generated by the stretch of flexor muscles as the limb is extended during stance has been shown to bring about an early transition from stance to swing (Hiebert et al. 1996).

These observations indicate that afferent feedback from muscle receptors signaling loading of the leg and extension of the leg are both involved in controlling the timing of the phase transitions.

Another possible role of afferent feedback is in the generation of the electromyographic (EMG) activity within a single phase of the locomotion cycle. The idea that monosynaptic excitatory feedback from muscle spindle afferents may contribute to muscle activity during stance arose when Severin et al. (1967) observed increased spindle activity during stance in treadmill walking decerebrate cats. Severin (1970) later provided evidence that afferent feedback produced by gamma activation of muscle spindles may contribute nearly 50% of the activity in ankle extensor muscles. A more recent study by Yang et al. (1993) also concluded that velocity-sensitive afferents reflexively contribute 30–60% of the activity in the soleus muscle during early stance in walking humans. Other studies in humans have shown that muscle spindles are involved in the generation of EMG activity during voluntary contractions. Blockade of fusimotor drive acts to decrease the number of motoneurons recruited (Hagbarth et al. 1986) and reduce their firing rates by one-third (Macefield et al. 1993) during maximal voluntary contractions.

Another group of afferents that may contribute to the generation of stance activity is the group Ib afferents arising from Golgi tendon organs in extensor muscles. Originally, extensor group Ib afferents were demonstrated to produce a disynaptic inhibition of homonymous and synergistic motoneurons (LaPorte and Lloyd 1952) and were therefore not considered to contribute to the excitation of motoneurons. However, over the past 15 yr, evidence has accumulated that the disynaptic group Ib inhibition is weakened during locomotion and replaced by excitation. Conway et al. (1987) showed that activation of extensor group I afferents during fictive locomotion in spinal cats enhances the amplitude of extensor activity, and if stimulated during flexor activity, resets the locomotor rhythm to extensor activity. This effect was attributed to the group Ib afferents in that a similar response could not be generated by selective activation of the Ia afferents by vibration of the muscle. The idea that the Ib inhibitory input observed in a passive, nonlocomoting state switches to excitation during an active, locomoting state has now been conclusively demonstrated (Gossard et al. 1994; Pearson and Collins 1993). In addition, in decerebrate animals feedback from group Ia muscle spindle afferents has recently been shown to influence extensor activity through locomotor-dependent disynaptic and polysynaptic pathways (Guertin et al. 1995; McCrea et al. 1995). This has given rise to the possibility that during locomotion the
group Ia and Ib afferents from extensor muscles converge onto common sets of interneurons that produce depolarization of synergistic motoneurons (see Fig. 8 in McCrea et al. 1995).

Although it is clear that muscle afferent feedback can influence the level of activity of extensor motoneurons during locomotor activity in cats, either via the group Ia monosynaptic reflex or the locomotion-dependent group I polysynaptic pathways (Gossard et al. 1994; McCrea et al. 1995), there are still a number of unresolved issues. One is the degree to which afferent feedback contributes to the generation of activity in different ankle and knee extensor muscles. The earlier study of Severin (1970) only estimated the contribution of group Ia afferents in the generation of activity in one ankle extensor muscle (lateral gastrocnemius). Another issue is the extent to which the magnitude of EMG activity in different muscle groups is regulated independently by afferent feedback during stance. In particular, we wish to know the extent to which feedback from a specific set of afferents influences the activity in one set of extensor muscles (e.g., knee extensors) without influencing activity in another set of extensor muscles (e.g., ankle extensors). Evidence suggesting some restriction of the influence of different muscle afferents on activity in different extensor motoneurons is that electrical stimulation of group I afferents from knee and ankle extensor muscles produces different effects on extensor motoneurons (Guertin et al. 1995). In this study we addressed these issues by using a variety of procedures to alter afferent feedback and measuring the associated changes in the magnitude of activity in knee and ankle extensor muscles during walking in decerebrate cats.

