Stumbling Corrective Reaction During Fictive Locomotion in the Cat

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Quevedo, Jorge, Katinka Stecina, Simon Gosgnach, and David A. McCrea. Stumbling corrective reaction during fictive locomotion in the cat. J Neurophysiol 94: 2045–2052, 2005. First published May 25, 2005; doi:10.1152/jn.00175.2005. An obstacle contacting the dorsal surface of a cat’s hind foot during the swing phase of locomotion evokes a reflex (the stumbling corrective reaction) that lifts the foot and extends the ankle to avoid falling. We show that the same sequence of ipsilateral hindlimb motoneuron activity can be evoked in decerebrate cats during fictive locomotion. As recorded in the peripheral nerves, twice threshold intensity stimulation of the cutaneous superficial peroneal (SP) nerve during the flexion phase produced a very brief excitation of ankle flexors (e.g., tibialis anterior and peroneus longus) that was followed by an inhibition for the duration of the stimulus train (10–25 shocks, 200 Hz). Extensor digitorum longus was always, and hip flexor (sartorius) activity was sometimes, inhibited during SP stimulation. At the same time, knee flexor and the normally quiescent ankle extensor motoneurons were recruited (mean latencies 4 and 16 ms) with SP stimulation during fictive stumbling correction. After the stimulus train, ankle extensor activity fell silent, and there was an excitation of hip, knee, and ankle flexors. The ongoing flexion phase was often prolonged. Hip extensors were also recruited in some fictive stumbling trials. Only the SP nerve was effective in evoking stumbling correction. Delivered during extension, SP stimulus trains increased ongoing extensor motoneuron activity as well as increasing ipsilateral hip, knee, and ankle hindlimb flexor activity in the subsequent step cycle. The fictive stumbling corrective reflex seems functionally similar to that evoked in intact, awake animals and involves a fixed pattern of short-latency reflexes as well as actions evoked through the lumbar circuitry responsible for the generation of rhythmic alternating locomotion.

INTRODUCTION

When the dorsum of a cat’s hind paw strikes an obstacle during forward walking, the paw is lifted in an attempt to clear the obstacle and avoid tripping. This is the stumbling corrective reaction (or reflex), evoked during the early swing phase of locomotion in intact (Buford and Smith 1993; Forssberg 1979; Prochazka et al. 1978; Wand et al. 1980) and decerebrate (Forssberg 1979) cats. Its occurrence in cats with chronic, lower thoracic, spinal lesions (Forssberg 1979; Forssberg et al. 1977) indicates that the stumbling reflex can be organized at the level of the lumbar spinal cord. An analogous response exists in adult humans (Eng et al. 1994; Schillings et al. 1996; Van Wezel et al. 1997; Zehr et al. 1997) and infants (Lam et al. 2003).

In the cat, the stumbling corrective reflex evoked during swing activates knee flexors, ankle extensors (Buford and Smith 1993; Forssberg 1979; Forssberg et al. 1977; Prochazka et al. 1978; Wand et al. 1980), and hip flexors (Pratt et al. 1996). In the ankle flexor tibialis anterior (TA), there is an initial brief excitation followed by a period of inhibition and finally a prolonged excitation (Buford and Smith 1993; Prochazka et al. 1978; Wand et al. 1980). Kinematically, knee flexion and initial ankle extension is followed by ankle flexion that raises the foot to clear the obstacle. The same sensory stimulation delivered during stance often increases ipsilateral extensor activity (Buford and Smith 1993; Forssberg et al. 1979). The increased extension facilitates propulsion (Forssberg 1979; Forssberg et al. 1977), and the pronounced flexion lifts the foot in anticipation of the obstruction during the next swing phase in this stumbling preventive reaction (Forssberg 1979).

The neuronal networks underlying the stumbling corrective and preventive reactions have not been identified. It is known that cutaneous afferents are important for evoking the reflex because stumbling correction is abolished by anesthesia of the skin of the foot (Forssberg et al. 1977; Wand et al. 1980) and can be evoked by puffs of air on the paw dorsum (Forssberg 1979). On the other hand, electrical stimulation of the skin has been reported to evoke some (Forssberg 1979) but not all (Buford and Smith 1993; Wand et al. 1980) features of stumbling correction, suggesting that participation of other sensory afferents is required to evoke full pattern of stumbling correction (discussed in Buford and Smith 1993).

