Degtyarenko, A. M., E. S. Simon, T. Norden-Krichmar, and R. E. Burke. Modulation of oligosynaptic cutaneous and muscle afferent reflex pathways during fictive locomotion and scratching in the cat. J. Neurophysiol. 79: 447–463, 1998. We have compared state-dependent transmission through oligosynaptic (minimally disynaptic) reflex pathways from low-threshold cutaneous and muscle afferents to some flexor and extensor lumbosacral motoneurons during fictive locomotion and scratching in decerebrate unanesthetized cats. As reported in earlier work, oligosynaptic cutaneous excitatory postsynaptic potentials (EPSPs) in flexor digitorum longus (FDL) and inhibitory postsynaptic potentials (IPSPs) in extensor digitorum (EDL) longus motoneurons were enhanced markedly during the early flexion phase of fictive locomotion. We show in this paper that, in contrast, these cutaneous reflex pathways were depressed markedly during both fictive scratching. On the other hand, disynaptic EPSPs produced by homonymous and synergist group I muscle afferents in flexor (tibialis anterior and EDL) motoneurons were present and strongly modulated during both fictive locomotion and scratching. During both actions, these disynaptic group I EPSPs appeared or exhibited the largest amplitude when the motoneuron membrane potential was most depolarized and the parent motor pool was active. There was an interesting exception to the simple pattern of coincident group I EPSP enhancement and motoneuron depolarization. During locomotion, disynaptic group I EPSPs in both FDL and flexor hallucis longus (FHL) motoneurons cells were facilitated during the extension phase, although FDL motoneurons were relatively hyperpolarized whereas FHL cells were depolarized. The reverse situation was found during fictive scratching: group I EPSPs were facilitated in both FDL and FHL cells during the flexion phase when FDL motoneurons were depolarized and FHL cells were relatively hyperpolarized. These observations suggest that the disynaptic EPSPs in these two motor nuclei are produced by common interneurons. Reciprocal disynaptic inhibitory pathways from group Ia muscle afferents to antagonist motoneurons were active and subject to phase-dependent modulation during both fictive locomotion and scratching. In all but one cell tested, reciprocal disynaptic group Ia IPSPs were largest during those phases (in which the motoneuron membrane potential was relatively hyperpolarized and the parent motor pool was inactive. Oligosynaptic IPSPs in motoneurons produced by stimulation of the mesencephalic locomotor region (MLR) were modulated strongly during fictive locomotion but were suppressed powerfully throughout fictive scratching. Large cord dorsum potentials generated by MLR stimulation were also suppressed markedly during fictive scratching. These results allow certain inferences about the organization of interneurons in the pathways examined. They also suggest that the central pattern generators that produce fictive locomotion and scratching are organized differently.

INTRODUCTION

There is ample evidence that the central pattern generators (CPGs) for rhythmic hindlimb locomotion and scratching are located in the lumbosacral segments (Grillner 1981; Rossignol 1996). It has been known for some time that transmission through cutaneous reflexes are modulated powerfully in a phase-dependent manner during locomotion in intact cats (Anderson et al. 1978; Duysens and Loeb 1980; Duysens and Pearson 1976; Forssberg et al. 1975) as well as during fictive stepping in immobilized, decerebrate animals (Schmidt et al. 1988; Schomborg and Behrends 1978a; Schomburg et al. 1981). Much, if not all, of this state-dependent modulation can be attributed to convergence of primary afferent inputs and output from the CPG for locomotion onto interneurons interposed in the specific pathways tested (Degtyarenko et al. 1996a; Moschovakis et al. 1991; Schmidt et al. 1988), as demonstrated for interneurons in the disynaptic group Ia reciprocal inhibitory pathway (Feldman and Orlovsky 1975). In earlier work from this laboratory, we used patterns of differential modulation during fictive locomotion of oligosynaptic cutaneous postsynaptic potentials (PSPs) in motoneurons to identify distinct categories of interneurons and to examine some details of their circuit organization (Burke and Flesham 1986; Degtyarenko et al. 1996a; Moschovakis et al. 1991). The present work was designed to apply this approach to fictive scratching. The CPG for hindlimb scratching also appears to be located in the lumbosacral segments, where many interneurons have been observed to fire in phase with motoneuron bursting (Baev et al. 1981; Berklinblit et al. 1978a, b), including interneurons belonging to the reciprocal Ia inhibitory pathway (Deligianna and Orlovsky 1980). These observations indicate that group Ia inhibition is modulated actively during rhythmic scratching (Deligianna and Orlovsky 1980). However, to our knowledge there have been no reports of phasic modulation of transmission through oligosynaptic cutaneous or excitatory muscle afferent pathways during fictive scratching. Some of the present results have appeared in abstract form (Degtyarenko et al. 1996b).

METHODS

The methods used for the present experiments were similar to those used in recent reports from this laboratory (Degtyarenko et al. 1996a; Floeter et al. 1993; Gossard et al. 1996). Some of the results reported in the present paper were extracted from data tapes
made during this earlier work but not previously reported. The experiments were conducted in accordance with the "Principles of Laboratory Animal Care" (National Institutes of Health Publication 86-23) and were approved by the National Institute of Neurological Disorders and Stroke Committee on Animal Care and Use.

Briefly, adult female cats (2.5–4.0 kg) were used. Halothane anesthesia was induced by mask and maintained (1–2% in air or oxygen) via a tracheal cannula during surgery. One common carotid artery was cannulated for blood pressure monitoring and the other was ligated. Intravenous catheters were placed in both cephalic veins for administration of noradrenaline and fluids as necessary to maintain blood pressure within physiological limits. The urinary bladder was catheterized. Rectal temperature was maintained near 38°C with a heating pad and lamp.

After extensive denervation of the left hindlimb, the following muscle nerves were cut and mounted on bipolar platinum wire electrodes for stimulation and recording: posterior biceps-semitendinosus (PBST), lateral gastrocnemius and soleus (LGS), medial gastrocnemius (MG), flexor digitorum longus (FDL), flexor hallucis longus (FHL), tibialis anterior (TA), and extensor digitorum longus (EDL). The caudal cutaneous sural (CCS) nerve was cut, and the cutaneous branches of the superficial peroneal (SP), medial plantar (MPL), and, in a few experiments, the saphenous nerves were freed but not cut; all were mounted on bipolar stimulating electrodes.

After a laminectomy exposing spinal segments L₄–S₁, the animals were transferred to a stereotaxic frame and skin flaps surrounding the spinal cord and the hindlimb nerves were used to construct paraffin oil pools. Precollricular postmammary decerebration was performed with a spatula and suction, after which anesthesia was discontinued. The dorsal surface of the first two cervical segments were exposed by opening the dura after removal of the dorsal arches of the C₁ and C₂ vertebrae. The animal was paralyzed with gallamine triethiodide (Flaxedil; 10 mg/kg supplemented every 40–60 min) and artificially ventilated to maintain the expired CO₂ near 4%.

