Asymmetry in the Pitch Control System of the Lamprey Caused by a Unilateral Labyrinthectomy

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Pavlova, E. L. and T. G. Deliagina. Asymmetry in the pitch control system of the lamprey caused by a unilateral labyrinthectomy. *J Neurophysiol* 89: 2370–2379, 2003; 10.1152/jn.00830.2002. A postural control system in the lamprey is driven by vestibular input and maintains a definite orientation of the animal during swimming. After a unilateral labyrinthectomy (UL), the lamprey continuously rolls toward the damaged side. Important elements of the postural network are the reticulospinal (RS) neurons that are driven by vestibular input and transmit commands for postural corrections to the spinal cord. We characterized the effect of UL on vestibular responses in RS neurons elicited by rotation of the animal in the pitch plane. The activity of RS neurons was recorded from their axons in the spinal cord before and after UL. The neurons can be classified into the Up and Down groups activated preferentially with nose-up or nose-down rotation, respectively. After UL, vestibular responses in the group Up changed only slightly on the damaged side and disappeared almost completely on the opposite side. In the group Down, responses on both sides persisted after UL. These results indicate that the left and right subgroups of the group Up neurons receive excitatory input mainly from the contralateral labyrinth. In contrast, the group Down neurons receive excitatory input from both labyrinths. We conclude that the UL-induced asymmetry in the bilateral activity of the group Up neurons seems to be an important factor contributing to the loss of equilibrium in UL animals and to their rotation during swimming.

**INTRODUCTION**

Ablation of one vestibular organ (unilateral labyrinthectomy, UL) evokes severe motor disorders in all classes of vertebrates. They include abnormal eye positions, spontaneous ocular nystagmus, asymmetry in the head and trunk posture, etc. Over time, these disorders gradually diminish. This process of the recovery of motor functions is usually referred to as "vestibular compensation" (for a review, see Dieringer 1995; Shaef er and Meyer 1974; Smith and Curthoys 1989; Vidal et al. 1998). Despite extensive studies of vestibular compensation, neuronal mechanisms of the origin of different UL-evoked symptoms and of the recovery of motor functions are still poorly understood. The main reason for this is that the corresponding neuronal networks are extremely complex.

We have been investigating the effect of UL on the neuronal postural networks, as well as the process of recovery of their function, by using a simple biological model—the lamprey, a lower vertebrate (cyclostome) (Deliagina and Pavlova 2002). One reason for this is that the basic organization of the lamprey CNS, and especially of the brain stem and spinal cord, is similar to that of higher vertebrates (Nieuwenhuys et al. 1998), yet simple enough to allow many more opportunities for analytical studies of the nervous mechanisms for postural control, including studies at the network and cellular levels (Macpherson et al. 1997; Orlovsky et al. 1992).

When swimming, the intact lamprey actively stabilizes its orientation in the gravity field due to the activity of two postural systems (de Burlet and Versteegh 1930; Deliagina 1995, 1997a,b; Ullén et al. 1995). The roll control system maintains a definite body orientation in the transverse plane (usually the dorsal-side-up one). The pitch control system maintains a definite body orientation in the sagittal plane (usually the horizontal one).

Important elements of the postural network in the lamprey are the reticulospinal (RS) neurons (Brodin et al. 1988; Busières 1994; Nieuwenhuys 1972) transmitting commands for postural corrections from the brain stem to the spinal cord (Deliagina et al. 1992a; Orlovsky et al. 1992). The RS neurons receive vestibular input through interneurons of the vestibular nuclei (Koyama et al. 1989; Northcutt 1979; Rovainen 1979; Rubinson 1974; Stefanelli and Caravita 1970). The RS neurons project ipsilaterally over long distances and affect different classes of spinal neurons (Buchanan and Cohen 1982; Ohta and Grillner 1989; Rovainen 1974). Individual RS neurons exert diverse effects on the spinal motor output (Zelenin et al. 2001).

