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Impairment and Recovery of Postural Control in Rabbits With Spinal Cord Lesions

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Lyalka, V. F., P. V. Zelenin, A. Karayannidou, G. N. Orlovsky, S. Grillner, and T. G. Deliagina. Impairment and recovery of postural control in rabbits with spinal cord lesions. *J Neurophysiol* 94: 3677–3690, 2005. First published July 27, 2005; doi:10.1152/jn.00538.2005. The aim of this study was to characterize impairment and subsequent recovery of postural control after spinal cord injuries. Experiments were carried out on rabbits with three types of lesion—a dorsal (D), lateral (L), or ventral (V) hemisection (HS) at T_{12} level. The animals were maintaining equilibrium on a platform periodically tilted in the frontal plane. We assessed the postural limb/trunk configuration from video recordings and postural reflexes in the hindquarters from kinematical and electromyographic (EMG) recordings. We found that for a few days after DHS or LHS, the animals were not able to maintain the dorsal-side-up position of their hindquarters. This ability was then gradually restored, and the dynamic postural reflexes reached the prelesion value within 2–3 wk. By contrast, a VHS almost completely abolished postural reflexes, and they did not recover for ≥7 wk. The DHS, LHS, and VHS caused immediate and slowly compensated changes in the postural limb/trunk configuration as well as gradually developing changes. After DHS, both hind limbs were placed in an abnormal rostral and medial position. After LHS, the limb on the undamaged side was turned inward and occurred at the abnormal medial position; LHS also caused a gradually developing twisting of the caudal trunk. VHS caused gradually developing extension of the ankle and knee joints. These findings show that ventral spinal pathways are of crucial importance for postural control. When a part of these pathways is spared, postural reflexes can be restored rapidly, but not the postural limb/trunk configuration. Spinal and supraspinal mechanisms responsible for postural deficits and their compensation are discussed.

INTRODUCTION

Maintenance of the basic body posture, upright in humans and dorsal-side-up in quadrupeds, is a vital motor function. Maintenance of this posture largely is a nonvolitional activity based on inborn neural mechanisms (Massion 1998). An efficient control of the basic posture is equally important for standing and walking (Horak and Macpherson 1996; Macpherson et al. 1997a; Orlovsky et al. 1999) as well as for providing support of voluntary movements (Massion and Dufosse 1988).

Damage to descending and ascending spinal pathways, caused by a spinal cord injury (SCI), results in a dramatic impairment of the postural system. This affects not only equilibrium when standing but also locomotion and voluntary movements that need postural support. Restoration of postural control is necessary for the recovery of other motor functions. Unfortunately the effects of SCI on postural mechanisms, as well as the recovery of postural functions after SCI, have received much less attention than the effects of SCI on locomotion (see e.g., Barbeau et al. 2002; Edgerton et al. 2004; Rossignol et al. 2002). Reasons for this discrepancy include the complexity of the postural system and a lack of detailed information on the organization of neural postural mechanisms. A general goal of the present study was to characterize the effect of selective lesions of the spinal cord on the postural system in an animal model, the rabbit, and to assess the adaptive capacities of this system.

Postural deficits depend on the location and degree of impairment of the spinal cord. A complete lesion of the spinal cord in the thoracic region isolates the spinal postural mechanisms of the hindquarters from the rest of the CNS. This has dramatic consequences. Studies on chronic spinal cats have shown that they exhibit very poor responses to disturbances of posture and as a rule are not able to maintain the dorsal-side-up orientation of the caudal part of their body (Macpherson and Fung 1999; Rossignol et al. 1999, 2002), although a reduced postural control may remain, and it can be improved by training (see e.g., Edgerton et al. 2001, 2004; Grillner 1973).

There are two major ways to account for the poor postural performance in the hindquarters after a complete lesion of the spinal cord. First, in the absence of a supraspinal excitatory tonic drive, the spinal postural reflexes are not sufficiently activated, and they cannot compensate for postural disturbances. Also the postural mechanisms of the hind limbs cannot coordinate their activity with the position of the anterior part of the body. Second, the spinal cord transection leads to a complete destruction of the spino-brain stem-spinal postural reflexes. The relative importance of these two factors is not clear, however (for discussion, see Deliagina and Orlovsky 2002; Deliagina et al. 2000; Horak and Macpherson 1996; Macpherson et al. 1997b).

In humans, the percentage of partial spinal lesions is high (Tator et al. 1993), and there is a need for a better understanding of such conditions using animal models. However, in contrast to complete SCI, the data on the effects of partial SCI on postural functions in quadrupeds are rather scarce. These effects have been roughly estimated for one type of lesion—the lateral hemisection of the spinal cord at the lower thoracic level. From the reports about locomotion of these animals, one can conclude that this type of SCI causes much less severe distortions of postural performance than a complete transection of the spinal cord. Cats subjected to a lateral hemisection, after some period of recovery, were able to maintain equilibrium of their hindquarters during locomotion and standing (Deliagina

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1977; Helgren and Goldberger 1993; Hultborn and Malmsten 1983; Kuhtz et al. 1996). A partial recovery of postural responses was also observed by Kato et al. (1985) in animals with two lateral hemisections performed on the opposite sides and at different levels.

A deterioration of lateral stability of the hindquarters was observed during locomotion in cats with ventral and ventrolateral spinal lesions; a loss of balance being correlated with the extent of the lesion (Brustein and Rossignol 1998, 1999). Dorsal spinal lesions caused only transient deficits in postural stability during locomotion (Jiang and Drew 1996). However, a quantitative analysis of postural deficits caused by the ventral and dorsal lesions was lacking in these studies.

In the present study, we used an animal model (rabbit) for which the functional organization of postural mechanisms has been characterized in our previous studies (Beloozerova et al. 2003b; Deliagina et al. 2000). In those studies, the rabbit was standing on a platform and maintained balance when the platform was tilted periodically in the frontal plane. It was found that a reaction to tilt included an extension of the limbs on the side moving down and a flexion on the opposite side, rabbits could effectively maintain the dorsal-side-up body posture when postural stimuli were applied separately to the anterior and posterior parts of the body, suggesting that the nervous mechanisms controlling positions of these parts could operate independently of each other, and somatosensory input from the limbs played a predominant role for the generation of postural responses to tilts.

Based on this experimental model and on the results of our previous studies, we analyzed the postural performance in rabbits with different lesions of the spinal cord—the lateral, dorsal, or ventral hemisection (LHS, DHS, and VHS, respectively) performed in the lower thoracic region. We characterized effects of spinal lesions on the balance control in the hindquarters and subsequent recuperation of postural functions. Special attention was given to the two principal aspects of postural control—the maintenance of limb/trunk postural configuration, and the postural reflexes that compensate for postural disturbances (Horak and Macpherson 1996). The main result of this study was that VHS caused much more severe damage of postural performance than LHS and DHS, suggesting an important role of the reticulo- and vestibulospinal pathways in postural control.

A brief account of part of this study has been published in abstract form (Lyalka et al. 2004).

METHODS

Experiments were carried out on 17 adult male New Zealand rabbits (weighting 2.5–3.5 kg). All experiments were conducted with the approval of the local ethical committee (Norra Djurförsöksetiska Nämnden) in Stockholm.

Surgical procedures

Each animal was subjected to two operations under Hypnorm-midazolam anesthesia, using aseptic procedures. During the first surgery, bipolar EMG electrodes (0.2 mm flexible stainless steel Teflon-insulated wires) were implanted bilaterally into muscle gastrocnemius (Gast, ankle extensor), m. vastus (Vast, knee extensor), and m. biceps femoris (Bic, knee flexor). The wires were led subcutaneously toward the head and then through a small incision in the skin on the dorsal aspect of the neck. The wound was sutured so that the wires were fastened to the skin. A small connector was soldered to each wire at a distance of 2–3 cm from the skin.

In 3–4 days, when the animal had recovered completely from the first surgery, its postural responses to tilts were tested (see following text), and afterward a second surgery was performed. An incision was made along the dorsal midline in the lower thoracic region. A laminectomy at T_{12} level was performed, and the dura was opened on the dorsum of the cord in T_{12} segment. A lesion of the spinal cord was performed under the dissecting microscope by means of spring scissors, microsurgery forceps, and small scalpel. For ventral lesions, we used a different technique. By means of a small surgical needle, a thin thread was led through the cord, from one side to the other, at half of its depth. The thread was then led back under the cord to form a loop with a knot. By tightening the loop progressively, we caused a lesion of the ventral part of the cord. Afterward, the incision was closed in anatomical layers.

Experimental design

Postural tests on a tilting platform have been described earlier (Beloozerova et al. 2003b, 2005; Deliagina et al. 2000). No special training of the rabbits was required prior to testing. For testing, an animal was positioned on the two platforms (P1 and P2 in Fig. 1*A*), so that P1 supported the fore limbs, and P2 supported the hind limbs. The sagittal plane of the animal was aligned to the axis of the platform rotation (Fig. 1, *B* and *C*). The surface of the platforms was covered with sandpaper to prevent sliding of the animal during tilts.

When standing, rabbits can adopt a variety of postures with different distance between the fore and hind limb feet. In the present experiments, intact animals usually kept this distance within the range of 7–12 cm (as in Fig. 1*A*). At such distances, the spine was not bent, and there was ~1–2 cm of clearance between the belly and the ground. If the inter-limb distance noticeably differed from the optimal value, we repositioned the limbs.

