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Corrective Responses to Loss of Ground Support During Walking I. Intact Cats

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SUMMARY AND CONCLUSIONS

1. In the cat step cycle the electromyographic (EMG) activity in ankle extensor muscles commences ~70 ms before foot contact. There is a sharp peak between 10 and 25 ms after contact and the EMG then declines for the remainder of the stance phase. It has been posited that the abrupt transition in EMG after contact is the consequence of reflexes elicited by the large barrage of afferent input that signals foot touchdown. However, it is also possible that the basic profile might be generated within the CNS, with little modification by afferent input.

2. These ideas were tested in 11 normal cats. We compared EMG responses and hindlimb kinematics in steps with normal ground support and steps in which an actuator-controlled trap door unexpectedly opened, withdrawing ground support just before foot contact.

3. In the absence of ground support the transition in EMG activity was still present. The averaged EMG pattern was similar for at least 30 ms after the foot passed through the plane of the floor. We conclude that the basic extensor activation profile in this part of the cycle is generated centrally and is not substantially altered by afferent input.

4. Between 35 and 200 ms after contact the stance phase was aborted and the foot was lifted smartly out of the hole. This reaction varied both in latency and kinematic detail, suggesting a fairly complex corrective response. For example, EMG and kinematic responses depended strongly on posture: if the contralateral leg was bearing weight a rapid withdrawal from the hole was seen, whereas if the contralateral leg was already in its swing phase, exaggerated and prolonged extension of the ipsilateral leg into the hole ensued. The speed of flexion out of the hole also depended on prior experience and expectancy. This raised the possibility of supraspinal involvement, an issue that is taken up in the subsequent paper.

INTRODUCTION

The extent to which peripheral afferents contribute to the central control of functional and adaptive locomotion remains a contentious issue. When it was demonstrated some years ago that the spinal cord alone, in the absence of motion-dependent feedback, could produce coordinated electromyographic (EMG) patterns similar to those seen during normal, unperturbed locomotion (Grillner and Zangger 1975, 1979, 1984), the importance of afferent input seemed in some doubt. However, it was noted from the outset that dorsal root, dorsal column, or perineal stimulation were often needed to produce sustained sequences of locomotor output in the spinal deafferented animal. Various other observations quite specifically demonstrated a significant role for sensory input, particularly that elicited by hip movements and weight bearing. For example, it has been known for some time that unilateral deafferentation in normal and chronic spinal cats makes stepping erratic and uncoordinated (Giuliani and Smith 1987; Goldberger 1977). The hindlimbs of spinal cats walking on a split-belt treadmill separately adapt their speed to that of their corresponding belt as a result of the sensory activity elicited by foot contact with the belt (Forssberg et al. 1980). Obstruction of hip movement (Andersson et al. 1978; Grillner and Rossignol 1978) or loading of extensor muscles (Duysens and Pearson 1980) can completely suppress rhythmicity in a leg while the other legs continue to be rhythmically active. The loading effect is precise and reproducible in that flexor phases of the cycle only commence when extensor load is reduced below a particular level (Conway et al. 1987; Pearson et al. 1992).

In our study we were specifically interested in any short-latency reflex effects of the afferent volley signaling hindlimb contact (Prochazka et al. 1989) and the compensatory reactions evoked in the absence of this information. During normal locomotion the EMG activity of ankle extensors commences 50–80 ms before foot contact and rises to a peak at 10–25 ms after contact. It then falls abruptly to a lower level and then declines slowly for the rest of the stance phase (Llewellyn et al. 1989; Trend 1987). From the extensive literature on short-latency reflexes in cat hindlimb muscles (Jankowska 1992) we posited that the abrupt transition was the result of reflexes elicited by ground contact. Proprioceptive input from the lower leg was suspected because cutaneous anesthesia of the foot pads did not substantially change the profile (Trend 1987). However, it is possible that the entire pattern could be generated within the CNS. Ankle extensor EMG bursts recorded in preparations in which there is no modulated afferent input rise rapidly, reach a peak, and decline (Grillner 1985; Grillner and Zangger 1979; Jordan et al. 1979; Pearson and Rossignol 1991). This shows that pattern generators in the CNS can produce an activation profile similar to that seen in the normal animal, although its precise timing in relation to footfall is unclear.

The pattern of EMG activity occurring after foot contact has been studied in human running (Dietz et al. 1979) and in landing from falls in cats and monkeys (Laursen et al. 1978; Prochazka et al. 1977). The human data indicated that reflexes just after foot touchdown played a significant role in running, but less so in walking (Dietz 1992). The monkey data were interesting because they indicated that the postcontact EMG patterns recorded after falls were centrally produced. The monkeys were trained to land either on a solid surface or on an identical-looking surface of tissue paper with the floor 8 cm below. No major difference

was seen in the EMG activity shortly after contact with either the solid or paper surfaces. Thus Laursen et al. (1978) concluded that input from limb receptors was not influential in shaping postcontact EMG patterns (but cf. Prochazka et al. 1977).

To test these ideas further we designed an experiment in which ground support was withdrawn just before foot contact in the intact, walking cat. This allowed us to compare EMG profiles with and without hindlimb contact. We found that the extensor activation in the early stance phase was similar in the two cases. We also found that the sudden loss of ground support evoked interesting compensatory reactions that depended on expectancy and the posture of the animal at the time the hindfoot entered the hole. The question as to how much these compensatory responses are mediated at the spinal level and how much they are supraspinally influenced or controlled is introduced here and further addressed in the following paper (Hiebert et al. 1994).