Recording and stimulation

The cord dorsal potential (CDP) was recorded with a platinum ball electrode placed near the dorsal root entry zone at the L₄–L₅ border. Stimulation intensity to peripheral nerves was expressed in multiples of the threshold for the most excitatory fibers in the nerve (usually at 2 times threshold, or 2 × T). Intracellular recordings from motoneurons in the L₄–L₅ segments were made with glass micropipettes (1.0–2.0 μm tip diam) filled with 2 M K⁺ acetate solution containing the local anesthetic QX314 (26 mM; Alomone Laboratories, Jerusalem, Israel) to suppress sodium-dependent action potentials (Frazier et al. 1970). Motoneurons were identified by antidromic invasion from muscle nerve stimulation during the several minute period required for spike blockade to occur.

To produce fictive locomotion, a monopolar tungsten electrode, insulated except at the tip, was placed into the mesencephalic locomotor region (MLR; nominal coordinates: P₂, Lι, HC–1). Constant current (100–150 μA) biphasic, charge balanced pulses (pulse duration 0.2–0.5 ms separated by an equal interval) were delivered in trains (10–40 Hz), referenced to a wire in the neck muscles. The optimal position of the MLR electrode was adjusted to produce rhythmic alternating activity in hindlimb muscle nerves (fictive locomotion), which usually was accompanied by distinct CDP waves (see Fig. 11C).

Fictive scratching was produced by placing a small pledget of cotton saturated with bicuculline in saline (1–2 mg/ml) on the exposed dorsal surface of the C₁ and C₂ spinal segments. Subsequent manual stimulation of the pinna ipsilateral to the recording in most cases elicited rapid rhythmic discharges in muscle nerves characteristic of fictive scratching (Berkinblit et al. 1980; Deligianna et al. 1975, 1981). The pledget was removed between bouts of scratching.

Data collection and analysis

Intracellular potential, CDP, and electroneurogram (ENG) activity in muscle nerves (usually LGS, FHL, FDL, TA or EDL, and PBST), were recorded with an eight-channel digital videotape recorder (Instrutech VR-100; band-pass DC-4 kHz for some early experiments or an Instrutech VR-100B; band-pass DC-9 kHz for the intracellular channel). Timing pulses that were synchronized with stimuli delivered to peripheral nerves and to the MLR were recorded on digital signal channels to synchronize averaging of selected synaptic potentials during off-line data analysis.

Synaptic potentials were evoked in motoneurons during fictive locomotion or scratching by alternating single pulse stimulation of the SP and MRL nerves (at 2 × T) or selected muscle nerves (at group I maximum, ~1.8 × T), usually at a rate of 10 Hz each (Fig. 1) (see Moschovakis et al. 1991 for detailed discussion of the technique). We attempted to obtain recordings of synaptic potentials during both fictive locomotion and scratching in the same motoneurons, although this was not always possible. In general, fictive scratching was produced more reliably than fictive locomotion.

The data from selected portions of the data tapes were digitized off-line (10 kHz) using an Apple Macintosh IIfx or PowerPC 8100 computer and National Instruments NB-MIO-16 A/D board. Data collection and analysis were done with "virtual instrument" programs written with the LabView software package (National Instruments, Austin, TX). In most cases, the phases of fictive locomotion were defined from the digitized ENG data streams, which were rectified and smoothed using decremental look-ahead exponential weighting with a time constant of ~34 ms. This algorithm provided a good match between the onsets and offsets of raw ENG bursts and the smoothed waveforms. The program allowed ENG burst onsets and offsets to be determined either automatically by threshold crossing or manually when signal-to-noise ratios were inadequate.

The start of each cycle during fictive locomotion was taken as the onset of firing in the flexor motoneurons, FDL and/or PBST or the offset of firing in extensor nerves (LGS and/or FHL). The flexion phases were defined between this onset and the termination of bursts in TA or EDL muscle nerves. The remaining time periods were defined as extension phases (e.g., Figs. 2A and 3A). The flexion and extension phases of fictive locomotion were each subdivided into three equal time bins. Because extension phases were generally more variable in duration than flexion phases, the time bins in the two phases usually did not have the same absolute durations (e.g., Fig. 2A) (see also Degtyarenko et al. 1996a; Moschovakis et al. 1991). In the case of fictive scratching, the onset of rhythmic cycles were taken as the offset of bursts in extensor motoneuron pools (LGS and/or FHL; Fig. 1, A and B) or the onset of bursts in the flexors, TA or EDL (e.g., Fig. 2C). Because scratching cycles were quite regular, the entire cycle period was simply divided into six equal increments for analysis (e.g., Fig. 1B).

The timing pulses associated with peripheral nerve stimuli were used to trigger the computer to average together the intracellular potentials and CDPs resulting from stimuli falling into the appropriate locomotion phase bins (see Degtyarenko et al. 1996a; Moschovakis et al. 1991). The MLR stimuli were not synchronized to these pulses. In cases where MLR stimulation produced significant PSPs, overlap between cutaneous and MLR PSPs was avoided during averaging by
FIG. 1. Suppression of oligosynaptic cutaneous excitatory postsynaptic potentials (EPSPs) in a flexor digitorum longus (FDL) motoneuron during fictive scratching. A: records of intracellular potential from the flexor digitorum longus cell (FDL IC), and electromyograms (ENGs) from lateral gastrocnemius and soleus (LGS), flexor hallucis longus (FHL), and FDL muscle nerves. Cutaneous superficial peroneal (SP) and medial plantar (MPL) nerves were stimulated alternately at 10 Hz each (→ in FDL IC trace) throughout fictive scratching. There were short bursts of FDL firing (✓) during the tonic phase coincident with SP nerve stimuli, suggesting that the SP nerve produced sufficient depolarization to cause synchronized firing in some FDL motoneurons when superimposed on the tonic depolarization. B: averages of the FDL baseline membrane potential (i.e., excluding evoked SP and MPL responses), and rectified and integrated ENGs, sampled at 20 equal time bins (○) during 82 cycles of rhythmic scratching. Computer program interpolated the sampling times of successive scratch cycles starting at the offset of firing in the LGS nerve. ‒ ‒ ‒ ‒ ‒ ‒ , 6 equal duration bins during which SP and MPL PSPs were averaged using a 2nd analysis program (see METHODS). C and D: averaged PSPs resulting from SP and MPL stimulation during the 6 cycle phases shown in B (numbered traces; between 43 and 52 sweeps each), superimposed on averages of responses during the tonic phase (7 sweeps) and a period without rhythmic activity (‘rest’; 133 sweeps). Minimum central latencies are indicated with thin arrows.

Using the stimulus timing pulses to include only those cutaneous PSPs that fell within an acceptance window with nearby MLR stimuli. The analysis program also allowed exclusion of responses that produced action potentials (Degtyarenko et al. 1996a). Averaged records of the evoked synaptic potential during fictive scratching were obtained in the same way. Scratch cycles were divided into six phases of equal duration to be analogous to the averaged PSPs obtained during fictive locomotion (Fig. 1C).

