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Asymmetric Changes in Cutaneous Reflexes After a Partial Spinal Lesion and Retention Following Spinalization During Locomotion in the Cat

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1Groupe de Recherche du Système Nerveux Central, Department of Physiology, Université de Montréal, Montreal, Quebec, Canada; 2Multidisciplinary Team on Locomotor Rehabilitation after Spinal Cord Injury (Strategic Initiative), Canadian Institutes for Health Research, Ottawa, Ontario, Canada; and 3Centre National de la Recherche Scientifique, Université Bordeaux 1, Bordeaux, France

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Frigon A, Barrière G, Leblond H, Rossignol S. Asymmetric changes in cutaneous reflexes after a partial spinal lesion and retention following spinalization during locomotion in the cat. J Neurophysiol 102: 2667–2680, 2009. First published September 2, 2009; doi:10.1152/jn.00572.2009. Locomotion involves dynamic interactions between the spinal cord, supraspinal signals, and peripheral sensory inputs. After incomplete spinal cord injury (SCI), interactions are disrupted, and remnant structures must optimize function to maximize locomotion. We investigated if cutaneous reflexes are altered following a unilateral partial spinal lesion and whether changes are retained within spinal circuits after complete spinal transection (i.e., spinalization). Four cats were chronically implanted with recording and stimulating electrodes. Cutaneous reflexes were evoked with cuff electrodes placed around left and right superficial peroneal nerves. Control data, consisting of hindlimb kinematics and electromyography (bursts of muscular activity and cutaneous reflexes), were recorded during treadmill locomotion. After stable control data were achieved (53–67 days), a partial spinal lesion was made at the 10th or 11th thoracic segment (T10–T11) on the left side. Cats were trained to walk after the partial lesion, and following a recovery period (64–80 days), a spinalization was made at T13. After the partial lesion, changes in short-latency excitatory (P1) homologous responses between hindlimbs, evoked during swing, were largely asymmetric in direction relative to control values, whereas changes in longer-latency excitatory (P2) and crossed responses were largely symmetric in direction. After spinalization, cats could display hindlimb locomotion within 1 day. Early after spinalization, reflex changes persisted a few days, but over time homologous P1 responses increased symmetrically toward or above control levels. Therefore changes in cutaneous reflexes after the partial lesion and retention following spinalization indicate an important spinal plasticity after incomplete SCI.

INTRODUCTION

Locomotion is often described as having a tripartite control system that involves dynamic interactions between the spinal central pattern generator (CPG), supraspinal signals, and peripheral sensory inputs (Rossignol et al. 2006). Following injury at any level, interactions are disrupted, and remnant structures must optimize function to maximize locomotion (Rossignol et al. 2008, 2009). After incomplete spinal cord injury (SCI), locomotor recovery can involve widespread plastic changes within remaining descending motor and ascending sensory pathways, within the spinal CPG, and within segmental spinal reflexes. The present work aims at evaluating some of these changes at the spinal level, more specifically.

Recently, the intrinsic spinal locomotor circuitry was shown to be involved in the recovery of hindlimb locomotion following incomplete SCI (Barrière et al. 2008). In that study, a partial spinal lesion was made at the 10th or 11th thoracic segment (T10–T11) on the left side, and following a recovery period during which cats were trained on a treadmill, a complete spinal transection (i.e., spinalization) was performed at T13. Within hours after spinalization, and without pharmacological stimulation (e.g., clonidine), cats displayed hindlimb locomotion, a process normally requiring 2–3 wk if no partial lesion is performed a priori (i.e., before spinalization) (Barbeau and Rossignol 1987; Bélanger et al. 1996). This “dual-lesion paradigm” demonstrated that the spinal CPG had reached a stage where it could function more or less independently from descending supraspinal inputs at the time of spinalization.

Spared pathways from supraspinal structures are most probably involved in reorganizing the spinal CPG during training after the partial spinal lesion. However, given the importance of locomotor training, peripheral sensory inputs are probably also critical in ensuring that the spinal CPG functions more effectively with diminished inputs, or no signals, from supraspinal structures. Changes in afferent inputs can be assessed partly by testing excitability changes in reflex pathways. Reflex changes following a lesion that primarily damages function on one side, such as a lateral spinal hemisection, could be asymmetric, to offer more compensatory inputs on the damaged side. Conversely, sensory inputs from the undamaged side could be increased to offset losses on the other side. In a series of studies, Hultborn and Malmsten showed that cats and rats with chronic partial spinal lesions displayed left/right reflex asymmetries in their hindlimbs following a complete spinal transection during an acute terminal experiment (Hultborn and Malmsten 1983a,b; Malmsten 1983). The chronic preparation offers the possibility to test changes in reflex pathways over time by recording reflexes from both hindlimbs before and after a lateral spinal hemisection in the same animal. The dual lesion paradigm has a further advantage: if reflex changes do occur after spinal hemisection and contribute to the recovery of hindlimb locomotion, there should be some retention of this plasticity within spinal circuits soon after spinalization because cats can walk within hours after the complete spinal transection.

In the present study, we used cutaneous reflexes evoked by stimulating skin afferents of the superficial peroneal (SP) nerve at the ankle, bilaterally. This is part of an ongoing series of
studies aimed at identifying changes in cutaneous reflexes after central or peripheral lesions of the nervous system (Frigon and Rossignol 2007, 2008a,b, 2009). Cutaneous inputs are strongly involved in the control of locomotion, and at least some inputs from the skin of the paw are required for proper locomotion after complete spinal transection in the cat (Bouyer and Rossignol 2003a,b). Moreover we recently showed that cutaneous reflexes from the paw were altered in specific phases of the locomotor cycle after spinalization, suggesting an important role of inputs from the skin of the foot in the recovery process after the total loss of supraspinal inputs (Frigon and Rossignol 2008a). The SP nerve was chosen because it is a purely cutaneous nerve and because it evokes coordinated and prominent reflex responses during locomotion in intact and spinal cats (Forsberg 1979; Forsberg et al. 1975). In addition, at low stimulation intensity, stimulating the SP nerve has little effect on the ongoing locomotion (Abraham et al. 1985) as opposed to stimulating a mixed nerve, which can perturb the rhythm by causing muscular contractions.