METHODS

Adult cats were anesthetized with halothane and the trachea cannulated for continued administration of the anesthetic. One of the carotid arteries was ligated, and a cannula to monitor blood pressure was placed in the other. A cannula was inserted in one jugular vein for the administration of fluids and drugs. Bipolar EMG electrodes were sutured into various flexor and extensor muscles of the hind legs: lateral gastrocnemius (LG), medial gastrocnemius (MG), soleus (SOL), vastus lateralis (VL), vastus medialis (VM), iliopsoas (IP), semitendinosus (ST), and/or tibialis anterior (TA). In some animals an E-shaped force-buckle (see Prochazka 1984) was placed on either the Achilles or the patellar tendon of the left hind leg, depending on the experiment.

Before decerebration, the animal was prepared for one of the four experimental paradigms described below. Decerebration was performed by transecting the brain stem at a 50° angle from the anterior edge of the superior colliculus, leaving the mammillary bodies intact. After the decerebration the anesthetic was discontinued, and within 1 h spontaneous locomotion activity usually occurred. If no spontaneous walking occurred, stimulation of the mesencephalic locomotion region (MLR) was used to induce locomotion (15 Hz, 0.6 ms duration, 30–100 mA; coordinates P2, L4, H6) (Shik et al. 1966). No consistent differences were observed in the responses of animals walking spontaneously and in animals walking in response to MLR stimulation.

All procedures were carried out with approval from the University of Alberta Health Sciences Animal Welfare Committee.

Extensor activity during the absence of ground support

The first procedure we used to assess the contribution of afferent feedback to the generation of extensor activity was to examine the magnitude of activity in extensor muscles in the absence of ground support. This was achieved by allowing a leg to step into a hole in a motorized treadmill belt (0.2–0.3 m/s) on which the animal was stepping. We have previously used this paradigm to assess the role of afferent feedback on the timing of locomotor activity in spinal and decerebrate cats (Hiebert et al. 1994, 1995). In ~85% of the trials, when the foot entered the hole, it was rapidly withdrawn. This rapid withdrawal was associated with termination of extensor activity and a rapid onset of flexor burst activity (Hiebert et al. 1995). In the remaining 15% of the trials, the leg was held extended in the hole for a longer period, usually being withdrawn following the completion of one step cycle of the contralateral leg (the animal’s weight was supported during these trials either by a sling under the abdomen or by holding the tail). Data from these trials were not included in Hiebert et al. (1995). In the present study, we chose to examine only those trials in which the leg was held extended in the hole. The relatively long delay before the onset of flexor activity allowed an easier assessment of the reduction in the level of activity in extensor muscles because activity could be averaged over a period of 500 ms (see Figs. 1 and 2). There are no indications that the effect on the level of extensor activity during these trials was substantially different from the immediate effect during the trials when the foot was withdrawn.
In eight animals the force generated by the ankle extensors was controlled when the animal encountered the hole in the treadmill belt (Fig. 3). A wire was attached to a clamp secured to the dorsum of the foot (the superficial and deep peroneal and the distal tibial nerves were cut to denervate the foot). The wire passed through a guide that was attached to the tibia by a bolt inserted through the bone. The other end was attached to a DC servo motor. A sheath inside of which the wire could freely move maintained the distance between the tibial attachment and the motor. Thus changing the position of the motor resulted in a change of ankle position. Tension in the wire was monitored and controlled when the animal encountered the hole in the treadmill belt. The motor was used to lift or drop the hindquarters after the onset of activity in the MG muscle. Custom-written computer programs were used to recognize the onset of MG EMG bursts.

**Loading or unloading of the hindquarters during walking**

In four animals the L₄–S₂ spinal processes were exposed and a length of strong surgical thread passed through the base of each spine. The thread was subsequently attached to a DC motor mounted above the animal. The motor was used to lift or drop the hindquarters after the onset of activity in the MG muscle. Custom-written computer programs were used to recognize the onset of MG EMG bursts.

**Feedback from ankle extensor muscles**

In eight animals the force generated by the ankle extensors was reduced when the animal entered the hole in the treadmill belt (Fig. 3A). A wire was attached to a clamp secured to the dorsum of the foot (the superficial and deep peroneal and the distal tibial nerves were cut to denervate the foot). The wire passed through a guide that was attached to the tibia by a bolt inserted through the bone. The other end was attached to a DC servo motor. A sheath inside of which the wire could freely move maintained the distance between the tibial attachment and the motor. Thus changing the position of the motor resulted in a change of ankle position. Tension in the wire was monitored and controlled when the animal encountered the hole in the treadmill belt. The motor was used to lift or drop the hindquarters after the onset of activity in the MG muscle. Custom-written computer programs were used to recognize the onset of MG EMG bursts.

