

# Corrective Responses to Loss of Ground Support During Walking II. Comparison of Intact and Chronic Spinal Cats

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## SUMMARY

1. The preceding study described a corrective response in cats when one hind leg steps into a hole. In this investigation we examine the extent to which this behavior is organized at the spinal level by comparing the responses elicited in intact and chronic spinal cats.

2. Adult cats were trained to step bipedally with their hind legs on a treadmill. After training, the responses to stepping into a hole cut in the treadmill belt were monitored with a video recorder and by recording electromyograms from muscles in both hind legs. The responses to stepping into the hole were also recorded in chronic spinal cats that had recovered the ability to step with their hind legs a few weeks after spinalization.

3. The behavioral responses in the two groups of animals differed in two respects. First, the latency of the onset of the flexion movement to remove the foot from the hole was shorter in intact animals (70–150 ms in intact vs. 130–350 ms in spinal animals). Second, the flexion movement in the intact animals was stronger. The exaggerated flexion movement in intact animals lifted the paw well clear of the hole and allowed support to be regained on the treadmill belt. The weaker flexion movement in spinal animals was usually insufficient to lift the paw completely from the hole.

4. Differences in the motor patterns recorded from flexor muscles during the corrective response in intact and spinal animals correspond with the differences in the kinematics. First, the onset of flexor activity after the foot entered the hole was delayed by ~100 ms in spinal animals relative to intact animals. Second, in intact animals the magnitudes of flexor bursts were increased relative to the flexor bursts associated with the swing phase during stepping, whereas in spinal animals flexor bursts during the corrective response resembled those occurring during swing.

5. Similarities in the duration and the timing of bursts in different flexor muscles in intact and spinal animals during the corrective response and during swing indicated that the corrective response involves activation of the spinal system that normally produces swing phase flexor activity. We conclude that activation of this system is facilitated by input from supraspinal structures during the corrective response in intact animals.

6. In all intact animals and three of five spinal animals, support of the hindquarters when the foot entered the hole was maintained by the contralateral leg. The stance phase of this leg was prolonged and extension significantly increased. The magnitude of extensor activity in the contralateral leg increased ~60 ms after the ipsilateral leg entered the hole, presumably because of the additional loading. These similarities indicate that the mechanism by which the contralateral leg maintains support during the corrective response is organized primarily at the spinal level.

## INTRODUCTION

One approach for determining the function of afferent feedback from peripheral receptors during walking is to perturb the step cycle and monitor electromyographic (EMG) responses in leg muscles. In humans, for example, sudden accelerations or decelerations of a treadmill evoke characteristic compensatory responses in leg muscles that appear to be triggered by input from group II muscle afferents (Berger et al. 1984; Dietz et al. 1985, 1987). Corrective reactions to tactile stimulation of the paws of cats have also been extensively studied (Drew and Rossignol 1987; Forssberg 1979; Wand et al. 1980). Two general issues have arisen from these types of studies. The first is whether the responses are simple reflexes or whether they are the result of triggering more complex motor programs. The second is whether the corrective reactions are organized at a spinal or supraspinal level. Current evidence indicates that the stumbling-corrective reaction in the cat is mediated primarily via spinal reflex pathways that are strongly modulated in a phase-dependent manner by the locomotor rhythm generator. Moreover, differences in the effectiveness of these reflex pathways in intact, mesencephalic, and chronic spinal cats have shown that the spinally organized reactions are modulated by supraspinal structures (Forssberg 1979).

To further investigate corrective reactions during walking in intact cats we have examined the behavior of the hind legs when one foot unexpectedly enters a hole in the supporting surface (Gorassini et al. 1994). In this situation the foot is quickly removed from the hole by strong flexion at the knee and ankle while support of the hindquarters is maintained by a prolongation of stance in the contralateral limb. The mechanisms regulating these reactions have not been identified. However, EMG recordings from hind leg muscles have shown that the latency to the onset of the flexor activity responsible for withdrawing the foot from the hole is in the range of 35–65 ms. Moreover, the absence of afferent signals normally generated by ground contact does not alter the profile of ankle extensor activity for 35–40 ms after the foot enters the hole (Gorassini et al. 1994). These relatively long latencies to the onset of changes in the motor pattern raise the possibility that the flexor burst activity required to remove the foot from the hole is initiated via supraspinal pathways. Thus the main objective of the present investigation was to assess the contribution of supraspinal structures by comparing the responses of intact and chronic spinal cats to the entry of one of their hind legs into

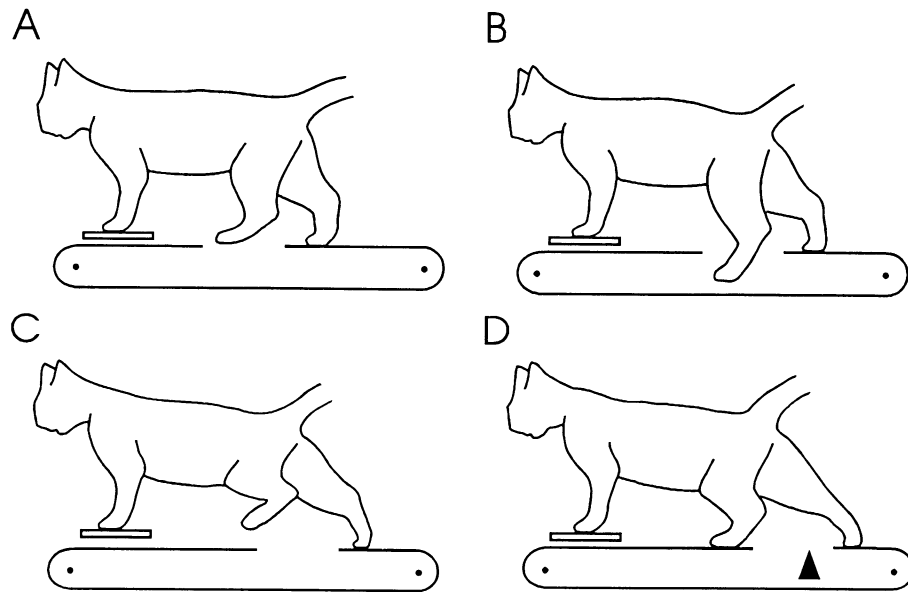


FIG. 1. Drawings showing the corrective reaction examined in this investigation. Intact and chronic spinal cats stepped bipedally with their hind legs on a treadmill while standing with their forelegs on a stationary platform. The belt of the treadmill contained a hole on the left side. This sequence of drawings shows the positions of the hind legs in an intact animal when (A) the left paw entered the hole, (B) the left leg began to flex and withdraw from the hole, (C) the left leg was maximally flexed, and (D) the left paw regained support on the treadmill belt (note the large extension of the right leg.  $\blacktriangle$ : position of right paw at the stance to swing transition when the left leg did not enter the hole).

a hole cut in the treadmill belt on which they were stepping. Our data show that the flexion responses of the leg entering the hole are more rapid in onset and larger in amplitude in the intact animal. We conclude that supraspinal pathways enhance the flexion response by modulating the spinal system that normally produces swing phase flexor activity.

#### METHODS

The primary objective of this study is to compare the reactions in the hind legs of intact and chronic spinal cats when one hind foot failed to contact the supporting surface at the end of the swing phase of walking. Because stepping of the hind legs in chronic spinal cats is bipedal and can only be elicited reliably on a treadmill, we considered it important to examine intact animals under similar conditions.

##### *Bipedal stepping in intact cats*

Four cats were trained with food rewards to step with their hind legs on a moving treadmill belt. Four hours before each training session, food was removed from the home cage. During training, food was presented at mouth level while the hind legs were stepping on the moving treadmill. The forelegs were supported on a platform 2 cm above the treadmill belt. Animals were trained once per day, with each training session lasting 10 min. Within a few days the animals learned to continuously step bipedally for many minutes and would adapt their rate of stepping to wide variations in treadmill speed (0.1–1 m/s). Two other cats with EMG electrodes implanted for an unrelated study were observed during bipedal stepping on the treadmill. Because these animals were not trained their performance was less reliable. Nevertheless the corrective responses were similar to those in the trained animals.