Several studies have shown changes in spinal reflex pathways following spinal cord injury (reviewed in Frigon and Rossignol 2006), but the majority of these studies compared reflex responses between groups of intact and spinal cord-injured animals. A major limiting factor in such studies is that reflex pathways can differ considerably between individuals (Loeb 1993; Zehr et al. 1997), making changes in reflex pathways difficult to interpret and assess by comparing intact and spinal cord-injured groups. A few studies have used chronic recordings to examine reflex changes after SCI in the same animal (Bennett et al. 1999, 2004; Lavrov et al. 2006), but, besides our study of complete spinal transection (Frigon and Rossignol 2008a), there are no data during locomotion. Most SCIs in humans are incomplete in nature, and consequently, studying the reorganization of reflex pathways during locomotion before and after an incomplete SCI in the same animal could provide important clues as to their intrinsic organization, reorganization after partial SCI, and possible involvement in locomotor recovery.

In the present study, we describe changes in cutaneous reflexes over time using the dual-lesion paradigm with a focus on homologous responses between hindlimbs. The results indeed show that cutaneous reflexes are modified after a partial spinal lesion. Specifically, changes in short-latency excitatory homologous reflex responses from the hindlimbs are largely asymmetric in direction (i.e., relative to control values) after the partial spinal lesion, with increased responses in one hindlimb accompanied by decreases in the other hindlimb. Furthermore, early after complete spinal transection, reflex changes persisted for a few days, and over time with further training, reflexes returned to or surpassed control levels. These plastic changes within the spinal cord probably represent one of the underlying mechanisms involved in the near immediate hindlimb locomotion observed after complete transection in cats that had a preceding partial spinal lesion.

**Methods**

**Animal care and general procedures**

The wellbeing of the cats was continuously ensured, and all procedures concurred with the guidelines of the animal Ethics Committee of the Université de Montréal, according to the Canadian Guide for the Care and Use of Experimental Animals. All animals were regularly monitored by a veterinarian. Cats were housed in large individual cages (104 × 76 × 94 cm) with food and water. After the lesions, foam mattresses were placed in the cages, and cats were attended to a few times a day to clean the head connectors, empty the bladder and clean the hindquarters when necessary. All surgical procedures were performed under general anesthesia and aseptic conditions. Prior to any surgery, cats were injected with an analgesic (Anafen 2 mg/kg sc) and premedicated (Atravef 0.1 mg/kg im, Glycopyrrolate 0.01 mg/kg im, ketamine 0.01 mg/kg im). Cats were then intubated and maintained under gaseous anesthesia (isoflurane 2%) while heart rate and respiration were monitored. After surgery, an analgesic (Buprenorphine 0.01 mg/kg) was administered subcutaneously. An oral antibiotic (cephalexin or cephalone, 100 mg/day) was given for 10 days following surgery.

The general experimental protocol pertaining to the dual lesion paradigm (i.e., partial lesion at T10–T11 followed by complete spinal transection at T13) was previously described (Barriere et al. 2008). In the present study, four cats were used (cats GB3, GB6, GB7, and GB8). These animals weighed between 2.5 and 4.5 kg at the time of implantation and were first selected for their ability to walk for prolonged periods on a treadmill. After a period of treadmill adaptation, cats were chronically implanted with electrodes for electromyographic (EMG) recordings and nerve stimulation and allowed to recover from the implantation, and then EMG (locomotor bursts of activity and cutaneous reflexes) and kinematics were recorded during locomotion. After stable control data were obtained, a partial unilateral lesion was performed at T16–T11 on the left side. The partial spinal lesion was respectively made in cats GB3, GB6, GB7, and GB8 at 53, 54, 67, and 53 days following the initial implantation. Recordings resumed as soon as the animals displayed somewhat stable hindlimb locomotion. The first recording sessions for cutaneous reflexes were at 3, 8, 3, and 4 days postpartial lesion for cats GB3, GB6, GB7, and GB8, respectively. It should be emphasized that the modulation of phase-dependent reflexes cannot be rigorously studied unless the animal is capable of achieving long periods of several hundred consecutive steps. All cats were trained after the partial spinal lesion. EMG (muscle bursts of activity and cutaneous reflexes) and kinematics were recorded throughout the duration of the study (i.e., before and after spinal lesions). Chronic implantation procedures enable direct comparisons between states (i.e., intact, partial spinal lesion and spinalized), while also minimizing the number of cats used. Each animal was tested for a period ranging from 6 mo to 1 yr.

**Kinematics**

Kinematics of both hindlimbs were captured before and after spinal lesions (i.e., partial and complete spinal lesions) using a Panasonic digital 5100 camera (1/1,000-s shutter speed, 30 frames/s or 60 fields/s, giving a final time resolution of 16.7 ms) and a Sony RDR-GX315 DVD recorder during treadmill locomotion. Foot contacts and lifts were determined to provide durations of stance, swing, and cycle period. Stance duration was measured as the time between contact and lift of the paw for the left and right hindlimbs. Stance duration relative to locomotor cycle period was measured for each hindlimb and the ratio between the left (partial spinal lesion) and right (approximately intact) sides was used as a rough measure of symmetry during hindlimb locomotion (Ying et al. 2008).

**Implantation of EMG electrodes**

Chronic EMG electrodes were implanted bilaterally in the following hindlimb muscles: semitendinosus (St: knee flexor/hp extensor), anterior part of sartorius (Srt: hip flexor/knee extensor), iliopsoas (IP, hip flexor), vastus lateralis (VL: knee extensor), lateral gastrocnemius (LG: ankle extensor/knee flexor), medial gastrocnemius (MG: ankle extensor/knee flexor), and tibialis anterior (TA: ankle flexor). A pair...
of Teflon-insulated multistrain fine wires (AS633; Cooner Wire, Chatsworth, CA) was directed subcutaneously from head-mounted 15-pin connectors (cinch connectors; TTI) and sewn into the belly of each muscle for bipolar EMG recordings. Over the course of the study some EMG recordings were lost in both hindlimbs, as determined by the disappearance of EMG signals and/or the loss of reflex responses. Moreover, some muscles discharged irregularly and the burst could not be delineated, although reflex responses could be recorded in these muscles. EMG recordings were band-pass filtered (100–3,000 Hz) and amplified (gains of 1,000–10,000) using two Lynx-8 amplifiers (Neuralynx, Tucson, AZ). EMG data were digitized (5,000 Hz) using custom-made acquisition software.