In our previous studies, responses of RS neurons to rotation in the roll and pitch planes were recorded in vitro preparations (Deliagina et al. 1992a; Orlovsky et al. 1992) and in intact animals (Deliagina and Fagerst edt 2000; Pavlova and Deliagina 2002). When tested by rotation in the roll plane, most RS neurons fall into one of the two groups, activated preferentially by the left tilt (group R) or by the right tilt (group L). When tested by rotation in the pitch plane, most neurons also fall into one of the two groups activated preferentially by the nose-up tilt (group Up) or by the nose-down tilt (group Down). The population of neurons responding to roll tilt and that responding to pitch tilt partly overlap (Pavlova and Deliagina 2002).

We suggest that these four groups of RS neurons, differing in their vestibular inputs, constitute an essential part of the roll and pitch postural systems in the lamprey, and elicit corrective...
motor responses to any deviation from the stabilized body orientation (Deliagina et al. 1992a, 1993; Deliagina and Fagerstedt 2000; Deliagina and Pavlova 2002; Pavlova and Deliagina 2002). The groups L and R, with opposite responses to roll tilts, cause rotation of the body around its longitudinal axis in opposite directions. The system will stabilize the pitch angle at 0°, that is, the activity on the side contralateral to UL disappeared almost completely, whereas the responses on the UL side changed only slightly. This asymmetry was considered an important reason for the loss of equilibrium: the dominating group of RS neurons will cause continuous rolling of the lamprey. This suggestion has been confirmed in the model studies (Zelenin et al. 2000).

Because postural mechanisms in the lamprey are driven primarily by vestibular input, the effect of UL in this animal is most dramatic. In the swimming lamprey, UL results in a complete loss of postural stability and in continuous rolling of the animal around its longitudinal axis toward the damaged labyrinth, so that the swim trajectory represents a spiral (de Burlet and Versteegh 1930; Deliagina 1995, 1997a). During a few weeks following UL, the animals gradually recover their capacity to maintain equilibrium (Deliagina 1995, 1997a).

Since the main UL-induced motor deficit in the lamprey is continuous rolling, it was suggested that the primary effect of UL is an impairment of the roll control system (Deliagina 1995, 1997a). In a recent study (Deliagina and Pavlova 2002) we examined the UL-induced changes in the roll control system. It was found that UL causes a substantial asymmetry in the responses in the groups L and R to rotation in the transverse plane, that is, the activity on the side contralateral to UL disappeared almost completely, whereas the responses on the UL side changed only slightly. This asymmetry was considered an important reason for the loss of equilibrium: the dominating group of RS neurons will cause continuous rolling of the lamprey. This suggestion has been confirmed in the model studies (Zelenin et al. 2000).

From the behavioral and electrophysiological experiments, it remained unclear, however, if the UL exerts any effect on the pitch control system, and if this effect contributes to the expression of the main UL symptom, the rolling. The aim of the present study was to examine the effect of UL on the pitch control system. For this purpose we recorded responses of RS neurons to rotation of the animal in the sagittal plane while its orientation in the transverse plane was unchanged. In each animal, the recordings were performed both before and after UL. The main result of this study is that UL affects the Up and Down groups of RS neurons differently and creates the left/right asymmetry in their activity. This will contribute to the main UL-induced motor deficit, the rolling.

METHODS

Experiments were carried out on seven adult (25–35 cm in length), intact lampreys (Lampetra fluviatilis), which were kept in an aerated freshwater aquarium at 7°C, with a 12 h:12 h light:dark cycle.

All experiments were approved by the local ethical committee (Norra Djurförsökssetiska Nämnden).

Electrodes

The activity of RS neurons was recorded from their axons in the spinal cord by means of chronically implanted macrolelectrodes as described in detail in the previous papers (Deliagina et al. 2000; Deliagina and Fagerstedt 2000). In short, the electrodes (2 silver wires 75 μm in diameter and 3 mm in length, separated by 1 mm, Fig. 1A) were glued to a plastic plate (6 mm long, 2 mm wide, and 0.25 mm thick) and were then oriented in parallel to the RS axons. They allowed an almost exclusive recording of the spike activity from larger fibers that have a conduction velocity of more than 2 m/s. In the lamprey, only RS pathways contain fibers with such a high conduction velocity.