The platforms supporting the rabbit could be tilted in the frontal (transverse) plane of the animal (angles $\alpha 1$ and $\alpha 2$, Fig. 1, *A* and *C*). The two platforms were tilted together, or only P2 was tilted while P1 was kept horizontal. The latter test allowed postural disturbances to be applied primarily to the hindquarters. Two types of tilt trajectories were used: a sine-like trajectory (Fig. 1*D*) with a frequency of 1 Hz and a trapezoidal trajectory (Fig. 1*E*) with transitions between stationary (extreme) positions lasting for 0.5–0.7 s and with each position being maintained for 2–3 s. In both sinusoidal and trapezoidal tests, tilts were symmetrical in relation to the horizontal position, with the peak-to-peak value of 40°. Smaller values (20 or 30°) were used only if an animal with a spinal lesion could not compensate for 40° peak-to-peak tilts.

In a previous study (Beloozerova et al. 2003b), it was shown that lateral displacements of the trunk in relation to the platform characterized the efficacy of stabilization of the dorsal side-up trunk position. In the present study, lateral displacements of the rostral and caudal parts of the trunk in relation to the corresponding platform (postural corrections) were monitored separately by the sensors S1 and S2, respectively. The sensors were positioned in the corresponding areas, at the half-height of the body (Fig. 1, *A–C*). Each sensor consisted of a high-resolution variable resistor the axis of which was rotated by means of a long lever; the latter was touching the lateral aspect of the body.

To characterize the body configuration in the standing animal, video recordings (25 frames/s) from the side or from the top were performed on the horizontal platform. The fur on the limbs and on the back of the animals was shaved, and black paper stickers were attached to the skin projections of the main hind limb joints (see Fig. 10*A*). Seven stickers were attached along the midline of the spine, neck, and head (see Fig. 8*C*). In addition, a view from below was video recorded using a transparent horizontal platform and a mirror

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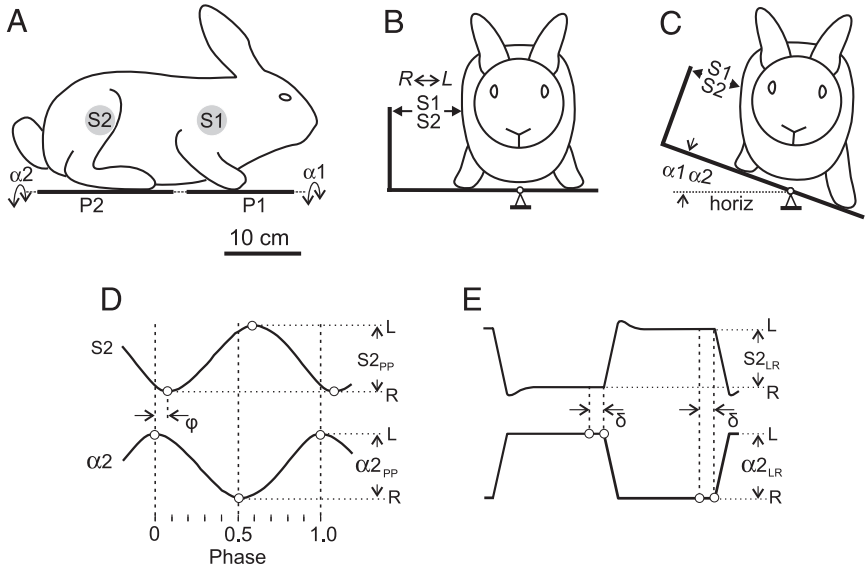

FIG. 1. Experimental design for testing postural responses to tilts. *A–C*: animal was standing on 2 platforms, 1 under the fore limbs (P1) and 1 under the hind limbs (P2). Each platform could be tilted in the transverse plane ($\alpha 1$ and $\alpha 2$, platform tilt angles). The sagittal plane of the animal was aligned to the axis of platforms rotation. Mechanical sensors S1 and S2, positioned at the half-height of the body, measured lateral displacements of the rostral and caudal parts of the trunk in relation to the corresponding platform. *D* and *E*: schematic representation of trajectories of the caudal platform angle ($\alpha 2$) and corrective movements of the caudal trunk (S2) in sinusoidal (*D*) and trapezoidal (*E*) tests. The following values were measured: $\alpha 2_{PP}$, the peak-to-peak value of tilt angle of the caudal platform; $S2_{PP}$, the peak-to-peak value of postural corrections in the hind quarters; φ, the phase shift between the peaks of $\alpha 2$ and S2 trajectories; $\alpha 2_{LR}$, the difference between the sustained tilt angles; $S2_{LR}$, the difference between the postural responses to sustained left and right tilts; the responses were measured and averaged during the last 0.5-s interval of a tilted position (δ).

(see Fig. 4 *A* and *B*). The video camera was positioned at a distance of ~2 m from the rabbit. The recordings were analyzed frame by frame. The resolution of video images was ~2 mm.

Recordings and data analysis

The signals from the EMG electrodes and from the position sensors were amplified, digitized with a sampling frequency of 5 kHz (EMGs) and 1 kHz (sensors), and recorded on a computer disk using the data-acquisition and analysis software (Power-1401/Spike-2, Cambridge Electronic Design, Cambridge, UK). The EMG signals were rectified and smoothed (time constant, 50 ms).

In tests with sinusoidal tilts, the EMG signals as well as the signals from mechanical sensors were averaged over 10–30 normalized successive tilt cycles (20 bins/cycle); the peak tilt to the left was taken as the cycle onset (Fig. 1*D*). To evaluate the postural performance in these tests, we calculated the gain of "dynamic" postural reflexes defined as: $GPR_D = S2_{PP}/\alpha 2_{PP}$ (cm/°), where $\alpha 2_{PP}$ is the peak-to-peak value of tilts of the caudal platform, and $S2_{PP}$ is the peak-to-peak value of postural corrections in the hindquarters (Fig. 1*D*). A phase delay of the postural corrective responses in relation to the postural stimuli was characterized by measuring the phase shift between the corresponding peaks (φ in Fig. 1*D*).

In tests with trapezoidal tilts, we characterized the gain for "static" components of postural responses: $GPR_S = S2_{LR}/\alpha 2_{LR}$ (cm/°), where $\alpha 2_{LR}$ is the difference between the sustained left and right tilt angles, and $S2_{LR}$ is the difference between the postural responses to sustained left and right tilts; the responses were measured and averaged during the last 0.5-s interval of a tilted position (δ in Fig. 1*E*).

All quantitative data in this study are presented as the means ± SD or SE. Student's *t*-test was used to characterize the statistical significance when comparing different means; the significance level was set at $P = 0.05$.

Histological procedures

At the termination of the experiment, rabbits were killed with pentobarbital sodium and perfused with isotonic saline followed by a 10% Formalin solution. Frozen sections of 30 μm thickness were cut in the region of spinal cord damage. The tissue was stained for Nissl substance with cresyl violet. The position and the extent of lesions were verified by observation of a series of magnified digital images of sections.

RESULTS

Evaluation of the extent of spinal lesions

Figure 2*A* shows the reconstructed lesion sites for five rabbits with dorsal lesions. In all cases, the dorsal columns and the dorsal part of the lateral funiculi were transected.

Figure 2*B* shows the reconstructed lesion sites for seven rabbits with lateral lesions. In all cases, the lesion occupied the most part of the gray and white matter on one side. However, in *rabbits 4, 5, 9,* and *15,* a small medial part of the ventral funiculus remained undamaged. In *rabbits 4, 9,* and *14,* a part of the dorsal column on the side of lesion was spared. By

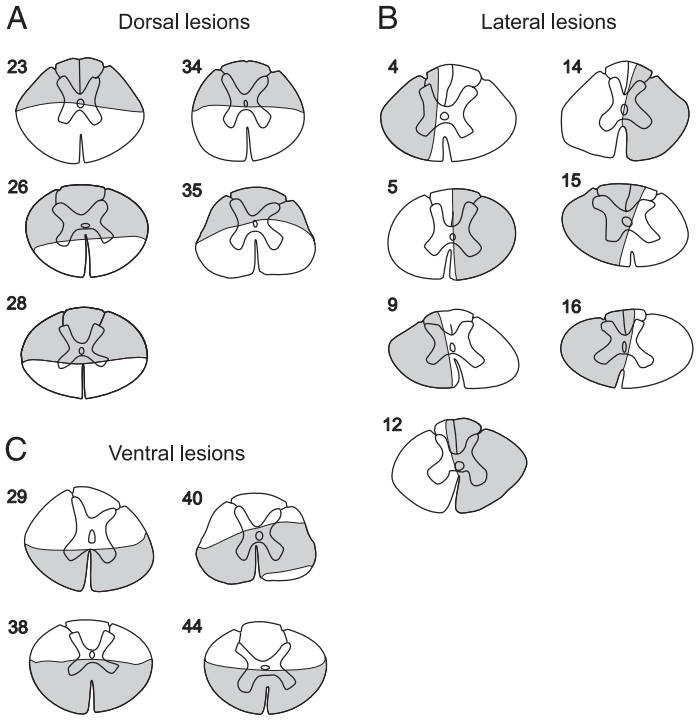

FIG. 2. Extent of the spinal cord damage in rabbits with dorsal lesions (*A*), lateral lesions (*B*), and ventral lesions (*C*). The total extent of the lesion is projected on a spinal cord section taken more rostrally after inspecting several consecutive sections.

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contrast, in *rabbits 12, 15,* and *16,* a part of the contralateral dorsal column was also damaged.

Figure 2*C* shows the reconstructed lesion sites for four rabbits with ventral lesions. In *rabbits 29, 38,* and *44,* the ventral funiculi and the ventral part of the lateral funiculi on both sides were transected. In *rabbit 40,* a strip of ventral funiculus on the right side remained undamaged. In *rabbit 41* (not illustrated), the ventral half of the spinal cord was undoubtedly destroyed, but histological control could not be done because of the damage to the spinal cord during histological procedures.