Nerve stimulation and reflexes

A chronic bipolar stimulating electrode composed of wires (AS633; Cooner Wire) embedded in a polymer (Densply International) cuff (Julien and Rossignol 1982) was placed around the left and right superficial peroneal (SP) nerves at the ankle. The SP nerves were stimulated with a Grass S88 stimulator connected through an optically coupled constant current isolation unit (Grass PSIU6) and a supplemental custom-made current measurement unit that monitors precisely the current delivered. Initially, stimulation was delivered at different intensities during locomotion with single 0.2-ms pulses 100 ms after ipsilateral (i.e., on the side of the stimulation) St burst onset to determine the reflex threshold for obtaining small yet consistent short-latency (~10 ms) responses in the ipsilateral TA. Stimulation current was then set at 1.5 times this threshold to evoke reflexes. The cats did not seem to notice the stimulation during locomotion, and as a result, long sequences (>10 min) of stimulation could be obtained. The stimulation did not visibly alter limb trajectory during intact or spinal locomotion, which is important because perturbations of the limb could introduce movement-related proprioceptive reflexes. A computer-generated pseudorandom sequence delivered stimuli throughout the step cycle for a total of ~120–200 stimuli per session, each stimulus being given once every three cycles. Once reflexes were qualitatively and quantitatively reproducible from one session to another for a few weeks (53–67 days before the partial spinal lesion), the same stimulation current was used for the remainder of the study before and after both spinal lesions.

Reflexes were measured as detailed previously (Frigon and Rossignol 2007, 2008a–c, 2009). The EMGs were grouped into stimulated or control (nonstimulated) locomotor cycles. The locomotor cycle was divided into 10 equal bins by beginning the cycle from the left or right St (or Srt if St was lost) burst onset for reflexes evoked by stimulating the left and right SP nerves, respectively. Averaged EMG responses with stimulation were separated into these 10 bins according to the time in the cycle they were evoked. An average of ≥50 control cycles provided a template of baseline locomotor electromyography (bEMG) during the step cycle. The latency of responses, denoted as prominent negative or positive deflections away from the bEMG, was determined manually using predefined latencies as guidelines (Abraham et al. 1985; Drew and Rossignol 1985; Duyssens and Stein 1978; Loeb 1993; Pratt et al. 1991). In the present study, for quantitative analyses, we focused on short (P1)and longer (P2)- latency excitatory response evoked during the swing phase in flexors and extensors bilaterally.

Fixed windows were determined after study termination for each muscle in each cat and were used to quantify responses before and after spinal lesions. For ipsilateral responses, windows of ~10–25 and 25–55 ms were used for P1 and P2 responses, respectively. For the ipsilateral Srt or IP, a time window of 20–50 ms was used because these muscles often show a prolonged reflex response during that time period and not two distinct responses. For contralateral muscles a time window of ~20–50 ms was used before and after spinal lesions.

To measure reflexes, the stimulated and nonstimulated EMG within the determined window was integrated. The nonstimulated integrated EMG within the same window was then subtracted from the integrated stimulated value. Because reflex amplitude is known to scale (i.e., automatic gain control) with the level of EMG activity (Matthews 1986), the subtracted value was then divided by the integrated value of the nonstimulated EMG (see Fig. 1 of Frigon and Rossignol 2008c for an example), thus giving a reflex amplitude normalized to the level of baseline locomotor activity. Inhibitory responses, in our context, can only be quantified when there is a baseline level of EMG. Often the inhibitory response scales with the level of bEMG, but the percent inhibition relative to bEMG remains the same. To use a single method to compare inhibitory and excitatory responses in two conditions with different levels of bEMG, we divide the subtracted responses by the level of bEMG. Therefore reflex responses are expressed as a percentage of the level of baseline locomotor activity before and after spinal lesions. A change in normalized reflex responses over time indicates that reflex responses did not simply scale with changes in bEMG that occurred after the spinal lesions, indicating a change in the “strength” of the reflex pathway.

To determine if changes in cutaneous reflex responses in both hindlimbs were symmetric following the partial spinal lesion and complete transsection, we compared homologous responses between hindlimbs. The term “symmetry” or “asymmetry” is used to denote directional changes in reflex responses relative to control values (i.e., an increase or a decrease of reflex amplitude). The term “homologous” is used to denote responses in the same muscle in the left and right hindlimbs evoked by stimulating the left or right SP nerves. For example, P1 responses evoked in the left St by stimulating the left SP nerve is homologous to P1 responses evoked in the right St by stimulating the right SP nerve. Responses evoked and recorded on the same side are termed “ipsilateral” responses. The spinal lesion is always on the left side, and the term ipsilateral is never used to describe the lesion. Homologous responses were also quantified in crossed pathways. For example, responses evoked in the left VL by stimulating the right SP nerve are homologous to responses evoked in the right VL by stimulating the left SP nerve. Responses recorded on one side but evoked by stimulating the SP nerve on the contralateral side are termed “crossed” responses.

Spinal lesions and histological analysis

To perform the spinal hemisection, a small laminectomy was performed at T10 or T11. After removing the dura, a local anesthetic...
(xylocaine, 2%) was applied on the surface of the exposed spinal cord and then injected into the segment to prevent axon discharges and brisk movements during the lesion. Using a microknife under microscope, the left side of the spinal cord was sectioned and an absorbable hemostat (Surgicel, oxidized regenerated cellulose) was used to fill to the lesioned area to prevent axonal regrowth through the gap. The wound was then sutured. The same methodology was used for the complete spinal transection at T13.

Postmortem histological analysis (40-μm serial sections using cresyl violet) of the spinal cord was used to evaluate lesion extent. The initial goal was to perform a lateral spinal hemisection on the left side of the spinal cord, but as Fig. 1 shows the hemisection was not fully complete, and in some cases, there was damage to the right side as well. Thus we refer to the lesion as a partial spinal lesion. The serial section showing the most extensive damage is shown schematically in Fig. 1 for each cat to illustrate the extent of the partial spinal lesion.