Surgery

All seven animals were operated on two times under MS-222 (Sandoz) anesthesia (100 mg/l). During the first surgery, implantation
of the electrodes was performed as previously reported by Deliagina et al. (2000) and by Deliagina and Fagerstedt (2000). The plate with electrodes was implanted at the level of the third gill. The electrodes were facing the dorsal aspect of the spinal cord (Fig. 1A). The UL was performed 1 to 2 days after implantation of the electrodes, as described in detail earlier (Deliagina 1995, 1997a). In short, a hole was made in the dorsolateral aspect of the vestibular capsule and the labyrinth was removed with a pair of fine forceps under visual control. After removal, the intact medial wall of the vestibular capsule and a stump of the eighth nerve could be seen. Postmortem investigation showed that, in all cases, removal of the vestibular organ was complete and that the medial wall of the capsule was undamaged.

**Experimental protocol**

In all animals, vestibular responses of RS neurons were examined two times—on the next day after implantation of the electrodes and on the next day after UL. The arrangement for vestibular stimulation and the characteristics of stimuli have been described in the previous paper (Pavlova and Deliagina 2002). In brief, the lamprey was rotated in the sagittal plane around the axis situated in the mid-body area. Two full turns (a and b in Fig. 1B) were performed in opposite directions. The initial orientation of the animal was with its dorsal side down (180°). Rotation was performed in 45° steps. The transition from one position to the next lasted approximately 1 s, and each position was maintained for approximately 3 s.

**Data processing**

Signals from the electrodes were amplified by conventional AC amplifiers, digitized with a sampling frequency of 10 kHz, and transferred to the hard disk of an IBM AT compatible computer by means of data acquisition hardware/software (Digidata 1200/Axoscope, Axon Instruments, Foster City, CA). The recorded multiunit spike trains were separated into unitary waveforms, representing the activity of individual axons, by means of data analysis software (Datapac III, Run Technologies, Laguna Hills, CA). The criteria for separation of units were the amplitude and shape of the signals recorded simultaneously by the two electrodes. The analysis procedure was described in previous papers (Deliagina and Fagerstedt 2000; Deliagina and Pavlova 2002). From 5 to 15 neurons were recorded in individual animals.

To determine the spatial zones of sensitivity of individual RS neurons, their vestibular responses were characterized quantitatively. For this purpose, each step of rotation was divided into three intervals (1–3, see inset in Fig. 1B), and the firing frequency of a neuron was measured separately for each of the intervals in each step.

The mediolateral position of individual axons in the spinal cord was estimated by comparing the amplitudes of the same spike recorded by the left and right electrodes (for details, see Deliagina and Fagerstedt 2000).

All the analytical procedures and possible sources of errors during the spike sorting have been described in detail in previous papers (Deliagina and Fagerstedt 2000, Pavlova and Deliagina 2002). Besides the possible errors introduced by spike sorting, an additional possible source of errors in the present study could have been a change in the recording conditions caused by displacement of the electrode arrays during the second surgical intervention (UL). However, special experiments have shown that there were no marked changes in spike waveforms following UL (Deliagina and Pavlova 2002).

All quantitative data in this study are presented as the mean ± SE. The paired *t*-test was used to characterize the statistical significance when comparing different means; the significance level was set at *P* = 0.05.

**RESULTS**

In seven animals, the activity in RS pathways was recorded both before and after UL. Normally, the resting activity in RS neurons was low or absent. Vestibular stimulation activated RS neurons. This is illustrated for animal MT3 in Fig. 2, where traces EL and ER show the mass activity in RS pathways recorded by the left and right electrodes, respectively, before UL (A) and after left UL (B). Using the spike sorting program, the activity of individual axons was separated from the mass activity. All neurons were divided into two groups according to the criteria formulated earlier (Pavlova and Deliagina 2002): group Up neurons were activated preferentially by the nose-up rotation, and group Down neurons were activated by the nose-down rotation. Each of the groups was farther divided into the left and right subgroups according to the position of the axon in the spinal cord. In animal MT3, before UL (Fig. 2A), the group Up included 11 neurons, of which 5 neurons (L1–L5) belonged to the left subgroup and 6 neurons (R1–R6) belonged to the right subgroup. Group Down included three neurons—one of the left subgroup (L6) and two of the right subgroup (R7, R8).