These experiments began during a survey of the effects of stimulation of hindlimb peripheral nerves during fictive locomotion in decerebrate cats. It was noted that when the cutaneous nerve innervating the dorsum of the foot [superficial peroneal (SP)] is stimulated during the flexion phase, hip and knee flexor activity increased, and a brief period of ankle extensor activity was produced. This study attempts to determine the extent to which these actions resemble the stumbling corrective reaction described by others during real locomotion. This paper reports the patterns of motoneuron activity recorded in the peripheral nerves after stimulation of the SP nerve during fictive locomotion. The effects of SP nerve stimulation recorded intracellularly in hindlimb motoneurons are presented in the companion paper (Quevedo et al. 2005). Preliminary results have been presented (McCrea 2002; McCrea et al. 1998).
METHODS

Observations were made in 28 purpose-bred cats (2.1–4.5 kg) following guidelines set by the Canadian Council on Animal Care and the University of Manitoba. Surgical procedures, preparations of hindlimb nerves for recording, and stimulation and brain stem stimulation were as described in Quevedo et al. 2000. Using halothane anesthesia delivered in a mixture of 70% NO₂ and 30% O₂, left hindlimb nerves were prepared for recording and stimulation. Remaining sciatic, obturator, and femoral nerve branches were cut in both hindlimbs, and tendons around the hip were sectioned bilaterally. A laminectomy exposed the L₅–L₇ segments of the spinal cord for intracellular recording from lumbar motoneurons. Those results are presented in the companion paper (Quevedo et al. 2005). The cat was transferred to a stereotaxic frame, and a precollicular–postmamillary decerebration was performed in which both cortices and all tissue rostral to the transection were removed. Anesthesia was discontinued, and the cat was paralyzed with gallamine triethiodide or pancuronium bromide and artificially ventilated.

Ipsilateral hindlimb nerves placed on bipolar electrodes for either stimulation or recording included combinations of posterior biceps (PB), semitendinosus (St), semimembranosus and anterior biceps (SmAB), medial gastrocnemius (MG), lateral gastrocnemius and soleus (LGS), plantaris (Plant), flexor digitorum longus (FDL), flexor hallucis longus (FHL), tibial (Tib), peroneus longus (PerL), TA, peroneous tertius and brevis, extensor digitorum longus (EDL), and SP nerves. Vasti, rectus femoris (separately or together as quadriceps, Q), and sartorius (Sart) were mounted in ventral cuff electrodes. In one experiment, Vas and rectus femoris were mounted together (Q), and the branches to medial and lateral sartorius (mSart and lSart) were separated. Branches of the psosas (Psosas) muscle nerve were dissected and mounted in two experiments. The adductor tendons of both hips were cut, and the right hindlimb was denervated by cutting the sciatic nerve and the nerves to SmAB and PBSt. In accord with the strong knee flexion that occurs during stumbling correction with the activity of PB and St (e.g., Pratt et al. 1996), these bifunctional (knee flexor, hip extensor) muscles will be referred to simply as knee flexors.

Fictive locomotion was elicited by unilateral or bilateral electrical stimulation of the mesencephalic locomotor region (MLR; 30–200 μA, 0.5- or 1.0-ms pulses, 15–20 Hz; see Guertin et al. 1995). Peripheral nerves were stimulated with 0.2-ms constant current stimuli pulses with intensities expressed in multiples of threshold (T) for the most excitable fibers as measured at the cord dorsum in L₆ or L₇. Trains of stimuli (9–45 shocks, typically 15; 200 Hz) were delivered to several cutaneous and muscle afferent nerves in each experiment to evoke stumbling correction and other reflexes during fictive locomotion. Rectified and integrated electroneurogram (ENG) activity was used to monitor fictive locomotor activity, and by means of a software-based window discriminator, to trigger stimulus delivery from a selected ENG at a particular phase of the step cycle. Trains were delivered every two or three cycles with intervening step cycles serving as control step cycles. Window discriminator trigger pulses were collected and used to construct off-line averages of ENG activity in steps with and without peripheral nerve stimulation. Latencies of changes in ENG amplitudes were measured from the arrival of the afferent volley at the spinal cord. The duration of the step cycle was defined as the time interval between the onset of consecutive burst activity in a selected nerve. Cycle durations were compared with a t-test (2 samples assuming equal variances, α = 0.05). Rectified and integrated ENGs were digitized (500 Hz) and analyzed using software developed by the Winnipeg Spinal Cord Research Centre (Pentium PCs running Linux).

The effects of SP nerve stimulation were examined in 28 experiments in which brain stem–evoked fictive locomotion was produced. Once fictive locomotion was established, the effects of SP nerve stimulation were observed on ipsilateral hindlimb ENG activity. In those preparations displaying all features of stumbling correction (n = 14), the effects of SP nerve stimulation were examined further during multiple runs of fictive locomotion (typically of 2-min durations). These results are reported here and in the companion paper (Quevedo et al. 2005). Preparations not displaying fictive stumbling correction and some with stumbling correction were also used for other purposes (McCrea et al. 2000; Quevedo et al. 2000; unpublished observations).