Because in each type of lesion, the damaged area occupied approximately a half of the spinal cord cross-section, in this paper, we use the term hemisection (lateral, dorsal, or ventral) to roughly characterize the type of lesion. A few animals, with an extent of lesion strongly differing from a hemisection, were excluded from the present analysis.

Postural performance in intact rabbits

KINEMATICAL DATA. The intact rabbits, intended for each type of spinal lesion (DHS, LHS, and VHS), served as controls when tested before the lesions. The rabbits were easily engaged in the main postural task, which was to balance on the platforms (P1 and P2 in Fig. 1*A*) that were periodically tilted together in the frontal plane. The animals exhibited stereotypic postural responses. A simultaneous tilt of the platforms caused an extension of both limbs on the side moving down and a flexion of both limbs on the opposite side, as shown schematically in Fig. 1, *B* and *C* (see also Beloozerova et al. 2003b; Deliagina et al. 2000). These flexion and extension limb movements made the trunk and head move in the transverse plane in relation to the platforms, in a direction opposite to the platform tilt. These corrective trunk movements reduced the deviation of the body from the dorsal-side-up position. The lateral displacements of the rostral and caudal parts of the trunk in relation to the platforms (monitored by the sensors S1 and S2, Fig. 1, *A–C*) are shown in Fig. 3*A* for parallel sinusoidal tilts of the two platforms. The corrective trunk movements (S1 and S2) were in-phase with each other and of similar amplitude. In relation to the platform tilting movements (α1 and α2), the corrective trunk movements occurred approximately in anti-phase. In some cases, however, a small shift of the peak movement in relation to the peak tilt was observed. In Fig. 3*A,* this shift is indicated by an arrow. Averaged over all intact rabbits, the shift was 0.02 ± 0.04 of the tilt cycle (mean ± SD).

In the tests with parallel trapezoidal tilts of the two platforms (Fig. 3*C*), the S1 and S2 trajectories also approximately mirrored the α1 and α2 trajectories. As in the test with sinusoidal tilts (Fig. 3*A*), the values of the postural corrections in the rostral and caudal parts of the trunk (S1 and S2) were similar.

Postural corrections in the hindquarters were also well expressed when only the caudal platform (P2) was tilted while the rostral platform (P1) was kept at the horizontal position and, therefore a posture of the rostral part of the body was not perturbed directly. This is illustrated for the trapezoidal tilts in Fig. 3*D.* One can see that the hindquarters compensated well for tilts of the caudal platform, and the S2 trajectory approximately mirrored the α2 trajectory.

To characterize postural performance in sinusoidal tests, we measured the gain of dynamic postural reflexes, GPR_D, whereas in trapezoidal tests, we measured the gain of the static component of postural reflexes, GPR_S (see METHODS). For the case illustrated in Fig. 3*A,* the GPR_D and GPR_S values were 0.12 and 0.11 cm/°, respectively, and the ratio of GPR_S to GPR_D was 0.92. Averaged over all intact animals, this ratio was 1.03 ± 0.25 (mean ± SD).

EMG DATA. In the previous study (Beloozerova et al. 2003b), it was shown that compensatory movements of the hind limbs were caused by a specific pattern of muscle activity. When the ipsilateral side of the platform was moving downward, the limb was extending due to activation of extensor muscles. When it was moving upward, the limb was flexing due to a reduction of the extensor activity and activation of flexor muscles. This result was reproduced in the present study and illustrated in Fig. 3*A,* where parallel sinusoidal tilts of the P1 and P2 platforms caused a periodical modulation of the EMGs of the left and right m. gastrocnemius (Gast, ankle extensor). There was increased activity during ipsilateral tilting and decreased activity during contralateral tilting. The peaks of activity on the left and right sides alternated. A different pattern was observed in m. biceps femoris (Bic, knee flexor), which peaked during contralateral tilting, that is, in anti-phase with Gast (Fig. 3*A*). The pattern similar to that in Gast was observed also in m. vastus (Vast, knee extensor). Figure 3*B* compares the EMG profiles for the three muscles of the right hind limb in one of the rabbits, averaged over 20 successive tilt cycles. The EMG patterns in both extensors (Gast and Vast) were very similar, with a peak slightly preceding the maximal ipsilateral tilt. In the flexor (Bic), the peak preceded slightly the maximal contralateral tilt.

Tests with trapezoidal tilts showed that, as in sinusoidal tests, the extensor muscles (Gast and Vast) were activated with the ipsilateral tilt, and the flexor muscles (Bic) with the contralateral tilt (Fig. 3, *C* and *D*). The EMGs had two components: a dynamic one with a peak during tilting and a static one with sustained activity when the tilted position was maintained. Typically the peak of the dynamic component was 1.5–2 times larger than the static component.

The phases of the EMG peaks were highly consistent across all subjects. Figures 7, *A* and *B,* show the peak position in the tilt cycle (±SE) for different muscles of the left and right hind limbs, averaged over each of the two groups of intact rabbits (controls for DHS and LHS). There was no statistically significant difference between the peak positions in the two groups (*t*-test). In contrast to the consistency of phase characteristics of EMG activity, the peak value of EMG activity and the degree of the tilt-related modulation were much more variable, both in repeated tests and even during the same test. For example, the mean value of the extensor EMG, or the amplitude of its periodic tilt-related modulation, or both values could spontaneously change ≤1.5–2 times. The EMGs of different extensors of a limb could exhibit these changes together or separately. It seems most likely that this variability was caused by changes in the postural body configuration not recorded here, such as small changes in the limb configuration or inter-foot distance. Because of the considerable variability of the EMG values in control tests, we did not use these characteristics for the evaluation of postural deficits caused by SCI.

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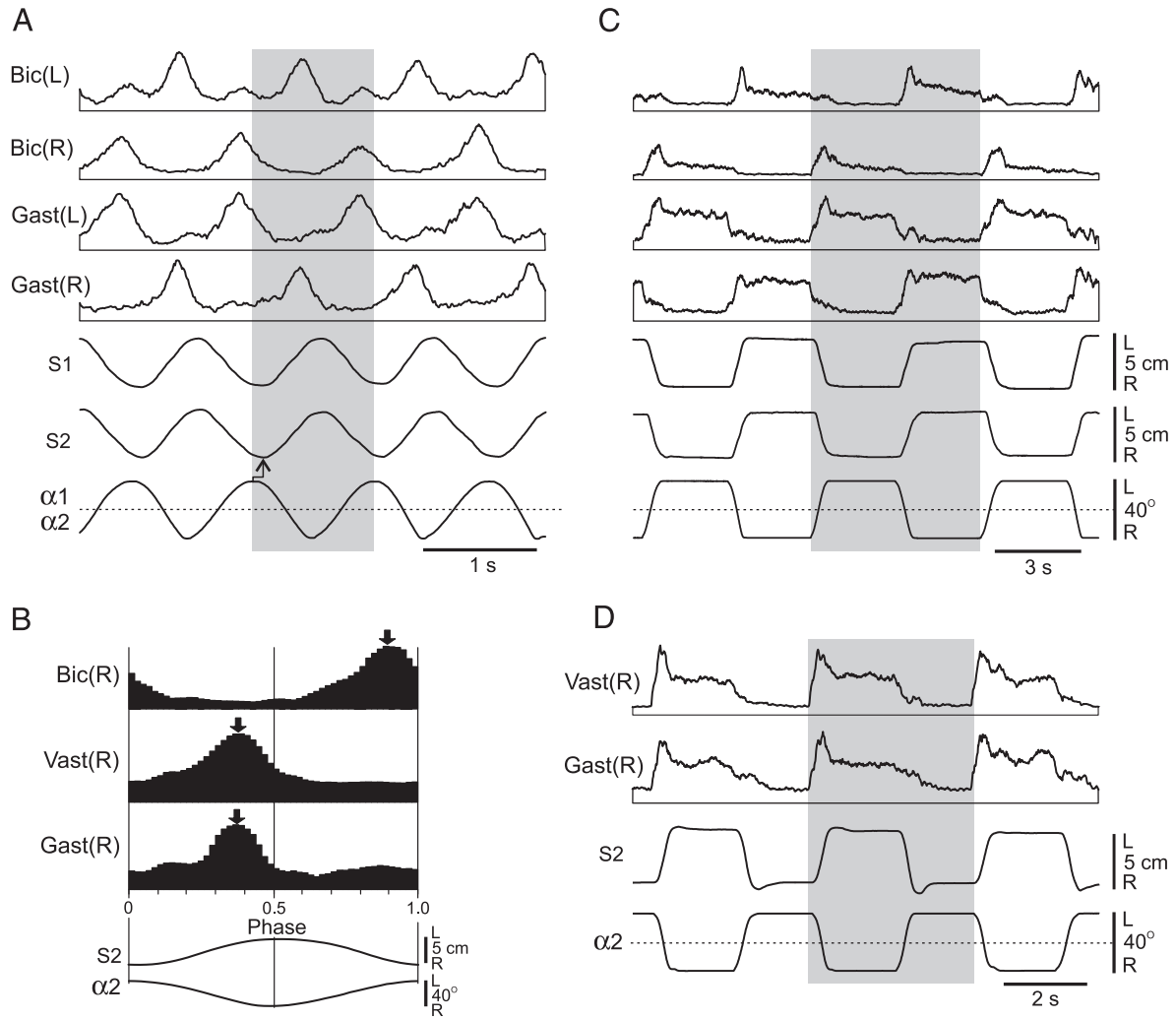

FIG. 3. Postural responses in intact rabbits. *A* and *B*: a representative example of responses to sinusoidal tilts of both platforms. *A*: kinematical and electromyographic (EMG) responses. *B*: an EMG profile for 3 muscles of the right hind limb averaged over 20 successive tilt cycles. The cycle was normalized, and the peak tilt to the left was taken as the cycle onset. ↓, the peak values of EMGs. *C*: responses to trapezoidal tilts of both platforms. *D*: responses to trapezoidal tilts of the posterior platform while the anterior platform was kept in the horizontal position. $\alpha 1$ and $\alpha 2$, tilt angles of the rostral and caudal platforms, respectively; S1 and S2, lateral displacements of the rostral and caudal parts of the trunk in relation to the corresponding platform. The EMGs of the following muscles were presented: left (L) and right (R) biceps femoris (Bic), gastrocnemius (Gast), and vastus (Vast). In *A, C,* and *D,* ▨, 1 of the tilt cycles to facilitate comparison between curves. In *A*, →, a phase shift between the α and S trajectories.