After the left UL (Fig. 2B), the responses in all neurons of the right Up subgroup (R1–R6) disappeared, and no new neurons appeared instead in this subgroup. By contrast, in the left Up subgroup, the activity in two neurons (L1, L4) disappeared, but four new neurons (L7–L10) appeared instead. In the Down group, the left subgroup increased in number due to the appearance of three new neurons (L11–L13). The right Down subgroup did not change in number after UL, but R7 was replaced by a new neuron (R9).

Altogether in seven animals, 83 neurons were recorded before UL. Of these, 65 neurons (78%) belonged to group Up, and 18 neurons (22%) belonged to group Down. The same proportion of group Up and Down neurons was observed in our previous study (Pavlova and Deliagina 2002). Of the 65 group Up neurons, 33 neurons were located on the side ipsilateral to the subsequent UL, and 32 neurons were located on the opposite side. Of the 18 group Down neurons, 7 neurons were located on the side ipsilateral to the subsequent UL, and 11 neurons were located on the opposite side.

The main effect of UL, that is a dramatic reduction in the number of responding group Up neurons on the side contralateral to UL, was observed in each of the seven animals investigated. In four animals, responses in this subgroup of neurons disappeared completely (as in Fig. 2). In the remaining three animals, of the 19 neurons responding before UL, 15 neurons disappeared after UL, and 1 neuron appeared instead.

Also, in each of the animals, the total number of group Up neurons on the side ipsilateral to UL changed only slightly. However, of the 35 neurons of this subgroup active before UL, 18 neurons disappeared after UL, and 17 neurons appeared instead.

In the group Down, the UL caused an increase in the number of responding neurons, from 7 to 14 in the ipsilateral subgroup, and from 11 to 17 in the contralateral subgroup. Of the 14 and 17 neurons active after UL in the ipsilateral and contralateral subgroup, respectively, only 2 neurons in each subgroup were active before UL.

To evaluate the effect of UL on different subgroups of RS neurons, each step of the angular change was divided into three
intervals (*inset* in Fig. 1B). Interval 1 corresponded to a movement from the preceding position to a new one, and the intervals 2 and 3 corresponded to a period when the new position was maintained. The activity in interval 1 was considered as a dynamic response, and the activity in intervals 2 and 3 were considered as early and late static responses, respectively.

For each of the three intervals of each step, two characteristics of population activity were calculated (Pavlova and Deliagina 2002): 1) the number of simultaneously active neurons, and 2) the mean discharge frequency of the active neurons. Both characteristics were calculated separately for each animal and then averaged over all seven animals. These calculations were performed separately for the ipsilateral and contralateral (to UL) subgroups of the group Up and group Down neurons under two conditions, that is, before and after UL.

Figure 3, A, and B, shows the histograms of the number of simultaneously active group Up neurons recorded before UL on the side ipsilateral to a subsequent UL (A) and on the opposite side (B). Along the horizontal axis, the successive angles of pitch tilt during two turns (a and b), performed in opposite directions, are indicated. From these graphs one can see that the responses recorded before UL on the two sides were qualitatively similar to each other; they were also similar to the responses described in a previous paper (see Fig. 4A, in Pavlova and Deliagina 2002). The neurons responded preferentially to the nose-up rotation (*turn a*). In this turn, any change of orientation evoked a dynamic response in many RS neurons.
To evaluate this directional sensitivity, for each of the animals, we calculated the mean number of neurons responding dynamically to sequential steps in turn a and then averaged this value over all seven animals. The responses are presented as a function of pitch angle. Angle of 0° corresponds to the horizontal, back-up orientation of the lamprey. Each step of rotation was divided into 3 intervals (see inset in Fig. 1B). In each of the steps, the dynamic response (activity during rotation) is shown by a black bar; the early and late static responses are shown by 2 successive shaded bars. A1, B1: responses before UL. A2, B2: responses after UL. Designations as in Fig. 1B.