RESULTS

Stumbling correction: stimulation of the SP nerve during flexion

Figure 1 shows rectified-integrated ENG recordings from selected hindlimb muscle nerves during MLR-induced fictive locomotion. Activity in a hip flexor ENG (Sart) was used to trigger a train of stimuli to the SP nerve every other step cycle with a delay of about 250 ms from the onset of flexor motoneuron activity. SP nerve stimulation produced a large burst of PB (knee flexor, hip extensor) and ankle extensor (LGS) activity and a delayed excitation of ankle flexors (TA). These bursts were bigger than those occurring during unperturbed step cycles. Averaged ENG activity (n = 5) occurring with (solid traces) and without SP stimulation (dotted) during a different run in the same experiment is shown in Fig. 2. Shortly after the beginning of the stimulus train there is 1) an increase in knee flexor (PB, St) ENG activity, 2) a burst of ankle (LGS) extensor activity, and 3) an inhibition of TA and PerL (ankle flexor) activity, followed by 4) ankle flexor excitation. Furthermore, there was 5) an increase in hip flexor (Sart) activity. The increased knee flexor activity, the excitation of ankle extensor motoneurons, and the inhibition followed by excitation of
ankle flexor activity are characteristics of real stumbling correction (see Fig. 1 of Prochazka et al. 1978; Fig. 3 of Wand et al. 1980) as is the evoked activity in Sart (Pratt et al. 1996). The pattern of ENG activity in Fig. 1 (also Figs. 2, 3A, 4A, and 6A) shows the features of what we will call the fictive stumbling corrective reaction. These features were produced in all 14 preparations reported here.

Knee flexor ENG activity was always increased by SP stimulation during fictive stumbling correction and when recorded separately in both the PB and St motoneuron pools (Fig. 2). In some cases, posterior biceps-semimembranosus (PBSt) activity was absent or minimal during control steps with no stimulation but was recruited with SP stimulation (Figs. 2 and 4A). Ankle flexor activity during the fictive stumbling corrective reflex always displayed a period of inhibition. In the case of TA and PerL motoneuron pools, this was often preceded by a very brief excitation (Figs. 1, 2, 4A, and 6A) that usually (9/12 cases) is absent in EDL (compare TA and EDL records; Figs. 2 and 4A). When EDL motoneurons were strongly activated at the time of SP stimulation, the inhibition of the EDL ENG was usually complete (Fig. 4A). Similar to records obtained during real locomotion (Trank et al. 1996), EDL activity is sometimes delayed from that of TA during fictive locomotion. In those cases, the degree of SP-evoked inhibition in EDL was difficult to assess (Fig. 2).

Figure 3 shows averaged ENG activity obtained with (continuous lines) and without (dotted lines) SP stimulation delivered during the flexion phase of fictive locomotion. In Fig. 3A, the longer stimulus train evoked excitation in the ankle plantar’s (Plant) and hip [semimembranosus and anterior biceps (SmAB)] extensors along with inhibition of hip (Sart) and ankle (TA) flexors. Step cycle period was 1,475 ± 147 ms in control steps (n = 15), increasing to 1,866 ± 151 ms with SP nerve stimulation (n = 5; P < 0.01). B: The shorter duration stimulus train did not change step cycle period (P > 0.05) and did not result in recruitment of ankle extensor motoneurons.

6A) shows the features of what we will call the fictive stumbling corrective reaction. These features were produced in all 14 preparations reported here.

Knee flexor ENG activity was always increased by SP stimulation during fictive stumbling correction and when recorded separately in both the PB and St motoneuron pools (Fig. 2). In some cases, posterior biceps-semimembranosus (PBSt) activity was absent or minimal during control steps with no stimulation but was recruited with SP stimulation (Figs. 2 and 4A). Ankle flexor activity during the fictive stumbling corrective reflex always displayed a period of inhibition. In the case of TA and PerL motoneuron pools, this was often preceded by a very brief excitation (Figs. 1, 2, 4A, and 6A) that usually (9/12 cases) is absent in EDL (compare TA and EDL records; Figs. 2 and 4A). When EDL motoneurons were strongly activated at the time of SP stimulation, the inhibition of the EDL ENG was usually complete (Fig. 4A). Similar to records obtained during real locomotion (Trank et al. 1996), EDL activity is sometimes delayed from that of TA during fictive locomotion. In those cases, the degree of SP-evoked inhibition in EDL was difficult to assess (Fig. 2).