Statistical analysis

To determine changes in reflex excitability over time, reflex responses pooled from the last 2–3 days before the partial lesion (i.e., in the intact state), evoked during the swing phase (i.e., the first 3 bins, see Fig. 3) were averaged and compared with each day postlesions (partial and complete) using a one-way ANOVA. A Dunnett’s post hoc test for many comparisons against a control group was performed if significant changes were detected by the ANOVA (Bouyer et al. 2001; Frigon and Rossignol 2008b). For example, P1 responses evoked during swing in the left St in the intact state were compared against P1 responses elicited during swing in the left St for all days after spinal lesions. Reflexes were only tested sporadically in cats GB6, GB7, and GB8 after the partial lesion and are not used in the analysis of reflex excitability over time. The same analysis was performed to determine changes in the stance/locomotor cycle ratio between the left and right hindlimbs. Only responses in which the homologue was present in the opposite hindlimb were retained for statistical analysis. Figures 5–8 show all homologous responses (24 in total) that were analyzed statistically in three animals.

We also compared reflex responses evoked on the last day before the spinalization with responses evoked during the first testing session following complete transection (i.e., ~1 wk after complete transection). This analysis was limited to ipsilateral P1 responses and crossed responses. Ipsilateral P2 responses in flexors such as St and TA were excluded because these responses are mostly abolished following the partial lesion. For the group, a one-way repeated-measures ANOVA was performed and, to determine specific differences for each response, we used unpaired t-tests. All values, unless otherwise stated, are means ± SE.

RESULTS

After a partial spinal lesion at T10–T11, all cats eventually regained a stable voluntary quadruped locomotion as shown previously (Barriére et al. 2008). The recovery of quadruped locomotion was associated with complex changes within cutaneous reflex pathways. Multiple changes in homologous reflex responses were asymmetric in direction between hindlimbs, particularly ipsilateral short-latency excitatory responses. After spinalization, hindlimb locomotion was evident within 24 h, and asymmetric reflex changes observed after the partial lesion persisted for some days and could be further modified with time.

Extent of partial spinal lesion

The extent of the partial spinal lesion of the left spinal cord at segments T10 or T11 is shown in Fig. 1. As can be seen, the lateral “hemisection” was nearly complete in cats GB7 and GB3, although some white matter was spared medioventrally. In cat GB8, the lesion was mostly confined to the left dorsolateral quadrant, whereas in cat GB6, there was extensive damage bilaterally to the dorsolateral spinal cord.

Asymmetry in the locomotor pattern

After the spinal lesions, cycle period was the same in both hindlimbs (1:1 rhythm), although there were changes in the duration of the locomotor cycle on both sides with time. However, the internal structure of the cycle could change differently on both sides. To quantify changes in the internal cycle structure after spinal lesions, the ratio of stance duration relative to locomotor cycle period between the left and right hindlimbs was measured over time. A ratio of 1 indicates a perfect symmetry, whereas a ratio <1 indicates that stance duration of the right (i.e., unlesioned side) hindlimb occupied a greater percentage of the locomotor cycle compared with the left hindlimb (Fig. 2). The left/right normalized stance ratio was significantly modified over time in each cat (P < 0.001, 1-way ANOVA). Cats GB6, GB7, and GB8 showed a similar profile of change over time. After the partial lesion, the ratio dropped <1, indicating that stance of the right hindlimb occupied a greater percentage of the locomotor cycle, which was most evident in cats GB7 and GB8, and returned toward control values over time. After spinalization, the ratio was >1 in all cats, indicating that stance of the left hindlimb occupied a greater percentage of the locomotor cycle. In cat GB8, the ratio dropped <1 over time after spinalization, whereas in the other cats, it remained >1. In cat GB3, there was no significant change of the ratio on any specific days.

Asymmetry in reflex responses after a partial spinal lesion

To determine changes in reflexes after spinal lesions, the left and right SP nerves were stimulated during locomotion before and after spinal lesions (i.e., partial and complete). In intact cats, stimulating the SP nerve evokes multi-phasic cutaneous reflex responses in muscles of the hindlimb ipsilateral and contralateral to the stimulation.

Some responses, not found in the intact state, appeared after the partial spinal lesion. For example, Fig. 3 shows the appearance of ipsilateral short-latency excitation, or P1, over time after the partial lesion in cat GB8 that was most prominent in the left LG. In the intact state, stimulating the left SP nerve during stance evoked short-latency inhibition, or N1, followed by longer-latency excitation, or P2, in ipsilateral extensors, such as LG. After the partial lesion, however, the inhibition could be replaced by short-latency excitatory responses. In cat GB8, the appearance of P1 responses during stance was evident by 29 days following the partial lesion in the left LG. Also note the appearance of P1 responses in the swing phase at 9 days after the partial lesion, coupled with the disappearance of P2 responses. At 29 days, P1 and P2 responses were prominent during swing, whereas stance was characterized by P1 responses and delayed N1 responses followed by P2 responses. Latencies of P1 responses during swing and stance were similar. With stimulation of the right SP nerve, ipsilateral responses during stance followed the usual N1/P2 pattern, and over time this pattern changed little despite the appearance of
a small “blip” (see open arrows), which might be considered weak P1 responses. Compared with the left ipsilateral LG, right ipsilateral P1 or P2 responses in LG were relatively absent during swing, a pattern that was maintained over time.

The rectified EMG burst, averaged from control locomotor cycles, for the left and right LG is shown for all days illustrated in Fig. 3, A and B, far right. The magnitude and duration of LG bursts, within the normalized locomotor cycle, changed little after the partial spinal lesion. Consequently, it is unlikely that the appearance of P1 responses was due to changes in the bursting behavior of LG during locomotion. In summary, Fig. 3 shows the appearance of short-latency excitatory responses during the stance phase of locomotion and a possible asymmetry in the time course of their appearance between hindlimbs.

The appearance of short-latency excitation during the stance phase was found in extensors of the left and/or right hindlimb of all cats and required a few weeks to develop (not shown). We had a small sample of homologous extensors, and more work is required to determine if the time course of appearance of P1 responses during stance is asymmetric between hindlimbs following incomplete SCI. We cannot pool all extensors (VL, MG, and LG) to make such a comparison because even close synergists, such as LG and MG, can show dramatically different reflex changes after spinal lesions (Frigon and Rossignol 2008a), probably owing to differential inputs from a given cutaneous nerve to motor pools of close synergists (LaBella et al. 1989), or to independent sources of modulation (Degtyarenko et al. 1996).