METHODS

Eleven adult cats (4 males, 7 females) weighing between 2.5 and 4 kg were used in this study. We recorded EMG and behavioral data from nine of these cats. In the remaining two, only behavioral data were collected. All procedures were carried out with the approval of the University of Alberta Health Sciences Animal Welfare Committee.

Implants

In one aseptic procedure performed with pentobarbital sodium anesthesia (40 mg/kg iv) pairs of intramuscular electrodes (Cooner Wire AS-632) were implanted into the lateral gastrocnemius (LG) and other hindlimb muscles. The wires were led subcutaneously to a dental acrylic headpiece that housed the EMG connectors and telemeter socket (Prochazka 1983). Postoperatively, under veterinary supervision, an analgesic (buprenorphine, 0.005-0.01 mg/kg) was administered for 1–2 days as needed and an antibiotic (Amoxil, 50 mg per tablet) was given twice daily for 10 days. The animals recovered within 1–3 days with no residual locomotor deficit.

Trap door walkway

An actuator-controlled trap door built into a walkway was triggered to open just before hindlimb contact (Fig. 1). The camouflaged trap door contained a force sensor to detect forepaw loading. The decline in the force signal at the end of the foreleg stance phase was used to trigger the trap door, which swung down very rapidly, reaching a vertical orientation within 30 ms (a slight delay was included to prevent opening before the forepaw was lifted). The precise time of hindpaw entry into the hole was detected by a photoelectric "light fence." This consisted of a linear array of light-emitting diodes, spaced 6 mm apart, whose collimated beams traversed the hole in a plane 1 mm below that of the walkway and activated a similar array of light-sensitive diodes on the far side. Individual signals from this array were encoded into a single multilevel output signal, allowing the beams interrupted by the cat's paw to be identified. Step changes in the output signal were subsequently used to align EMG traces for averaging. The moment of foot contact during normal steps was obtained in separate trials using a different detector. A piece of metal foil taped to the pad of the hindpaw made electrical contact between adjacent, interlaced tracks of a printed circuit sheet stuck to the floor of the walkway. This produced a sudden change in voltage of one of the sets of tracks, signaling foot contact. In two animals foot contact data were obtained during foot-in-hole trials.

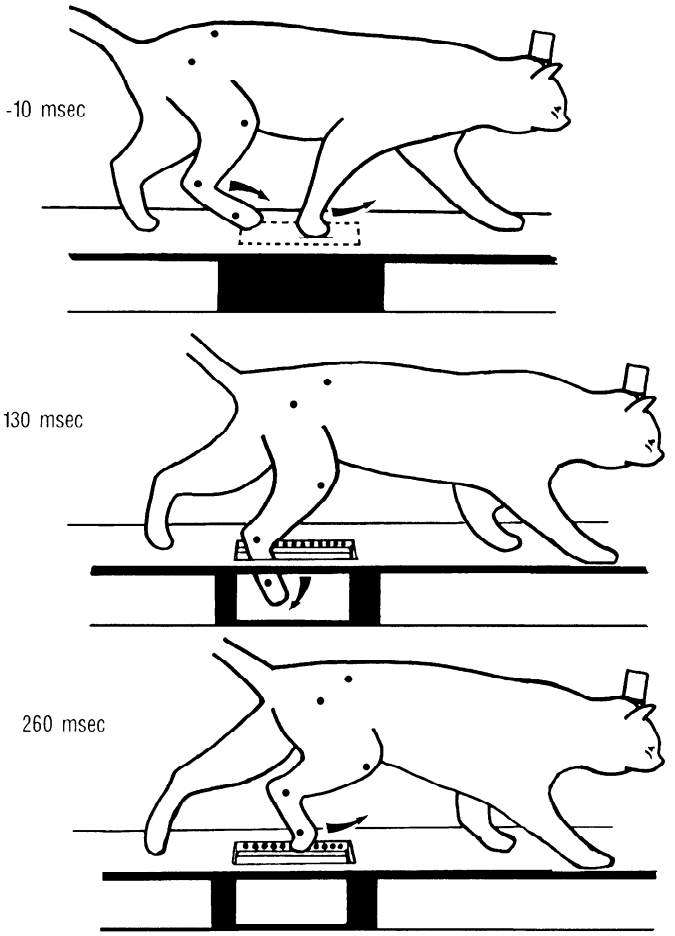

FIG. 1. Video tracings illustrating the "foot-in-hole" experiment. Loading of the forelimb was detected by a force sensor built into the camouflaged trap door (- - -). A decline in the force signal at the end of forelimb stance was used to trigger the opening of the spring-loaded trap door, which swung down to a vertical position within 30 ms. The precise timing of foot entry into the hole was detected by the interruption of beams of light emitted from a linear array of light-emitting diodes on 1 side of the hole (*middle panel*) and directed at a similar array of light-sensitive diodes under the near rim of the hole. The tracings are from the video frame just before foot entry (*top*), the 4th frame after entry (*middle*), and the 8th frame after entry (*bottom*). Corresponding times relative to entry are accurate to ±16 ms.