Bouts of rhythmic fictive scratching were analyzed with an additional program that allowed comparison among averaged integrated ENG signals, baseline intracellular potentials, and peak amplitudes of reflex PSPs with finer temporal resolution. This program sampled rectified and integrated ENG signals from up to five muscle nerves, as well as the intracellular potential, each averaged during 0.3-ms windows (i.e., 3 data points at 10 kHz digitizing rate) excluding the evoked PSPs. The calculated average and standard deviation of these values were placed into 10 or 20 equal increment time bins, which were chosen to provide continuous estimates of with a reasonable number of data points per bin (Fig. 1B). The program also sampled the membrane potential immediately before and at selected delays after each stimulus trigger pulse (averaged within 0.3-ms windows) to give a continuous estimate of the peak PSP amplitudes. These means and standard deviations of these values were placed into the appropriate time bins (see Fig. 4, B and E). Although the same bouts of fictive scratching were analyzed by the two programs, there were sometimes small discrepancies between the peak PSP amplitudes obtained with the two programs in a given scratch cycle phase. However the basic structure of the PSP modulation cycle was the same (see Fig. 4).

RESULTS

The present results are based on a study of 50 motoneurons in 12 cats in which fictive scratching was elicited during intracellular recording. In some of these animals, fictive locomotion also was examined, enabling comparison of modulation of reflex pathway PSPs during both states in the same motoneurons. Much of the fictive scratching data to be reported were obtained from FDL, EDL, and TA motoneurons, for which patterns of modulation of cutaneous reflex pathway PSPs during fictive locomotion have been documented in a larger sample of cells (Degtyarenko et al. 1996a; Moschovakis et al. 1991; Schmidt et al. 1988). Fictive locomotion was either spontaneous or produced by stimulation of the MLR (see METHODS).

Some general features of fictive scratching are shown in
Fig. 1, A and B, obtained during intracellular recording from an FDL motoneuron. Application of bicuculline to the C1–C2 spinal segments, combined with manual stimulation of the pinna (see METHODS), usually produce an initial tonic firing in flexor muscle nerves and depolarization in the intracellular record (Figs. 1A and 2C, tonic phase). This was followed by stereotyped rhythmic firing in muscle nerves (rhythmic phase) (Deligianna et al. 1981) and rhythmic depolarizing waves (scratching drive potentials, or SDPs, analogous to locomotor drive potentials, or LDPs, found in motoneurons during fictive locomotion) (Jordan 1983). Sometimes the troughs between SDPs remained positive to the pre-existing resting membrane potential (Fig. 1A), whereas in other examples the membrane potential returned to near the baseline level (Fig. 2C).

Oligosynaptic cutaneous excitation is suppressed during fictive scratching

FDL MOTONEURONS. This account begins with FDL motoneurons because distal skin nerves SP and MPL produce especially prominent disynaptic excitatory PSPs (EPSPs) in these cells that are modulated powerfully during fictive locomotion (Moschovakis et al. 1991; Schmidt et al. 1988). An example of enhancement of the initial SP EPSP (central latency = 1.5–1.8 ms, measured from the first afferent volley recorded in the CDP) (Moschovakis et al. 1991) during the early flexion phase (F1) is shown in Fig. 5 (see also Moschovakis et al. 1991). Similar modulation has been found in a total of 29 FDL motoneurons recorded during fictive locomotion.

Figure 1B shows the average membrane potential in an FDL motoneuron, along with averages of rectified and integrated ENG potentials in the LGS, FHL, and FDL muscle nerves, sampled at 20 time periods during each scratch cycle (Avg FDL MP, ○; see METHODS). To examine phase-related PSP modulation during fictive scratching, the same rhythmic scratch cycles were divided into six equal duration time bins (Fig. 1B, −−−−−−). The PSPs falling within each bin during a long series of scratch cycles were averaged together (see METHODS). Figure 1, C and D, show that the oligosynaptic SP and MPL EPSPs observed when there was no rhythmic motor activity (‘‘rest’’) were suppressed markedly (↓) during both tonic and rhythmic portions of fictive scratching, with little phase-related modulation of the residual late inhibitory PSPs (IPSPs). The same suppression of oligosynaptic SP and MPL EPSPs during fictive scratching was found in all nine FDL motoneurons studied in this way. Unfortunately, it was not possible to elicit fictive locomotion during recording from any of these FDL cells to permit direct comparisons.

Flexor MOTONEURONS. Low-threshold afferents in the SP nerve generate oligosynaptic IPSPs during the early flexion phase of fictive locomotion in EDL motoneurons (Degtyarenko et al. 1996a), in contrast to the EPSPs that are generated in its antagonist FDL (Fig. 5C) (Moschovakis et al. 1991; Schmidt et al. 1988). In the EDL cell shown in Fig. 2, small SP EPSPs observed at rest and during extension were replaced during the flexion phase of locomotion by a disynaptic (central latency = 1.8 ms; see DISCUSSION) IPSP.
that was largest during the first third of the flexion phase (Fig. 2B, F1). These IPSPs exhibited an inflection (arrow), suggesting the presence of a later trisynaptic component. On the other hand, the MPL EPSPs seen during extension simply were depressed during the flexion phase but returned to rest level during extension (Fig. 2B) (Degtyarenko et al. 1996a), as seen also in FDL cells (Moschovakis et al. 1991). In contrast, the SP and MPL EPSPs seen at rest were both suppressed throughout fictive scratching, with essentially no phase-related modulation (Fig. 2D). Similar findings were obtained in a total of nine EDL motoneurons, five of which were studied only during fictive scratching.

As discussed elsewhere (Degtyarenko et al. 1996a), the interpretation of IPSP modulation during fictive locomotion is complicated because of the sensitivity of IPSP amplitude to changes in membrane potential (Coombs et al. 1955), especially when enhanced IPSPs are superimposed on depolarizing LDPS as in Fig. 2. We have shown elsewhere that SP IPSP enhancement can occur in EDL motoneurons even without membrane depolarization (Degtyarenko et al. 1996a). Several properties of the IPSPs in Fig. 2B suggest that the enhancement was not due entirely to the depolarization. First, the short latency SP IPSPs were not evident at all during extension or rest but rather appeared suddenly in early flexion (Fig. 2B, F1). Second, IPSP enhancement diminished during F2 and F3 even though the membrane potential reached maximum during F2. Third, no IPSPs were evident in the SP responses during fictive scratching, even though the scratching drive potentials (SDPs) were about twice as large as during locomotion (c.f. Fig. 2, A and C). In any case, it was clear that the SP and MPL pathways both were suppressed powerfully during fictive scratching.

EXTENSOR MOTONEURONS. Although most extensor motoneurons do not exhibit prominent PSPs from the SP and MPL nerves (Fleshman et al. 1984), examination of SP input to FHL motoneurons during fictive locomotion and scratching produced one surprising observation. In 9 of 11 FHL motoneurons tested during fictive locomotion, oligosynaptic SP IPSPs (central latencies ranging from 1.9 to 3.2 ms) appeared during early and midextension. In three cells, the central latency was ≤2 ms, indicating disynaptic connection (see DISCUSSION). An example is shown in Fig. 3.

