Abstract
Interneuronal convergence of corticospinal and segmental pathways involved with the generation of extensor activities during locomotion was investigated in decerebrate and partially spinalized cats. L-dihydroxyphenylalanine (L-DOPA) was slowly injected until long-latency, long-lasting discharges could be evoked by the stimulation of contralateral flexor reflex afferents (coFRA) and the group I autogenetic inhibition was reversed to polysynaptic excitation in extensor motoneurons. Under these conditions, we stimulated in alternation the contralateral pyramidal tract (PT), group I afferents from knee and ankle extensor muscles, and both stimuli together. We did the same for the stimulation of PT and of coFRA. Clear polysynaptic EPSPs could be evoked from all three sources in 32 extensor motoneurons. Convergence was inferred from spatial facilitation, which occurred when the amplitude of the EPSPs evoked by the combined stimuli was notably larger than the algebraic sum of the EPSPs evoked by individual stimulation. Spatial facilitation was found between PT and extensor group I inputs in 30/59 tests (51%) in 20 motoneurons and in all cases (6/6) between PT and coFRA in six motoneurons. When fictive locomotion was induced with further injection of L-DOPA, PT descending volleys from the same stimulating site could reset the stepping rhythm by initiating bursts of activity in all extensors. These results indicate that at least some of the corticospinal fibers project onto interneurons shared by the coFRA and the polysynaptic excitatory group I pathways to extensors. The implications of such convergence patterns on the organization of the extensor “half-center” for locomotion are discussed.

Keywords
Locomotion · Convergence · Pyramidal tract · Spinal cord · Central pattern generator

Introduction
The role of the motor cortex has been the subject of intensive study, but its role in motor control remains controversial. Studies on the role of corticospinal neurons in the control of locomotor processes have contributed tremendously to a better understanding of the voluntary control of movements in general (Armstrong 1986; Armstrong and Drew 1984, 1985; Drew 1988, 1991; Drew et al. 1996; Widajewicz et al. 1994). In the decorticate or decerebrate cat, it is possible to have a normal locomotor pattern on a flat surface, making clear that the motor cortex is not an essential structure in creating synergies required for simple locomotion (Armstrong 1986; Whelan 1996). However, it was shown that finer locomotor tasks, such as walking along the rugs of a horizontal ladder, were impossible after a complete bilateral lesion of the bulbar pyramids (Liddel and Phillips 1944). More recently, it has been shown that the rate of discharge of the pyramidal tract neurons was markedly increased when there was a need for a precise placement of the foot in freely moving cats (Beloozorova and Sirota 1993). Moreover, pyramidal tract neurons in the hindlimb representation of the motor cortex increased their activity when the cat voluntarily modified its gait in order to step over an obstacle attached to a treadmill (Widajewicz et al. 1994).

Pyramidal-tract neurons send their projections towards a wide range of areas in the central nervous system, including structures that govern head, trunk, and limb musculature (Armand 1984 and Canedo 1997 for reviews). Even if half of the pyramids are made of corticobulbar fibers, the medullary pyramids are also the most discrete source of direct corticospinal fibers in the brain (Wiesendanger 1969; cf. Canedo 1997). There is compelling evidence demonstrating that the pyramidal tract has a strong influence on locomotor movements not only through spinal networks, but also via other structures in the brainstem. Shik et al. (1968) showed that, in the decerebrate cat walking on a treadmill, pyramidal stimulation could profoundly influence the ongoing locomotion...
by either halting the rhythm or by initiating it. Moreover, they showed that, after a bilateral lesion of the pyramids, stimulation of the pyramidal tract rostral to the lesion was able to initiate a locomotor pattern that did not differ from the one elicited by stimulation of the mesencephalic locomotor region (MLR; Jordan 1983). Pyramidal stimulation ceased to elicit walking after a bilateral destruction of the MLR. Thus, as far as initiation of locomotion is concerned, the pyramidal systems need the participation of other structures in the brainstem and cannot do it directly through corticospinal fibers.

Later, Orlovsky (1972) showed that electrical stimulation (at 150 Hz) of the pyramidal tract at the medullary level in the decerebrate walking cat could increase the level of activity in the contralateral flexor muscles if the stimulation was given during swing phase of the stepping cycle. If a train of stimuli of higher frequency (at 300 Hz) was given during the stance phase, it was possible to affect the stepping rhythm by initiating a burst of activity in ankle flexors. More recently, it was reported that stimulation of the motor cortex in awake cats walking steadily on a treadmill frequently produced significant changes in the mean cycle duration when applied during stance, primarily by reducing its duration and by initiating a new period of swing (Armstrong and Drew 1985; Rho et al. 1999). These effects were absent one week after pyramidecтомy at mid-olivary level (Armstrong and Drew 1985), suggesting that they were primarily mediated by direct corticospinal projections. The available evidence thus suggests that at least part of the corticospinal pathway projects to a population of spinal circuits responsible for the timing of the stepping cycle.