Effects of dorsal hemisection

The effects of dorsal hemisection of the spinal cord were studied in five rabbits (see Fig. 2*A* for the extent of lesion). Observations of these animals in the cage and on the floor have shown that, during some period after DHS, the rabbits were not able to maintain the dorsal-side-up orientation of their hindquarters. They moved around using their forelimbs only. When the animal was positioned on the tilting platform during this period, its hindquarters passively followed the platform movements and with larger tilts the rabbit could fall sideways. Later on, freely behaving animals managed, for short periods of time, to regain equilibrium and to maintain the dorsal-side-up position of their hindquarters. Starting from this moment, the rabbits were subjected to repeated tests that included video recording of the body configuration on the horizontal platform and recording of postural reflexes on the tilting platform. The period of inefficient postural control in the hindquarters lasted from 1 to 2 days postlesion with a mean value of 1.6 ± 0.2 (SE) days.

POSTURAL CONFIGURATION. As compared with control (Fig. 4*A*), DHS caused a considerable change in the position of the hind limbs in relation to the trunk. The effects of DHS are shown schematically in Fig. 4*B* and include a decrease of the distance between the left foot and the right foot, an abnormal rostral position of both feet, and a decrease of the angle between the feet. To characterize these effects, for each limb we measured the foot angle (ϵ) as well as the coordinates (x and y) of the rostral point of the foot in relation to the mid-body axes (Fig. 4*A*).

Figure 4*C* shows the distance and the angle between feet, as well as their anterior-posterior (A-P) position for one of the rabbits, in control (day 0) and on different days after DHS. The inter-foot distance was reduced by almost 50% in the first test after DHS. Later on, this distance gradually increased but did not reach the control value even on day 22 postlesion. The same relates to the inter-foot angle. Furthermore, in the first test after DHS the limbs appeared protracted forward by 4 cm

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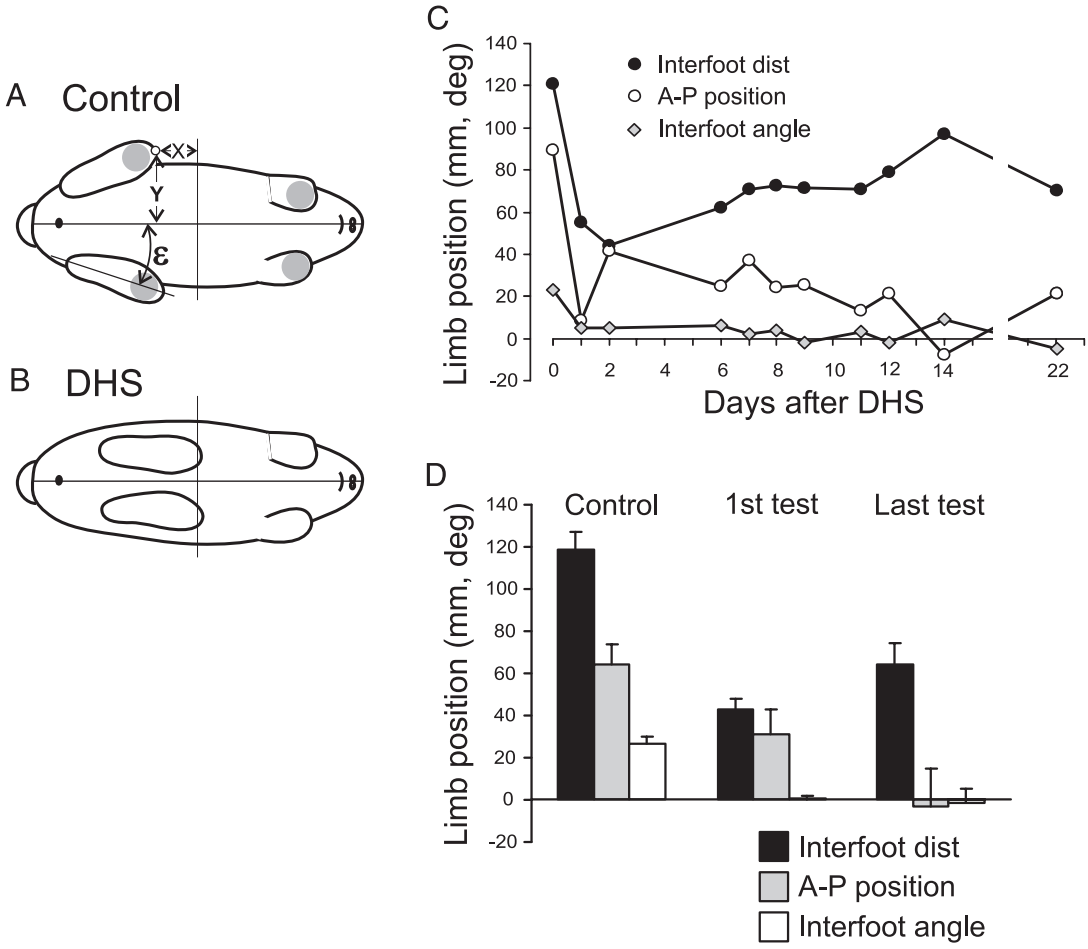

FIG. 4. Distortions of postural configuration caused by dorsal hemisection (DHS). *A*: postural configuration of the intact rabbit on the horizontal platform, view from below. ◎, the areas of foot-ground contacts. To characterize the position of each hind limb, the following values were measured: the foot angle (ϵ), as well as the coordinates (*x* and *y*) of the rostral point of the foot in relation to the mid-body axes. *B*: a schematic representation of distortions in the hind limbs configuration caused by DHS. *C*: foot position in the hind limbs of rabbit 14 in control (day 0) and on different days after DHS. The distance between the rostral points of feet, the angle between the feet, and the mean A-P position of the 2 feet are shown. *D*: foot position values averaged over the group of DHS animals; the values are presented for the control, for the 1st test after DHS, and for the last test (means ± SE). The differences between the corresponding values in control and after DHS were statistically significant.

(as compared with control). This protraction remained unchanged even on day 22.

The decrease of inter-foot distance and angle, and the abnormal rostral feet position, as well as a very slow compensation of these deficits (as illustrated in Fig. 4C) were observed in all five DHS rabbits. Figure 4*D* shows the population averages of the inter-foot distance, the inter-foot angle, and the A-P feet position in the control, in the first test after DHS, and in the last test. A reduction of these values as compared with control was statistically significant.

Normally the rabbits, in addition to the quadrupedal standing posture, can adopt a vertical bipedal standing posture, which is often associated with exploratory or grooming behavior. After DHS, this ability recovered in four of five animals as early as in 8 days (illustrated for LHS-animal in Fig. 8D).

POSTURAL REFLEXES. Immediately after the re-appearance of equilibrium control (days 1–2), all animals were able to compensate for the standard, 40° peak-to-peak tilts. Initially, the value of postural corrections was reduced. Later on, this value gradually increased to reach the control level in 1–2 wk after DHS. Figure 5*A* shows a representative example of postural responses to sinusoidal tilts of the caudal platform in a rabbit tested on day 15 after DHS. The control test for this rabbit was presented in Fig. 3. The tilts caused postural corrections in the hindquarters with a peak-to-peak value of ~5 cm, which is similar to control. The corrective movements occurred approximately in anti-phase to tilts: the phase shift was 0.01 ± 0.04 and did not differ significantly from the control value (0.02 ± 0.04). The pattern of periodic modulation of the left and right Gast EMG was also close to normal, with the peaks of activity slightly preceding the maximal ipsilateral tilt (compare Figs. 3*A* and 5*A*).

Responses to trapezoidal tilts in the same rabbit (day 15 postlesion) are shown in Fig. 5*B*. Similar to that in control animals (Fig. 3*B*), tilts of the platform caused anti-phase corrective movements and activation of the ipsilateral Gast muscles. Postural corrections after DHS, however, differed from control in that they gradually decayed during the stationary phase of the tilt. This is clear from the superposition of the two S2 trajectories (control and DHS in Fig. 5*C*).