During fictive locomotion (Fig. 3A), the FHL membrane potential exhibited sharp depolarizing LDPS coincident with FHL and LGS ENG bursts. The SP PSP at rest consisted of a small oligosynaptic EPSP and a late IPSP with central latency of ~4 ms, which showed minor changes throughout the flexion phase of fictive stepping (Fig. 3B). However, during the first two-thirds of extension (E1 and E2), an oligosynaptic (central latency 2.0 ms) IPSP appeared, only to disappear completely in E3. This possibly disynaptic IPSP (see DISCUSSION) resembled those found in EDL motoneurons during flexion (Fig. 2B), except that its enhancement was in the opposite phase of stepping. In this case, IPSP enhancement was correlated more closely with LDP depolarization than it was in the EDL cell in Fig. 2. However, the fact that the locomotor SP responses at rest, during flexion, and in E3 showed absolutely no sign of any early IPSP suggests that it appeared because of premotoneuronal facilitation in the last-order interneurons in a hitherto unrecognized cutaneous reflex pathway (see DISCUSSION). The importance of this response in the present context is that it was entirely absent in the SP PSPs obtained in the same FHL cell during fictive scratching (Fig. 3D), despite substantial SDP depolarizations (Fig. 3C). As in FDL and EDL motoneurons, transmission in the oligosynaptic SP pathway to FHL motoneurons was suppressed markedly throughout the scratch cycle.

Our observations of cutaneous PSPs can be summarized as follows. Suppression of oligosynaptic transmission from low-threshold SP and MPL afferents during fictive scratching shown in Figs. 1–3 was typical of all motoneurons studied in which distal skin nerves produced PSPs of reasonable amplitude. The sample included 16 typical flexor cells (9 EDL, 4 TA, and 3 PBST), 10 typical extensor cells (1 LGS, 2 MG, 4 FHL, and 3 plantaris), 9 FDL motoneurons, and 15 other motoneurons, 5 of which were identified tentatively as flexor or extensor by activity and other PSP patterns and 10 were unidentified. In some of these (mostly the extensor cells), the SP and/or the MPL nerve produced small or negligible PSPs at rest, making reduction during scratching a moot issue. We also examined a few motoneurons (1 PBST, 1 MG, and 2 of unknown identity) during fictive locomotion in which the CCS nerve produced oligosynaptic EPSPs of reasonable amplitude (2–4 mV). In all four cases, CCS EPSPs were depressed or abolished during fictive scratching. In summary, when present, oligosynaptic cutaneous PSPs from the hindfoot in hindlimb motoneurons were suppressed during fictive scratching in all motoneurons examined in this study.

Group I muscle afferent pathways are modulated during locomotion and scratching

We also examined the behavior of two oligosynaptic muscle afferent pathways in some of the same motoneurons: disynaptic group I excitation in homonymous and heteronymous motoneurons (Angel et al. 1996; McCrea et al. 1995; Schomburg and Behrends 1978b) and the well-known disynaptic group Ia pathway that produces reciprocal inhibition in antagonist motoneurons (Eccles et al. 1956; Jankowska 1992). In sharp contrast to the behavior of oligosynaptic cutaneous reflex pathways, both of these disynaptic group I pathways were not only active but were modulated powerfully during fictive scratching as well as fictive locomotion.

Disynaptic group I EPSPs

EXTENSOR MOTONEURONS: FHL. Figure 4 illustrates the behavior of homonymous disynaptic EPSPs, superimposed on a monosynaptic EPSP, produced in an FHL motoneuron by group I muscle afferents in the FHL nerve during fictive locomotion (Fig. 4, A–C) and fictive scratching (Fig. 4, D–F). During locomotion, the disynaptic group I EPSP component (Fig. 4C; central latency ~1.6 ms; inflection at arrow) varied from maximum in early extension (E1) to undetectable in midflexion (F2). It is possible that no disynaptic component was present in any of the EPSPs during F1–F3 and E3; the small changes in shape of the EPSP
FIG. 3. Comparison of SP PSPs in a FHL (extensor) motoneuron during fictive locomotion (A and B) and scratching (C and D; format as in Fig. 2). At rest, the SP nerve produced a small EPSP and a longer latency (4 ms) IPSP in this cell (B and D, thick gray trace; 86 sweeps). However, during fictive locomotion (B), an oligosynaptic IPSP (central latency 2.0 ms) appeared during the first two-thirds of the extension phase (E1 and E2; 7–17 sweeps). In contrast, the late SP IPSPs were simply depressed throughout fictive scratching (D; 17–29 sweeps).

falling phase might have been due to changes in postsynaptic membrane conductance and effective time constant (see also Fig. 5B). On the other hand, these superimposed records suggest that a small disynaptic component may have been present at rest, although without a clear inflection (thick gray trace). When the EPSP potential at 2.5-ms central latency was averaged during 63 step cycles (second trace in Fig. 4B; see METHODS), there was a clear modulation in amplitude that was parallel with the average LDPs in the motoneuron (Avg FHL MP), as well as with the integrated ENG burst in the LGS muscle nerve. This in-phase pattern of disynaptic EPSP modulation has been found in other extensor motoneurons during fictive locomotion (Angel et al. 1986; McCrea et al. 1995). Figure 4, E and F, shows that the disynaptic group I EPSP also was modulated strongly during fictive scratching, from undetectable in phase 1 to maximum in phase 5. In fact, the range of modulation appears somewhat wider during scratching than locomotion. However, in contrast to the pattern of modulation during locomotion (Fig. 4B), the disynaptic group I EPSPs were modulated out-of-phase with the FHL motoneuron membrane potential during fictive scratching (Fig. 4E). The group I EPSPs were largest during the scratching phases when the flexor TA was active (phases 2–5), and the FHL membrane potential was relatively hyperpolarized. All of these observations were confirmed in three other FHL motoneurons in which FHL group I EPSPs were compared during fictive locomotion and scratching.

FDL MOTONEURONS. Figure 5 illustrates modulation of heteronymous FHL group I EPSPs in a FDL motoneuron during fictive locomotion. The FDL motor pool exhibited its normal locomotion firing pattern (Fig. 5A, —), with activity in early flexion (O’Donovan et al. 1982) accompanied by a small depolarizing LDP (Fleshman et al. 1984; Moschovakis et al. 1991; Schmidt et al. 1988). In this case, a heteronymous disynaptic group I EPSP (central latency = 1.3 ms) produced by FHL stimulation appeared only during the extension phase of fictive locomotion (Fig. 5B: E1–E3), with maximum amplitude in late extension (E3) when the LGS nerve showed maximum activity (Fig. 5A). The disynaptic EPSP component disappeared during the flexion phase, where it was possibly replaced by a small IPSP in F2. The pattern of EPSP facilitation was completely out of phase with the depolarizing LDP in the motoneuron and FDL activity in the early flexion phase. Figure 5C shows the typical facilitation of a disynaptic cutaneous SP EPSP (central latency = 1.5 ms) in the same cell during F1 (Moschovakis et al. 1991). The fact that the FHL and SP disynaptic EPSPs were modulated in completely different patterns demonstrates that they are produced by independent sets of last-order excitatory interneurons (see DISCUSSION).