**Data analysis**

Eight channels of EMG data were recorded on VHS tape using a Vetter 4000A PCM recording unit. A hard copy of the data was subsequently displayed on a Gould ES1000 electrostatic chart record.
corder. Selected sequences were digitized and stored on disk using the Axotape data acquisition system (Axon Instruments). Custom-written software capable of reading and displaying the Axotape data files was used to display, store, and analyze averaged EMG data. The average extensor EMG activity during a single step was calculated for a 500-ms period beginning 100 ms after the onset of activity in SOL or \( \sim 50\) ms after entering the hole (as indicated by the light fence). The initial 100 ms was not included because this period corresponds to the E1 phase of the locomotion step cycle where the limb is not in contact with the ground. The extensor EMG activity during the E1 phase is not dependent on the afferent signals generated by foot contact (Gorassini et al. 1994). The activity during undisturbed walking was taken to be 100% and the level of EMG activity during a perturbation to the stance phase calculated as a percentage of the control. A Student’s t-test (unpaired) was used to calculate significant changes from the control.

To record kinematic data, surface markers were placed over the iliac crest, greater trochanter, lateral epicondyle of the femur, lateral malleolus and metatarsophalangeal joint and the relative joint angles of the leg monitored with a video recorder (shutter speed 1 ms; 60 frames/s). Triangulation was not used to determine knee joint angles. Stepping sequences were later digitized and saved on computer disk using Video-Blaster software (Creative Labs). Custom-written software was then used to display the kinematics and plot the joint angles of the limb. The graphics program used to display the plots of joint angles (CorelDraw 5.0) partially smoothed the traces.

**RESULTS**

**Extensor EMG activity is reduced during the absence of ground support**

The first objective of this investigation was to estimate the total contribution of afferent feedback to the generation of extensor burst activity during walking. We did this by measuring the reduction of EMG activity in ankle and knee extensor muscles of one hind leg when the foot unexpectedly entered a hole in the treadmill belt on which the animal was walking. Figure 1 illustrates the EMG activity recorded from two different animals during trials when the foot was not rapidly withdrawn from the hole (we will refer to these trials as “foot-in-hole” trials). While the limb was in the hole, there was a large reduction of activity in both knee and ankle extensor muscles. The initial burst of activity in LG and MG is largely unaltered because this burst begins before ground contact and thus before the foot enters the hole. It has previously been shown that this initial activity is not dependent on afferent feedback generated by limb contact with the ground (Gorassini et al. 1994).

Figure 2A further illustrates the reduction in knee and ankle extensor EMG activity during the absence of ground support by superimposing the average EMG activity in the knee extensor VL and the ankle extensor MG during normal steps (thin traces) and during foot-in-hole trials (thick traces). A quantitative assessment of the reduced EMG activity was made by measuring the total level of EMG over a period of 500 ms beginning 50 ms after the foot entered the hole, and comparing it with the EMG activity in the two preceding normal steps (the averaging period is indicated in Fig. 2A by the interval between the vertical lines). The scatter plot in Fig. 2B summarizes the results of this analysis. The average reductions in the EMGs were 68 ± 13% (mean ± SD), 68 ± 8%, and 74 ± 9% for VL, MG, and LG, respectively.

In most trials the step cycle period changed during foot-in-hole trials. For example, Fig. 1A shows a trial in which the cycle period was prolonged (indicated by the delayed activity in ST). However, there was no correlation between the increased/decreased cycle period and the reduction in EMG amplitude as shown in the scatter plot of the normalized EMG amplitude versus cycle period (Fig. 2B). Note that in foot-in-hole trials where little or no change occurred in the cycle period (normalized value of 100%) there was a reduction in EMG amplitude similar to trials in which the cycle period changed.

**Loading extensor muscles restores EMG activity**

Although the above experiment demonstrates that afferent feedback generates a considerable portion of activity in extensor muscles during stance, it does not reveal which afferents (i.e., muscle spindle, tendon organs, or cutaneous receptors from the footpad) are involved. To assess whether feedback from proprioceptors in the ankle extensor muscles contributes to the generation of extensor activity, we selectively loaded these muscles as the limb entered the hole in the treadmill (Fig. 3A).