##### *Stepping in chronic spinal cats*

Three of the animals that had been trained to step bipedally and three other animals were spinalized at the T<sub>12-13</sub> level under aseptic conditions using procedures described elsewhere (Barbeau and Rossignol 1987; Pearson et al. 1992). During the week after spinalization each animal received antibiotic daily (Ayeracillin, 1 cc) and, if required, an analgesic (buprenorphine, 0.005–0.01 mg/kg). A dietary supplement (Aminolean, 10 cc in saline) was administered subcutaneously for the first few days after spinaliza-

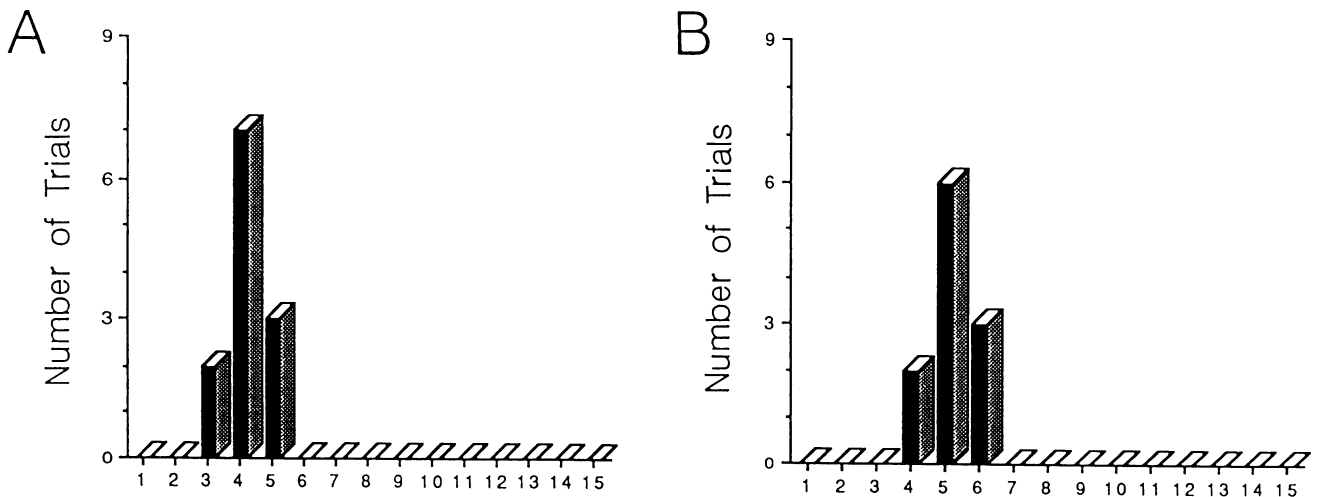
tion. The bladder was emptied using a catheter for the 1st wk after spinalization and subsequently expressed manually twice a day. Each animal was housed individually on a heated fleece.

Training commenced a few days after spinalization. The animal was positioned so that its hindlimbs were situated on a treadmill while the forelegs rested on a stationary plate. Movements of the hind legs were evoked by pinching the skin of the perineal region, and the posture of the animal was adjusted so that the legs thrust against the moving treadmill belt during leg extension. Balance and weight support was maintained by holding the tail. In five of the six animals the strength and coordination of the stepping movements progressively improved over a period of 2–3 wk to the point where stepping in the hind legs could occur in the absence of perineal skin stimulation and be sufficiently powerful to support completely the weight of the hindquarters. Training sessions lasted ~20 min and were performed twice daily. Food rewards were given to three of the animals (those previously used in the intact group) to minimize distractions.

##### *Responses to loss of ground support*

When either an intact cat had been trained to step bipedally or a chronic cat had been trained to step with weight support of the hindquarters during perineal stimulation, the treadmill belt was changed to one containing a hole (10 × 30 cm) on the left side. This resulted in the left hind leg occasionally stepping into the hole. The entry of the paw into the hole was monitored by an array of infrared light-emitting diodes directed across the hole onto an array of phototransistors (spacing 1.0 cm) located 1.5 cm below the top surface of the treadmill belt. This device was similar to that described by Gorassini et al. (1994) and it will be referred to as the "light fence." Disruption of any part of the light fence resulted in a 5-V pulse for the duration of light obstruction. Because of the 1.5-cm drop the light fence signal was delayed from the time the paw was level with the top of the treadmill belt. From measurements of the foot velocity at the end of the swing phase from video recordings we estimate that the interval from the time the foot entered the hole to activation of the light fence was 30 ms. Up to 20 successful trials were recorded in each testing session. A trial was judged to be successful if the paw entered the hole without contacting the rim. Trials where the limb did make contact were discarded because of the possible activation of cutaneous afferents. All animals were fed during the testing sessions in the same manner as during training. An important parameter during testing was the treadmill speed. This was usually kept low (0.2–0.3 m/s)

## INTACT



## SPINAL

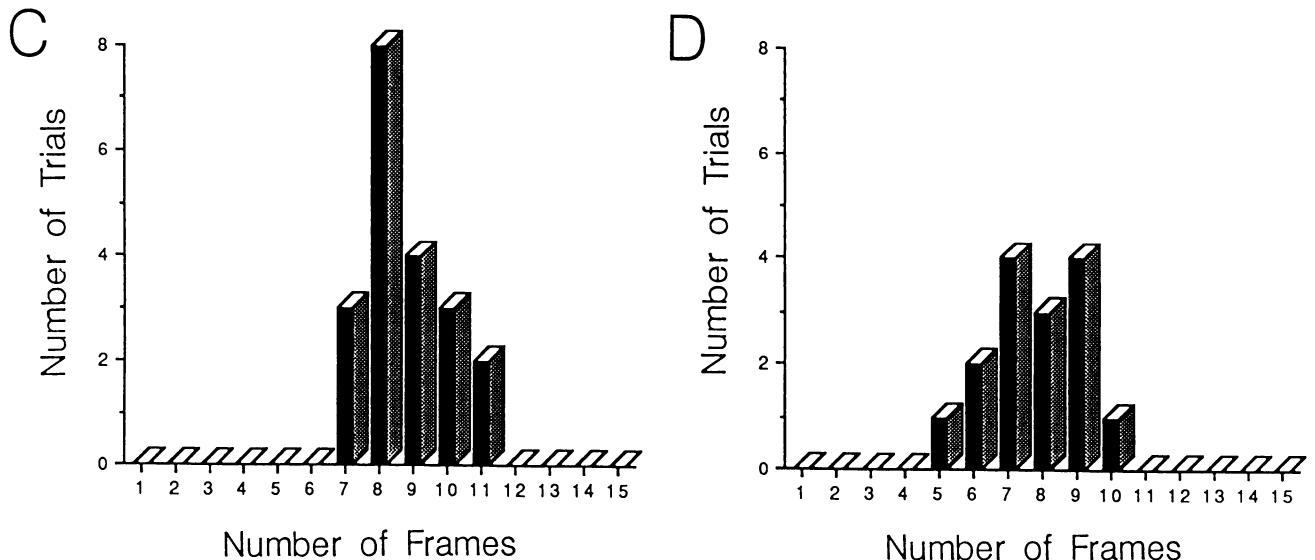


FIG. 2. Histograms showing the latency of the onset of leg flexion after the paw of the left leg entered the hole in the treadmill. *A* and *B*: intact animals. *C* and *D*: spinal animals. The latencies were measured from the video recordings as the number of frames from the frame immediately before the paw entered the hole to the frame showing the 1st observable flexion movement at either the hip, knee, or ankle. The camera speed was 30 frames per second. Each histogram represents data from a single test session. On average the minimum latency of the flexion response in the spinal animal was 130 ms longer than that observed in the intact animal.

so that the contralateral (right) leg could support the hindquarters during the period that the left leg was in the hole. If the left foot was not withdrawn quickly to regain support on the treadmill and/or the right leg initiated swing before the left leg support had been achieved, the animal was prevented from collapsing by supporting the abdomen by hand (intact animals) or lifting the hindquarters by the tail (spinal animals). During testing in spinal animals the tail was held to maintain balance of the hindquarters but not to support the animal's weight.