Modulation of FHL disynaptic group I EPSPs during fictive scratching in a different FDL motoneuron is shown in Fig. 6. The FDL motor pool was coactive with the EDL (Fig. 6A) and the membrane potential exhibited irregular ramp-like depolarizations during the active phase (Fig. 6, A...
FIG. 4. Modulation of homonymous (FHL) disynaptic group I EPSPs during fictive locomotion (A–C) and fictive scratching (D–F) in an FHL motoneuron. A and D: intracellular potential (FHL, IC) and LGS, tibialis anterior (TA), and posterior biceps-semitendinosus (PBST) ENGs during 2 locomotion cycles (A) and 5 scratch cycles (D) on the same time base. Note that PBST fired in phase with TA during locomotion (A) but out of phase during scratching (D). B and E: graphs as in Fig. 1B of the average membrane potential (top) and the rectified and integrated ENGs (bottom 3 traces). The 2nd trace shows the amplitude above baseline of the averaged group I EPSPs measured at a central latency of 2.5 ms ("FHL EPSP @ 2.5 ms"; denoted by dashed arrows in C and F) obtained from 63 and 73 step or scratch cycles, respectively. C and F: averaged FHL group I EPSPs at rest (thick gray trace; 86 sweeps) and during the cycles of locomotion (C; 61–110 sweeps each) and scratching (F; 38–44 sweeps) indicated in B and E. The oblique arrows in C and F indicate inflections at the onset of disynaptic EPSPs (central latency 1.6–1.8 ms). Note that the disynaptic EPSP peak amplitude varied almost exactly in phase with membrane depolarization during fictive locomotion (B; double headed arrows) but was almost entirely out of phase during fictive scratching (E).

and C). Unlike the pattern in fictive locomotion (Fig. 5), modulation of the FHL disynaptic EPSP here was generally in phase with the average membrane potential (Fig. 6, B and C). The same pattern was observed in all four FDL motoneurons studied in this way, irrespective of whether the homonymous (FDL) or heteronymous (FHL) nerve was stimulated. Note that the central latencies of the smaller disynaptic EPSPs (phases 1 and 2) were ≈0.5 ms longer than those of the larger responses (note dashed arrows in Fig. 6B; see DISCUSSION).

FLEXOR MOTONEURONS: EDL. Modulation of homonymous disynaptic group I EPSPs also occurred during both fictive locomotion and scratching in typical flexor motoneurons, as illustrated in Fig. 7. In this EDL motoneuron, no disynaptic EPSP component was evident at rest but one appeared in all phases of fictive locomotion (Fig. 7A), with maximum amplitude in F3 coincident with peak intracellular depolarization (Fig. 7B). The trajectories of membrane potential and disynaptic EPSP amplitudes during fictive scratching were also parallel (Fig. 7D), although the range of disynaptic group I EPSP modulation was about twice as wide during fictive scratching, as compared with locomotion. Similar observations were made in 4 of 4 EDL motoneurons and in 2 of 2 TA cells.

Group Ia reciprocal disynaptic IPSPs

Interneurons in the disynaptic inhibitory pathway between group Ia afferents and antagonist motoneurons (the reciprocal group Ia inhibitory pathway) are driven phasically during both fictive locomotion (Feldman and Orlovsky 1975; see also Pratt and Jordan 1987) and fictive scratching (Deliagina and Orlovsky 1980). As expected from these findings, this inhibitory pathway was modulated strongly during both types of rhythmic motor activity.

Figure 8 shows a comparison of reciprocal disynaptic group Ia IPSPs in a typical flexor (TA) motoneuron during fictive locomotion (Fig. 8, A–C) and scratching (Fig. 8, D–F). Stimulation of the antagonist MG nerve at 1.8 × T produced relatively large IPSPs with disynaptic central
modulation of the reciprocal Ia IPSPs in Fig. 8 depends on premotoneuronal mechanisms (see DISCUSSION). The same observations were made during scratching in 4 of 4 TA motoneurons (MG, LGS, and/or FHL stimulation) and 4 of 4 EDL cells (LGS, FHL, or plantaris stimulation).

Group Ia reciprocal IPSPs were studied during fictive locomotion in four extensor motoneurons (3 FHL cells with EDL stimulation and 1 plantaris motoneuron with TA stimulation). Figure 9 illustrates findings typical of all these extensor motoneurons. During the flexor (F) phase, the cell was hyperpolarized markedly (Fig. 9A), reaching maximum negative potential during the last third (F3). The peak amplitudes of the disynaptic reciprocal Ia IPSP showed a parallel change (i.e., increase in negativity from F1 to F3), exactly opposite to what would be expected on the basis of IPSP driving potential. The only motoneuron in which this was not true was the only FDL cell tested, in which the amplitude of the disynaptic group I IPSP (EDL stimulation) changed in strict accord with membrane potential changes, implying no premotoneuronal modulation (see Pratt and Jordan 1987).

Modulation of reciprocal disynaptic Ia IPSPs in the only extensor motoneuron ( MG ) in which these PSPs were examined successfully during fictive scratching is illustrated in Fig. 10. The IPSPs produced by stimulation of group I afferents in the EDL nerve were as large as or larger than the resting response (Fig. 10B), even though this MG cell exhibited only modest membrane potential swings during scratching (Fig. 10, A and C). As in Figs. 8 and 9, the largest IPSPs were found during the scratch cycle phases (2–4) when the membrane potential was relatively hyperpolarized (Fig. 10C).

**Oligosynaptic MLR PSPs**

Shefchyk and Jordan (1985) have shown that stimulation of the MLR can produce short-latency PSPs in lumbosacral motoneurons that are modulated strongly during fictive locomotion produced by that stimulation. Such MLR PSPs were compared during fictive locomotion and scratching in three flexor motoneurons (1 TA and 2 EDL). Figure 11 illustrates findings representative of all three cases, from the same TA motoneuron shown in Fig. 8. The MLR was stimulated repetitively at 30 Hz and 100 µA to induce fictive locomotion. The averaging program was triggered by the MLR stimulus pulses, producing the averaged PSPs shown in Fig. 11A. The MLR EPSP had a central latency of ~2 ms from the first deflection in the CDP (Floeter et al. 1993). It was largest during the F1 phase of rhythmic stepping, when the TA membrane potential was most depolarized (Fig. 8, A and C). This EPSP disappeared throughout the extension phase, to be replaced by a small IPSP with slightly longer latency, when the membrane potential was relatively hyperpolarized (Fig. 8A) (see also Shefchyk and Jordan 1985).

The same stimulation of the MLR was continued while fictive scratching was elicited by pinna stimulation (bicuculline already had been applied to the cervical segments; see METHODS). This elicited a bout of fictive scratching like that shown in Fig. 8D. The MLR PSPs were essentially abolished throughout scratching (Fig. 11B). In addition, the large
MLR-evoked CDP during fictive locomotion was markedly reduced, with relatively little change in the initial deflection that represented the arrival of the fastest descending volley (Fig. 11C). Similar observations were made in the other two motoneurons.