Five out of eight animals used in this set of experiments stepped sufficiently regularly to allow a comparison of the level of extensor activity during normal steps and during foot-in-hole trials when the ankle extensors were loaded. The data from all five animals were consistent. First, compared with steps with the cable unattached, there was no noticeable change in the EMG activity of flexors and extensors when the wire was attached and the motor was maintaining a low level of background force (25 g). This was taken to indicate that the motor was following the natural movement of the ankle and not interfering with the generation of the stepping motor pattern. Second, the reduction in extensor EMG activity during foot-in-hole trials in these animals was very similar to that reported in the previous section. Figure 3B illustrates the low level of activity in SOL and LG when the limb was in the hole. Because the cutaneous afferents from the foot were transected in this set of experiments, the large reduction in extensor activity during normal foot-in-hole trials (Figs. 1 and 2) is not primarily due to the loss of cutaneous feedback. Finally, increasing the force in the cable spanning the ankle joint during foot-in-hole trials increased the magnitude of activity in ankle extensor muscles (compare Fig. 3, B and C). The magnitude of the activity in ankle extensors could be increased to a level similar to that during weight-bearing steps (Fig. 3C). In addition to increasing the magnitude of bursts in the ankle extensors, loading the extensor muscles also increased the duration of the extensor activity, provided the duration of the force command exceeded the normal duration of the stance phase (Fig. 3C).

To obtain more quantitative information on the effects of loading the ankle extensor muscles during foot-in-hole trials, in three animals we measured the level of EMG activity in SOL, LG, MG, and VL when the reference force level was set to different values. Examples of averaged EMGs from the three ankle extensor muscles in one animal are shown in Fig. 4, and scatter plots of individual trials for the four extensor muscles are shown in Fig. 5. Although loading increased the magnitude of activity in all four muscles, the characteristics of the responses differed somewhat. These differences are seen most clearly in the scatter plots in which the magnitude of the EMGs...
in the LG, MG, SOL, and VL muscles is plotted against the force in the Achilles tendon for individual trials at four different reference forces (Fig. 5). The regression lines for LG, MG, and SOL are for data from trials when the ankle extensors were loaded, i.e., the data for trials when the foot entered the hole and the muscles not loaded (crosses) were excluded from the regression analysis. Three differences between muscles can be seen in Fig. 5. First, the level of activity in LG and MG increased progressively as the Achilles force increased. Second, the level of activity in SOL increased with relatively small
AFFERENT CONTRIBUTION TO EMG ACTIVITY

The observation that activity in VL decreased as the force required for weight support decreased suggests that afferent feedback is acting to continuously regulate the level of activity to match loading conditions. If this is true, then transient changes in the load carried by the animal should be mirrored by changes in EMG activity. Figure 7A illustrates the change in EMG activity after a 3-cm lift, which unloads the hindquarters, triggered 50 ms after the onset of activity in SOL. Similar to the observations described above, a distinct feature was that EMG activity in the knee extensor VL was reduced, whereas there was a negligible effect in the amplitude of activity in ankle extensor muscles when the animal was lifted. In one animal in which the activity in all four quadriceps muscles was recorded, there was a reduction of activity in all muscles (data not shown). This reduction was greatest in VL and VM, whereas the deeper, more postural vastus intermedials muscle was the least affected by the lifting.

A rapid 3-cm drop of the hindquarters, midway through stance phase, produced a distinct increase in VL activity 20–30 ms after the onset of the drop, which then lasted for the remainder of the stance phase (Fig. 7B). A transient increase in SOL activity was also observed 20–30 ms after the onset of the drop (arrow in Fig. 7C). However, after this short burst, SOL EMG returned to the amplitude observed during undisturbed stepping.

Combining the lift and drop of the hindquarters produced a combination of the two responses described above (Fig. 7C). The initial lifting of the hindquarters produced a reduction in VL activity that was similar to that illustrated in Fig. 7A. The drop, applied approximately halfway through the same stance phase, produced an increase in EMG activity similar to that in Fig. 7B. The rapid, short-duration increase in SOL activity is distinct (arrow in Fig. 7C), as well as the rapid prolonged increase in VL activity. Note that as the patellar force returned to normal levels, the VL EMG also returned to control levels. An important feature to note in Fig. 7, B and C, is that the increase in EMG, which occurred during the drops, preceded any detectable change in muscle force. This phenomenon indicates that an afferent signal other than one related to muscle force is generating the initial change in muscle activity.