Figure 3 shows averaged ENG activity obtained with (continuous lines) and without (dotted lines) SP stimulation delivered during the flexion phase of fictive locomotion. In Fig. 3A, there is an excitation...
of knee flexors and ankle extensors characteristic of fictive stumbling. The most common response in Sart (11/14 experiments) was an increase in activity soon after the onset of SP stimulation (Fig. 2). A similar response was observed in Psoas for the two times it was studied. The other response in the Sart motor pool (3 experiments) was an initial inhibition during SP stimulation that was followed by a large increase in activity (Fig. 3A). The records in Fig. 3B from the same preparation show that reducing the number of shocks in the stimulus train decreased the duration of the inhibition of ankle and hip flexor motoneurons. An excitation of hip and ankle flexors that follows or persists after the termination of SP stimulation is shown in Figs. 2, 3, A and B, 4A, and 6A.

Ankle extensor excitation was evoked by SP stimulation during flexion in all 14 preparations. This evoked activity was often larger than that occurring during the extensor phase of the fictive locomotor step cycle (e.g., Figs. 1, 2, and 4A). Unlike the activation of limb flexors, ankle extensor activity did not persist after the end of the stimulus train to the SP nerve. When recorded separately, excitation during stumbling correction was seen in the Plant, MG, and LGS nerves; the branches to lateral gastrocnemius and soleus were not separated.

Recruitment of other extensors during fictive stumbling correction was variable. In the hip extensors, SmAB, small bursts were evoked with SP stimulation during flexion in 2/14 experiments (Fig. 3A) and very small (almost undetectable) bursts (Fig. 4A) in 7 experiments. In one experiment, both rectus femoris and vasti motoneurons were recruited during stumbling correction (data not shown). In two experiments, no activity was evoked in the combined Q nerve (Fig. 4). FDL activity was often similar to that of TA and PerL in that there was usually a brief period of excitation followed by inhibition (Fig. 2). As stimulation of the SP nerve during flexion produced no effect on the FHL ENG (e.g., Fig. 2), it is assumed that the effects evoked on the flexor digitorum and hallucis longus (FDHL) nerve in Fig. 4 are caused by a recruitment of FDL motoneurons by SP stimulation (Degtyarenko et al. 1998). In some experiments, the response in FDHL was weak, and the period of inhibition was missing.

The latencies of the increases and decreases in ENG activity occurring during fictive stumbling correction are shown on the left of the respective traces in Fig. 2. Average latencies were calculated from runs in eight experiments in which all features of the stumbling reflex were observed and without an initial inhibition of Sart activity. The latency for the increase in both hip and knee flexor ENG activities was 4 ±1 (SD) ms (n = 8). The latency of ankle extensor activation (16 ± 2 ms) was always greater than that of the flexor motoneurons acting at the hip or knee. The brief increase of ankle flexor activity that often preceded the prominent inhibition had an average latency of 5 ± 1 ms in these eight experiments. As mentioned, the period of inhibition of ankle flexors was variable (e.g., Fig. 3) but often similar to the duration of the stimulus train (Figs. 2 and 4A). The large increase in ankle flexor activity occurred soon after the termination of SP stimulation. The evoked activity in SmAB in Fig. 3A had a latency of ~4 ms.

**Stumbling preventive reaction: SP nerve stimulation during extension**

Figure 4B shows the effects of SP nerve stimulation delivered during the extension phase in one of the two experiments where responses were compared with those evoked during flexion (Fig. 4A). During extension (Fig. 4B), SP stimulation evoked short-latency increases in hip (SmAB), knee (Q), and ankle (GS) extensor activity as well as in the combined FDHL nerve. These bursts were also larger than those occurring during control steps. Activity was also evoked in PBSt but was of shorter duration than that occurring during stumbling correction (Fig. 4A) and shorter than the stimulus train duration. The latency of SP-evoked GS activation during extension was 4 ms. Large, short-latency activity was evoked in FDHL in both the flexion and extension phases of fictive locomotion.

A particularly striking feature of SP stimulation during extension was, however, its effects on the amplitude of hip (Sart), knee (PBSt), and ankle (TA, EDL) ENGs in the subsequent step cycle. These bursts (Fig. 4B, stars) were considerably larger than those occurring without SP nerve stimulation. This pattern of motoneuron activity observed during fictive locomotion resembles that occurring during the stumbling preventive reaction in chronic spinal (Forsberg 1979; Forsberg et al. 1975) and intact (Buford and Smith 1993) cats. Thus stimulation of the SP nerve during fictive locomotion in cerebrate cats evokes ipsilateral hindlimb motoneuron activity much like that occurring during both the stumbling corrective and preventive reflexes observed during real walking.