Data recording and analysis

The cats were encouraged to walk along the platform with food rewards. A telemeter attached to the implanted headpiece transmitted EMG activity that was monitored by a nearby radio receiver. In two animals connections to the recording amplifiers were made via long flexible cables attached to the headpiece of the cat. Light fence and EMG signals were stored on a TEAC R-71 cassette or DR-890 Neuro Data digital tape recorder. Data analysis was performed off-line. Only those trials in which there was a clear and unobstructed entry into the hole, as judged by viewing the relevant videotaped sequence, were included in the averages. Relevant segments were replayed and digitized with a CED 1401 (Cambridge, UK) interface and an 80486 Express microcomputer running CED data acquisition software. The raw EMG data were rectified and filtered (DC, 1 kHz) and digitized at a sampling rate of 2 kHz for each channel. Sigma Stat 5.0 and Matlab Software

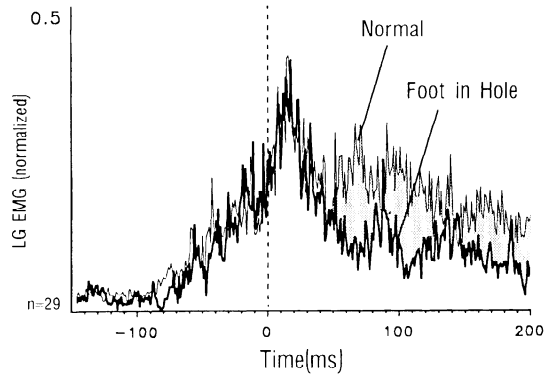

FIG. 2. Averaged lateral gastrocnemius (LG) electromyogram (EMG) profile of 29 steps into the hole (thick trace) superimposed on 29 normal steps (thin trace) from 9 cats. Averages were aligned around the moment the foot entered the hole and at the time of foot contact, respectively, as indicated by the dashed vertical line at time 0. Both averages reach a peak at 15 ms from time 0 and then show a sharp decline. The difference in the EMG profiles between the 2 averages is highlighted by the stippled area.

were used to perform the statistical analysis on the LG EMG profiles.

The side wall of the walkway in front of the trap door was made of transparent Plexiglas to allow for video filming (30 frames per second). Relative joint angles of the leg entering the hole were obtained by placing surface markers over the iliac crest, greater trochanter, lateral epicondyle of the femur, lateral malleolus, and metatarsophalangeal joint. Joint angles and hindlimb position in individual video frames were analyzed using Video-Blaster software (Creative Labs) and with custom programs written by Keir Pearson. The time of onset of leg flexion was estimated from a frame-by-frame analysis of the video recordings and was therefore accurate to within 33 ms.

RESULTS

Figure 2*A* shows the averaged LG EMG firing profile of 29 steps into the hole (thick trace) superimposed on the average of 29 normal steps with ground contact (thin trace) from nine cats. The latter were aligned to the moment of contact and the former were aligned to the moment the foot penetrated the surface of the hole, as indicated by the dashed vertical line. To compare EMG profiles from different cats, all traces were normalized to peak EMG amplitude that occurred shortly after ground contact or after the foot entered the hole. Peak EMG amplitudes for both conditions were similar (foot-in-hole steps were on average only 7% greater), so we felt that normalizing both conditions separately would not give misleading amplitude information. Note that the two LG EMG profiles were quite similar for the first 100 ms of rise, and more importantly, both averages underwent a sharp transition in activity after reaching a peak 15 ms after the moment of ground contact or after the foot penetrated the surface of the hole. The data do not support the hypothesis of Llewellyn et al. (1989) that the EMG transition between 10 and 25 ms after normal foot contact is reflexly mediated. The stippled area highlights the difference in EMG activity after the foot entered the hole, compared with steps with ground contact: LG activity decreased substantially in the foot-in-hole trials starting at ~50 ms after time 0.

Figure 3 provides a more detailed statistical analysis of the two individual averaged profiles from Fig. 2. The solid traces are the mean EMG traces and the dashed traces show the computed $\pm 2 \times$ SE envelopes (95% confidence limits about the mean). If one superimposes the two traces (not shown), the SE envelopes in the period of 0–40 ms overlap. Thus we can be 95% confident that the means lie between these boundaries for both conditions. To examine the time course of the decline in EMG that ensued shortly after peak activity, we fitted regression lines through the data points as shown and compared slopes. A slightly higher slope was obtained in the foot-in-hole trials as compared with normal steps with ground contact (5.60×10^{-3} vs. 6.48×10^{-3}, respectively). The difference between the two slopes (14%) was tested for significance using Student's t test and was found not to be significantly different at the 95% confidence level ($P > 0.05$) [a significant difference was only obtained at the 50% level ($P > 0.5$)]. The latency of peak EMG activity was quite variable: 16.2 ± 7.7 ms (mean ± SD) after normal contact and 13.4 ± 7.6 ms for foot-in-hole trials. This discrepancy may be attributed to a small delay in the alignment point of foot-in-hole trials caused by the light fence being located 1 mm below the surface of the walkway.