Motor pool firing patterns during fictive scratching

Figure 12 illustrates the ENG firing patterns that were typical of most examples of fictive scratching in the present work. The alternating activity between LGS and FHL on the one hand and EDL and TA on the other during scratching was the same as found during locomotion, except that the “extension” phase was shorter than the “flexion” phase during scratching (Deliagina et al. 1975, 1981) (see also Figs. 6A, 8D, and 10A), whereas in fictive locomotion the extension phase was usually even longer than flexion (Figs. 2A, 5A, and 9A) or about the same duration (Figs. 3A and 4A). In addition, in most bouts of fictive scratching, FDL was coactivated with TA and EDL, whereas there was often little overlap in activity between FDL and either flexor in fictive locomotion (Fig. 5) (Degtyarenko et al. 1996a; Fleshman et al. 1984; Moschovakis et al. 1991). The PBST motor pool also showed differences, in that it was often coactive with the extensors LGS and FHL during fictive scratching, thus appearing roughly inverse to the FDL pattern (see also Figs. 3 and 4). In our experience, PBST was usually coactive with FDL during the early flexion phase of fictive locomotion (e.g., Fig. 5). It should be noted that the activity pattern reported for “FHL” by Deliagina and coworkers (1981) resembles that of FHL in the present work, presumably because of a difference in terminology.

DISCUSSION

The main result of the present work is that, in contrast to fictive locomotion, transmission in oligosynaptic excitatory and inhibitory pathways activated by low-threshold cutaneous afferents from the distal hindlimb was either blocked completely or markedly reduced during fictive scratching. In contrast, the pathways for disynaptic excitation and inhibition from group I muscle afferents to flexor and extensor motoneurons were not only open but exhibited strong phase-dependent modulation during both types of rhythmic motor output. It is important to note that the data came largely from motoneurons of the long plantar flexor (FDL and FHL) and plantar extensor muscles (EDL and TA) because of our interest in oligosynaptic cutaneous pathways.
dramatically in fictive locomotion and scratching. Although double-burst activation sometimes has been observed during locomotion in thalamic preparations (Perret and Cabelguen 1980), in our experience with fictive locomotion, the PBST motor pools usually exhibit only one burst per step cycle during early flexion phase, coincident with the burst in FDL (Figs. 3A, 4A, and 5A) (Degtyarenko et al. 1996a; Fleshman et al. 1984; Moschovakis et al. 1991). In contrast, PBST firing during fictive scratching often tended to overlap with extensor activity (Figs. 3C, 4D, and 12; but cf. 6A) (cf. also Berkenblit et al. 1980).

The patterns of modulation in interneuronal pathways described in this paper provide additional markers for CPG transitions during fictive scratching that were not available in earlier work. For example, in addition to the marked suppression of oligosynaptic cutaneous pathways during fictive scratching (Tao et al. 1997; Mileusnic et al. 1999), disynaptic pathways also provide additional information about the switch to fictive scratching (see text). The activity of the PB and ST motor pools also differs dramatically in fictive locomotion and scratching. Although double-burst activation sometimes has been observed during locomotion in thalamic preparations (Perret and Cabelguen 1980), in our experience with fictive locomotion, the PBST motor pools usually exhibit only one burst per step cycle during early flexion phase, coincident with the burst in FDL (Figs. 3A, 4A, and 5A) (Degtyarenko et al. 1996a; Fleshman et al. 1984; Moschovakis et al. 1991). In contrast, PBST firing during fictive scratching often tended to overlap with extensor activity (Figs. 3C, 4D, and 12; but cf. 6A) (cf. also Berkenblit et al. 1980).

The patterns of modulation in interneuronal pathways described in this paper provide additional markers for CPG transitions during fictive scratching that were not available in earlier work. For example, in addition to the marked suppression of oligosynaptic cutaneous pathways during fictive

Other differences between fictive locomotion and scratching: muscle activation patterns

Many typical flexor (e.g., TA, EDL) and extensor (e.g., MG, LGS, FHL) muscle groups display the same alternating activation during both rhythmic fictive scratching and locomotion (e.g., Figs. 2–6, 8, 9, and 12) (see also Berkinblit et al. 1980; Deliagina et al. 1975, 1981; Kuhta and Smith 1990). However, this is not the case for FDL and PBST. During fictive locomotion, the FDL motor pool is usually (but cf. Fig. 2A) active in the early flexion phase, when it is coactive with PBST but shows less overlap with TA and EDL activity (Fig. 5A) (Degtyarenko et al. 1996a; Fleshman et al. 1984; Moschovakis et al. 1991; Schmidt et al. 1988). During fictive scratching, however, the FDL pool is usually coactive with TA and EDL, whereas PBST usually (but cf. Fig. 6A) (see also Deliagina et al. 1975, 1981) switches to become coactive with the extensors, LGS and FHL (Figs. 2, 7, and 12). Coactivation of FDL and EDL with unloaded digits produces claw protrusion (Straus and Sprague 1944), which is an integral part of the scratching behavior but is not normally seen during walking in intact cats (Goslow et al. 1972; Trank and Smith 1996).

The activity of the PB and ST motor pools also differs dramatically in fictive locomotion and scratching. Although double-burst activation sometimes has been observed during locomotion in thalamic preparations (Perret and Cabelguen 1980), in our experience with fictive locomotion, the PBST motor pools usually exhibit only one burst per step cycle during early flexion phase, coincident with the burst in FDL (Figs. 3A, 4A, and 5A) (Degtyarenko et al. 1996a; Fleshman et al. 1984; Moschovakis et al. 1991). In contrast, PBST firing during fictive scratching often tended to overlap with extensor activity (Figs. 3C, 4D, and 12; but cf. 6A) (cf. also Berkenblit et al. 1980).
In earlier publications from this laboratory (Degtyarenko et al. 1996a; Flesham et al. 1984, 1988; Floeter et al. 1993; Gossard et al. 1996; Moschovakis et al. 1991, 1992), it was concluded that central latencies of 1.2–1.8 ms can be regarded as diagnostic of disynaptic connection in cutaneous excitatory pathways (e.g., Fig. 5C) but that PSPs with latencies as long as 2.0 ms also might be disynaptic. This conclusion is supported by additional observations in the present work.

We have observed examples of group I EPSPs (Fig. 7, A and C) and reciprocal group Ia IPSPs (Fig. 8, B and E) in which central latencies decreased incrementally from 2.0 to 1.5 ms as PSPs were facilitated. There is little doubt that scratching, oligosynaptic EPSPs from the MLR, as well as the characteristic CDPs generated by MLR stimulation, are suppressed markedly during fictive scratching (Fig. 11, B and C). Shefchyk and Jordan (1985) first showed that such oligosynaptic MLR PSPs in both flexor and extensor motoneurons, presumably produced by segmental interneurons, were largest during the phase of fictive stepping when the recorded motoneurons were depolarized. The observed suppression during fictive scratching suggests that a major fraction of the segmental interneurons activated by descending MLR volleys during fictive locomotion are inhibited during fictive scratching.