Removal of feedback by deafferentation decreases extensor activity

The experiments where the animal’s hindquarters were lifted or lowered indicated that changes in afferent feedback could selectively affect the level of activity in the quadriceps without affecting ankle extensor activity. To extend these results showing an independent regulation of muscles acting at the knee, we examined the effects of partially deafferenting the quadriceps muscle. The quadriceps afferents enter the spinal cord primarily through the L5–L6 dorsal roots, whereas the ankle extensor afferents enter mainly through the L7 and S1 roots. If the independent regulation of muscle activity were true, then selective deafferentation by cutting the more rostral roots (L4–L6) should preferentially reduce activity in the quadriceps.

All of the animals used in this series of experiments (n = 4) produced periods of reliable decerebrate locomotion with weight support when dorsal roots were intact. Three animals...
walked spontaneously after decerebration, while one animal required constant stimulation of the MLR to produce a locomotor pattern. In all four animals, transection of the L₄–L₆ dorsal roots reduced the magnitude of EMG activity in VL without having a large influence on the magnitude of activity in the ankle extensors (Fig. 8). The average reduction in EMG amplitude in VL following partial deafferentation for each experiment was 53 ± 31%, 53 ± 28%, 21 ± 39%, and 51 ± 11% compared with the amplitude before dorsal root transection. All but the third value represents a statistically significant decrease in VL EMG. This animal required constant stimulation of the MLR to produce locomotion.

In general, partial deafferentation produced a slight trend toward an increase in the amplitude of ankle extensor activity. LG activity increased slightly in all animals (Fig. 8C). In two animals, SOL activity also increased slightly, but decreased in the other two. Thus the relatively selective removal of afferent feedback to the quadriceps reduced activity in at least one of these muscles (VL), whereas extensor muscles acting at the ankle joint were not adversely affected by the loss of input.

Given the reduction of quadriceps activity to ~50% of normal, deficits in the kinematics of walking were not unexpected. Following partial deafferentation, one of the animals was not capable of supporting its body weight, in that the deafferented limb would collapse at the knee joint if no vertical support was provided. The other three animals were still capable of walking with independent weight support following partial deafferentation, but there was a distinct increase in the yield of the knee joint observed at the beginning of the stance phase. It was qualitatively noted that the yielding of the knee joint was smallest in the animal that required MLR stimulation to produce walking (this animal also showed the least reduction in VL activity).

The kinematics of leg movements of one animal capable of weight support after partial deafferentation are illustrated in Fig. 9A. The lower position of the hindquarters during stance indicates that the deafferented limb was in a more flexed position than normal. The joint angle plots (Fig. 9B) better illustrate this phenomenon. In the intact state, there was a slight yield of the knee after the onset of stance phase as the weight of the animal was transferred onto the limb. In the partial deafferented state the yield at the knee was dramatically increased. The knee became more flexed during the initial period of stance and extended only after the limb was moved out from underneath the animal. Movements around the hip and ankle joint were much less affected than movements around the knee joint. In the example shown in Fig. 9B, the hip angle was shifted toward flexion, while yielding at the ankle joint during early stance was slightly increased.

A contribution of afferent feedback to the generation of activity in the ankle extensors was assessed by transecting the ipsilateral L₇, S₁, and S₂ dorsal roots in three of the animals in which the L₄–L₆ dorsal roots had been cut. In all three animals the locomotor rhythm was severely impaired for ~5–10 min immediately following the additional transection of the L₇–S₂ dorsal roots. The deafferented limb was typically held rigid in extension, while the other three limbs continued to step on the treadmill. After the initial period of tonic extensor activity, a more regular locomotor rhythm eventually resumed in two animals (one of which was the animal requiring MLR stimu-
lation to evoke locomotion). The third animal was at best only capable of making four or five poorly coordinated steps at a time, interrupted by periods of maintained extensor tonus. This prevented an analysis of burst magnitudes in this animal.