We previously described the existence of short-latency crossed inhibitory pathways in extensor muscles during locomotion in intact cats, and there was preliminary evidence that crossed inhibition was also present in flexor and bifunctional muscles (Frigon and Rossignol 2008c). In the present study, crossed inhibition became prominent in some flexor and bifunctional muscles after the partial spinal lesion. For example, Fig. 4 shows reflex responses evoked in the right (top) and left (bottom) St evoked by stimulating the left and right SP nerves, respectively. In the intact state, crossed responses, inhibitory or excitatory, in St are weak and not easily discernible. However, after the partial spinal lesion, a prominent short-latency crossed inhibition appeared. Crossed inhibition appeared very early on the right side (i.e., stimulation of left SP nerve) at ~13 days postpartial lesion, whereas on the left side (i.e., stimulation of right SP nerve), crossed inhibition developed more slowly. Thus as with the appearance of P1 responses in ipsilateral extensors, there appears to be an asymmetry in the time course of changes within crossed inhibitory pathways.

There were, however, prominent changes in the locomotor bursts of St bilaterally over time after the partial spinal lesion, as shown Fig. 4, A and B, far right. Locomotor bursts occupied a larger percentage of the normalized locomotor cycle after the partial spinal lesion. Inhibition cannot be revealed unless there is a background level of EMG and the widening of St bursts could be a contributing factor in the appearance of crossed inhibition. Crossed inhibition was found in “pure flexors,” such as TA and iliopsoas, in most cats (not shown). The appearance of crossed inhibition appears to be a robust effect, but again the
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**FIG. 3.** Appearance of short-latency excitatory responses (i.e., P1) in ipsilateral extensors after a partial spinal lesion in cat GB8. A: reflex responses in the left lateral gastrocnemius (LG), evoked by stimulating the left superficial peroneal (SP) nerve before and at 9 and 29 days postpartial spinal lesion. B: reflex responses in the right LG, evoked by stimulating the right SP nerve before and at 50 and 70 days after the partial spinal lesion. The locomotor cycle was divided into 10 equal bins, synchronized to the left sartorius (Srt) burst onset in A and to the right St burst onset in B. Thus in Fig. 2, A and B, the 1st horizontal trace for a given graph is the 1st bin of the locomotor cycle, which approximately corresponds to swing onset for the left and right hindlimb, respectively. Values in a given bin are at the same scale in microvolts (µV) before and after the partial spinal lesion. The dotted vertical line indicates the time of stimulation, whereas solid vertical lines delineate a time window of 10–25 ms, which is generally used for P1 responses. Each reflex trace is the average of ~10–40 stimuli. The template of background locomotor electromyography (biEMG) is the average of 100–150 nonstimulated cycles. The rectified, averaged, locomotor burst for the left and right LG are shown on the far right for all illustrated days, at the same scale. Open arrows in B are used to show the appearance of weak P1 responses.
small sample of homologous muscles prevents a more quantitative analysis of this phenomenon.

Evolution of reflexes after spinal lesions

To determine if cutaneous reflexes changed after spinal lesions, we plotted values of reflex responses evoked during the swing phase for selected muscles over time. Only reflex responses in muscles that retained a clear activity were retained for analysis. Figures 5–8 show changes in cutaneous reflexes over time with the dual-lesion paradigm. The dashed horizontal colored lines in each graph indicate the average of two to three control sessions before the partial spinal lesion (i.e., the intact state) for responses evoked in the left (i.e., left axes) and right (i.e., right axes) hindlimbs. There was a significant change in reflexes over time for all responses (1-way ANOVA, \( P < 0.001 \)). Responses that were significantly greater or smaller than the reference line (Dunnett’s test, \( P < 0.05 \)) are indicated by an asterisk of the corresponding color. Note that the scales for muscles of the left (left axes) and right (right axes) hindlimbs are different, with the exception of Figs. 6, B and C, 8C, which have identical y axes. The gray area in Figs. 5–8 is for days after spinalization to highlight retention and continuing reflex changes.

Partial lesion

Figure 5 shows ipsilateral short-latency excitatory responses (i.e., P1), evoked in St or TA bilaterally, in cats GB3 and GB7. The directional asymmetry in reflex changes relative to control
P2 reflex responses are largely abolished after a partial spinal lesion, which is maintained over time.

Figure 7 shows ipsilateral, short-latency excitatory responses, evoked in Srt or LG, bilaterally, in cats GB7, GB8, and GB3. In the left Srt of cat GB7 (Fig. 7A), responses decreased after the partial lesion relative to control values before showing a gradual increase. In the right Srt of cat GB7 (Fig. 7A), responses were mostly unchanged after the partial lesion compared with control values. Responses of the right Srt of cat GB8 (Fig. 7B: red triangles) decreased postpartial lesion, followed by a gradual increase. In the left Srt of cat GB8 (Fig. 7B: black circles), note that responses at a time window of (20–50 ms) were inhibitory, and for simplicity, the reference line is at −100%. After the partial lesion, large excitatory responses appeared showing why responses move above the 0

values between homologous muscles after the partial spinal lesion is clear in all cases. For example, the left St (Fig. 5A) and right TA (B) of cat GB3 and the right TA (C) of cat GB7 increased over time after the partial lesion, relative to control values. On the other hand, the right St (Fig. 5A) and left TA (B) of cat GB3 and left TA (C) of cat GB7 decreased early after the partial lesion. Thus reflex changes in St and TA are largely asymmetric in direction between hindlimbs after a partial lesion of the spinal cord.