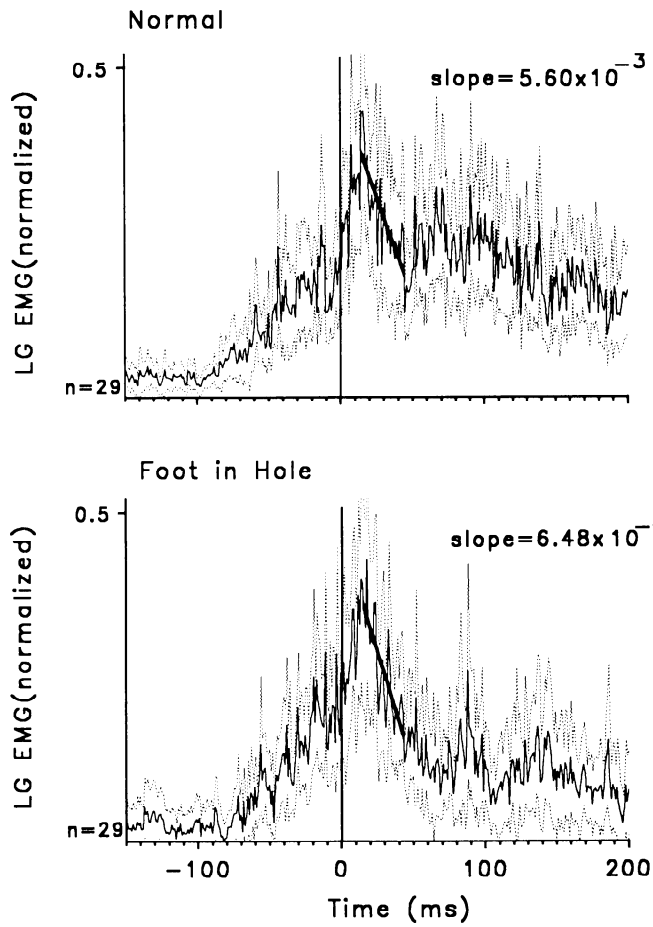

FIG. 3. Mean (solid traces) and $\pm 2 \times$ SE values (dashed traces) of the averages from Fig. 2. The SE envelopes of both averages overlap in the period of 0–40 ms, which indicates that the means during this interval are not significantly different at a 95% confidence level. The slope of the regression line through the data points during the declining EMG is slightly higher in the foot-in-hole trials (slopes are significantly different at $P > 0.5$, Student's t test). Latency of peak EMG activity: foot-in-hole trials, 13.4 ± 7.6 ms (mean ± SD); normal contact trials, 16.2 ± 7.7 ms.

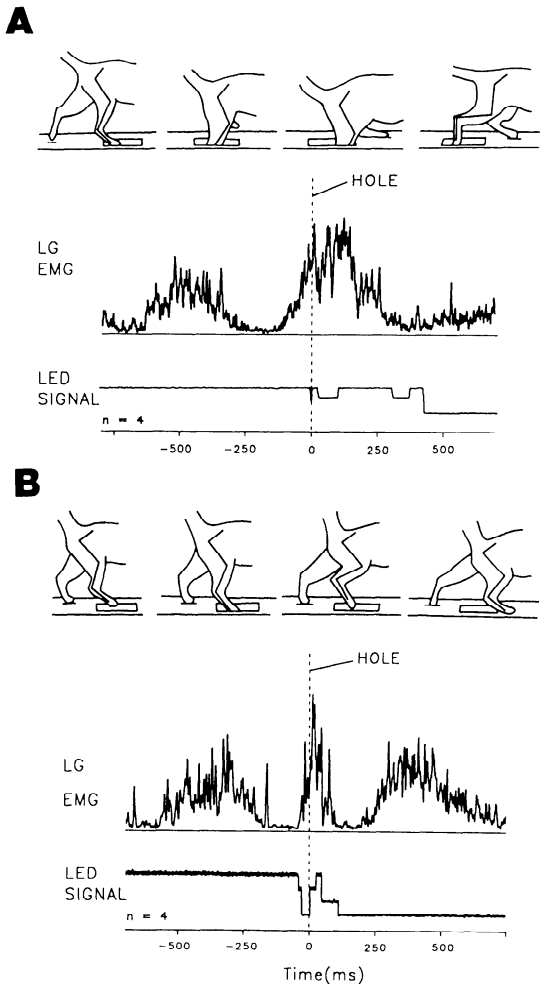

FIG. 4. *A*: behavioral and EMG responses to loss of ground support without contralateral limb support. The video tracings show that the contralateral leg had just commenced its swing as the ipsilateral paw entered the hole. There was a marked extension of the ipsilateral leg into the hole, which was accompanied by a prolonged LG EMG burst. *Lower trace*: light fence [light-emitting diode (LED)] signal, which was also monitored on the video images, allowing precise synchronization. *B*: during trials in which contralateral support was maintained throughout, the cat quickly flexed its limb out of the hole and the extensor EMG activity was aborted at ~140 ms after entry. It then resumed as the foot was placed on the surface beyond the hole.

The averaged foot-in-hole profiles of Figs. 2 and 3 were composed of trials in which the corrective responses ranged from rapid flexion out of the hole (Fig. 4*B*) to prolonged extension into it (Fig. 4*A*). In the four-trial average of Fig. 4*A* the hindlimb extended into the hole for >250 ms and LG EMG was elevated from 40 to 200 ms after entry, compared with steps with normal ground contact. On the other hand, in the four examples averaged in Fig. 4*B*, extensor activity was aborted within 80 ms. Note, however, that in both types of response, the EMG profiles in the periods corresponding to late swing and early stance (±40 ms of entry) resembled those of steps with ground contact and showed comparable sharp transitions shortly after the foot passed through the plane of the floor.