**Effects of SP stimulation on step cycle timing**

The overlaid averaged records in Fig. 2 show an example of fictive stumbling correction in which there was little effect on either the duration of flexor activity or the time of onset of the subsequent extension phase. In this case, the increase in hip and knee flexor motoneuron activities evoked by SP stimulation during flexion was not accompanied by changes in step cycle timing. In the example shown in Fig. 3A, however, SP stimulation increased the duration of ongoing flexor activity, delayed the subsequent extensor phase, and hence delayed the onset of the next step cycle (compare solid and dotted traces). Figure 3B shows the effects of decreasing the duration of SP stimulation from 105 (21 shocks) to 50 ms (11 shocks). The shorter duration train did not affect the onset time of the next step cycle (see TA and Sart records) and did not evoke the excitation of ankle (and in this case hip) extensors characteristic of stumbling correction. Both stimulus trains increased the amplitude of hip, knee, and ankle flexor activity. Figure 4A shows another example in which cycle duration was prolonged by SP stimulation during flexion. In this case, the stimulus train duration was relatively long with respect to the short step cycle period. Because the primary goal of this study was to mimic an obstacle striking the foot briefly during swing, the effects of altering the duration of SP stimulation on the timing of the step cycle were not systematically explored. It is clear, however, that SP stimulation can affect the timing of the step cycle. Presumably this is achieved by excitatory actions evoked through the flexor portion of the central pattern generating networks (Quevedo et al. 2005).
In 14 of 28 preparations tested, SP stimulation during the flexion phase failed to evoke all features characteristic of stumbling correction in any trial. In three preparations in which other features of stumbling correction were present, ankle extensor excitation recorded in the peripheral nerves was absent. In five animals, SP stimulation prolonged ongoing flexor activity and delayed the onset of extensor activity. In these cases, this resetting of the timing of the step cycle was not accompanied by an inhibition of ankle flexor activity or excitation of ankle extensors. In five preparations, SP stimulation terminated ongoing flexion and initiated a premature extensor phase, i.e., reset the locomotor cycle to extension. Although not systematically explored, when the stimulation of the SP nerve was delivered during late flexion, the effects were more likely to be resetting to extension. In one experiment in which stumbling correction could not be evoked, SP stimulation was about equally effective in prolonging flexion or resetting to extension in the same run of fictive locomotion.

Which afferents evoke the stumbling corrective reflex?

In this series of experiments, 2-T intensities of SP stimulation using trains typically of 15 shocks (200 Hz) were used to evoke the stumbling corrective reflex. As evident from the short latencies, the excitation of knee and sometimes hip flexors occurred with the first shock in the train. While systematic efforts were not made to determine minimum stimulus intensities or the effects of higher intensity stimulation, in one experiment, stimulation at 1.6 T evoked stumbling correction. This indicates that the large diameter cutaneous afferents in the SP nerve are sufficient for evoking these reflexes during fictive locomotion in denervated and paralyzed cats. To determine if other afferents from around the foot might contribute to stumbling correction, the effects of stimulation of several other nerves during the flexion phase of fictive locomotion were examined.

Figure 6, A–F, compares the effects of stimulation during flexion of the SP nerve, the nerves from the ankle flexors shown in Fig. 5A, the SP nerve was stimulated twice (15 shocks, 200 Hz, 2 T) during the flexion phase of fictive locomotion. The first stimulus presentation resulted in a rapid termination of flexion (note the truncated durations of bursts in the Sart and EDL ENGs) and the onset of extensor (LGS) activity. This is a resetting to, or a premature initiation of, extension (see also Fig. 6, D–F). The next SP stimulus train resulted in essentially the opposite effect. There was a large increase in the amplitude and duration of flexor (Sart and EDL) activity. Variable effects of SP stimulation in this experiment were also produced at higher stimulus intensity (5 T). This is shown in Fig. 5B, where the first SP stimulation shortens the ongoing flexion phase, whereas the second increases the amplitude and duration of flexor activity. In other trials, 5-T SP stimulation evoked no extensor activity but prolonged the flexion phase (data not shown). Thus in this experiment, SP stimulation produced seemingly at random, stumbling correction, a resetting to extension, or a flexion phase prolongation (Stecina et al. 2005).