Figure 6*A* shows how the GPR (for sinusoidal tests) changed over time after the lesion in five individual DHS rabbits,

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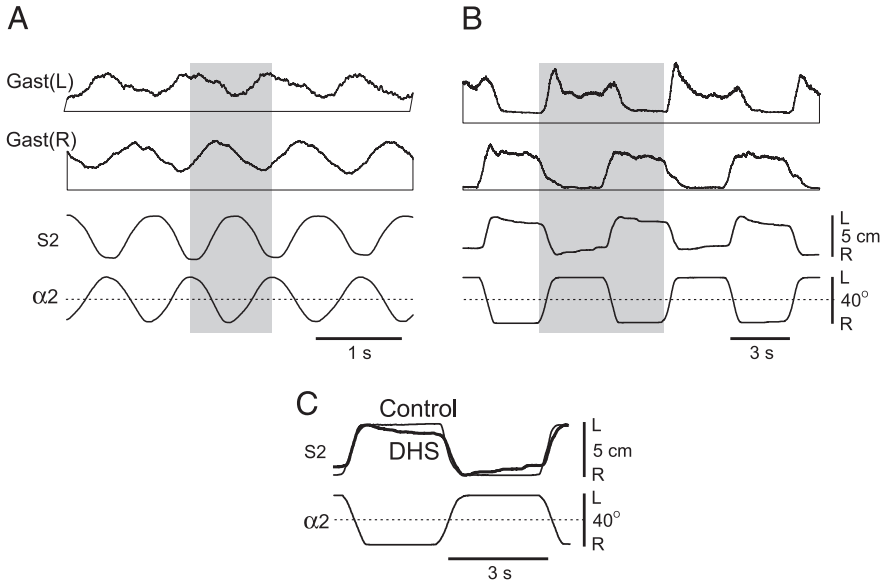

FIG. 5. *A–C*: a representative example of responses to tilts on day 14 after DHS (testing this rabbit before DHS was presented in Fig. 3). *A*: responses to sinusoidal tilts of the caudal platform. *B*: responses to trapezoidal tilts of the caudal platform. *C*: superposition of the tilt angle and response trajectories for the same rabbit in control and after DHS.

starting from the moment of re-appearance of postural reflexes. In *rabbits 26* and *34,* the GPR occurred with a close-to-normal value already in the first test (day 2). In *rabbits 23, 28,* and *35,* the GPR was initially considerably reduced, and then gradually increased to reach a close-to-normal value on days 3, 8, and 5 postlesion, respectively.

In contrast to intact animals, for which GPR in trapezoidal and sinusoidal tests were similar, in DHS animals a decay of the corrective responses to stationary tilts (Fig. 5*C*) led to a reduction of GPR in trapezoidal tests. Figure 6*B* compares, for one of the rabbits, the GPR in sinusoidal tests with that in trapezoidal tests in control (day 0) and on different days after DHS. In control, the GPR values in the two tests were almost the same. After DHS, the GPR in trapezoidal tests was lower than in sinusoidal tests in all trials. Similar results were obtained for all five DHS rabbits. The population average of the ratio between the GPRs in the two tests (0.77 ± 0.16) was significantly smaller than in control (1.02 ± 0.2).

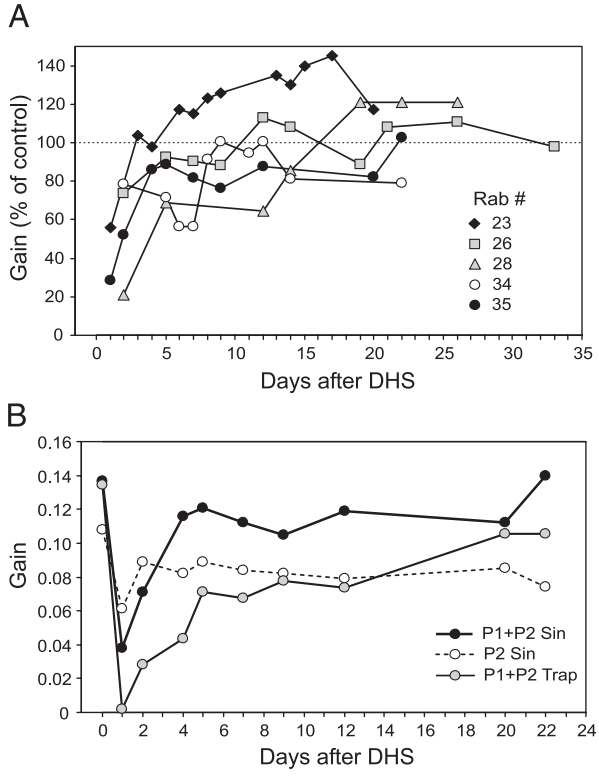

FIG. 6. Recovery of postural corrections in DHS animals. *A*: the gain of postural reflexes in 5 individual rabbits (normalized to control) as a function of time after DHS (sinusoidal tilts). *B*: the gain of postural reflexes in 1 of the rabbits as a function of time after DHS under different conditions: sinusoidal P1+P2 tilts, sinusoidal P2 tilts, and trapezoidal P1+P2 tilts.

In intact rabbits, the GPR caused by tilts of only the caudal platform (P2) was close to or slightly lower than the GPR caused by tilts of both platforms (P1+P2) (see also Beloozerova et al. 2003b). In Fig. 6*B,* the GPR for the tests with P1+P2 sinusoidal tilts is compared with the GPR for the tests with only P2 sinusoidal tilts in control (day 0) and on different days after DHS. In almost all trials, the GPR caused by P2 tilts alone was 20–30% lower than that caused by P1+P2 tilts, and similar results were obtained in all five DHS rabbits.

Figure 7*A* shows that the position of the EMG peak in the tilt cycle remained unchanged after DHS for six hind limbs muscles (averaged over 5 rabbits). This applies to the whole period of observations after DHS.

Effects of lateral hemisection

The effects of a lateral hemisection of the spinal cord were studied in seven rabbits (see Fig. 2*B* for the extent of lesion). In LHS animals, a period of inefficient postural control in the hindquarters lasted from 2 to 9 days postlesion with a mean value of 4.7 ± 1.0 (SE) days. This value was significantly longer and more variable than the corresponding value in DHS rabbits (1.6 ± 0.2 days). Immediately after re-appearance of equilibrium control, some animals were able to compensate for the standard, 40° peak-to-peak tilts (see Fig. 9*A*), whereas others were able to compensate only for 30° peak-to-peak tilts or even 20°. No relation was found between the degree of the spinal lesion (Fig. 2*B*) and the time needed to recover postural control (Fig. 9*A*).

POSTURAL CONFIGURATION. A lateral hemisection of the spinal cord caused considerable changes in the body configuration,

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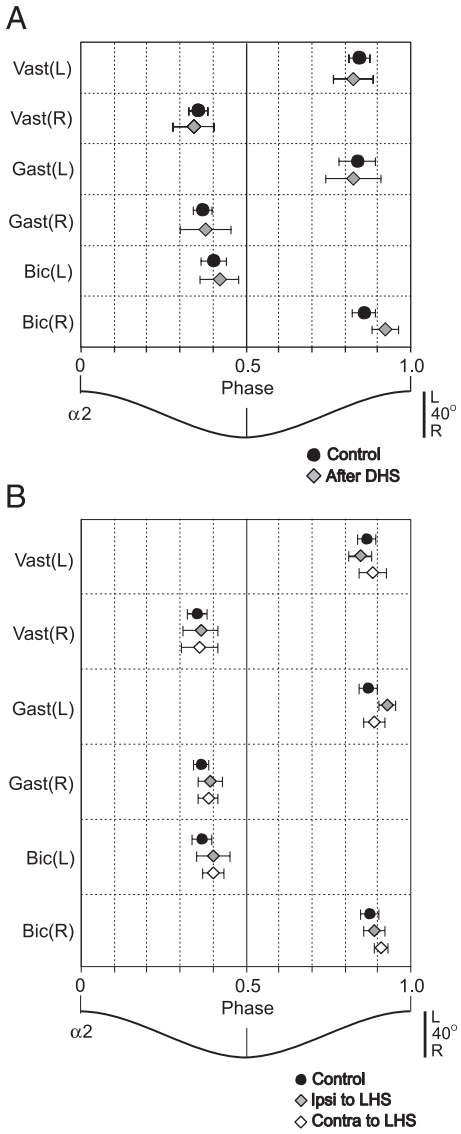

FIG. 7. Effects of DHS and lateral hemisection (LHS) on the temporal EMG patterns. The mean phases (± SE) of the peak EMG of different muscles in the normalized tilt cycle are indicated. Data are presented for intact animals (●) and lesioned animals (◇). *A*: effects of DHS (days 10–20). *B*: effects of LHS (days 5–15). In *B*, data are presented separately for the limbs ipsilateral and contralateral to LHS.

that is, in the position of the hind limbs in relation to the trunk, as well as in the shape of the trunk. The main effect of LHS on limb position was an inward turn of the hind limb contralateral to LHS (Fig. 8*B*). In control (Fig. 8*A*), both feet were turned outward, and the foot angles were positive as illustrated for one of the rabbits in Fig. 8*E*. In the first test after LHS(R) (day 9), the foot angle on the damaged side was reduced but remained positive, whereas the foot angle on the contralateral side became negative. Over time, the foot angle on the damaged side returned to its normal value (day 26), whereas the absolute value of the angle on the undamaged side was slightly reduced, but the angle remained negative until the end of the observations (day 47). The inward turn of the foot on the nondamaged side, as well as a very slow compensation of this deficit was observed in all seven LHS rabbits. Figure 8*F* shows the population average of the foot angles in control, in the first test and in the last test.

The limb on the LHS side was slightly deflected caudally (Fig. 8*B*) as compared with control (Fig. 8*A*), but this difference was not statistically significant, however. It is interesting to note that in some cases, during the initial period of recovery after LHS, this limb was positioned very far backward; this hampered its use for supporting the body. In these cases, the experimenter could help the rabbit to regain postural control in the hindquarters by repositioning the limb to its normal (more rostral) position.

In all LHS rabbits, the trunk was twisted and/or bent laterally, and distortions of the trunk configuration were obvious from the top view as illustrated in Fig. 8*C* for the rabbit with an LHS on the right side. The dorsal midline, defined by markers attached against the skin projections of the dorsal vertebral appendages, clearly deviated to the left, i.e., contralaterally to the LHS, with a maximal deviation (q) in the caudal part of the trunk. Similar changes of body shape were observed in all LHS rabbits. The relative contribution of body twisting and bending to the midline distortion was difficult to assess, however. This deficit developed gradually during 1–3 wk postlesion and did not decrease afterwards, as illustrated in Fig. 8*G*. Figure 8*H* shows the midline deviation in the first and last test for the three animals in which this deficit was best documented.