From these data and the accompanying video sequences it became apparent that the overall behavioral responses to loss of ground support depended greatly on prior experience, posture, and the speed of locomotion. For example, in naive cats or in cats trotting across the platform, the leg remained extended in the hole for ~200 ms or more after entry (Fig. 4*A*: the contralateral hindlimb had already commenced its forward swing at the time the ipsilateral foot entered the hole). This suggested that the prolonged ipsilateral LG EMG burst and extension into the hole were part of a compensatory reaction to a sudden, complete lack of support of the hindquarters. In such trials, initiation of flexion out of the hole was always delayed until the contralateral leg regained support. In contrast, if the contralateral leg *was* weight bearing at the time of the perturbation (Fig. 4*B*), ipsilateral extension into the hole was aborted within as little as 80 ms and quick flexion ensued. Initiation of swing of the contralateral leg generally commenced after the ipsilateral leg had regained ground support beyond the hole, although in a few cases the two hindlimbs flexed together so that the animal hopped to the other side of the hole.

Latencies to initiation of flexion, measured from the time the foot penetrated the plane of the floor until the first flexion response at the ankle as detected in frame-by-frame video replays, progressively shortened on repeated exposure to the hole. Figure 5 shows a representative sequence from one cat in which the response time to initiation of flexion changed in consecutive trials over a 3-day period. In all trials, except for the one circled, the cat walked at relatively constant, slow speeds (0.3–0.4 m/s). At these speeds the contralateral leg remained supported when the ipsilateral foot entered the hole. One data point is well separated from the rest (circled square, day 3). In this trial the cat trotted at a speed of 0.7 m/s. The contralateral leg started its swing phase just at the moment of ipsilateral entry into the hole and consequently flexion was delayed until the ninth

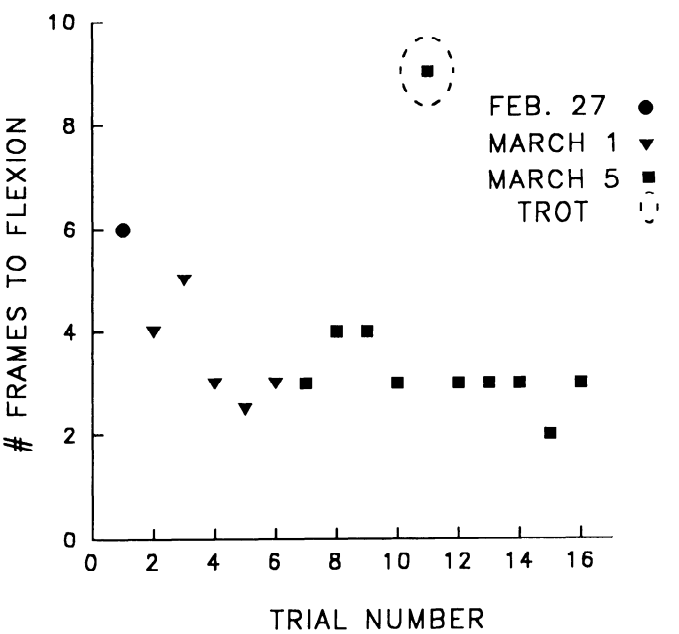

FIG. 5. Speeding up of flexor response in repeated trials. Latency from entry into hole was estimated by counting frames to the onset of flexion. Note that after 3 stimulus presentations the cat's responses had stabilized at ~3 frames (100 ms). The one very long-latency response was a trial in which the contralateral leg had entered swing phase just before the entry of ipsilateral paw into the hole (this is an example of the dependency of the reactions on postural state).