In the two animals that eventually used their deafferented leg, regular stepping sequences lasting 1 min sometimes occurred without the need for any external support of the hindquarters. During these sequences the magnitude of activity in flexor and extensor muscles remained reasonably constant throughout. This is illustrated in Fig. 10, B and D. Each panel shows a 10-s segment of raw EMGs recorded during a long sequence of stepping in the deafferented hind leg in the two animals. During these sequences, the magnitude of activity in ankle and knee extensors was obviously reduced compared with the magnitudes of activity when the dorsal roots were intact, as can be seen by comparing panels A and C with panels B and D in Fig. 10. This reduction in magnitude was associated with abnormal yielding at the knee and ankle during stance (not shown). A quantitative analysis of bursts in the ankle extensors LG and SOL before and after deafferentation revealed that deafferentation reduced burst magnitudes by >50% (Fig. 11). LG burst magnitude was reduced by 78 ± 9% in one animal and by 67 ± 11% in the other. Corresponding values for SOL were 65 ± 14% and 56 ± 8%, respectively. The reductions in the magnitude of the ipsilateral VL activity in these two animals were 69 ± 13% and 64 ± 24%, respectively. Deafferentation did not noticeably influence the magnitude of activity in hip (IP) and knee (Sart) flexor muscles, but it did increase the rate of stepping slightly (Fig. 11, A and C).

**DISCUSSION**

In this investigation we have used a variety of procedures to demonstrate that feedback from peripheral receptors contributes significantly to the activation of extensor motoneurons during the stance phase of walking in decerebrate cats. We have estimated that, when the animals are walking on a horizontal surface, between 50 and 80% of the activity in knee and ankle extensor muscles is produced by afferent signals. Our data confirm and extend the results of the only other study aimed at quantifying the afferent contribution to extensor burst generation in walking cats (Severin 1970). By reducing afferent feedback from muscle spindles by blocking the activity of γ-motoneurons, Severin observed a large reduction in the intensity of activity in ankle extensor muscles. He concluded that afferent feedback contributed ~50% to burst generation in ankle extensor motoneurons. More recently, the contribution of feedback from primary muscle spindles to soleus muscle activation has been estimated to be between 30 and 60% during the early part of stance in walking humans (Yang et al. 1991). During running, the afferent contribution to activation of ankle extensor muscles may be even higher (Dietz et al. 1979).

**Afferent contribution to extensor burst generation**

Our estimate of the afferent contribution to extensor burst generation was based initially on the large reduction in EMG
magnitude in the knee and ankle extensor muscles when the hind leg unexpectedly stepped into a hole (Figs. 1 and 2). In earlier studies on spinal (Hiebert et al. 1994) and decerebrate (Hiebert et al. 1995) cats, a reduction in the amplitude of extensor activity was also noted before the onset of flexor activity when the foot was withdrawn quickly from the hole. However, the short duration of the reduced extensor activity preceding the onset flexor activity (see Fig. 6D in Hiebert et al. 1994) made it difficult to quantify the reduction of extensor activity at the time the foot entered the hole. For this reason we chose in the present study to examine only those trials in which the foot was not withdrawn quickly from the hole. In addition, we examined the effects of increasing proprioceptive feedback on the amplitude of extensor EMG activity while the limb was in the hole.

The first issue we wish to consider is whether the reduction in extensor EMG magnitude during foot-in-hole trials is a reasonable measure of the afferent signal contributing to extensor activity during the stance phase. It is conceivable that the reduction in EMG activity during foot-in-hole trials is due to a rapid reprogramming of the central pattern generating (CPG) network rather than being due to the withdrawal of afferent signals that sum with central signals to activate extensor motoneurons. Given that in 85% of the foot-in-hole trials there is normally rapid initiation of flexion (Hiebert et al. 1994), the 15% of trials where the limb is held in the hole may have been due to changes in the functioning of the CPG.

However, there are a number of reasons for believing that the reduced EMG activity is not due to a reprogramming of the CPG. First, the reduction in extensor burst amplitude was independent of the cycle period during the foot-in-hole trials (Fig. 2B) and occurred where there was no change in cycle period. Thus the effect of the loss of afferent feedback on the magnitude of extensor activity is independent of the effects on the timing of burst generation. Previous experiments have also concluded that extensor burst magnitude may be regulated to some degree by afferent pathways not influencing the CPG (Guertin et al. 1995; McCrea et al. 1995; see review of Pearson 1995).