Figure 6 shows ipsilateral longer-latency excitatory responses (i.e., P2), evoked in St or TA bilaterally, in cats GB3 and GB7. In all muscles, P2 responses were decreased postpartial lesion relative to control values and tended to remain decreased following complete transection. However, in cat GB7, P2 responses of the left TA (Fig. 6C; black circles) returned to intact values late after the partial lesion. Therefore

![Figure 5](https://example.com/image1.png)

**FIG. 5.** Changes in ipsilateral P1 responses during the swing phase in St and tibialis anterior (TA) after spinal lesions in cats GB3 and GB7. The dotted blue and black lines indicate the reference lines (i.e., 100% of control values) for responses in the left hindlimb, whereas dotted green and red lines indicate the reference lines for responses in the right hindlimb. Blue and black asterisks indicate significant differences from control values (Dunnett’s test, \( P < 0.05 \)) for the left St and TA, respectively. Green and red asterisks indicate significant differences from control values (Dunnett’s test, \( P < 0.05 \)) for the right St and TA, respectively. Each data point is the average of ~30–100 P1 responses evoked during the swing phase.

![Figure 6](https://example.com/image2.png)

**FIG. 6.** Changes in ipsilateral P2 responses during the swing phase in St and TA after spinal lesions in cats GB3 and GB7. The dotted blue and black lines indicate the reference lines (i.e., 100% of control values) for responses in the left hindlimb, whereas dotted green and red lines indicate the reference lines for responses in the right hindlimb. Blue and black asterisks indicate significant differences from control values (Dunnett’s test, \( P < 0.05 \)) for the left St and TA, respectively. Green and red asterisks indicate significant differences from control values (Dunnett’s test, \( P < 0.05 \)) for the right St and TA, respectively. Each data point is the average of ~30–100 P2 responses evoked during the swing phase.
increased after the partial lesion relative to control values. Crossed excitatory responses in the left and right Srt of cat GB7 (Fig. 8B) also showed a similar profile change post-partial lesion with a gradual increase followed by a return to control values over time. In cat GB8, changes in crossed responses of Srt bilaterally were largely asymmetric in direction (Fig. 8C). In the left Srt, crossed responses increased immediately after the partial lesion followed by a return to intact values, whereas, in the right Srt, crossed responses increased gradually, which was maintained over time. In all three homologous muscles tested, changes in crossed responses were larger in magnitude on the right side (i.e., sensory pathway coming from the left side). Thus contrary to ipsilateral reflexes, homologous crossed responses (e.g., VL and Srt of GB7) can display a directional change that is symmetric following a partial spinal lesion.

FIG. 7. Changes in ipsilateral P1 responses during the swing phase in Srt and LG after spinal lesions in cats GB7, GB8, and GB3. The dotted blue and black lines indicate the reference lines (i.e., 100% of control values) for responses in the left hindlimb, whereas dotted green and red lines indicate the reference lines for responses in the right hindlimb. Black and blue asterisks indicate significant differences from control values (Dunnett’s test, \( P < 0.05 \)) for the left Srt and LG, respectively. Red and green asterisks indicate significant differences from control values (Dunnett’s test, \( P < 0.05 \)) for the right Srt and LG, respectively. Each data point is the average of \( \sim 30–100 \) P1 responses evoked during the swing phase.

point before returning toward intact values. Short-latency excitatory responses evoked in LG during swing were also quantified in cat GB3 (Fig. 7C). On the left and right sides, P1 responses were decreased in the first few days postpartial lesion relative to control values. On the left side, responses remained decreased over time, whereas on the right side, responses increased before returning to intact values. Thus similar to changes in ipsilateral short-latency excitatory responses in St and TA (Fig. 5), responses in Srt and LG are largely asymmetric in direction, relative to control values, between hindlimbs after a partial lesion of the spinal cord.

Prominent cutaneous reflex responses are also evoked in the limb opposite the stimulation (i.e., crossed reflexes). Figure 8 shows crossed excitatory responses, evoked in VL or Srt, bilaterally, in cats GB7 and GB8. Crossed excitatory responses in the left and right VL of cat GB7 (Fig. 8A) gradually
Complete spinal transection

Figures 5–8 also show the evolution of reflexes over time after complete spinal transection (gray area). In many ipsilateral responses, there was a bilateral increase. This was the case for P1 responses in St, TA, and LG of cat GB3 (Figs. 5, A and B, and 7C, respectively) and Srt of cats GB7 and GB8 (Fig. 7, A and B, respectively). For P2 responses of St and TA, responses remained mostly abolished after spinalization (Fig. 6). In cat GB3, P2 responses of the left St (Fig. 6A) increased early after complete transection before returning toward control values.

Figure 9 provides a summary of all reflex response values shown in Figs. 5–8 for recording sessions just before and after spinalization, except for P2 responses in St and TA. For the group, there was no significant difference between reflex responses for the last partial lesion day and the first spinalization session (P = 0.90, repeated-measures 1-way ANOVA). To determine specific differences for each response, we used unpaired t-tests. Overall, 11/18 reflex responses were altered with 6 showing an increase and 5 a decrease. Overall, increased responses after transection were very small, whereas decreases could be more substantial.

Therefore after complete transection, reflex changes incurred after a partial spinal lesion, were largely retained or slightly increased (13/18 responses). Over time after complete transection, cutaneous reflexes continued to evolve, and these changes were largely symmetric in direction (i.e., an increase relative to control values).

DISCUSSION

Following a mostly unilateral partial spinal lesion, the internal structure of the locomotor cycle became asymmetric in some cats. At the same time, cutaneous reflexes evoked during locomotion by stimulating SP nerves bilaterally, were considerably altered, with some changes exceeding 1,000% of control values. The vast majority of changes in homologous reflex responses were asymmetric in direction following the partial lesion, and these changes were largely retained early after complete spinal transection. However, a few days after spinalization, reflexes could change, and these changes were mostly symmetric in direction. To our knowledge, this is the first study to investigate changes in reflex pathways during locomotion in the same cat after a partial spinal lesion and the retention of such changes following a complete transection of the spinal cord.

Methodological considerations

We strongly believe that our method of recording reflexes in the same animal before and after a given lesion is the best approach to study changes incurred after injury. First it minimizes the number of experimental animals used because each cat is its own control. Second, it permits investigating the evolution of reflex changes across time in the same animal and not solely at specific time points. Third, chronic implantations enable testing during normal movement, such as locomotion. Finally, as stated in the INTRODUCTION, reflexes can be highly variable from one cat (Loeb 1993) or human (Zehr et al. 1997) to another, which probably extends to most species, including rats and mice. Moreover, variability in reflexes is not confined to inter-individual variability because there is evidence that homologous reflexes between hindlimbs are not the same in intact cats (Hultborn and Malmsten 1983a). If an asymmetry between homologous reflexes is present in both hindlimbs in the intact state, which appears to be the case, a lateral hemisection should further compound this asymmetry because of asymmetric descending influences from structures rostral to the partial spinal lesion. Add to this interanimal variability in cutaneous reflexes (Loeb 1993), and making interpretations between control and lesioned groups becomes increasingly problematic if recordings are not made before and after a given lesion in the same animal.