It is generally believed that resetting of the locomotor rhythm can only be achieved by inputs transmitted through interneurons of the rhythm generator (see Conway et al. 1987; Hultborn et al. 1998). One such input is the signal from the group I afferents of extensor muscles. Indeed, during fictive locomotion, stimulation of these afferents is able to reset the locomotor rhythm by interrupting the burst activity in flexor nerves and simultaneously triggering activity in extensor nerves (Conway et al. 1987; Gossard and Hultborn 1991; Gossard et al. 1994). Moreover, repetitive stimulation of extensor group I fibers was found to entrain the stepping rhythm (Conway et al. 1987; Pearson et al. 1992). Together with other observations, these findings were taken as evidence that extensor group I pathways share some interneurons of the extensor half-center for locomotion (Gossard and Hultborn 1991; Hultborn et al. 1998).

All of the studies quoted in the preceding paragraphs reported that the activity of flexors in both the forelimbs and the hindlimbs was more strongly modified by cortical stimulation than that of extensors. Nevertheless, one might expect that descending commands from the motor cortex would also have direct access to the spinal networks generating the activities related to the stance phase of stepping. The aim of this study was thus twofold: (1) to determine whether the corticospinal pathway can also influence the timing of locomotor activities in extensors, and (2) to determine if this effect is mediated through the same interneuronal circuits used by the group I afferents from extensor muscles. The spinal convergence between corticospinal and extensor group I fibers was investigated with the spatial facilitation technique (Lundberg and Voorhoeve 1962) in lumbosacral extensor motoneurons following injection of L-DOPA (Gossard et al. 1994; Leblond et al. 2000). The results presented here confirm that the corticospinal pathway has the ability to initiate and increase locomotor activities in hindlimb extensors and further indicate that corticospinal fibers project onto interneurons of the excitatory polysynaptic group I pathways from extensors. Some of this material has been reported in abstract form (Leblond et al. 1998a).

**Materials and methods**

**Surgical procedures**

All animal experiments were conducted in accordance with the “Principles of Laboratory Animal Care” (NIH publication no. 86–23, revised 1985) and the “Guide for the Care and Use of Laboratory Animals” (1996, National Research Council), using protocols approved by the Ethics Committee of the Université de Montréal. Adult cats of both sexes (n=12; 2.5–4.5 kg) were used. Halothane anesthesia was induced during the surgery. The right common carotid artery was cannulated for monitoring blood pressure and the left one was ligated. One jugular vein and one cephalic vein were cannulated for administration of fluids. Temperature (37°C) and blood pressure (80–120 mm Hg) were maintained within physiological limits.

The following flexor and extensor muscle nerves from the left hindlimb were used for recording (electroneurogram, ENG) and stimulation: posterior biceps-semimembranosus (PBSt), lateral gastrocnemius-soleus (LGS), medial gastrocnemius (MG), plantaris (Pl), flexor digitorum longus and flexor hallucis longus together (FDHL), tibialis anterior (TA), extensor digitorum longus (EDL), superficial peroneal (SP), and sciatic (Sci). The nerve to quadriceps (QUAD) was mounted in a polymer cuff electrode. In six experiments, three nerves were also mounted from the right (contralateral, co-) hindlimb: coPBST, coSmAB, and coSci.

A laminectomy was performed of the L4–L7 and T13 vertebrae and the cats were then transferred to a stereotaxic frame. A precollicular, post-mamillary decerebration was performed with a spatula and the rostral nervous tissues were aspirated. Anesthesia was discontinued and the cats were paralyzed with gallamine triethiodide (Flaxedil; 10 mg/kg supplemented every 45 min) and artificially ventilated, maintaining the expired pCO₂ near 4%. The spinal cord was partially transected at T13 to eliminate as much as possible irrelevant descending pathways, leaving the ipsilateral ventral and lateral funiculi intact.

**Recording and stimulation**

The cord dorsum potential (CDP) was recorded with a silver-chloride ball electrode located close to the dorsal root entrance at the L6–L7 border. The stimulation intensity for peripheral nerves was expressed in multiples of the threshold (T) for the most excitable fibers in the nerve as monitored from the CDP. Motoneurons were recorded intracellularly with glass micropipettes filled with 100 mM QX314 and 2 M K⁺-acetate to prevent sodium spikes. The impaled cells were identified by antidromic invasion from the motor cortex in awake cats walking steadily on a treadmill frequently produced significant changes in the mean cycle duration when applied during stance, primarily by reducing its duration and by initiating a new period of swing (Armstrong and Drew 1985; Rho et al. 1999). The right common carotid artery was cannulated for monitoring blood pressure and the left one was ligated. One jugular vein and one cephalic vein were cannulated for administration of fluids. Temperature (37°C) and blood pressure (80–120 mm Hg) were maintained within physiological limits.

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