#### EMG recordings

EMG electrodes were implanted in hind leg muscles of the six intact cats used in this study as described in the preceding paper

(Gorassini et al. 1994). Connections to the recording amplifiers were made via a long flexible cable attached to the headpiece. Three of these intact animals were subsequently spinalized and the same electrode array was used to monitor EMG activity during the recovery of stepping and during experimental trials when the left foot entered the hole in the treadmill. In the other chronic spinal animals EMG recordings were made percutaneously from a limited number of muscles (usually 4) on the day of the recording session.

#### Data recording and analysis

Leg movements were monitored using a video recorder (30 frames per second). Surface markers on the iliac crest, greater

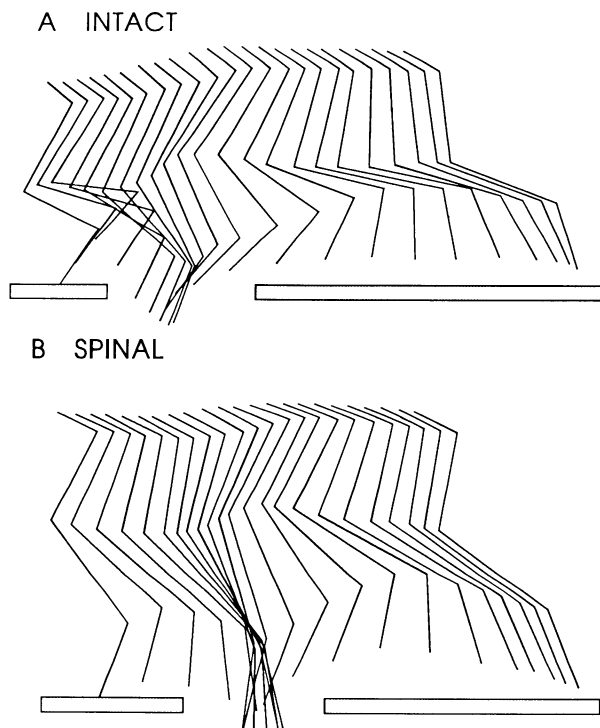


FIG. 3. Stick figures showing the movement of the left leg during normal swing phase, extension into the hole, and the corrective response lifting the foot from the hole. *A*: intact animal. *B*: spinal animal. The stick figure on the far *right* of each set of figures shows the leg position at the onset of swing. For clarity, sequential traces in the figures have been shifted to the *left* by the distance moved by the treadmill belt.

trochanter, and lateral epicondyle of the femur, lateral malleolus, and metatarsophalangeal joint were used to derive relative joint angles. In some sessions a split screen in the video record allowed the display of selected EMG recordings, thus allowing the synchronization of leg movements with the associated EMG records. EMGs and the output from the light fence were recorded on tape with a Vetter PCM recorder (Model 4000A).

All data analysis was done off-line. EMG records were rectified and low-pass filtered and, together with the signals from the light fence, were stored on computer disc using the Axotape data acquisition system (Axon Instruments) installed in an Express 80 486 microcomputer. The sampling rate for each channel was 700 Hz. Custom-written programs were used to average EMG records and to establish the timing of events relative to the triggering of the light fence signal.

A frame-by-frame analysis of the video recordings was used to determine the time of onset (to within 33 ms) of the first visible flexion movement at the hip, knee, or ankle after the entrance of the limb into the hole. Joint angles and limb position in individual video frames were analyzed using Video-Blaster software (Creative Labs.) and custom-written programs.

## RESULTS

The responses to stepping into the hole in the treadmill belt were recorded in six intact and five chronic spinal cats. Three of the intact animals were subsequently spinalized and thus included in the spinal group.

All the intact animals quickly lifted their feet from the hole and, provided the treadmill speed was low, maintained support of the hindquarters by prolonging the stance phase of the contralateral leg (Fig. 1). These responses to the ab-

sence of ground support closely resembled those made by intact animals when walking slowly on a walkway containing a trap door (Gorassini et al. 1994). The withdrawal of the paw from the hole was rapid in onset. The latency from the time the foot entered the hole to the beginning of leg flexion measured from the video recordings was in the range of 70–150 ms, i.e., flexion at hip, ankle, or knee was detected in the third to sixth frame after the foot entered the hole. Histograms showing the distribution of the latencies for two intact animals are shown in Fig. 2, *A* and *B* (data from the other 4 animals were similar). The data in each of these histograms were obtained during a single test period.

Chronic spinal cats also flexed their legs soon after the foot entered the hole in the treadmill, but compared with intact animals the latencies to the onset of this response were longer and more variable (range 165–350 ms). Histograms of latencies observed in two spinal animals are shown in Fig. 2, *C* and *D*. Although the latencies in some trials in spinal animals were within the range of the latencies in intact animals, in none of the spinal animals was the latency ever as brief as the shortest latencies observed in intact animals. The mean latency in spinal animals was about four frames ( $\approx 130$  ms) longer than the mean latency in intact animals.

Another clear difference in the responses of intact and spinal animals was observed in the kinematics of the flexion movements initiated after the foot entered the hole. In intact animals the maximum flexion of the limb out of the hole was exaggerated compared with normal swing phase movements (Figs. 3*A* and 4). The paw was lifted high above the treadmill surface, whereas during swing the limb is flexed just sufficiently to clear the surface. The result of this deliberate flexion movement was that the foot almost always regained support on the treadmill. By contrast, the flexion movements in chronic spinal animals were weak and usually not sufficient to lift the paw above the level of the treadmill belt. The leg would then re-extend into the hole, to be succeeded by a second flexion movement. Even on those occasions when the first flexion led to the regaining of support on the treadmill belt it was clear that the response was qualitatively different from that occurring in intact animals. Figure 3*B* shows an example in which foot support was regained after the first flexion movement. Note the weak elevation of the paw above the treadmill belt compared with the elevation achieved in an intact animal (Fig. 3*A*).

The quantitative features of the joint movements during swing and during the corrective response were determined from plots of the joint angles versus time in three intact and two spinal animals (see examples in Fig. 4). In the intact animals the average value of the peak flexion at the knee increased by  $6^\circ$  ( $n = 10$ ),  $9^\circ$  ( $n = 6$ ), and  $16^\circ$  ( $n = 5$ ) relative to peak flexion during swing, whereas peak flexion at the ankle correspondingly increased by  $19^\circ$ ,  $16^\circ$ , and  $23^\circ$ . In the spinal animals, by contrast, there was no obvious increase in peak flexion at either the knee or the ankle joints. The differences compared with peak flexion during swing were  $-2^\circ$  ( $n = 10$ ) and  $1^\circ$  ( $n = 10$ ), respectively. Another difference between the two groups of animals was that hip extension at the onset of the corrective response was noticeably less (mean of  $20^\circ$ ,  $n = 21$ ) than at the onset of swing in intact animals, whereas in spinal animals the hip