In addition, despite our best efforts, performing a perfect lateral hemisection is not feasible (see Fig. 1) because of uncontrollable secondary damage. Consequently, varying amounts of spared matter and morphological changes over time could have profound effects on reflex changes. In the present study, we do not attempt to draw parallels between the extent of the partial lesion and changes in cutaneous reflexes. With the present approach, changes in reflex pathways after a partial spinal lesion cannot be attributed to specific structures, and more work is required to elucidate the relative contribution of different structures in the observed changes.

One potential limitation of using chronic recordings in the same animal before and after injury is that results must be expressed on a case-by-case basis. However, this does not weaken the overall impact of the results. On the contrary, using a case-by-case analysis permits consistencies between cats to be revealed while highlighting unique adaptive strategies. Unique adaptive strategies, or interanimal variability, do not indicate that reflex changes after incomplete SCI are not involved in locomotor recovery. It illustrates that multiple compensatory mechanisms can produce the desired outcome (Marder and Goaillard 2006), in this case, an adapted locomotion. Variability in the intrinsic circuitry and adaptive mechanisms should not be dismissed as experimental error but...
explored further because they constitute inherent components of how networks operate and adapt to different situations, including SCI (Gorassini et al. 2009).

Recording reflexes over time for a period ranging from 6 mo to 1 yr is not trivial in nature. Over time, some EMG recordings are lost, and re-implanting a muscle after a lesion is not an option because the same part of the muscle will not be sampled. Despite this problem, we obtained a fairly large number of homologous reflex responses between hindlimbs in three cats to make general conclusions regarding reflex changes using the dual lesion paradigm.

Asymmetry in the locomotor pattern and reflex changes after partial spinal lesion

After a partial lesion that damaged primarily the left side of the spinal cord (Fig. 1), although both hindlimbs operated at the same rhythm, there were some asymmetries in the internal structure of the locomotor cycle between limbs (Fig. 2). For example, percent time spent in stance by the left hindlimb decreased compared with the right hindlimb as shown previously (Helgren and Goldberger 1993). Early after complete spinal transection, this pattern reversed with the left hindlimb contacting the support surface a greater percentage of the locomotor cycle than the right hindlimb. Reversal of the asymmetry suggests that descending inputs were involved in maintaining the initial shift to the right hindlimb after incomplete SCI. After spinalization, the bias within the spinal locomotor circuitry was removed, and the pattern shifted to the left hindlimb. Over time after complete transection, the pattern could change.

Asymmetry in locomotion, after the partial spinal lesion, was paralleled by asymmetric changes (i.e., the direction of change relative to control values) in homologous P1 responses. For example, ipsilateral P1 responses in St and TA increased on one side, while decreasing on the other (Fig. 5, A–C). Over time, reflexes could remain altered or return toward intact values. Early after spinalization, asymmetric reflex changes were largely retained (Fig. 9). This “memory trace” within spinal circuits (Wolpaw and Lee 1989) could be a major contributing factor in the immediate locomotion observed after complete spinal transection in cats with a partial spinal lesion (Barriere et al. 2008). As the animal is voluntarily attempting to regain hindlimb function following incomplete SCI, asymmetric changes are induced within the spinal CPG via spared descending motor pathways, which is reflected by asymmetric changes within cutaneous reflex pathways during locomotion. Early after spinalization, changes within the spinal cord are largely retained and animals can display hindlimb locomotion within hours.

In a series of studies, Hultborn and Malmsten investigated reflex changes by recording ventral root activity during an acute terminal experiment in cats and rats with a chronic hemisection of the left spinal cord (Hultborn and Malmsten 1983a,b; Malmsten 1983). In the terminal experiment, a complete spinal transection was performed one segment below the hemisection to eliminate asymmetric descending influences from rostral structures. They found that most ipsilateral reflexes, including those evoked by stimulating the SP nerve, were greater on the hemisected side (Hultborn and Malmsten 1983a), indicating that persistent changes occurred within spinal circuits.

After complete spinal transection, however, reflexes do not remain fixed entities. On the contrary, after ~1 wk, some cutaneous reflexes displayed considerable changes, which were largely symmetric in direction (Figs. 5 and 7). Hultborn and Malmsten (1983a) also reported that reflex responses increased bilaterally in chronic spinal hemisected cats after complete spinal transection in their acute experiment. Removing tonic descending inhibitory influences that project bilaterally within the spinal cord could be responsible for bilateral reflex increases due to a strong reticulospinal inhibition of some spinal reflex pathways in decerebrate animals (Holmqvist and Lundberg 1959, 1961). However, in the present study, large increases in reflexes after complete spinal transection were not observed immediately after spinalization, which argues against a simple release from inhibition. Continuing changes in cutaneous reflexes after complete spinal transection could be critical in ensuring sufficient excitability within spinal circuits and in further improving locomotion.

Not all changes in reflex responses were asymmetric in direction following the partial spinal lesion. For example, ipsilateral P2 responses in St and TA decreased symmetrically (Fig. 6, A–C). Previously, we showed that ipsilateral P2 responses were decreased following a complete transection of the spinal cord (Frigon and Rossignol 2008a), and we suggested that pathways mediating P2 responses in ipsilateral flexors, contrary to ipsilateral extensors, rely heavily on supraspinal contributions because spino-bulbo-spinal reflex pathways project primarily to flexors (Shimamura et al. 1991). Symmetric decreases in P2 responses in both hindlimbs suggest that supraspinal pathways project bilaterally to interneurons intercalated in P2 pathways. Crossed responses could also change symmetrically after partial spinal lesions (e.g., Fig. 8, A and B). In all cases, the magnitude of change was greater for responses of the left hindlimb (Fig. 8). Hultborn and Malmsten (1983a) also showed that crossed responses on the hemisected side were larger than their counterparts in the other limb.