The segment shown in Fig. 5A, the SP nerve was stimulated twice (15 shocks, 200 Hz, 2 T) during the flexion phase of fictive locomotion. Rectified and integrated ENGs from hip (Sart), knee (Ph, St), and ankle (EDL and LGS) muscle nerves during MLR-evoked fictive locomotion. The SP nerve was stimulated with trains of 15 shocks at 200 Hz during the flexion phase. A: SP stimulation on the left cuts short ongoing flexion (see Sart and EDL ENGs) and evokes a premature onset of extensor (LGS) activity. There is a large but brief excitation of the knee flexor, St. The other SP stimulus presentation prolongs ongoing flexion in Sart and EDL (after a brief period of inhibition) and delays the onset of the subsequent extensor phase. During this flexor phase prolongation, there is a prolonged excitation of St motoneurons. B: similar variations in responses are seen with increased intensity of SP stimulation (5 T) during another run from the same experiment. The 1st stimulus presentation (SP 5 T) resets to extension and the 2nd prolongs the flexion phase. Note the small bursts of activity evoked in the LGS nerve during flexion.

In this series of experiments, 2-T intensities of SP stimulation during the flexion phase of fictive locomotion were sufficient for evoking these reflexes during fictive locomotion in denervated and paralyzed cats. To determine if other afferents from around the foot might contribute to stumbling correction, the effects of stimulation of several other nerves during the flexion phase of fictive locomotion were examined.

FIG. 5. Spontaneous reversals of effects of SP nerve stimulation during the flexion phase of fictive locomotion. Rectified and integrated ENGs from hip (Sart), knee (Ph, St), and ankle (EDL and LGS) muscle nerves during MLR-evoked fictive locomotion. The SP nerve was stimulated with trains of 15 shocks at 200 Hz during the flexion phase. A: SP stimulation on the left cuts short ongoing flexion (see Sart and EDL ENGs) and evokes a premature onset of extensor (LGS) activity. There is a large but brief excitation of the knee flexor, St. The other SP stimulus presentation prolongs ongoing flexion in Sart and EDL (after a brief period of inhibition) and delays the onset of the subsequent extensor phase. During this flexor phase prolongation, there is a prolonged excitation of St motoneurons. B: similar variations in responses are seen with increased intensity of SP stimulation (5 T) during another run from the same experiment. The 1st stimulus presentation (SP 5 T) resets to extension and the 2nd prolongs the flexion phase. Note the small bursts of activity evoked in the LGS nerve during flexion.

FIG. 6. Only SP afferents and not other cutaneous or muscle afferents from the foot evoke the stumbling corrective response. Averages of rectified and integrated ENGs from Sart, TA, LGs, St, and SmAB nerves during fictive locomotion. Average of control step cycles are indicated by dotted lines. A–F show data from 2 different preparations, respectively. The number of step cycles averaged is 15, 17, 39, 5, 3, and 24 for A–F, respectively. All nerves were stimulated with trains of shocks at 200 Hz delivered during flexion. A: stimulation of the SP nerve during flexion with 25 shocks produced a short-latency increase of Sart, St, and TA, followed by an increase of LGS and a concurrent decreased of TA ENG activities. B and C: stimulation of the EDL and PerL nerves with 25 shocks of 5 T produced an increase of ongoing flexor activity in Sart and TA nerves. D–F: stimulation of the TA (5 T), MG (5 T), and Tib (2 T) nerves with 25, 25, and 15 shocks, respectively, terminated ongoing flexion (Sart and TA) and advanced the onset of extension, i.e., reset to extension.
(EDL, PerL, TA), and an ankle extensor (MG) nerve. All panels except F were obtained in the same experiment. Stimulation of EDL or PerL at 5 T (an intensity sufficient to recruit group II afferents) increased hip and ankle flexor activity when either EDL or PerL nerves were stimulated (Fig. 6, B and C) (Stecina et al. 2005). The small, flat response in the LGS nerve in Fig. 6, B–D, is stimulus artifact made evident by the on-line rectification and integration.

Stimulation of the ankle flexor nerve, TA (Fig. 6D), cuts short ongoing Sart activity and produces prominent excitation of both ankle (LGS) and hip extensors (SmAB) before Sart activity occurs again. This is a resetting of the step cycle to extension (Perreault et al. 1995). A similar termination of ongoing flexion and initiation of a premature extensor phase is also seen after stimulation of an ankle extensor nerve (Fig. 5E, MG) or, in another experiment, of the mixed muscle and cutaneous plantar foot nerve (Fig. 6F; Tib) (Guertin et al. 1995). These results show that, while activation of EDL and PerL group II afferents might contribute to hip flexion, only stimulation of SP afferents is able to evoke the full pattern of stumbling correction.