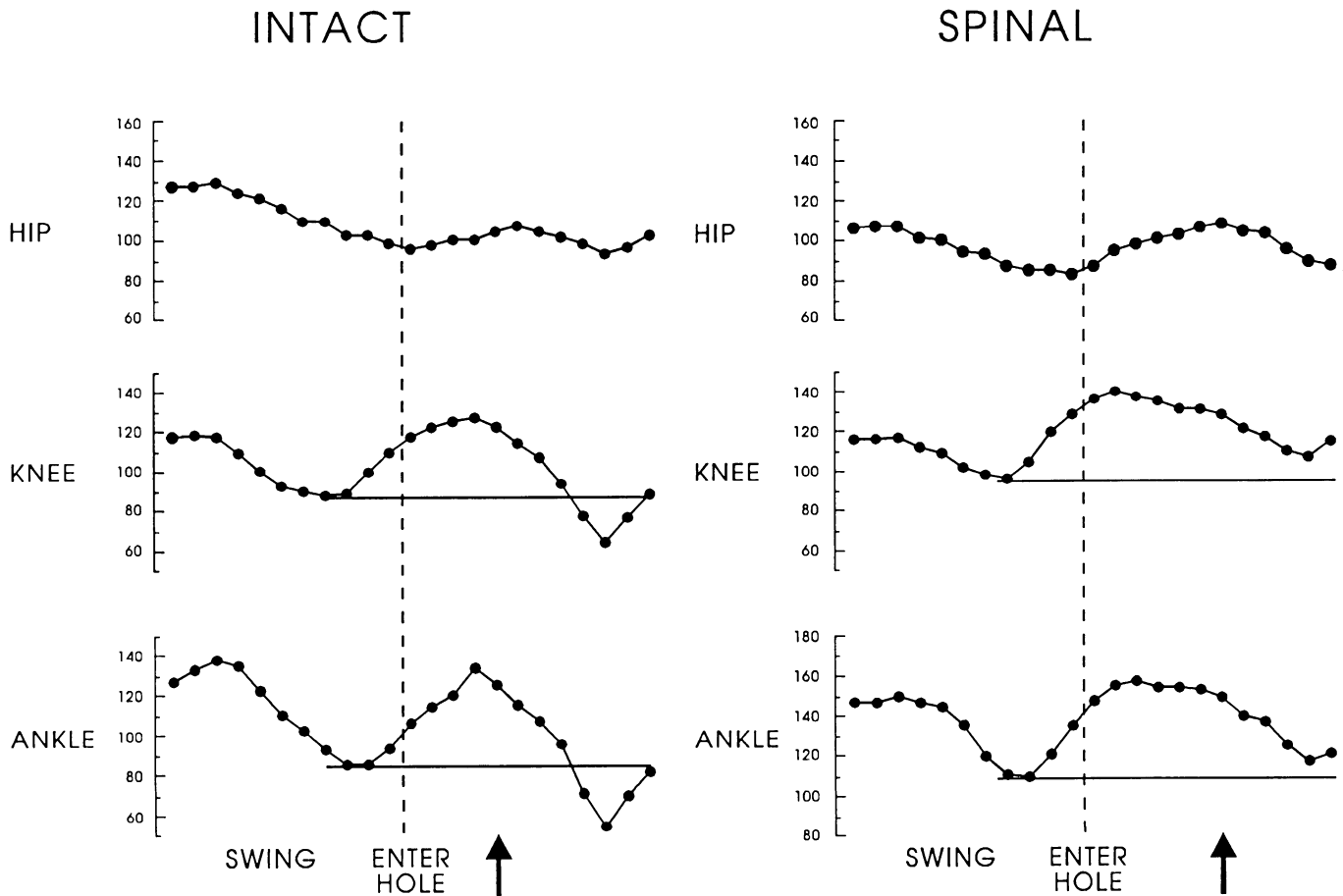


FIG. 4. Plots showing the joint angles at the hip (*top*), knee (*middle*), and ankle (*bottom*) during the swing phase of walking and during the subsequent corrective response in an intact (*left*) and a spinal (*right*) animal. The plots begin at the moment of foot liftoff and end when the foot regained support on the treadmill belt. Vertical line: point at which the limb would normally have contacted the surface of the treadmill. Arrow: onset of the corrective response. Horizontal lines: peak flexion of the knee and ankle during swing.

extension at the beginning of the response was usually larger than at the onset of swing (range 0–14°). In general the corrective responses with the longest latencies were associated with the larger values of hip extension (not shown).

EMG recordings revealed a characteristic pattern of motor activity in response to the paw entering the hole (Figs. 5–7). In the intact animal the ankle extensor lateral gastrocnemius (LG) muscle commenced activity ~70 ms before the foot entered the hole and ceased abruptly 60–100 ms after foot entry. This burst of activity in LG resembled the initial part of the LG bursts generated during stance when walking bipedally (Fig. 6*A*) and during quadrupedal walking (see Fig. 2, Gorassini et al. 1994). When the foot entered the hole there was little activity in LG for ~150 ms after the initial burst. It was during this period that the flexors were maximally active (Fig. 5*A*, *top 2 traces*). Regaining of foot support on the treadmill belt was associated with a resumption of LG activity that was often noticeably larger in amplitude than the stance phase bursts (Fig. 5*A*, *middle trace*). Vastus lateralis (VL) muscle, a knee extensor, demonstrated a similar pattern of activity. A small burst of activity occurred just before the limb entered the hole. The EMG activity then ceased as the limb flexors became active (Fig. 5*A*).

A qualitatively similar pattern of extensor activity was seen in spinal animals (Figs. 5*B* and 6, *C* and *D*). Activity in LG commenced ~60 ms before the foot entered the hole and declined abruptly soon after entry. This initial burst of activity was similar to that occurring around the time of ground contact during stance (Fig. 6*C*). The decline in this initial burst after the foot entered the hole was followed by a period of either inactivity or low-level activity lasting for ~250 ms. The latter part of this period corresponded to the generation of burst activity in flexors (Fig. 5*B*). A similar pattern of activity was seen in VL (Fig. 6*D*).

In both intact and spinal cats bursts of activity occurred in ipsilateral flexor muscles shortly after the limb entered the hole [see examples for semitendinosus (ST) and iliopsoas (IP) in Figs. 5 and 7]. These bursts of activity in flexors were associated with the flexion of the limb out of the hole. Although the general features of the patterns of activity in flexors during the corrective response were similar in intact and spinal animals, a number of consistent differences in the timing and amplitude were observed. One of the most noticeable was the longer latency to the onset of flexor burst activity in spinal animals. This latency was in the range of 40–80 ms in intact animals and 100–250 ms in spinal animals (relative to the estimated time the foot entered the hole). The difference corresponded to the behavioral obser-