The ability to adopt a vertical, bipedal standing posture recovered in five of seven LHS animals within 10 days (Fig. 8*D*). The stability of this posture was not tested, however.

POSTURAL REFLEXES. As in control (see e.g., Fig. 3*A*), the restored corrective movements occurred approximately in antiphase to the tilts. In the group of LHS rabbits, the shift between the tilt and the corrective response trajectories was 0.01 ± 0.04 of the tilt cycle (mean ± SD), which did not differ significantly from the control value (0.02 ± 0.04).

The effectiveness of the postural corrections changed over time in most LHS animals. Figure 9*B* shows the gain of postural reflexes in individual animals (sinusoidal tilts) as a function of time. To facilitate the comparison between individual animals, which differed markedly in their rate of recovery, the *abscissa* indicates the time intervals after the first test, when postural control had reappeared. If there was more than one test in a given interval, an average was calculated and taken as the data point. The values of gain were normalized to control.

In two animals (*5* and *9*), the gain was close to the control value as early as in the first test and remained at this high level in the subsequent tests. In three animals (*12, 14,* and *15*), the gain in the first test was only 20–40% of control, but within a few days, it reached the control level. In the remaining two animals (*4* and *16*), the gain was initially low with a tendency to grow although the number of data points was insufficient for quantitative comparisons.

In LHS animals, postural corrections in trapezoidal tests gradually decayed during the stationary phase of the tilt, as described in the preceding text for DHS animals (Fig. 5*C*). Because of this phenomenon, the GPR in trapezoidal tests was reduced. The population average of the ratio between the GPRs in the trapezoidal and sinusoidal tests (0.74 ± 0.18) was significantly smaller than in control (1.04 ± 0.23).

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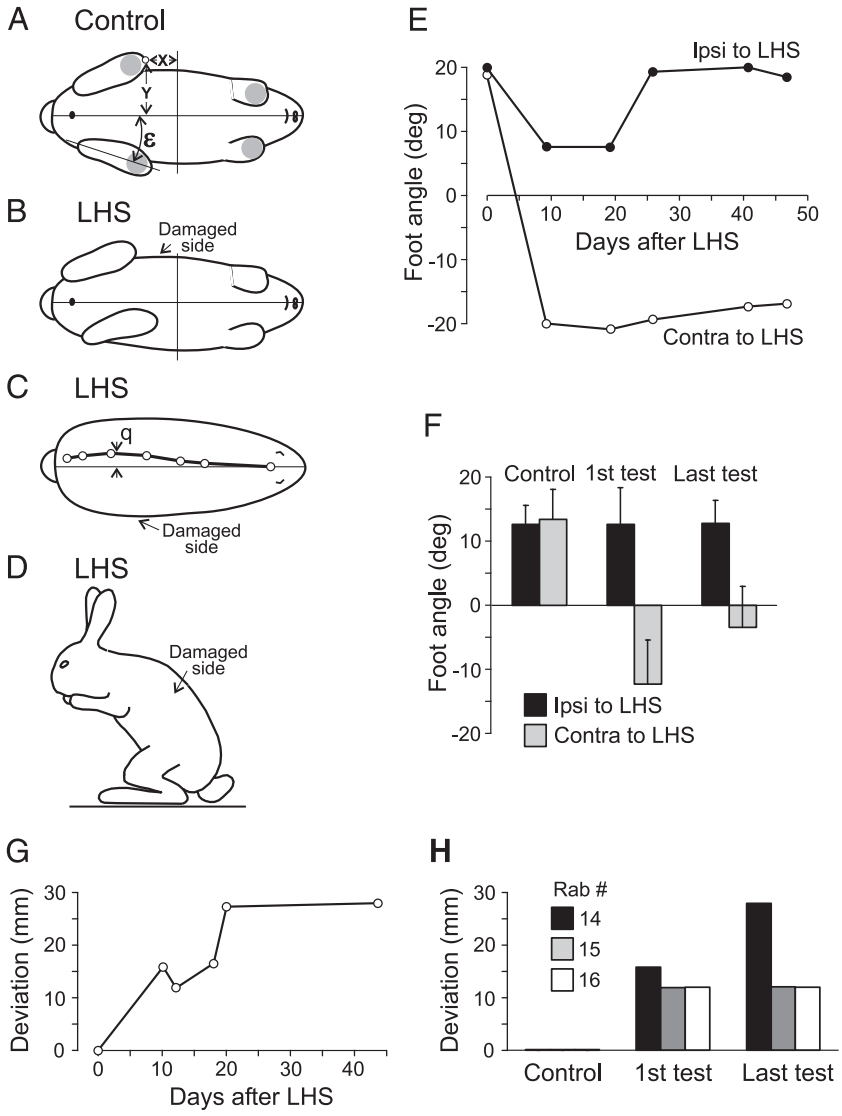

FIG. 8. Distortions of postural configuration caused by LHS. *A*: postural configuration of the intact rabbit on the horizontal platform, view from below (designations as in Fig. 4*A*). *B*: a schematic representation of distortions in the hind limbs configuration caused by a right LHS. *C*: a representative example of distortions in the trunk configuration (10 days after the right LHS). The view from above shows the dorsal midline; the maximal deviation from the control is indicated (q). *D*: restoration of bipedal standing (10 days after the left LHS). *E*: foot angles in the hind limbs of 1 of the rabbits in control (day 0) and on different days after the right LHS. *F*: foot angles averaged over the LHS animals; the values are presented for the control, for the 1st test after LHS, and for the last test (means ± SE). The changes in angles of ipsilateral limb were statistically significant. *G*: deviation of the dorsal midline (q in *C*) in 1 of the rabbits as a function of time after the right LHS. *H*: deviation of the dorsal midline in different LHS rabbits in the 1st and the last tests.

Figure 7*B* shows the position of the EMG peak in the tilt cycle for six muscles of the hind limbs (averaged over 5 rabbits) before LHS (control) and for the whole observation period after LHS. As for DHS (Fig. 7*A*), LHS had practically no effect on the phases of EMG activity during postural corrections.

Effects of ventral hemisection

The effects of ventral hemisection of the spinal cord were studied in five rabbits (see Fig. 2*C* for the extent of lesion). In all these animals, postural corrections and the ability to maintain equilibrium in the hindquarters were dramatically reduced or disappeared altogether after the lesion and did not recover during the whole observation period (≤49 days).

POSTURAL CONFIGURATION. No voluntary movements of the hindlimbs were observed after VHS, and these limbs usually were deviated backward due to the forward body movement caused by stepping of the forelimbs (Fig. 10*B*). The initial backward deviation (day 3) increased over time (days 15 and 30) because of gradually growing extensor tone in the knee and ankle joints, defined as an increasing resistance to passive flexion of the joints.

Because of the backward deviation of the hind limbs and absence of active hip flexion, the rabbits could not use these limbs for supporting their body even on the horizontal surface. If the experimenter positioned the hind limbs to their normal, more rostral site (Fig. 10, *C* and *D*), this could allow the animal to remain in the standing position for some period of time. However, the hindquarters fell to the side when the rabbit tried to change position of its fore quarters. With time, the lateral stability worsened because of the increasing height of the hindquarters above the supporting surface caused by the knee and ankle extension. Figure 10*G* shows how the height of the hindquarters in the standing position (Ht in Fig. 10*C*) changed over time in individual VHS rabbits. In control (day 0) the height was 15–17 cm. For a few days after VHS, the height was reduced to 10–12 cm, but then it increased to reach almost a twofold value within a few weeks.

With the strong limb extension at the knee and ankle joints, it was also possible for the experimenter, by strongly flexing the hip, to position the hind limbs of the rabbit very rostrally,

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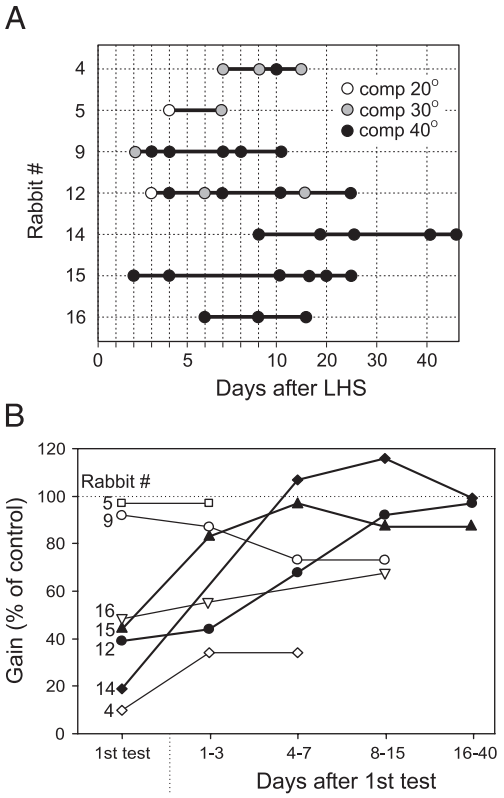

FIG. 9. Recovery of postural corrections in LHS animals. *A*: sequences of tests of individual animals. Each sequence starts with the 1st successful trial. ●, ◍, and ○: the rabbit was able to compensate for 40, 30, and 20° peak-to-peak tilts, respectively. *B*: the gain of postural reflexes in individual rabbits (normalized to control) as a function of time after the 1st successful postural test (sinusoidal tilts).

in between the forelimbs (Fig. 10, *E* and *F*). Such a semi-sitting posture was also unstable, however.