The UL affected differently the RS neurons on the ipsilateral and contralateral sides. On the ipsilateral side, the basic pattern of response persisted after UL, that is, the neurons responded dynamically in turn a much stronger than in turn b (compare Fig. 3, A1 and A2, as well as Fig. 4, A1 and A2). When
characterized by the number of active neurons, the mean response was 4.1 ± 0.3 neurons in turn a versus 1.9 ± 0.4 neurons in turn b (P < 0.0003). Similarly, the mean frequency of response in turn a was 3.2 ± 0.4 Hz versus 0.8 ± 0.2 Hz in turn b (P = 0.0002). The UL caused, however, a statistically insignificant increase in the value of dynamic response in turn a: the mean number of active neurons was 3.7 ± 0.6 before UL versus 4.1 ± 0.3 after UL. Similarly, the mean frequency in turn a was 2.9 ± 0.3 Hz before UL versus 3.2 ± 0.4 Hz after UL (P = 0.29). An increase of the responses in turn b after UL (compare Fig. 3, A1 and A2) was also insignificant.

On the side contralateral to UL, the number of neurons responding in turn b dramatically decreased (compare Fig. 3, B1 and B2), and responses in turn a only weakly increased. The mean number of responding neurons in turn a was 3.6 ± 0.7 before UL versus 0.8 ± 0.2 after UL (P = 0.0006). The few neurons that remained active after UL responded with a frequency of 1–2 Hz (Fig. 4B2).

The patterns of responses of the group Down neurons recorded before UL on the side ipsilateral to a subsequent UL and on the opposite side were qualitatively similar to each other and mirrored that of the group Up neurons. As shown in Fig. 5, A1 and B1, and Fig. 6, A1 and B1, the neurons responded dynamically to the nose-down rotation (turn b) much stronger than to the nose-up rotation (turn a). The patterns of responses were also qualitatively similar to those described in a previous paper (see Fig. 4B2 in Pavlova and Deliagina 2002).

On the side ipsilateral to the subsequent UL, the mean number of dynamically responding neurons in turn b was 0.8 ± 0.2 versus 0.4 ± 0.1 in turn a (P = 0.014). On the side
contralateral to UL, the mean number of dynamically responding neurons in turn b was 1.1 ± 0.3 versus 0.5 ± 0.2 in turn a ($P = 0.0074$). In turn b, 10 to 20% of neurons on both sides also exhibited a static response.

On the side ipsilateral to the subsequent UL, the mean frequency of the dynamic response in turn b was 3.1 ± 0.6 Hz versus 0.6 ± 0.2 Hz in turn a ($P = 0.0031$). On the side contralateral to UL, the mean frequency of the dynamic response in turn b was 2.4 ± 0.7 Hz versus 0.5 ± 0.3 Hz in turn a ($P = 0.005$). The frequency of a static response in both turn a and turn b was very low (0.1–0.2 Hz).

The UL similarly affected the RS neurons on the ipsi- and contralateral sides. On both sides, the basic pattern of response persisted after UL, that is, the neurons responded dynamically in turn b much stronger than in turn a (compare Fig. 5, $A_1$ and $A_2$, as well as Fig. 6, $A_1$ and $A_2$). When characterized by the number of active neurons, the mean response on the side ipsilateral to UL was 1.6 ± 0.7 neurons in turn b versus 0.6 ± 0.2 neurons in turn a ($P = 0.04$). On the side contralateral to UL, the mean response was 1.5 ± 0.3 neurons in turn b versus 0.5 ± 0.1 neurons in turn a ($P = 0.0083$). The mean frequency of the dynamic response on the side ipsilateral to UL in turn b was 1.6 ± 0.4 Hz versus 0.5 ± 0.2 Hz in turn a ($P = 0.02$). On the side contralateral to UL, the mean frequency was 2.3 ± 0.7 Hz in turn b versus 0.4 ± 0.1 Hz in turn a ($P = 0.0011$).

Although the basic pattern of responses in the group Down neurons persisted after UL, some changes in the magnitude of the responses were observed. As shown in Fig. 5, the dynamic responses in turn b, when characterized by the number of active neurons, increased on both sides after UL (compare Fig. 5, $A_1$ and $A_2$, as well as Fig. 5, $B_1$ and $B_2$). On the side ipsilateral to UL, the mean number of active neurons in turn b

![Diagram](https://example.com/diagram.png)
was $0.8 \pm 0.2$ before UL versus $1.4 \pm 0.6$ after UL. On the side contralateral to UL, the mean number of active neurons in turn $b$ was $1.1 \pm 0.3$ before UL versus $1.5 \pm 0.3$ after UL. In both cases, however, the increase of response after UL was not statistically significant.