Functional plasticity of reflex pathways

Due to the multitude of interconnections within the nervous system (Sherrington 1906), injury at any level causes functional reconfiguration within the spinal circuitry that can subsequently be accompanied by structural changes (Maier and Schwab 2006). Consequently, the synaptic weight of excitatory and inhibitory connections within the spinal cord changes and some pathways, not present in the intact state, can be uncovered, as is the case for short-latency excitation in ipsilateral extensors during stance. Short-latency excitatory responses are generally absent during stance but present during swing. However, over time, after incomplete spinal cord lesions or complete spinal transection (Frigon and Rossignol 2008a), P1 responses appear during stance (Fig. 3). The existence of short-latency excitatory and inhibitory reflex pathways to flexor and extensor motoneurons was demonstrated in anesthetized or paralyzed animals (Forssberg et al. 1975; LaBella and McCrea 1990; Quevedo et al. 2005; Wilson 1963), and evidence is building that various pathways, difficult to activate in otherwise intact cats can be uncovered after SCI and/or peripheral nerve lesions (Frigon and Rossignol 2007, 2008a–c).
Functional reflex changes after incomplete SCI undoubtedly result from altered interactions between different levels of the nervous system, but some interactions might be relatively more important. For instance, the corticospinal tract and sensorimotor cortex are critical in inducing operant conditioning of the soleus H-reflex in rats (Chen and Wolpaw 2002; Chen et al. 2002, 2006), and supraspinal inputs influence cutaneous reflex excitability in anesthetized cats (Fleischer et al. 1988) and in intact cats during locomotion (Bouyer and Rossignol 2003a; Bretzner and Drew 2005a). Cerebellar lesions in dogs and rabbits also generate reflex asymmetries within the spinal cord, which are retained following complete spinal transection (reviewed in Patterson 2001). After incomplete SCI, supraspinal inputs to spinal circuits are asymmetric, which could induce disproportionate bilateral changes within the spinal cord to ensure symmetrical gait. Conversely, there could be a dampening of supraspinal influences so that the spinal CPG “learns” to function more autonomously.

Propriospinal neurons are used to relay locomotor command signals from the brain stem to the lumbarosacral spinal cord (Cowley et al. 2008; Zaporozhets et al. 2006), and the propriospinal circuitry coupling cervical and lumbarosacral spinal segments could play a role in reflex changes after SCI. Movement of the arms influences reflex excitability in the legs (Frigon et al. 2004) and vice versa (Balter and Zehr 2007). Functional changes in propriospinal pathways coupling fore- and hindlimbs correlated with the voluntary recovery of hindlimb locomotion, and/or posture, following lateral hemisection in cats and mice (Courtine et al. 2008; Kato et al. 1985). Changes in cutaneous reflexes could also result from changes in other segmental reflex pathways (Frigon and Rossignol 2006; Hultborn and Malmsten 1983a,b). Reflex pathways have powerful influences on one another, at rest and during locomotion, and disrupting their control changes segmental interactions (Pierrot-Deseilligny and Burke 2005).

Functional significance and clinical implications

At present, an effective approach to facilitate locomotor recovery after SCI is to train patients on a motorized treadmill. Patients are suspended with a harness, and therapists move the legs to provide sensory cues consistent with normal walking (Gorassini et al. 2009; Harkema 2001). In effect, reflex pathways entrain the spinal locomotor CPG to function more autonomously with diminished inputs from supraspinal structures. However, not all SCI patients respond to treadmill training (Gorassini et al. 2009). Individuals that respond show changes in their pattern of muscle activity following treadmill training that are vastly different from nonresponders (Gorassini et al. 2009). Variable changes in reflex pathways from one individual to another after incomplete SCI could explain why some will respond to training while others will not.

Recently, we showed that there was considerable variability in the recovery of locomotion after complete spinal transection in cats that had a partial denervation of ankle extensors in the intact state (Frigon and Rossignol 2009). Specifically, in two of three cats denervated prior to spinalization, the ability for hindlimb locomotion after complete spinal transection was either severely compromised or nonexistent. In the two cats that showed spinal locomotion, some changes in reflex pathways, particularly to knee and ankle flexors, were not the same as in cats spinalized but without denervation (Frigon and Rossignol 2008a). Together, these data suggest that persistent changes within the spinal circuitry, initially incurred after denervation, adversely influenced the ability to express hindlimb locomotion after complete spinal transection, by preventing “normal” changes in reflex pathways or in how reflexes interact with the spinal CPG, which could be a prerequisite for locomotor recovery after SCI. In the present study, all cats walked fairly well after the partial spinal lesion, which was a prerequisite to evaluate how reflex changes evolve after incomplete SCI and complete spinal transection.

Future studies could actively modify reflex changes to determine if this is beneficial or detrimental to locomotor recovery after incomplete SCI. One approach would be to use classical conditioning and induce precise changes in reflex pathways (Dupovic 1975, 1983; Durkovic and Damianoopoulos 1986) during locomotion. Specific reflex changes might also predict whether animals or humans recover the ability to walk after incomplete SCI.

Large changes in cutaneous reflexes observed in the present study during locomotion highlight the role of sensory inputs from the skin of the foot following incomplete SCI. It was shown that cutaneous inputs from the hindpaws are particularly critical for spinal locomotion (Bouyer and Rossignol 2003b) and that phasic cutaneous inputs from the foot can facilitate locomotor recovery following incomplete SCI (Muir and Steeves 1995). Changes in cutaneous reflexes could partially offset the loss of excitability normally provided by supraspinal structures and might be critical for locomotor recovery. A reorganization of spinal circuits following SCI could also be a requirement enabling descending motor pathways to more easily re-establish connections with spinal motor programs, thus allowing voluntary control of motor functions. Further investigations are required to determine changes in the interactions at multiple levels of the nervous system and how these impact locomotor recovery after incomplete SCI. The present study highlighted the role of cutaneous reflexes from the dorsal surface of the foot but multiple reflex pathways that interact with one another, with the spinal CPG, and with supraspinal structures are undoubtedly involved in optimizing remnant locomotor functions following incomplete SCI.

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