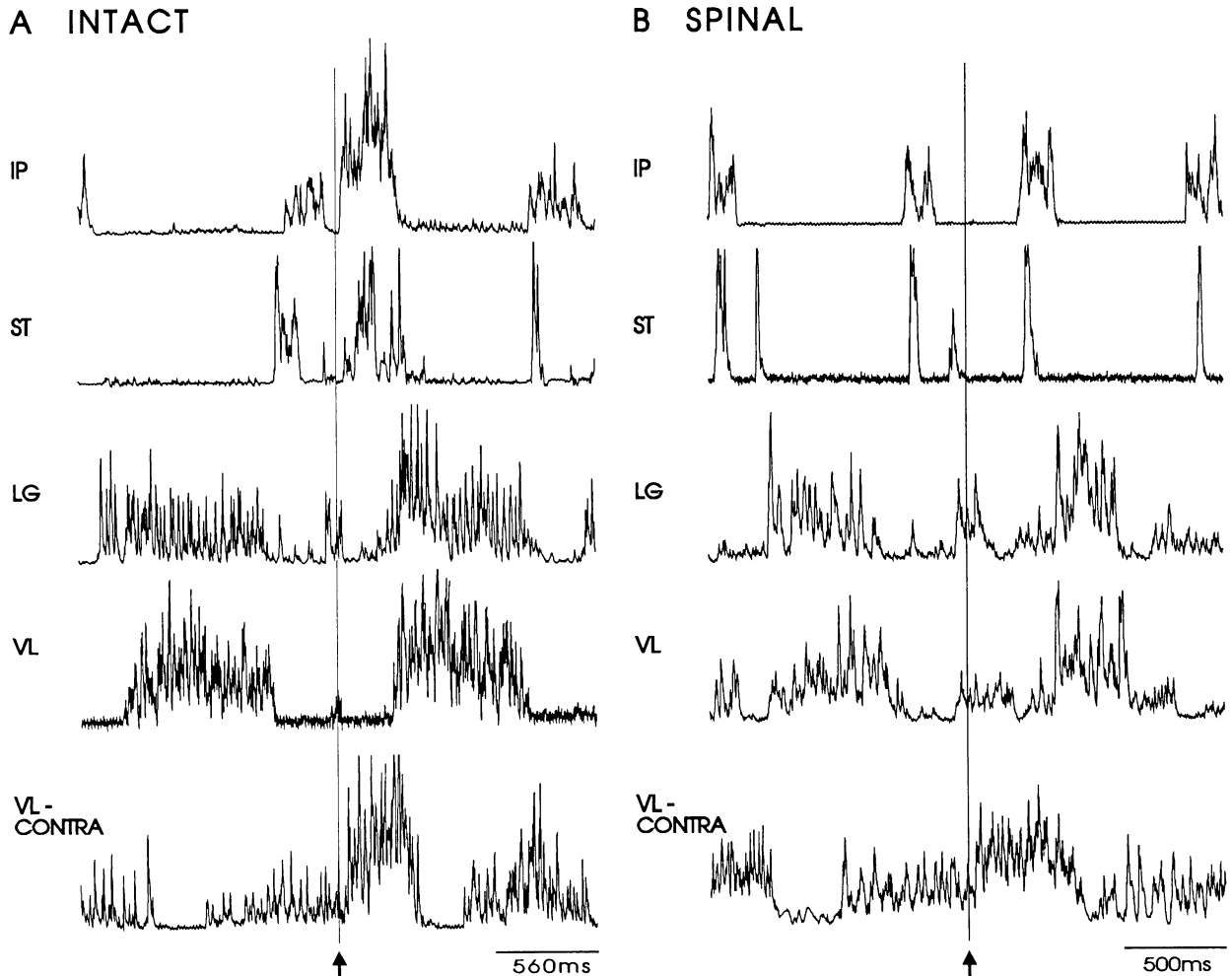


FIG. 5. Electromyogram (EMG) records from hind leg muscles of (A) an intact cat and (B) a chronic spinal cat showing the characteristic muscle activity in response to stepping into the hole in the treadmill belt. Vertical lines: times that the paw passed through the light fence. The limb is estimated to have passed beneath the plane of the treadmill belt 30 ms before the light fence signal. IP, iliopsoas; ST, semitendinosus; LG, lateral gastrocnemius; VL, vastus lateralis; CONTRA, contralateral.

vation that the time of onset to flexion in spinal animals was delayed (Fig. 2).

Another difference was that in intact animals the magnitude of the bursts in the IP were larger relative to that of the swing phase IP bursts, whereas in spinal animals only a slight increase in relative magnitude was observed (Figs. 5B and 7, C and D). Also the magnitude of the bursts in semitendinosus (ST) during the corrective response in intact animals was quite variable. In some trials it was noticeably larger than the swing phase bursts, whereas in others it was of similar magnitude. By contrast, in spinal cats the magnitude of ST bursts during the corrective response usually did not differ markedly from that observed during swing (Figs. 5 and 7, C and D).

Finally, a difference was observed in the profile of the flexor bursts. In intact animals the magnitude of the bursts in IP and in some trials in ST often increased noticeably during the burst (see  $\rightarrow$  in Fig. 7B). The timing of the onset of this increase in activity was variable and in the range of 130–180 ms. This augmentation of the flexor bursts was not observed in spinal animals, nor did it occur during the swing phase of stepping in either group of animals.

It is also important to note that some features of the

flexor bursts during the corrective response were similar in intact and spinal animals. One was that in both groups of animals the durations of the bursts in IP were increased relative to the durations of the IP bursts during swing (Fig. 7). Another was that the onset of ST activity followed the onset of IP activity during the corrective response (Fig. 7) (note that ST activity usually precedes IP during swing in the intact cat). A third was that the ST bursts were shorter than the IP bursts, and ST often became weakly active at the time extensor activity resumed. This second, smaller burst of ST activity at the onset of stance is a characteristic feature of this muscle's activity pattern during walking (Fig. 7, A and C).

At low treadmill speeds (<0.4 m/s) intact cats maintained support of their hindquarters during the corrective response by prolonging the stance phase of the contralateral leg (Figs. 1 and 8). This resulted in the contralateral leg extending further than normal. At higher treadmill speeds the flexion of the contralateral leg began while the ipsilateral foot was in the hole, thus causing a collapse of the hindquarters. Because this disturbed the subsequent behavior of the animal (manifested by a reluctance to walk with a stable bipedal pattern in the center of the treadmill) we

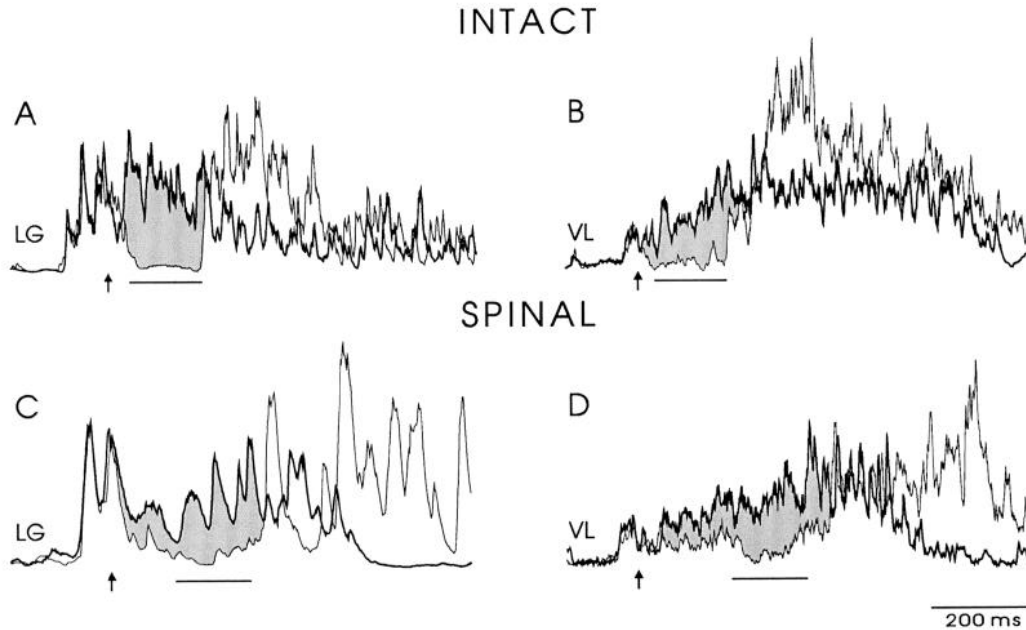


FIG. 6. Average rectified and filtered EMGs ( $n = 4$ ) recorded from the left LG (A and C) and VL (B and D) muscles in an intact cat (A and B) and a chronic spinal cat (C and D). Thick traces: EMG activity during normal stance. Thin traces: EMG patterns during trials where the left limb entered the hole. Arrows: times the paw passed through the light fence. Horizontal bars: periods of inactivity associated with flexor burst activity. Shaded areas: large decrease in extensor activity associated with the limb entering the hole.

deliberately kept the treadmill speeds low. Thus insufficient data were obtained to analyze responses at high treadmill speeds.