Including the example shown, the stumbling correction evoked by SP stimulation was compared to the effects of TA stimulation in four experiments, EDL in five experiments, Tib in two experiments, and to stimulation of ankle extensors in three experiments. In all cases, only SP stimulation evoked stumbling correction. The effects of the other nerves were in keeping with the repertoire of effects shown in Fig. 6. In conclusion, in these preparations, only the cutaneous SP nerve (Fig. 6A) evoked ankle extensor activity and briefly silenced ankle flexor (TA) activity while exciting hip and knee flexors, i.e., evoked the stumbling corrective reflex.

**Discussion**

The main result of this study is that the pattern of motoneuron excitation and inhibition occurring during real stumbling corrective and preventive reactions can be evoked during fictive locomotion by brief trains of stimuli to the SP nerve. The sequence of excitation of knee flexors, initial inhibition of ankle flexors and the delayed excitation of ankle extensors is strikingly similar to that described in intact (Buford and Smith 1993; Forssberg 1979; Prochazka et al. 1978; Wand et al. 1980) and in chronic spinal cats (Forssberg 1979; Forssberg et al. 1980) during real stumbling correction.

Functionally, stumbling correction during swing consists of knee flexion coupled with activation of ankle extensors and an initial inhibition of ankle flexors that assists in removing the paw from further contact with the obstacle. This is followed by a period of ankle flexion. During the swing phase of locomotion in adult cats, the latencies of knee flexor (Kt) activation during stumbling correction are 9.2–9.7 ms (Forssberg 1979) when evoked with electrical stimulation and 10 ms when evoked with mechanical stimulation (Forssberg 1979). Those latencies measured in the electromyogram include the afferent conduction times from the distal SP nerve and the efferent conduction to PBSt (3 + 2 ms) plus a delay at the neuromuscular junction (1 ms) for an estimated 6-ms delay. In these experiments, latencies were measured from the cord dorsum (no afferent delay) to the response in the ENG (efferent conduction time but no neuromuscular delay). Adding a 4-ms correction to these data to the 4-ms latency of increased PBSt ENG activity during fictive stumbling correction gives a value (8 ms) comparable with that reported during real stumbling correction (Forssberg 1979). Similarly, the mean 20-ms (16 + 4 ms correction) increase in ankle extensor ENG activity found here compares favorably with the mean 25-ms latency of ankle extensor activity using mechanical stimulation (Prochazka et al. 1978; Wand et al. 1980).

Stimulation of the SP nerve during the extension phase of fictive locomotion also produced actions similar to those occurring during real locomotion. This includes the increase in ongoing extensor muscle activity and the increase in flexor activity in the subsequent step cycle (Fig. 4B). Thus the effects of SP nerve stimulation in decerebrate cats during fictive locomotion occur with a pattern and at latencies similar to those reported in awake animals during real stumbling corrective and preventive reactions.

Some of the variations in the reflex pattern reported during real stumbling correction were also encountered during fictive stumbling. For example, hip extensor EMG activity is often but not always a feature of the real stumbling reaction (Buford and Smith 1993). While strong SmAB activation was not a feature of fictive stumbling, activity was sometimes evoked in SmAB (Fig. 3A). The duration of flexor activity during the swing phase may be prolonged in real (Buford and Smith 1993) and fictive stumbling correction. Excitation of hip flexors (Sart and Psoas) is a prominent feature of fictive stumbling correction even when preceded by inhibition (Fig. 3). Kinematic analysis shows that there can be an increased flexion at the hip during real stumbling (Forssberg et al. 1977; Pratt et al. 1996; Wand et al. 1980). On the other hand, the powerful activation of the bifunctional PBSt (hip extensor, knee flexor) and the hip extensor, anterior biceps, muscles during real stumbling can produce extension or reduced flexion at the hip that may be absent in weaker stumbling reactions (Buford and Smith 1993).

During stumbling correction in forward locomotion, there is an initial excitation of the unifunctional (knee and hip flexor) medial Sart, while the lateral Sart (knee extensor and hip flexor) is inhibited (Pratt et al. 1996). These differing actions may explain the mixture of effects recorded when the lateral and medial branches were combined (Sart) during fictive stumbling correction (Sart excitation in Fig. 2, initial inhibition in Fig. 3, and mixed initial effects in Fig. 4). The excitation of knee extensors that occurred in one of two experiments during fictive stumbling correction has not been reported during real stumbling correction and requires further study. The companion paper (Quevedo et al. 2005) reports the existence of very small, short-latency SP-evoked excitation in two Q motoneurons.