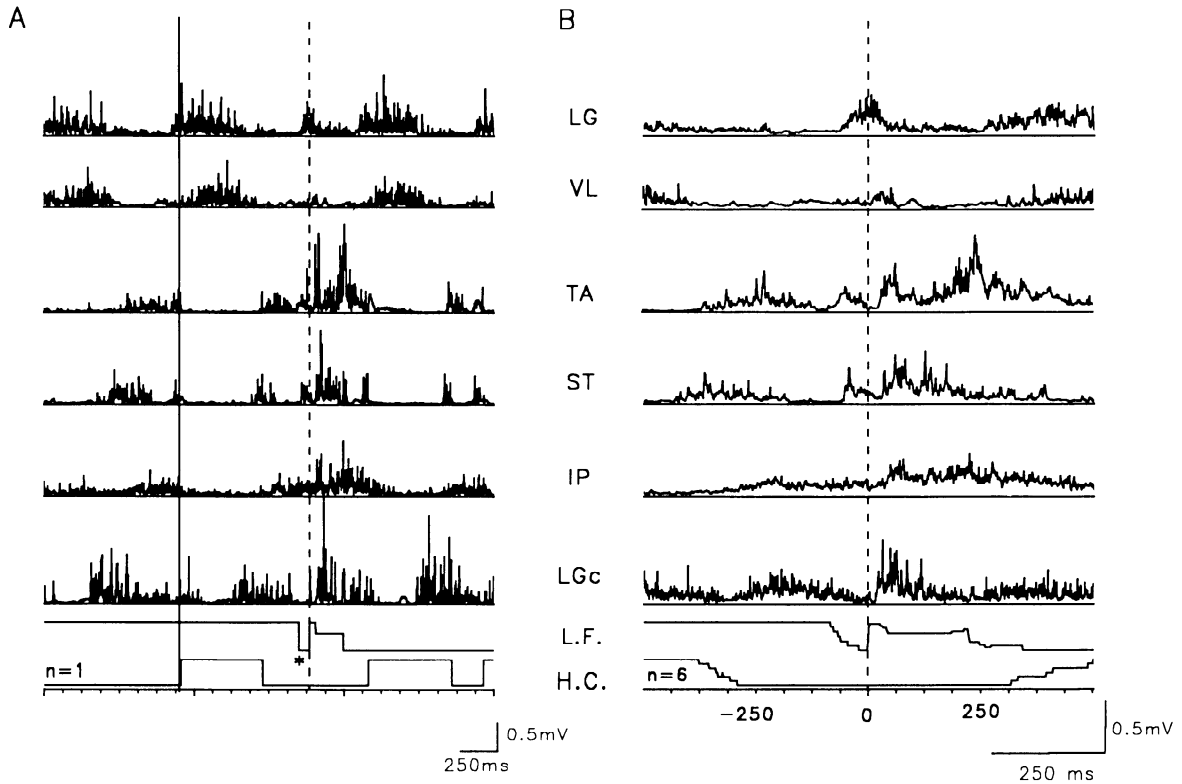

FIG. 6. *A*: LG (ipsilateral and contralateral), vastus lateralis (VL), tibialis anterior (TA), semitendinosus (ST), and iliopsoas (IP) activity recorded in 1 cat during a single foot-in-hole trial. Hindlimb contact (H.C.) is indicated by the solid vertical line and moment of foot entry into hole by the dashed vertical line. Drop in light fence (L.F.) signal (*) marks trap door opening; the subsequent rise in this trace marks foot entry into hole. Ipsilateral LG and VL EMG declined shortly after foot entry, as flexor EMG commenced: TA at 30 ms, ST at 34 ms, IP at 39 ms. Contralateral LG activity increased concomitantly with increased loading of the contralateral hindlimb as the ipsilateral foot entered the hole. *B*: average of 6 foot-in-hole trials recorded during the same session as those from Fig. 6*A* on an expanded time scale.

video frame, corresponding to a latency between 265 and 300 ms.

In one cat we recorded EMG from the ipsilateral hip flexor iliopsoas (IP), the knee flexor semitendinosus (ST), the ankle flexor tibialis anterior (TA), the knee extensor vastus lateralis (VL), and the ipsilateral and contralateral ankle extensor LG. Ipsilateral flexor activity was also recorded in two other cats, TA in one animal and IP in the other. Figure 6*A* shows a single foot-in-hole trial in which the moment of foot contact (solid vertical line) was also recorded for the step before the foot entered the hole. Note that in this step the activity in all three flexor muscles suddenly declined at the moment of foot contact. The dashed vertical line indicates the moment the foot entered the hole. The drop in the light fence signal (*) indicates the moment the trap door opened and the subsequent rise the moment the foot entered the hole. The latency of the corrective flexor response in this animal was very short, 30 ms for TA, 34 ms for ST, and 39 ms for IP, and corresponded to the time that the ipsilateral extensor activity decreased (LG and VL). Support of the contralateral hindlimb was maintained until the ipsilateral foot regained support on the other side of the trap, as evidenced by the abrupt increase in contralateral LG EMG activity as a result of the increased loading on this leg.

Latencies of the corrective flexor response in the other two cats were somewhat longer: IP ~65 ms in one cat, TA ~40 ms in the other. In both cases flexor initiation occurred before the termination of LG EMG activity, as seen in Fig. 6. Similar hip flexor responses are reported in foot-in-hole experiments in intact cats walking on a treadmill in the following paper, with latencies as low as 45 ms (Hiebert et al. 1994). Taken together with the other results above, this indicates that from 30 ms after hole entry onward, corrective responses can occur that are not part of the normal locomotor cycle. It also shows that extensors may remain active during strong corrective flexor responses. Figure 6*B* shows the average of six foot-in-hole trials recorded during the same session as in Fig. 6*A* but on a more expanded time scale. Here the onset of flexor activity after the foot enters the hole is 30 ms for TA, 30 ms for ST, and 40 ms for IP.

Figure 7 shows the kinematic variations that occurred during the foot-in-hole trial of Fig. 6. Each measurement was separated by 33 ms (1 video frame). The time of foot entry is indicated by the asterisk and the dashed vertical line. In this trial, the commencement of flexion out of the hole is just detectable four stick figures later (arrow) (~130 ms after entry). Subsequent knee and ankle flexion were exaggerated compared with the immediately preceding swing phase of the normal step (—— shows maximal flexion in the latter; in the corrective response knee flexion was 56% greater, ankle flexion was 65% greater). The hip did not change its excursion dramatically. It is interesting to note that the corrective flexion response was initiated at a hip extension 20° less than that at the onset of a normal swing (—— shows hip angle at onset of normal swing).