POSTURAL REFLEXES. When tested on the tilting platform, postural reflexes were small, if present at all. Figure 11, *A* and *B,* shows postural responses in one of the rabbits before VHS and on day 5 after VHS, respectively. Before VHS, lateral displacements of the caudal trunk (S2) occurred in anti-phase to tilts of the caudal platform ($\alpha 2$), indicating the presence of efficient postural corrections. After VHS, caudal trunk displacements instead occurred in-phase with tilts, indicating that the hindquarters passively swayed toward the side tilting downward. Tilts of the platform, however, caused activation of extensors in the appropriate (ipsilateral to tilts) hind limbs, but of a smaller magnitude than in control, and with a much smaller dynamic component (compare Fig. 11, *A* and *B*). Apparently, these postural reflexes, despite their correct phase relations to tilts, appeared insufficient to counteract the passive body sway. In Fig. 11*C,* ● shows the tests of individual VHS rabbits in which the normal phase of motor and EMG responses were observed (as in Fig. 11*A*). This occurred only in control (day 0). The ◍ shows the tests in which the EMG responses were normally phased in relation to tilts, whereas the motor responses did not (as in Fig. 11*B*). Such responses were observed only on days 1–7, when the extensor tone had not yet been strongly expressed, as judged from a moderate hind limbs extension (Fig. 10*G*). Later, episodes with normally phased EMG responses were not observed (Fig. 11*C,* ○). These responses seem to disappear in parallel with the development of extensor tone.

DISCUSSION

Maintenance of the dorsal side-up body position and equilibrium on a tilting surface is a complex postural task. In quadrupeds, it requires the participation of the closed-loop postural mechanisms of the fore and hindquarters. An analysis of postural performance in this task was carried out earlier, using two experimental models—the rabbit (Beloozerova et al. 2003b, Deliagina et al. 2000), and the cat (Beloozerova et al. 2005). In the present study, we characterized the effects of

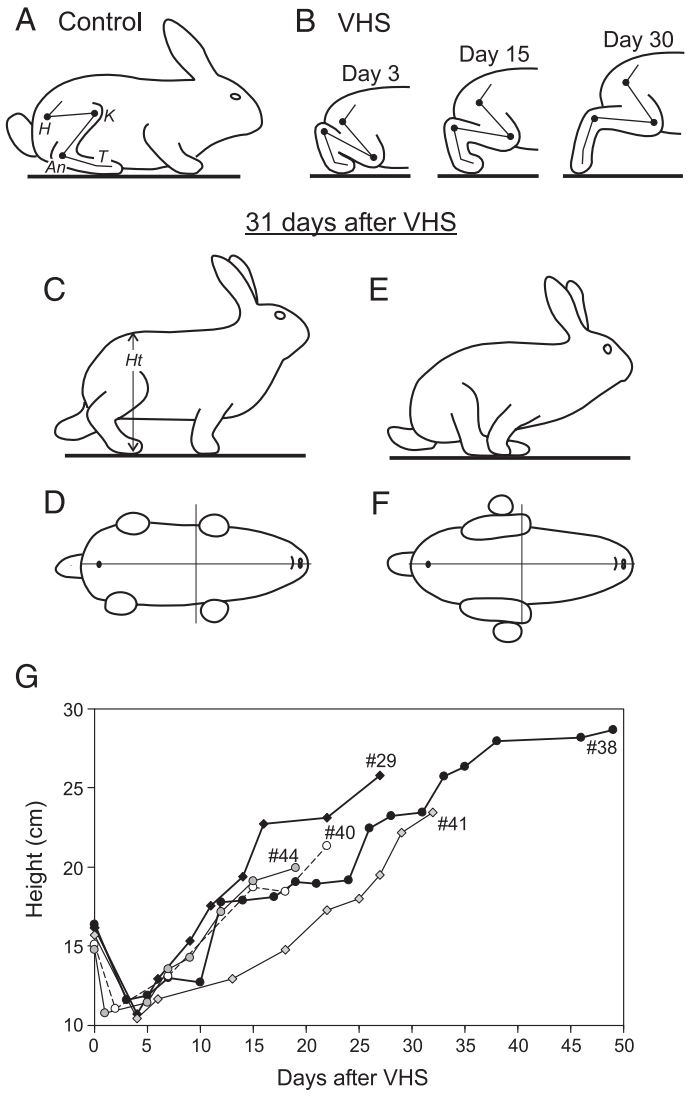

FIG. 10. Distortions of postural configuration caused by VHS. *A*: postural configuration of the intact rabbit on the horizontal platform, view from the right side; position of the main limb joints is indicated (H, hip; K, knee; An, ankle; T. toes). *B*: a schematic representation of distortions in the hind limb configuration in one of the rabbits caused by VHS (postlesion days are indicated). *C–F*: 2 different postural configurations in the VHS rabbit that allow the animal to maintain the dorsal-side-up position of the hindquarters with an abnormal caudal position of the hind limbs (*C* and *D*), and an abnormal rostral position (*E* and *F*). *G*: the height of the hindquarters in the standing position (Ht in Fig. 10*C*) for individual rabbits in control (day 0) and on different days after VHS.

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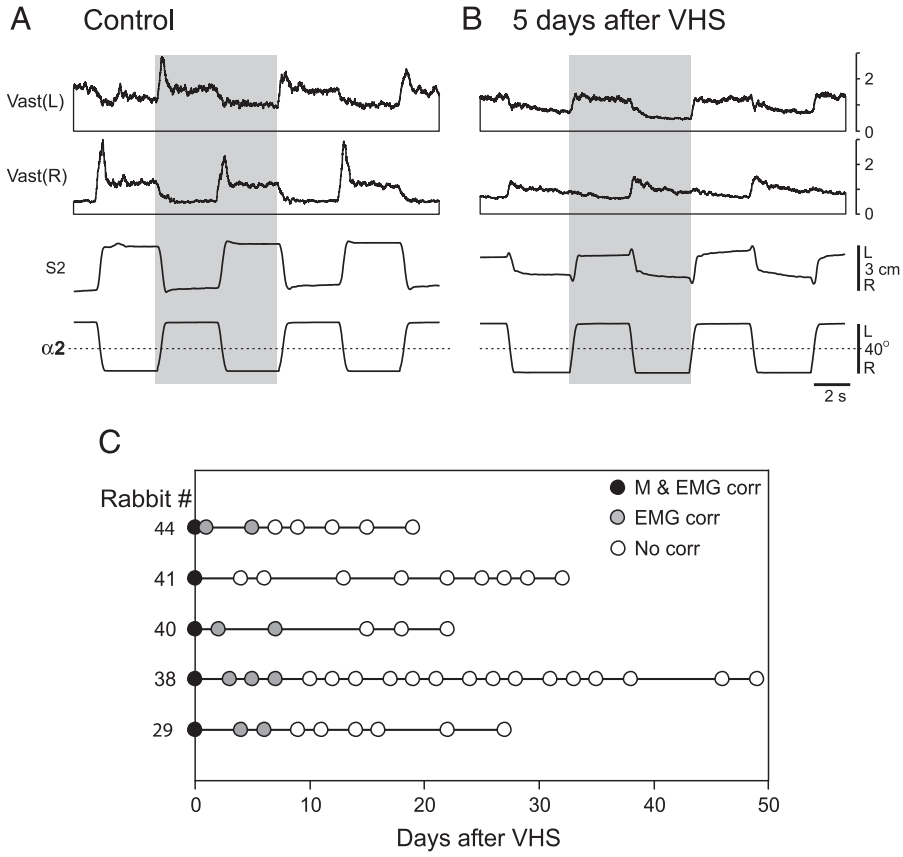

FIG. 11. Effect of VHS on postural reflexes. *A* and *B*: a representative example of responses in one of the rabbits. *A*: responses to trapezoid tilts before VHS. *B*: responses to trapezoidal tilts after VHS (day 5). Abbreviations as in Fig. 3. *C*: characteristics of responses to tilts in individual rabbits in control and on different days after VHS. ●, normal phase of motor and EMG responses; ◍, normal phase of only EMG responses; ○, abnormal phase of both motor and EMG responses or no responses.

different types of SCI on the postural control in rabbits balancing on a tilting platform. This study was aimed, *first*, at a better understanding of the basic postural mechanisms in quadrupeds. In particular, the study has provided some information about the distribution of postural functions between different parts of the CNS. *Second*, for clinical applications it is important to characterize the effects of different types of SCI on the postural system in an animal model and assess the adaptive capacity of this system.

In the present study, we considered separately the effects of different types of SCI on two principal aspects of postural control—the maintenance of postural body configuration and the maintenance of equilibrium.

Maintenance of postural body configuration

Like other quadrupeds (see e.g., Lacquaniti et al. 1984; Maioli and Poppele 1991), standing rabbits can adopt a great variety of postures differing, for instance, in the distance between the fore and hind limbs, in the degree of limb extension, and in the number of supporting limbs as in quadrupedal (Fig. 1*A*) or bipedal standing (Fig. 8*D*). Selection of an appropriate posture is based on many factors such as environmental conditions and behavioral state of the animal and is a function of higher levels of control. One can assume that special tonic supraspinal commands are sent to the spinal cord for eliciting different postural body configurations (Fig. 12*A*, control of

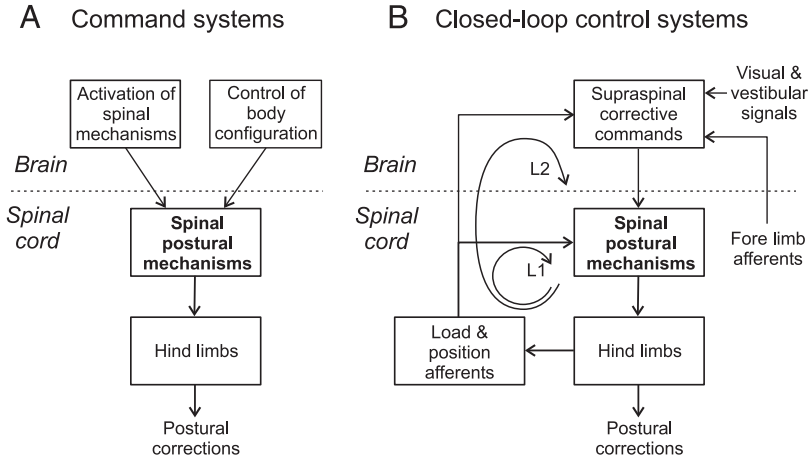

FIG. 12. Presumed basic mechanisms of postural control in hindquarters. *A*: two supraspinal command systems are responsible for the activation of spinal postural mechanisms and for the control of postural body configuration. *B*: functional organization of the feedback mode of postural control. Two closed-loop control systems (loops L1 and L2) stabilize the body orientation (see text for explanations).