**Effects of SP stimulation on the timing and amplitude of locomotor ENG bursts**

SP stimulation evokes a net inhibition of ankle flexors that ends with the termination of stimulation. We suggest that the emergence of strong activity after the termination of the SP stimulus train is caused by an active excitation of flexor motoneurons and not by an escape from inhibition. The parsimonious explanation is that SP afferents access and can facilitate the operation of the central circuitry producing the locomotor excitation of flexors, i.e., the flexor portion of the central
pattern generator for locomotion. After a mixture of stimulus-
locked short-latency postsynaptic potentials in motoneurons
during the stimulus train, this locomotor related excitation
produces an enhancement of limb flexor activity. The finding
that the duration of the flexion phase of the fictive step cycle is
often increased during both fictive and real stumbling reactions
(Buford and Smith 1993) is in keeping with this explanation.
This interpretation is further strengthened by the excitation of
the subsequent flexor phase that occurs during both fictive and
real stumbling correction when stimulation occurs during
stance.

The question arises as to whether the ankle extensor activity
that is evoked during stumbling correction represents a prema-
ture initiation of the extension phase of the step cycle (i.e., a
resetting). We consider this unlikely because 1) the activation
of ankle extensors is rarely accompanied by strong activation
of hip or knee extensors, 2) except for ankle flexors, other
flexors are often active during the period of ankle extensor
activity, and 3) the enhancement of flexor activity can occur
without changes in the timing of the step cycle. We suggest in
the companion study (Quevedo et al. 2005) that ankle extensor
activation occurs by activation of specialized short-latency
reflex pathways from SP afferents to ankle extensor motoneu-
rons and not as a result of a brief resetting to a new extension
phase of the step cycle.

It was possible to show reliable fictive stumbling correction
in one-half of the preparations in this series of experiments
(14/28). We have no adequate explanation for the failure to
produce the activation of knee flexors and ankle extensors
characteristic of stumbling correction in the others. The results
in Fig. 5 suggest that excitatory SP pathways to both flexor and
extensor portions of the locomotor pattern generator exist, but
unknown mechanisms normally control the expression of the
functionally appropriate response.

Is the stumbling corrective reaction a cutaneous reflex?

Our observations on the effects of stimulating other
nerves around the foot (Fig. 6) and throughout the hindlimb
(Guertin et al. 1995; McCrea 2001; Perreault et al. 1995;
Stecina et al. 2005) have not identified any other nerve that
can evoke the full sequence and pattern of motoneuron
activation characteristic of fictive stumbling correction. We
suggest that stumbling corrective reflexes during fictive
locomotion are evoked mainly by activation of low thresh-
hold cutaneous afferents in the SP nerve. During real loco-
motion, electrical stimulation of the dorsal foot during
flexion activates knee flexors but does not inhibit ankle
flexors or evoke substantial ankle extensor activity (dis-
cussed in Buford and Smith 1993). Thus it has been argued
that, in addition to cutaneous SP afferents, activation of other
afferents is needed to produce stumbling correction
(Buford and Smith 1993). During fictive locomotion, 75-ms
trains of shocks (e.g., 15, 200-Hz pulses) to the SP nerve
alone produced all the features of stumbling correction
described in intact animals. We suggest that failure of
electrical stimulation to evoke the full stumbling corrective
reaction in intact preparations could result from the use of
different stimulus parameters and often single shock stim-
ulation used in those studies (Buford and Smith 1993;
Forsberg 1979; Wand et al. 1980). The abolition of real

stumbling correction after local anesthesia of the skin of the foot (Wand et al. 1980; see also Forsberg et al. 1975; 1977)
is in keeping with the cutaneous nature of the stumbling
corrective reflex. After anesthesia, mechanical stimuli pro-
duce only brief short-latency responses in ankle and knee
flexors (Wand et al. 1980).

Even if cutaneous afferents can produce the complete pattern
of stumbling correction, mechanical stimuli to the foot dorsum
during a real swing phase would activate a variety of afferents
in other nerves and could modify responses evoked by SP
nerve activation. For example, activation of group II afferents
from EDL and PerL muscles could contribute to hip and ankle
flexion. One difficulty in assessing such potential contributions
in our preparation is that, during fictive locomotion, stimula-
tion of flexor afferents may sometimes evoke opposite reflex
effects (McGrea et al. 2000). Presumably activation of group I
ankle extensors and plantar foot afferents (Tib) during flexion
would not contribute to stumbling correction since these nerves
reset the step cycle to extension (Fig. 6F; Guertin et al. 1995).

The present and accompanying (Quevedo et al. 2005) results
show that the adult decerebrate cat preparation can serve as
a useful model of the stumbling corrective reactions described
during real locomotion. It would be of great interest to char-
acterize interneurons involved in SP reflex pathways that also
access the rhythm generator and shape the output of the
locomotor networks.

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