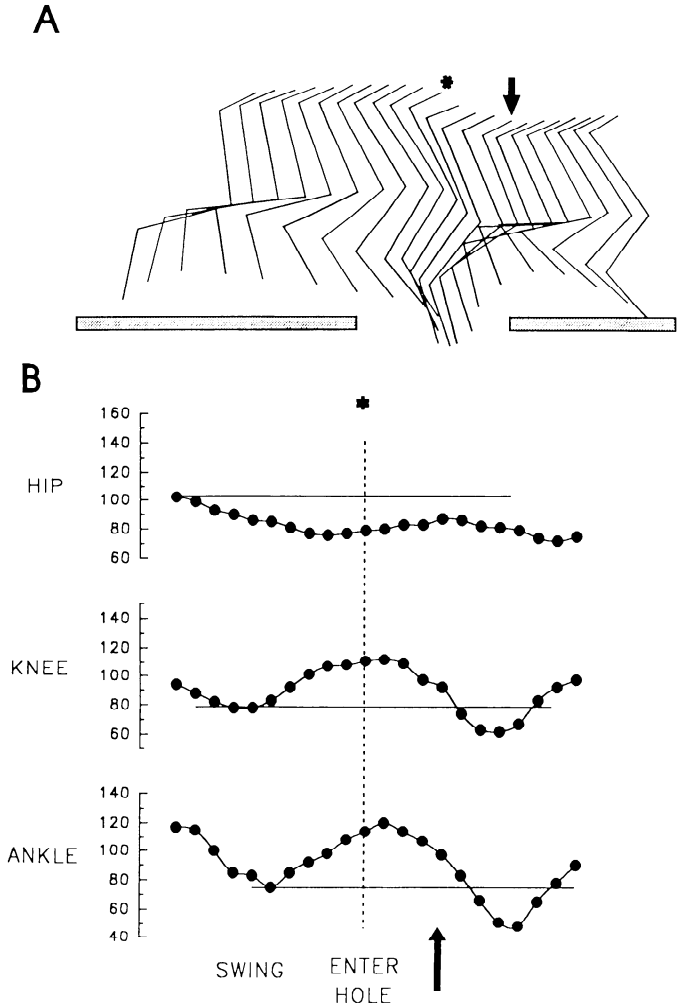

FIG. 7. *A* and *B*: kinematic data from the foot-in-hole trial of Fig. 6. Each measurement is separated by 33 ms (1 video frame). *A*: 1st flexion response (arrow) was detected 4 frames after foot entry (~130 ms). *B*: joint angle changes corresponding to stick figures of Fig. 7*A*. Note exaggerated flexion of knee and ankle compared with immediately preceding swing (: maximal flexion during swing). Hip angle at onset of corrective flexion response was 20° less than that at the onset of swing in preceding normal step.

DISCUSSION

During locomotion, the firing of thousands of proprioceptive and cutaneous afferents suddenly changes when the foot contacts the ground (Prochazka et al. 1989). A sharp transition in EMG activity of various hindlimb muscles occurs 10–25 ms later (Trend 1987). The relative timing of these events led us to posit that they were causally linked (Llewellyn et al. 1989). However, the present results show that in the absence of contact (and therefore of a synchronized modulation of afferent input) the EMG profile of LG was indistinguishable from that in normally supported steps for at least 30–40 ms after contact would have occurred. Thus the hypothesis is not supported.

This was very surprising in view of the vast literature on short-latency reflex actions of many groups of afferent on motoneurons of the muscles we studied (for review see Jankowska 1992). Several explanations are possible. First, reflexes elicited by the multimodal input signaling contact, some being excitatory and some inhibitory, might sum more or less to zero. Second, the afferent "cocktail" generated after the foot enters the hole, although different from that when contact occurs, might have a similar net reflex action [for example, the abrupt stretch of pretibial flexors elicited by the rapid plantar flexion (Fig. 7) would be expected to generate inhibition in LG]. Third, the centrally generated motor pattern might include a sudden reduction in extensor activation and/or presynaptic inhibition just when the afferent volley arrives, rendering motoneurons reflexly inexcitable (Baev and Shimansky 1992; Dubuc et al. 1988). Note that this explanation is not supported by Akazawa et al. (1982), who found *increased* monosynaptic reflex transmission in early stance in decerebrate walking cats. On the other hand, in a *normal* cat, Duenas et al. (1990, Fig. 2*A*) found that monosynaptic reflexes in gastrocnemius were suppressed 20–30 ms after foot contact. Fourth, our expectation of the latency of spinal reflex action might be wrong. Although EMG responses in cat hindlimb muscles can occur within 9–18 ms of very rapid mechanical perturbations (MacPherson et al. 1986; Prochazka et al. 1977; Sinkjaer and Hoffer 1990), the afferent transients elicited by footfall might be less synchronized (Trend 1987). The EMG responses they evoke might therefore have a slower time course, and, because they are superimposed on a rapidly changing and variable EMG signal, they may not be detectable until 30–40 ms after first contact. Furthermore, segmental reflexes in cats have been shown to occur at latencies >40 ms (Ghez and Shinoda 1978; MacPherson et al. 1986; Pearson et al. 1992).