Corrective responses in the contralateral leg in three of five chronic spinal cats were generally similar to those in intact animals, i.e., the stance phase of this leg was prolonged to support the hindquarters when the ipsilateral leg entered the hole (Fig. 8). In most trials the additional extension of the contralateral leg was sufficient to support the animal's weight during the initial period when the ipsilateral foot was being removed from the hole. In the event that the ipsilateral leg failed to gain support at the end of the flexion response and re-entered the hole, the contralateral leg reached maximum extension and swing was initiated, thus leading to a collapse of the hindquarters. In a few trials in all three of these spinal animals the swing phase of the contralateral leg began before the initiation of the flexion of the ipsilateral leg. When this occurred, the ipsilateral flexion response was delayed until the swing phase of the contralateral leg was completed. This delayed onset of flexor activity is presumably due to mutual inhibitory coupling between the flexor burst generating systems of the two legs (Grillner 1981). In the other two spinal animals the contralateral leg always initiated its swing phase while the ipsilateral leg was in the hole. Thus the flexion of both legs occurred almost synchronously. The hindquarters of these two animals had to be supported by the experimenter when the foot entered the hole.

In all intact animals and in the three spinal animals that maintained contralateral support during the corrective response, EMG recordings from extensor muscles in the contralateral leg (the right leg) showed an increase in the intensity of extensor activity  $\sim 70$  ms after the left paw entered the hole (Fig. 9). This latency was similar in both spinal

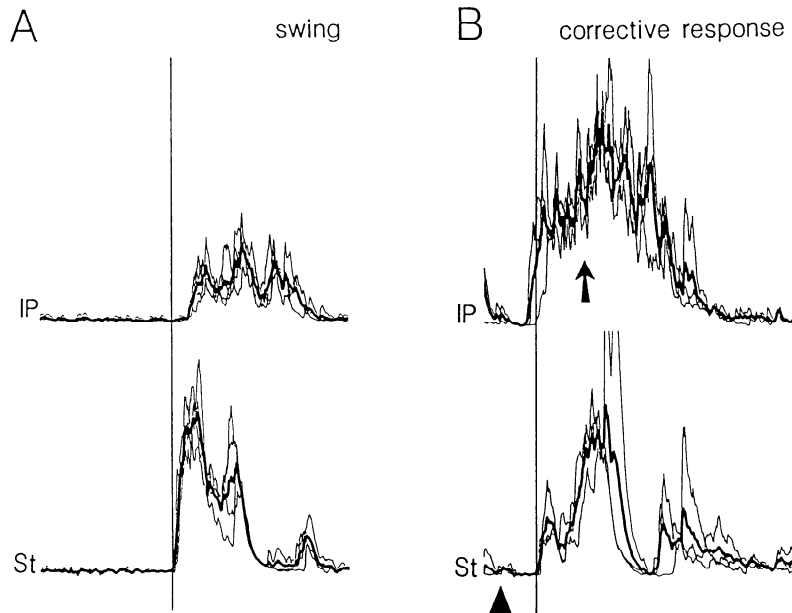
and intact animals, but the magnitude of the increase was usually larger in intact animals. The higher level of activity was maintained for the duration of support.

#### DISCUSSION

In the preceding paper (Gorassini et al. 1994) the corrective response of a hind leg to the unexpected loss of ground support was examined in normal cats walking on a surface containing a hidden trap door. One of the issues arising from that study was whether the response was organized at a spinal level or whether supraspinal mechanisms were involved in its initiation and regulation. In the current investigation we address this issue by comparing the reactions to loss of hind leg support in intact and chronic spinal cats walking bipedally on a treadmill with a hole in the belt. Before discussing this comparison it is important to note that the responses evoked in intact cats stepping bipedally on a treadmill were similar to those occurring during normal quadrupedal walking. This was characterized by a strong flexion of the leg commencing 70–150 ms after the foot entered the hole and, at low speeds, a marked prolongation of the stance phase of the contralateral leg to support the hindquarters. The flexion movements of the leg were exaggerated compared with these movements during swing. Finally, the motor pattern associated with the corrective response was similar during bipedal and quadrupedal walking. In particular, activity in leg extensors was abruptly terminated soon after the paw entered the hole and at approximately the same time bursts of activity were evoked in flexor muscles.

One of the main findings of the present study is that the reaction to a loss of ground support in chronic spinal cats was not the same as that in intact animals. The most obvi-

## INTACT



## SPINAL

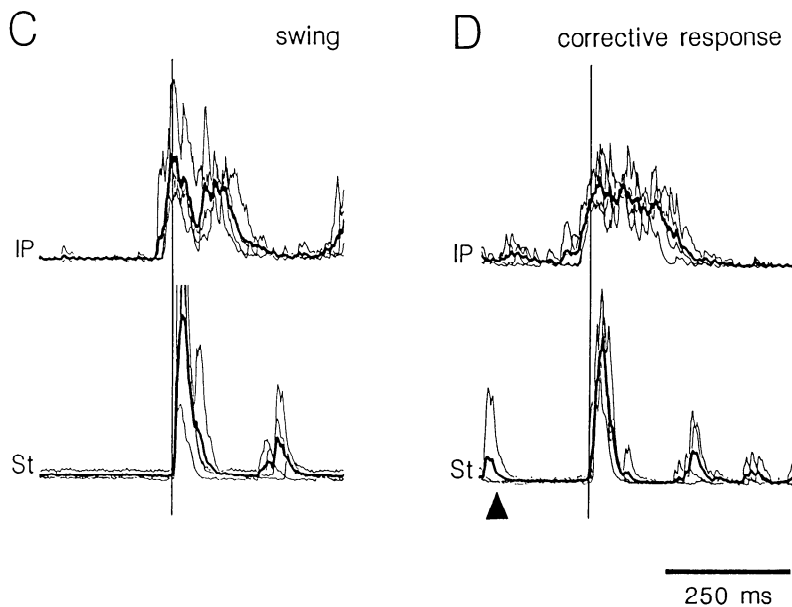


FIG. 7. Averaged ( $n = 4$ ) rectified and filtered EMGs (thick lines) from flexor muscles showing flexor bursts during the swing phase of stepping (*A* and *C*) and the corrective response (*B* and *D*). Thin lines: individual trials. Vertical lines: onset of ST activity. Arrowheads in *B* and *D*: estimated time that the foot entered the hole. *A* and *B*: intact animal. *C* and *D*: spinal animal. Note the increase in the magnitude of the IP bursts ( $\rightarrow$ ) during the corrective response in the intact animal and the longer latency in the onset of flexor burst activity in the spinal animal.

ous differences were 1) in chronic spinal cats the latency of onset of the flexion movement was variable and significantly longer (by  $\sim 130$  ms on average: Fig. 2) and 2) the exaggerated flexion movement of the leg did not occur in the spinal cats (Fig. 3). Corresponding to these kinematic observations, we found that the latency of onset of flexor EMG activity in spinal cats was delayed significantly compared with that recorded in intact animals (Figs. 5*B* and 7) and in spinal animals there was little increase in the magnitude of flexor burst activity relative to the magnitude of swing-phase flexor bursts. These observations clearly demonstrate that supraspinal mechanisms are participating in the production of the corrective response in intact animals.