Central latencies of cutaneous and group I PSPs

There are two issues that should be discussed before the data on reflex pathway modulation is considered. The first concerns central latency, which is the primary physiological criterion used to determine the number of interneuron layers interposed between an afferent input and the recorded motoneuron. Inferences that can be drawn from PSP modulation data are most secure when the afferent inputs under test converge onto last-order interneurons that make direct contact with motoneurons (i.e., disynaptic pathways) (Lundberg 1979).
these group I muscle afferent PSPs are disynaptic (Angel et al. 1996; Jankowska 1992). The existence of examples of group I EPSPs and Ia IPSPs with central latencies of as much as 2.0 ms strengthens the case for accepting this value as an upper limit for disynaptic connection. The smooth decreases in central latency that are observed during facilitation of cutaneous and muscle afferent PSPs (see also Degtyarenko et al. 1996a; Moschovakis et al. 1991) are most easily explained by Sherrington’s notion of the “subliminal fringe” (Creed et al. 1932). Previously silent interneurons in a disynaptic pathway can be made to fire to an afferent volley by convergence from another source of excitation onto the same cells (Lundberg 1979). Such background excitation will raise the transmembrane potential in reflex pathway interneurons closer to threshold, recruiting more cells and allowing already recruited interneurons to fire earlier on the rising phase of composite EPSPs produced in them by synchronous volleys in the tested peripheral afferents. The result will be a net decrease in central latency as well as increased PSP amplitude in the target motoneurons.

**Premotoneuronal control of oligosynaptic pathways**

For reasons discussed elsewhere (Degtyarenko et al. 1996a; Moschovakis et al. 1991; Schmidt et al. 1988), we assume that most of the modulation of cutaneous PSPs described in those papers is due to premotoneuronal facilitation of transmission through pathway interneurons. With regard to the present results, it seems unlikely that purely postsynaptic interactions with synaptic or intrinsic membrane conductances (e.g., Gillessen and Alzheimer 1997) can explain all of the observed changes in the shape and central latency in...
the disynaptic group I EPSPs during fictive locomotion and scratching (Figs. 4–7). In these examples, the monosynaptic component serves as monitor of postsynaptic interactions in the recorded motoneuron as well as possible presynaptic modulation at the first-order group I afferent synapses. Although the early monosynaptic peak sometimes exhibited phase-related changes in amplitude (e.g., Fig. 5B, E1 vs. F1; Fig. 6B, phase 3 vs. phases 1 and 2) (see also Shefchyk et al. 1984), these were not consistently related to the amplitude of the disynaptic component. Although postsynaptic factors such as the effect of depolarization on IPSP amplitudes clearly can affect the test PSPs, the key observations can best be explained by events that occur in the premotor input system. Two premotoneuronal mechanisms, not mutually exclusive, are possible: convergence of CPG control onto the last-order pathway interneurons or phasic modulation of transmission at first order afferent synapses by varying levels of presynaptic inhibition.

The patterns of PSP modulation that we have observed in different reflex pathways do not fit the stereotyped temporal pattern of primary afferent depolarization (PAD; the only convenient marker for presynaptic inhibition) that has been found in cutaneous and muscle afferents during fictive locomotion (Gossard et al. 1989, 1990) or scratching (Baev and Kostyuk 1981). Shefchyk and coworkers (1984) found little evidence for locomotion-related modulation of presynaptic inhibition in group Ia monosynaptic transmission (see preceding text regarding monosynaptic components). In a remarkable recent experiment, Gossard (1996) examined this issue directly by recording PAD in individual group Ia afferents and, simultaneously, singlefiber EPSPs produced by the same afferent in postsynaptic motoneurons during fictive locomotion. He concluded that there was no apparent connection between intra-axonal PAD and EPSP amplitudes. Although it is impossible to rule out some contribution from presynaptic inhibition in the highly structured PSP modulation seen during rhythmic motor activities (e.g., Angel et al. 1996), we suggest that control of the CPG(s) onto pathway interneurons best explains the experimental data available. This explanation also fits the finding that many segmental interneurons are rhythmically activated and deactivated during fictive locomotion (Edgerton et al. 1976; Orlovskii and Fel’dman 1972) and scratching (Baev et al. 1981; Berkenblit et al. 1978a) in the absence of phasic sensory inflow.

Circuit analysis

CUTANEOUS PATHWAYS. The powerful suppression of oligosynaptic cutaneous PSPs during fictive scratching is the most obvious point of difference from locomotion. The strength of this suppression and the fact that it affects disynaptic cutaneous EPSPs and IPSPs (Figs. 1–3) suggests that it results from profound postsynaptic inhibition of the last-order interneurons in the distal cutaneous pathways that have been described previously (Degtyarenko et al. 1996a; Moschovakis et al. 1991). The available data do not allow identification of the possible premotor circuits that suppress transmission in these cutaneous pathways during fictive scratching. It should be noted that the last-order inhibitory interneurons in the pathway that produces oligosynaptic SP inhibition in some FHL motoneurons (Fig. 3) must be different from the cells that produce SP inhibition in EDL motoneurons (e.g., Fig. 5C) because the two IPSPs are facilitated in different phases of the step cycle. To our knowledge, disynaptic cutaneous inhibition of hindlimb extensor motoneurons has not been previously described.

GROUP I MUSCLE AFFERENT PATHWAYS. It has been known for some time that group I muscle afferents can produce di- and trisynaptic EPSPs in cat lumbosacral motoneurons (Jankowska et al. 1981) and that transmission through excitatory interneurons in these pathways is modulated during fictive locomotion (Schomburg and Behrend 1978b; cf. Shefchyk et al. 1984). Recently, McCrea and coworkers (Angel et al. 1996; McCrea et al. 1995) showed that stimulation of group I afferents in hindlimb extensor muscle nerves produces disynaptic EPSPs (mean central latency 1.55 ms) in certain hip, knee, and ankle extensor motor pools as well as in some PBST motoneurons. These EPSPs either appeared or were enhanced considerably during the extension phase of fictive locomotion and in the extensor cells could be attributed at least in part to group Ia afferents. The present work has confirmed this finding for FHL motoneurons (Fig. 4, A–C) and extended it to typical flexor motor pools (EDL and TA), in which disynaptic group I EPSP facilitation occurred during the flexor phase of locomotion when the motoneurons were depolarized (Fig. 7, A and B). The same was true for disynaptic group I EPSPs in flexor motoneurons during scratching, but, unfortunately, we did not examine this point in extensor motoneurons other than FHL (which could be a special case).

The only exception to this simple pattern was found in the long toe flexors, FDL and FHL. During fictive locomotion, disynaptic group I EPSPs in both FDL and EDL motoneurons were enhanced during the extension phase, out of phase with the FDL depolarization (Fig. 5) but in phase with FHL depolarization (Fig. 4, A–C). These relations were exactly opposite during fictive scratching; disynaptic group I EPSPs were enhanced during the flexor phase, in phase with FDL depolarization (Fig. 6) but out-of-phase with the depolarization of FHL motoneurons (Fig. 4, D–F). A suggested explanation for this seemingly paradoxical observation is shown in Fig. 13.