Second, similar decreases in EMG amplitude have been observed under situations where the timing of the locomotor rhythm was experimentally controlled. We have previously shown that the timing of extensor activity can be controlled by artificially introducing afferent signals, by electrically stimulating group I muscle afferents, when the foot is in the hole (Hiebert et al. 1995). In that situation, afferent feedback from a single extensor muscle strongly influenced the cycle period (increasing extensor burst duration) without affecting the re-
duced EMG activity (see Fig. 3 in Hiebert et al. 1994). Third, in the present study we found that by loading the extensor muscles with a force pulse matching the normal extensor duration, we increased extensor burst amplitude without altering the stepping cadence (Fig. 4). Hence we believe that the CPG was operating in a similar manner during loaded and unloaded trials. As the force applied to the muscles increased, the level of activity in the ankle extensors also increased (Figs. 4 and 5). The fact that the magnitude was increased with small increases in Achilles force indicates that the afferent feedback has ready access to the extensor motoneurons.

Another reason for believing that rapid reprogramming is not responsible for the reduced extensor activity during foot-in-hole trials is that the reductions in magnitude were similar to those produced by other procedures that did not alter the timing characteristics of the motor program. For example, when the hindquarters were partially supported, the level of knee extensor activity was reduced by >50% without changing the cycle period or extensor burst duration (Fig. 6). Moreover, the fact that activity in the ankle extensors was not reduced significantly by this procedure indicates that there was not a global reduction in central drive to all leg extensor motoneurons. Similar effects were observed following partial deafferentation (Fig. 8). Complete deafferentation also reduced extensor activity by amounts similar to those observed during foot-in-hole trials (ranging from 55 to 78%) without noticeably influencing the level of activity in flexor muscles. IP, iliopsoas; Sart, sartorius.

A factor that could be responsible for reducing activity in extensors during foot-in-hole trials is reciprocal inhibition from primary muscle spindles in flexor muscles. When the foot entered the hole, the leg was often fully extended before being withdrawn from the hole. This resulted in stretching of flexors acting at the hip, knee, and ankle, presumably activating the group Ia afferents from these muscles (Prochazka et al. 1989). Although we cannot exclude the possibility that inhibitory feedback from flexor group Ia reduces extensor activity during foot-in-hole trials, a number of observations indicate that it may not be a major factor. The clearest is the large reduction in the level of activity in the knee extensors when the hindquarters of the animal were lifted. This reduction was associated with only minor increases in the magnitude of knee extension, hence stretch of flexors (Fig. 6), yet the magnitude of the reductions in knee extensor activity were often comparable with those produced during foot-in-hole trials. Another observation indicating that stretch of flexors was not the primary factor reducing extensor EMGs during foot-in-hole trials was the large reduction in the magnitude of knee extensor EMGs following transection of the L₄-L₆ dorsal roots (Fig. 8). In this situation, there was an exaggerated flexion at the knee (Fig. 9), hence shortening of flexors. In addition, it is probable that partial deafferentation also eliminated some feedback from knee flexor muscles. Thus it seems very unlikely that the reduction in knee extensor activity following partial deafferentation is due to inhibitory feedback from flexor Ia afferents. The most likely explanation is the loss of afferent signals from knee extensors that normally contribute to the activation of the knee extensor motoneurons.
Afferents regulating extensor burst activity

During foot-in-hole trials there is a loss of sensory signals from a large number of sensory receptors in the leg. Pressure-sensitive cutaneous receptors in the foot are not activated, and there would be reduced feedback from the force-sensitive Golgi tendon organs in extensor muscles. In addition, primary muscle spindle activity typically associated with early stance (Prochazka et al. 1989) would be reduced because there is no lengthening of extensor muscles. The present study suggests that it is the loss of feedback from muscle receptors that is primarily responsible for the reduction in extensor activity. This conclusion comes mainly from our observation that loading the ankle extensors when the foot entered the hole could increase activity in the ankle extensors to levels similar to those during normal steps (Fig. 4). Loading the ankle extensors would be expected to increase activity in Golgi tendon organs and primary muscle spindles, the latter as the result of a decrease in the rate of shortening of the muscles. Stimulation of afferents from both these groups of afferents has been shown previously to enhance extensor activity during fictive locomotion (Guertin et al. 1995). We consider it unlikely that changes in feedback from secondary spindle endings makes any significant contribution to the enhancement of extensor activity. Previous studies have failed to detect any excitatory effects on extensor motoneurons when extensor group II afferents are electrically stimulated during locomotion (Perreault et al. 1996; Whelan et al. 1995), whereas in passive preparations extensor group II afferents inhibit extensors (Jankowska 1992).