The issue now is whether the corrective response in intact animals is generated by a distinct motor program organized at the supraspinal level or whether supraspinal pathways modulate the spinal system that produces the flexion response in spinal animals. The first point to be made is that the spinal system generating the flexor bursts in spinal animals appears to be the same as the system that produces the swing phase flexor activity in gait. This follows from the fact that the pattern of flexor activity is similar in the two situations (Fig. 7, *C* and *D*). The main difference we observed was that the durations of the IP bursts were longer during the corrective response. This may be because of the differences in the leg position at the beginning of the corrective



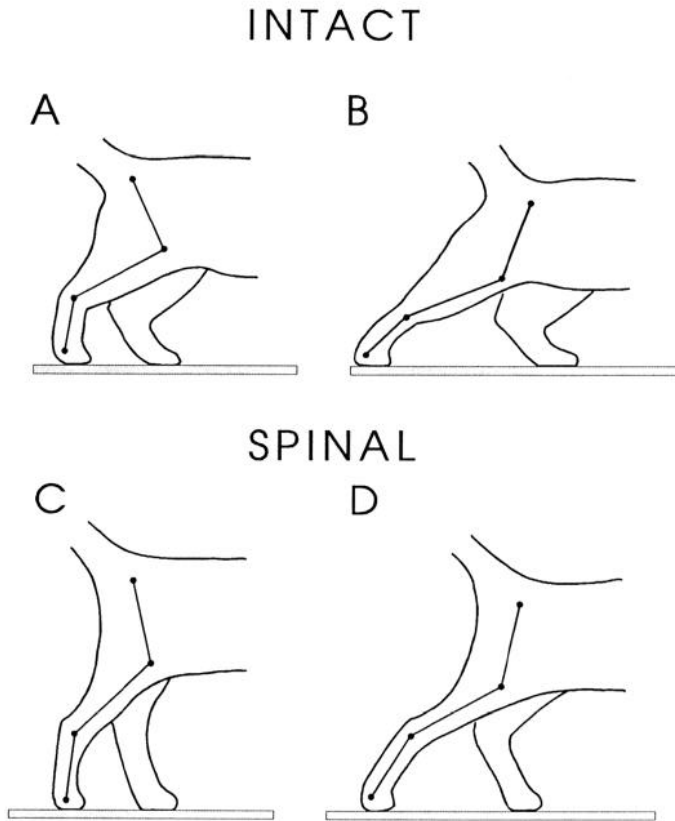


FIG. 8. Drawings of the positions of the hind legs of an intact cat (*A* and *B*) and a chronic spinal cat (*C* and *D*) at the end of stance phase of the right leg when stepping normally on a treadmill (*A* and *C*) and immediately after the left leg had been withdrawn from the hole in the treadmill (*B* and *D*). Note in both animals that after the left leg was withdrawn from the hole the right leg was extended well beyond normal.

response and at the beginning of stance [in chronic spinal cats it has been reported that the duration and intensity of flexor bursts is dependent on leg position (Pearson and Rossignol 1991)]. The next point is that there are several similarities in the pattern of flexor activity during the correc-

tive response in intact and spinal cats: 1) the ST bursts were shorter in duration than the IP bursts, 2) ST often generated a second weaker burst at the time that extensor activity resumed, 3) the onset of ST activity was delayed slightly compared with the onset of IP activity, and 4) the duration of the IP bursts was prolonged relative to the duration of swing phase flexor activity. These similarities indicate that the same system is used to produce the corrective response in both groups of animals. Because we have concluded that this is probably the same system generating swing phase flexor bursts in spinal animals (see above), it follows that the corrective response in intact animals also involves activation of the swing phase flexor burst generating system. The question now arises as to how this system in intact animals is modulated by supraspinal input. Before considering this we must discuss the mechanisms regulating the initiation of flexor activity during stepping and during the corrective response in chronic spinal cats.

Two factors are considered to be important in regulating the transition from stance to swing during stepping in spinal cats. The first is extension of the hip (Grillner and Rossignol 1978) and the second is unloading of extensor muscles (Conway et al. 1987; Duysens and Pearson 1980; Pearson et al. 1992). Because the hip joint angle at the onset of the corrective response was variable and was usually greater (more extended) than at the onset of swing, we feel that hip angle is not a critical factor in the initiation of the corrective response in spinal animals. A more likely cause of the reinitiation of flexor burst activity is the absence of any loading of leg extensor muscles. In decerebrate and spinal animals loading of ankle extensor muscles inhibits the generation of flexor bursts and prolongs the duration of extensor activity (Conway et al. 1987; Duysens and Pearson 1980; Pearson et al. 1992). Thus the complete absence of loading of extensors when the foot enters the hole would be expected to lead to a reduction in the interval between flexor bursts, exactly as we have observed (Figs. 5 and 7). Consistent with this proposal is that increasing the intensity of stimulation to the perineum increases the frequency of air stepping in spi-

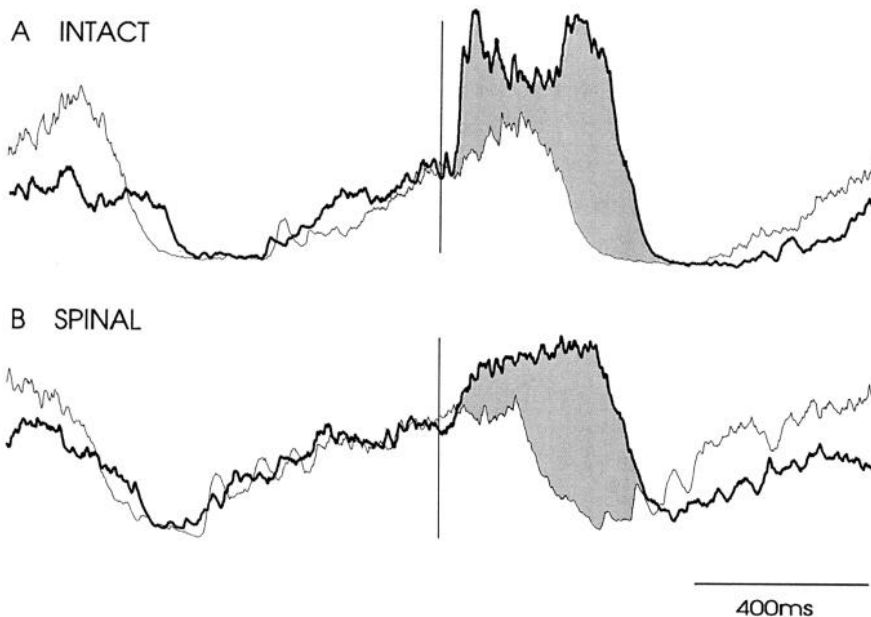


FIG. 9. Averaged rectified and filtered EMGs from the VL muscle of the contralateral leg in an intact (*A*) and a chronic spinal (*B*) cat during normal stepping (thin traces,  $n = 5$ ) and at the time the left leg entered a hole in the treadmill (thick traces,  $n = 5$ ). Vertical lines: times the left paw passed through the light fence. Shaded areas: increase in the magnitude of the EMGs commencing soon after the left leg entered the hole and the increase in the duration of the EMG activity.