The circuit diagrams in Fig. 13 represent an attempt to summarize the main findings about modulation of disynaptic group I excitation and reciprocal Ia inhibition during fictive locomotion (Fig. 13A) and scratching (Fig. 13B). The CPGs for locomotion and scratching are each represented as a rhythm generator connected to target motoneurons and pathway interneurons by undefined pattern-formation networks (arrows) that distribute excitatory drive to specific groups of last-order interneurons and motoneurons in appropriate spatial and temporal sequences.

In EDL and TA motoneurons, the pattern of modulation of disynaptic group I EPSPs was the same in scratching as in locomotion: enhancement of homonymous and synergist group I EPSPs when the parent motor pool was active and its motoneurons depolarized (Fig. 7). This pattern is the mirror image of the situation described for many ankle exten-
FDL and FHL motoneuron pools, where two patterns of disynaptic group I EPSP modulation were evident depending on the nature of the cyclic motor activity. The observations can be explained if FDL and FHL group I afferents project to common last-order excitatory interneurons that in turn project to both FDL and FHL motoneurons (Fig. 13). Control of these interneurons by the CPG is switched from the extensor half-center during locomotion to the flexor half-center during scratching (Fig. 13, asterisk), while the direct excitatory drive to FDL and FHL motoneurons does not. However, the LDP depolarizing drive does switch in the case of PBST motoneurons (left-most asterisks), which are driven by the flexor half-center in fictive locomotion but by the extensor half-center during fictive scratching.

**Functional significance**

Phase-dependent modulation of transmission through reflex pathways is a subject of considerable interest (Lennard and Hermanson 1985; Sillar 1991; VanWezel et al. 1997). We have suggested that the facilitation of disynaptic SP EPSPs in FDL and SP IPSPs in EDL motoneurons during the early flexion phase of fictive locomotion (Figs. 2B and 5C) (Degtyarenko et al. 1996a; Moschovakis et al. 1991; Schmidt et al. 1988) are likely part of the “stumbling corrective reaction”, an exaggerated flexion response that occurs in actual stepping when the foot encounters an obstacle in early swing (Forssberg 1979). The segmental locomotor CPG thus apparently contains a built-in system to enhance sensitivity to unexpected sensory input from the skin on the dorsum of the foot (the SP nerve territory) as the foot begins its forward swing.

At the same time, transmission through the disynaptic pathway from MPL afferents to both FDL and EDL is suppressed during the flexion phase of locomotion (Degtyarenko et al. 1996a; Moschovakis et al. 1991), which resembles the suppression of disynaptic SP and MPL EPSPs throughout the fictive scratching cycle. What could be the functional meaning to such reflex pathway suppression? It seems reasonable, albeit entirely speculative, to suggest that some sensory pathways may be inhibited by the CPG(s) to suppress expected cutaneous sensations. Stereotyped movements of the toes during the swing phase presumably activate cutaneous receptors on the plantar surface of the foot, possibly producing unwanted reflex perturbations unless centrally suppressed. By the same token, the act of scratching would be expected to generate a large volume of cutaneous input from the distal foot when it contacts the cat’s head (Kuhta and Smith 1990). Again, such expected input might produce counterproductive reflexes unless suppressed by the scratching CPG (see Deliagina et al. 1981).

It is important to note that distal cutaneous pathways were not completely closed during scratching (e.g., residual late IPSPs can be seen in Figs. 1, C and D, and 3D). It is known that strong stimulation such as pinching the toes disrupts fictive scratching in the cat (Deliagina et al. 1981) and cutaneous stimuli produce specific modifications in the complex scratch rhythms in the turtle (Currie and Stein 1989). It is obviously difficult to extrapolate from effects produced (or not produced) by synchronous electrical stimulation of
mixed afferent populations to those generated by natural activation of the skin. It is also difficult to compare fictive scratching in an immobile, reduced preparation to actual scratching in an intact behaving cat. Kuhta and Smith (1990) have shown that the short durations of extensor muscle activity that characterize most examples of fictive scratching (Fig. 13) (Deliagina et al. 1975) are only observed during air-scratching in normal, intact cats, when the foot makes no contact. In normal scratching, they found that firm contact with the head prolongs extensor muscle activity. Kuhta and Smith (1990) postulated that cutaneous feedback is important in this regulation of action during the scratching in intact cats, and the present data do not rule this out. Nevertheless, the relative balance of potential sensory throughput in oligosynaptic distal cutaneous pathways certainly appears to be different during fictive scratching as compared with fictive locomotion, at least among motoneurons in which these pathway are powerfully modulated during locomotion.

It is possible, of course, that amplified disynaptic excitation from group I muscle afferents plays a major role in regulating the intensity and duration of the extension phase of scratching, as occurs during locomotion in decerebrate and intact cats (e.g., Whelan et al. 1995). Disynaptic excitation from group I muscle afferents is modulated strongly during fictive scratching, possibly to a greater degree than during fictive locomotion (Figs. 4 and 7). Except for the interesting (partial) exception of FDL and FHL (see preceding text), the available evidence shows that EPSP amplification usually occurs when the target motoneurons are depolarized and active (Figs. 4 and 7) (Angel et al. 1996; McCrea et al. 1995; Schomburg and Behrends 1978b). There is also evidence that muscle spindle afferents are driven during scratching by alpha-gamma coactivation (Feldman et al. 1977). Thus information from muscle spindle (and possibly Golgi tendon organ) afferents apparently is amplified by the scratching CPG.

The simultaneous enhancement of disynaptic group I excitation to FDL (and EDL) motoneurons during scratching (Fig. 13B) might assist delicate proprioceptive control of claw protrusion during scratching, whereas group I feedback to FHL motoneurons through the same interneurons might be more important during locomotion.

Are fictive stepping and scratching generated by the same neuronal circuits?

As a result of extensive studies of fictive locomotion and scratching, the Moscow and Kiev groups proposed that fictive locomotion and scratching are produced by the same spinal mechanism operating in different regimes (Bayev 1978; Berkinblit et al. 1978b; Deliagina et al. 1975, 1981). They based this conclusion on three observations: many of the muscle groups associated with flexion and extension phases of both cyclic movements are the same despite a few notable exceptions (Deliagina et al. 1975, 1981), some examples of fictive scratching begin with slow cycles in which synergies similar to that in locomotion gradually change to those characteristic of scratching (Berkinblit et al. 1978b), and individual spinal interneurons can exhibit similar behaviors during both movement patterns (Bayev et al. 1981; Berkinblit et al. 1978a).

The present results provide only partial support for this conclusion. We find what appear to be major circuit realignments in the two rhythmic states, both in the distribution of CPG drive to specific motoneuron pools (e.g., FDL and PBST) and to specific populations of cutaneous and muscle afferent pathway interneurons (Fig. 13). In addition, there appears to be powerful suppression of a population of interneurons that are activated by the MLR during fictive scratching (Fig. 11). Such realignments are presumably accomplished by activating different systems of segmental interneurons that distribute the output of rhythm generating circuits to motoneurons and last-order interneurons. The CPGs for locomotion and scratching certainly could include common interneurons (Bayev et al. 1981), but whether or not they are fundamentally the same remains an open question.

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