The relative importance of the contributions from Golgi tendon organs and primary muscle spindles is difficult to assess. Severin’s finding of an ~50% reduction in ankle extensor activity following conduction block in γ-motoneurons suggests that feedback from primary muscle spindles makes a major contribution. Primary muscle spindles have also been implicated in the generation of extensor activity during walking and running in humans (Dietz et al. 1979; Yang et al. 1991). Some of our data also support the idea that feedback from primary muscle spindles makes a significant contribution to the generation of extensor burst activity. For example, when the ankle extensor muscles were loaded, there was an initial short-latency increase in EMG activity in the ankle extensors that preceded the relatively slow increase in force (Fig. 4). Similarly, when the hindquarters were dropped, a large increase in EMG activity in quadriceps muscles preceded any substantial force increase in the muscles (Fig. 7, B and C). Our evidence for a contribution from Golgi tendon organs is less compelling. One observation suggesting that feedback from these receptors makes a contribution to the generation of activity in knee extensors was that slight lifting of the hindquarters produced large reductions in the force and the activity in these muscles. These changes were associated with only small changes in the joint kinematics (Fig. 6) and therefore in muscle length. Even so, it is conceivable that small changes in primary spindle feedback produce large changes in EMG activity and a corresponding large change in muscle force.
Distribution of afferent feedback

If the activity in knee and ankle extensor muscles is regulated to a large degree by afferent feedback, then an interesting issue is the extent to which this regulation occurs independently. A number of observations indicate that afferent feedback from the knee extensors is primarily restricted to knee extensors and does not influence activity in ankle extensors. Rapidly lifting or dropping the hindquarters resulted in decreased or increased quadriceps activity, respectively, without producing a noticeable change in the activity of the ankle extensors (Fig. 7). Furthermore, transecting the L₅-L₆ dorsal roots decreased quadriceps activity by ~50% without producing a marked change in the magnitude of activity in ankle extensors (Fig. 8). Guertin et al. (1995) also noted a restriction of the influence of group I afferents from knee extensors during fictive locomotion. However, during fictive locomotion in spinal animals, stimulation of group I afferents from quadriceps can strongly excite motoneurons innervating ankle extensor muscles (Conway et al. 1987; Gossard et al. 1994).

Previous studies have indicated that the distribution of feedback from ankle extensor muscles may be quite widespread. Electrical stimulation of group I afferents from ankle extensors strongly enhances activity in knee extensors during fictive locomotion in decerebrate animals (Guertin et al. 1995) and often increases knee extensor activity in walking decerebrate cats (Whelan et al. 1995). On the other hand, there are data indicating a more restricted influence of feedback from ankle extensors. First, selectively loading the ankle extensor muscles during foot-in-hole trials (Fig. 3) only increased knee extensor activity at high forces (Fig. 5). Second, electrical stimulation of group I afferents in the lateral gastrocnemius-soleus (LGS) nerve in conscious walking animals has no effect on the level of activity in knee extensors (Whelan and Pearson 1997). And finally, modifying feedback from the medial gastrocnemius muscle by cutting the LGS nerve does not influence the level of activity in knee extensors in normal walking animals (unpublished observations).

It is now apparent that the distribution of afferent signals from knee and ankle extensor muscles differs in different preparations. Afferent feedback from both knee and ankle extensors appears to be widespread during fictive locomotion in spinal animals, less widespread during fictive locomotion in decerebrate animals and during walking in decerebrate animals, and probably restricted to homonymous and synergist muscles in conscious walking animals. The reason for these differences in different preparations is a matter of speculation. An obvious possibility is that supraspinal structures regulate the contributions different afferent pathways make to the generation of extensor burst activity. Three excitatory pathways have now been identified from extensor group I afferents to extensor motoneurons (reviewed by Pearson 1995).

Functional considerations

The main conclusion of this investigation is that activity in extensor motoneurons during the stance phase of walking in decerebrate cats is continuously regulated by afferent feedback from proprioceptors in extensor muscles. An important functional issue is whether this also occurs during walking in normal animals. There are some indications that afferent con-


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REFERENCES