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body configuration). The specific role of different descending pathways (reticulospinal, vestibulospinal, etc.) responsible for the transmission of these commands remains unclear. This aspect of postural control is severely damaged with the SCI-caused reduction of supraspinal influences and is absent in animals with a complete spinal lesion. In addition, an interruption of descending pathways, not related directly to the induction of a postural configuration, may cause an imbalance in supraspinal tonic drive to different components of the spinal postural network, which will also result in a distortion of the limb/body configuration.

In the present study, it was found that the distortions in postural configuration, caused by different types of SCI, were very different and were badly compensated during the whole period of observations. In DHS rabbits, the distance and the angle between the left and right feet decreased after the lesion, and the feet were kept at the much more rostral position than in control (Fig. 4). These distortions in the limb position were very slowly compensated or did not change. One can suggest that they were caused by a prevalence of the tonic activity of hip adductors and flexors over the activity of hip abductors and extensors, correspondingly. No such postural distortions have been reported in cats with dorsal spinal lesions (Jiang and Drew 1996).

In LHS rabbits, abnormalities were observed in the configuration and in the position of both hind limbs and of the trunk. The LHS effects were asymmetrical: the limb on the LHS side was positioned more caudally compared with control, whereas the opposite limb was turned inward, and the trunk was bent and twisted (Fig. 8). Distortions in the limb position decreased very slowly over time, whereas the trunk bending and twisting increased. The latter can be considered as scoliosis of neurogenic origin (Herman et al. 1985).

Normally, rabbits can adopt a vertical, bipedal standing posture, which is often associated with exploratory or grooming behavior. After DHS or LHS, this ability recovered (Fig. 8*D*), and some animals became capable of bipedal standing as early as 8 days postlesion. This finding indicates that each of the two groups of spinal descending pathways—the ventral pathways and the unilateral pathways—is sufficient for transmitting the commands for substantial changes in the postural body configuration, such as the switch from quadrupedal to bipedal standing. It seems likely that the overlapping part of the two groups of pathways (located in any one of the ventrolateral quadrants) is sufficient to cause this bilateral switch of postural mechanisms.

In VHS rabbits, an extensor tone gradually developed after SCI in the knee and ankle joints (Fig. 10). The limb extension was so strong that the rabbit was unable to position the hind limbs under the trunk for body support. An increase of the extensor activity in the hind limbs during locomotion was also reported for cats with ventral lesions of the spinal cord (Brustein and Rossignol 1998). In the cats, this increase, however, did not prevent them from using the limbs to support the body.

Maintenance of equilibrium

Maintenance of equilibrium on the tilting platform is achieved via postural reflexes, that is, corrective motor responses caused by the perturbation of posture. The initial effect of all three types of SCI performed in the present study, DHS, LHS, or VHS, was the same—the rabbits were unable to maintain the dorsal-side-up position of their hindquarters, whereas they maintained the normal position of the fore quarters and of the head. In rabbits with DHS or LHS, after an initial period of inability to maintain equilibrium of the hindquarters, the postural functions gradually recovered. In VHS rabbits, these functions did not recover, although small EMG responses with normal phasing could be observed during 1 wk postlesion.

In DHS rabbits, the period of inability to maintain the hindquarters in the standing position lasted for only 1.6 ± 0.2 days after SCI. When this ability reappeared, the gain of the dynamic postural reflexes (revealed in sinusoidal tests) was initially reduced as compared with control, and then it gradually recovered to approach the control value within 1–2 wk (Fig. 6*A*).

In LHS rabbits, the period of inability to maintain the hindquarters in a standing position lasted for 4.7 ± 1.0 days, that is, three times longer than in DHS rabbits. When this ability reappeared, the gain of the dynamic postural reflexes in two rabbits occurred close to the normal one as early as in the first test after SCI. In the remaining five rabbits, the gain was initially considerably reduced and then gradually recovered to approach the control value within 1–5 wk (Fig. 9*B*).

The early reappearance of full-scale dynamic postural reflexes in one part of DHS and LHS rabbits, and the rapid restoration of these reflexes in the other part strongly suggest that the basic nervous mechanisms generating postural reflexes had been only slightly affected by these two types of SCI. Also supporting this view is the striking similarity between the temporal patterns of EMG responses in controls and DHS or LHS rabbits (Fig. 7, *A* and *B*). Further, the restored responses were well pronounced with tilting of only the hindquarters, suggesting that the responses were mainly due to the activity of the hind limb postural mechanisms.

The main deficiency in the operation of reflex postural mechanisms in DHS and LHS rabbits was the significant decrease of the gain of static reflexes revealed in trapezoidal tests. The gain was reduced both in its absolute value and in its relation to the gain of dynamic reflexes. This reduction was primarily caused by a gradual decay ("fatigue") of the corrective postural responses to stationary tilts (Fig. 5, *B* and *C*). It could be observed at any stage of the postlesion recovery (Fig. 6*B*). A rapid fatigue of anti-gravity (extensor) muscle activity in the hind limbs has been reported for cats with complete lesion of the spinal cord. Interestingly, regular weight-support training reduces the fatigability of extensors and improves a weight-supporting function of the limbs during standing (Edgerton et al. 1997, 2001, 2004).

These data can be discussed in relation to the important problem of the distribution of postural functions between spinal and supraspinal structures. Two closed-loop nervous mechanisms controlling posture of hindquarters can be considered [Fig. 12*B* (based on Deliagina and Orlovsky 2002; Horak and Macpherson 1996)]. One of the mechanisms (loop L1) resides in the spinal cord. It is driven by input from limb mechanoreceptors and compensates for postural disturbances by generating corrective motor responses. The other mechanism contains a "long" reflex loop (L2) involving higher centers (spino-brain stem-cerebellar-cortico-spinal loop). This mechanism receives sensory signals from the hind limb mech-

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anoreceptors and, in addition, information about head orientation from visual and vestibular systems and signals from fore limb mechanoreceptors. It is hypothesized that these different sensory inputs are integrated to provide a generalized characteristic of the body posture. If the posture differs from the desired one, corrective commands will be sent to the spinal cord via different descending pathways (reticulospinal, corticospinal, etc.). Participation of the corticospinal pathways in the transmission of posture-related signals has been demonstrated for the rabbit (Beloozerova et al. 2003a) and cat (Beloozerova et al. 2005; Drew 1993). Posture-related correcting signals are also mediated by reticulospinal and vestibulospinal pathways of the cat (Matsuyama and Drew 2000a,b; Prentice and Drew 2001).

The relative contribution of the spinal and supraspinal mechanisms to the generation of postural corrections is not clear, however. On one hand, the animals with a complete SCI in the lower thoracic region exhibit very poor postural responses and as a rule are not able to maintain the dorsal-side-up orientation of their hindquarters (Macpherson et al. 1997b; Macpherson and Fung 1999). These results were interpreted as evidence to suggest a minor role for spinal reflexes (loop 1 in Fig. 12*B*) in postural control (Horak and Macpherson 1996).

An alternative interpretation that we favored (Deliagina et al. 2000) is that spinal postural networks normally are responsible for a considerable part of the postural corrections. However, complete SCI deprives the networks of a necessary supraspinal tonic drive (Fig. 12*A,* activation of spinal mechanisms), which results in a dramatic reduction of their activity. Indirect evidence for this hypothesis was obtained in the present study: the animals subjected to DHS and LHS exhibited rapid recovery of postural reflexes (Figs. 6 and 9). Moreover, the temporal characteristics of their EMG patterns were similar to those in normal rabbits (Fig. 7). Because these lesions evidently cause dramatic changes both in the ascending sensory signals and in the descending commands, it would be very difficult to explain the persistence of the principal features of postural responses in DHS and LHS animals by the operation of heavily damaged long-loop mechanisms (loop L2 in Fig. 12*B*). It seems more likely that the spinal reflex machinery plays an important role in postural control both under normal conditions and after DHS or LHS. After these lesions, the spinal circuits could be activated by a tonic drive through the remaining ventral descending pathways. We suggest that the recovery of postural reflexes in DHS and LHS animals is associated with increased efficacy of the activating drive. If so, attempts to substitute the natural drive in SCI subjects by electrical or pharmacological stimulation of the cord below the lesion seem plausible. In the context of humans with partial SCI, an important consideration would be which spinal pathways remain functional. Presumably, different rehabilitation schemes could be envisaged for patients with different types of lesions.

A striking similarity can been found between the effects of different types of SCI on the postural and locomotor systems. As in the postural task investigated here in the rabbit, damage to the ventral spinal cord caused dramatic impairment of locomotion in the cat (Afelt 1974; Brustein and Rossignol 1998; Eidelberg et al. 1981) and hampered initiation of locomotion by stimulation of the mesencephalic locomotor region (Noga et al. 1991; Steeves and Jordan 1980). These findings suggest that reticulospinal and vestibulospinal pathways, which descend predominantly in the ventral quadrants, are important for initiation and performance of both motor behaviors. They activate the spinal controllers for stepping and postural corrections, and provide the necessary muscle tone (see e.g., Mori 1987; Orlovsky et al. 1999).

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