In contrast to the increase of the number of responding neurons after UL, their firing frequency decreased after UL (compare Fig. 6, $A_1$ and $A_2$, as well as Fig. 6, $B_1$ and $B_2$). On the side ipsilateral to UL, the mean frequency in turn $b$ was $3.1 \pm 0.6$ Hz before UL versus $1.6 \pm 0.4$ Hz after UL ($P = 0.01$). On the side contralateral to UL, the mean frequency in turn $b$ was $2.4 \pm 0.7$ Hz before UL versus $1.7 \pm 0.6$ Hz after UL; this decrease was not statistically significant, however.

**DISCUSSION**

Two groups of RS neurons ($Up$ and $Down$), activated by the nose-up and nose-down tilts, respectively, were first characterized in our previous study (Pavlova and Deliagina 2002). The most likely source of their activation is the two groups of vestibular afferents in each of the labyrinths that have corresponding angular zones of activity (P2 and P1 in Deliagina et
These groups are designated as VA(Up) and VA(Down) in Fig. 7A. In the present study, we addressed the question of what the role is of the ipsilateral and contralateral labyrinths in the elicitation of vestibular responses in the Up and Down groups of RS neurons. For this purpose, we examined responses of RS neurons to pitch tilts before and after UL. The main result of this study is that UL produced different effects on the Up and Down groups of RS neurons. In the group Up neurons, the responses remained almost unchanged on the side ipsilateral to UL and were dramatically reduced on the opposite side (Fig. 3). This finding indicates that the group Up neurons receive their excitatory input predominantly from the contralateral labyrinth, whereas the input from the ipsilateral labyrinth is very weak. The two inputs are designated by the large and small arrows, respectively, in Fig. 7.

In the group Down neurons, the effects of UL on both sides were similar: the number of responding neurons increased, although their discharge frequency decreased (Figs. 5 and 6). This finding indicates that the group Down neurons receive similar inputs from both labyrinths. The inputs probably contain both excitatory and inhibitory components (designated by plus and minus in Fig. 7). A reason for the opposite effects of UL on the number and the frequency of responding group Down neurons is not clear, however.

It is interesting to compare these results with those obtained when studying the effect of UL on vestibular responses caused by roll tilts (Deliagina and Pavlova 2002). It was concluded that RS neurons receive their roll-dependent excitatory inputs from both labyrinths, although the contralateral input plays a predominant role. In this respect, the origin of roll responses is similar to that of pitch responses in the group Up neurons, but not in the group Down ones.

In previous studies it was found that a left/right asymmetry in the commands transmitted by RS neurons of the roll control system is a cause for roll turns; in particular, a cause for continuous rolling induced by the roll control system impaired by UL (Deliagina and Fagerstedt 2000; Deliagina and Pavlova 2002; Deliagina et al. 2002). Can a left/right asymmetry in the commands transmitted by RS neurons of the pitch control system play a similar role and initiate rolling? Most likely it can since there is a considerable overlap between the roll and
pitches populations (Pavlova and Deliagina 2002), which suggests a polyfunctional role of individual RS neurons (Deliagina et al. 2002). It seems therefore highly probable that any nose-up pitch tilt in UL animals, because of incorrect functioning of the pitch control system, will evoke rolling. Thus the motor deficits caused by the impaired roll and pitch control systems will summate, enhancing the main UL symptom, the rolling.

In mammals, UL causes numerous motor disorders including abnormal head and body posture, instability of posture, distortions of voluntary head and limb movements, distortions of locomotor movements, etc. (see, e.g., Deliagina et al. 1997; Smith and Curthoys 1989). Usually, each of these deficits is considered to be a consequence of the impairment of the corresponding nervous mechanism. The present study suggests, however, that a distortion of a particular motor function after UL can be caused not only by a damage to its own control mechanism but also by a damage to the mechanism that normally controls a different motor function. This may be due to the overlap between the command systems controlling different motor patterns (Deliagina et al. 2002).

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