Nonetheless, the results do suggest that the rise and the subsequent sharp decline in LG EMG activity occurring just before and shortly after the moment of foot contact are inherent in the motor program generated by the central locomotor pattern generator. This being the case, a similar time course should be apparent in records of LG activity from previous studies of locomotion in the absence of afferent input. In reviewing the literature we found it hard to make direct comparisons because the exact moments of foot contact were either not documented or were totally lacking (in fictive locomotion studies) and cycles were not averaged. In some preparations the extensor activation profiles in early stance seemed to conform well with those described in our study (e.g., Grillner and Zangger 1984: decerebrate, deafferented cats; Giuliani and Smith 1987: chronic spinal, deafferented cats). In others, particularly in deafferented or fictive spinal preparations, the profiles either rose more slowly or more rapidly than in normal cats (e.g., Grillner and Zangger 1979; Pearson and Rossignol 1991). Nevertheless, because normal-looking patterns were seen in some of the deafferented preparations we conclude that the CNS does seem capable of generating the basic profile for at least the first 100 ms or so of the extensor burst.

Although the motor pattern in the first 30–40 ms after contact seems to be centrally preprogrammed, this is not to say that afferent input is unimportant in shaping the locomotor pattern. In particular the afferent input activated by extensor loading is almost certain to contribute reflexly to the overall EMG activity during the remaining stance phase. Both muscle spindle (Yang et al. 1991) and tendon organ (Pearson et al. 1993) afferents have been implicated. A small contribution might also come from cutaneous af-

ferent input, because local anesthesia of the foot pads results in a small reduction of LG EMG amplitude in this part of stance (Trend 1987). Our results do not allow us to estimate such contributions because in the foot-in-hole trials a major corrective activation of flexors occurred as early as 30 ms after foot entry (Fig. 6 and Hiebert et al. 1994). Thus any deviations in extensor EMG from that in normally supported steps (e.g., Figs. 2 and 3) may have been due to the corrective response rather than a withdrawal of reflex support. It could even be argued that all foot-in-hole trials involved a corrective response starting some 30–40 ms after hole entry, but that this response often mimicked the activation profile in supported steps and so was not recognizable as a separate motor program (e.g., Fig. 4*A*).

Is the corrective flexion response to a loss of ground support a segmental reflex, a preprogrammed response comparable to the postural reflexes described by Dufossé et al. (1985), MacPherson et al. (1986), and Rushmer et al. (1987), or a voluntary response? In previous recordings of the spinal locomotor rhythm under isometric conditions the latency from mechanical unloading of extensor muscles to onset of flexor EMG was between 100 and 300 ms (mean 200 ms: Pearson et al. 1993). This is much slower than the quickest responses we observed in the IP muscle of normal cat in foot-in-hole trials (39 ms: Fig. 6*A*). In unrestricted spinal locomotion minimal latencies in IP were 120 ms (Hiebert et al. 1994), which is still 80–90 ms short of the quickest responses in normal animals. The difference seems attributable to expectancy of the stimulus in the intact cat. We noticed that the latency decreased in consecutive trials and cats began avoiding the trap door by stepping over it. After many trials we would see the most rapid reactions (e.g., flexor EMG bursts within 30 ms). In principle the speeding up of responses could be due to spinal learning (Wolpaw and Herchenroder 1990), expectancy-related "priming" of segmental circuitry, or a switch from segmental reflexes to long-loop, voluntary reactions. There are numerous examples in the literature of reflexlike responses speeding up or otherwise changing as a consequence of attention arousal or expectancy (for review see Prochazka 1989). Some of the best-known examples are those described by Nashner et al. (1976, 1979), who found that the postural responses of human subjects to perturbations of stance could adapt dramatically over a few stimulus presentations, speeding up the restoration of balance (see also Dietz et al. 1991).

In the foot-in-hole reactions in the present study it could be that the lack of expected skin input was detected at supraspinal levels and led to voluntary intervention. This follows from studies on the stumbling corrective reaction, where anesthesia of the skin led to a remarkable change in the overall response: the obstacle impeding forward swing could push the limb into extreme extension before the corrective reaction was finally initiated (Prochazka et al. 1978). Short-latency, proprioceptive stretch reflexes were preserved, but they were relatively ineffective in triggering a voluntary response. The role of skin input in triggering corrective flexion responses could be tested by anesthetizing the footpads in foot-in-hole trials.

The posture of the animal at the time of the perturbation was also an important determinant of the foot-in-hole response. Rapid flexion out of the hole was observed only if the contralateral hindlimb was bearing weight (i.e., conditional "IF-THEN" control: Prochazka 1993). During faster locomotion, when neither hindlimb was in ground contact at the time of the perturbation, the ipsilateral limb usually extended deep into the hole, as though searching for support. This makes functional sense because in the animal's normal environment support would often be regained in this way. Concomitantly, the contralateral limb is rapidly flexed forward and placed. There are well-established precedents for posture- or phase dependence of spinal reflexes (e.g., the phase dependence of responses to perturbations of the step cycle of the spinal cat (Forssberg et al. 1975, 1979). The issue of the interaction between limbs in foot-in-hole responses in spinal animals is addressed by Hiebert et al. (1994).

In conclusion, our results show that the large transients in afferent input signaling foot contact are surprisingly ineffective in shaping extensor activation immediately after the onset of stance. This activation appears to be predominantly centrally programmed. However, as the stance phase evolves afferent input may play an increasingly important role. Expectancy plays a big part in speeding up the corrective response that lifts the foot from the hole, suggesting a supraspinal mediation of sensory input. Finally, the corrective response is heavily dependent on whether the contralateral limb is in stance or swing phase, indicating state dependence or conditionality in the choice of motor program.

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