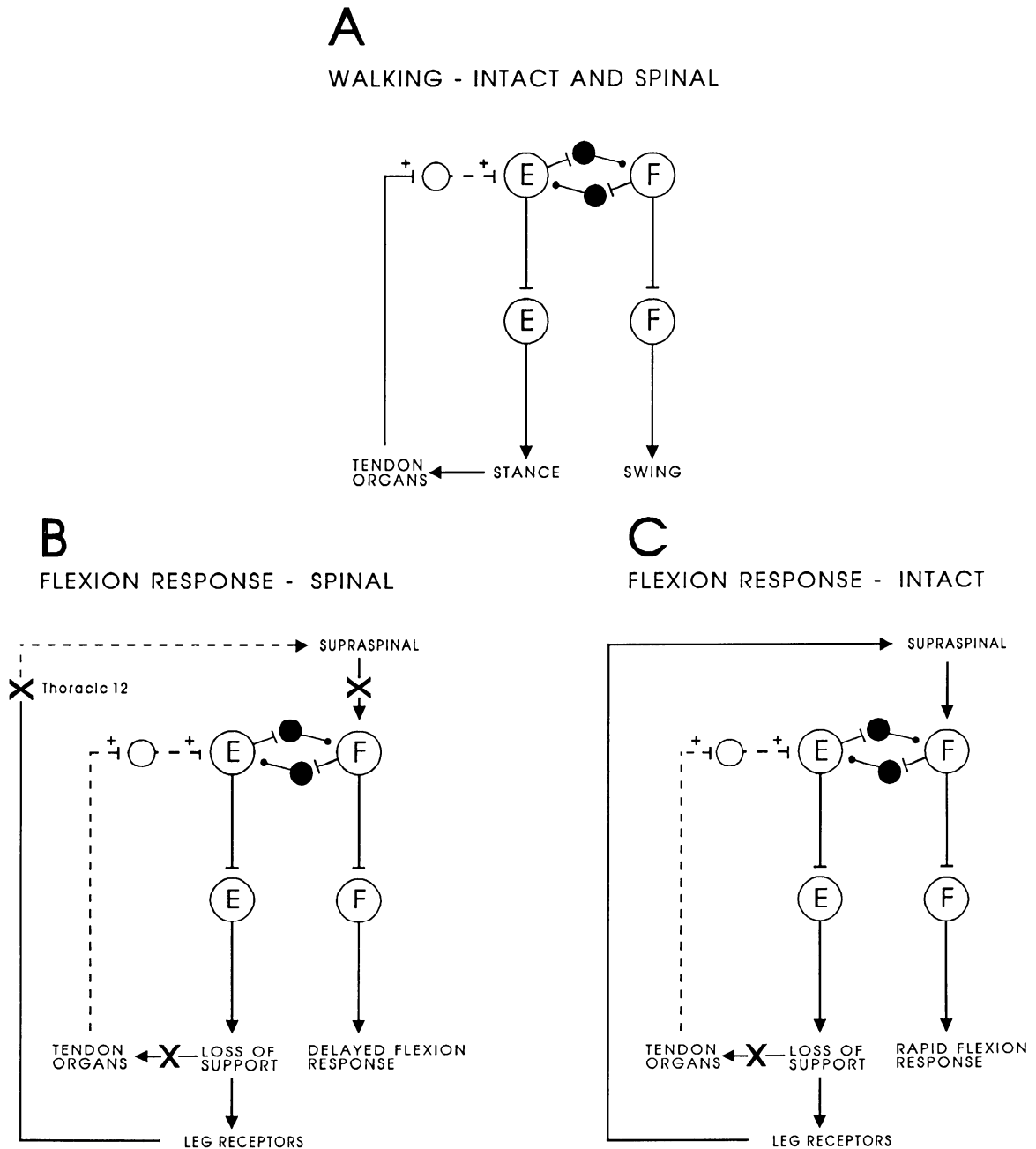


FIG. 10. Diagrams showing possible schemes for neural pathways involved in walking (*A*) and in the corrective response to a loss of ground support in spinal (*B*) and intact (*C*) animals. During normal walking proprioceptive feedback from the tendon organs (group Ib afferents) ensures that flexor activity is not initiated until the leg is unloaded near the end of the stance phase by inhibiting the flexor half-center via an excitatory pathway to the extensor half-center [the locomotor rhythm generator is represented as mutually inhibiting flexor (*F*) and extensor (*E*) half-centers]. *B*: in spinal animals when a leg enters the hole the tendon organs are not activated, thus reducing the excitatory input to the extensor half-center, which allows flexor burst activity to be reinitiated earlier. *C*: when a leg enters a hole in intact animals there is also an absence of tendon organ feedback but in this case either the absence of normal stimuli and/or the generation of novel signals activates supraspinal structures to facilitate the flexion response. Crosses in *B* and *C*: pathways not contributing to the regulation of flexor burst activity.

nal cats and decreases the latency of the corrective response (unpublished data). The afferents responsible for preventing the initiation of flexor activity when extensor muscles are loaded are most likely the group Ib afferents from the tendon organs (Conway et al. 1987; Pearson et al. 1992). Thus we propose that the flexion response in spinal animals consists of a much earlier onset of swing phase flexor activity because extensor activity generated as the foot entered

the hole was not maintained by any input from group Ib afferents (Fig. 10, *A* and *B*).

Although we propose that the lack of tendon organ feedback is the primary cause of the early flexor response, other proprioceptors could also contribute. For instance, at the transition between E1 and E2 phases of stance the contracting extensor muscles lengthen as they yield at ground contact, thus increasing Ia afferent activity from the muscle

spindles (Prochazka et al. 1989). However, when the limb enters a hole there would be no yield and thus a marked decrease in Ia activity. This would result in less excitation of extensor motoneurons and perhaps a reduction in the level of excitation of the interneuronal network generating extensor bursts (Pearson and Collins 1993). The latter may then facilitate the transition from extensor to flexor activity.

How could the mechanism functioning in spinal animals (Fig. 10B) be modified, and/or what additional mechanisms must function to account for the shorter latency and enhanced magnitude of the corrective response in intact animals? We consider it unlikely that the flexor burst-generating system is tonically facilitated by supraspinal inputs in intact animals because this would result in enhanced flexor activity during the swing phase of stepping in intact animals compared with spinal animals. We know from the work of others (Belanger et al. 1990) and from our own observations that flexor activity during swing is similar in intact and chronic spinal cats. However, it is conceivable that tonic supraspinal signals could facilitate spinal pathways that transmit the novel sensory signals elicited when the foot enters the hole to the network generating flexor bursts. Alternatively, novel phasic sensory signals generated when the foot enters the hole (or the absence of an expected sensory signal) could act via supraspinal pathways to trigger a descending command to facilitate flexor burst generation (Fig. 10C). It should be noted that fairly complex processes could be involved in intact animals because the latency of the onset of flexor activity after the foot entered the hole was between 40 and 80 ms. The possibility of phasic supraspinal modulation of spinal pattern-generating networks is plausible, because this is known to occur during predictive changes in stepping movements to avoid visually perceived obstacles (Drew 1991). In this case it has been found that the activity of neurons in the motor cortex is facilitated during the enhanced flexion response. It would be interesting to determine whether a similar facilitation occurs in cortical neurons during the corrective response to a foot entering a hole. Enhancement of flexor burst activity by phasic signals from subcortical structures must also be considered likely because postural reactions in response to a sudden drop of support to one hindlimb are not altered by unilateral ablation of the sensorimotor cortex (Dufossé et al. 1985).

One consistent observation in both intact and spinal animals was an increase in the magnitude of extensor activity in the contralateral leg ~70 ms after the ipsilateral leg entered the hole (Fig. 9). This increase may have been due to increase in the load carried by this leg. Other observations demonstrating the dependence of extensor activity on loading conditions are: an increase in the magnitude of extensor activity when intact animals walk up an incline (Pierotti et al. 1989) and a reduction in the magnitude of extensor activity during stance in spinal animals when the animal is lifted slightly to reduce loading of the hind legs (unpublished data). In humans afferent feedback is also considered to be involved in the generation of activity in the soleus muscle during the stance phase of walking (Yang et al. 1991). Alternatively, this contralateral response may have been triggered by sensory signals from the ipsilateral leg. One obser-

vation of potential relevance is that in standing animals a drop in the support of one hindlimb results in an increase in extensor activity in the contralateral hindlimb that precedes the additional loading of the limb. (Rushmer et al. 1987).

Another effect of the increased loading of the contralateral leg during the corrective response was a prolongation of the stance phase in all intact animals and in three of the five spinal animals. The simplest explanation for this phenomenon is that additional loading of extensor muscles inhibited the generation of flexor burst activity. The similar prolongation of the extensor phase in intact and spinal animals and the qualitatively similar profiles of activity in the VL muscle (Fig. 9) indicate that the mechanism regulating the stance phase of the contralateral leg during the corrective response is organized primarily at the spinal level.

In conclusion, we have shown that the corrective response to the loss of ground support during walking depends to a large extent on the integrity of supraspinal structures. Our data also indicate that descending signals from these structures facilitate the spinal network that normally generates flexor burst activity associated with the swing phase of walking. The identity of the relevant supraspinal structures and the mechanisms by which they exert the facilitatory influence on the locomotor pattern generator remain to be established.

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