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Review

Nervous mechanisms controlling body posture

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Abstract

This paper briefly summarizes the studies of nervous mechanisms controlling the body posture, which were performed in the Department of Neuroscience of the Karolinska Institute during the last decade. Postural mechanisms were investigated in "animal models" of different complexity — the mollusk, lamprey, rabbit, and cat. The following problems were addressed: (1) functional organization of the postural system; (2) localization of postural functions in the mammalian CNS; (3) postural networks; (4) impairment of postural control caused by vestibular deficit. These studies have significantly expanded our knowledge of how the postural control system operates, how the stabilized body orientation can be changed, and how the postural functions are distributed within different parts of the CNS. For simpler animal models (mollusk, lamprey), the neuronal networks responsible for the control of body posture have been analyzed in considerable detail, with identification of the main cell types and their interactions. Also, alterations in the activity of postural mechanisms caused by the vestibular deficit were investigated to better understand the process of recovery of postural function.
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Keywords: Posture; Equilibrium control; Vestibular control; Cortical control; Reticulospinal system

Contents

1. Introduction

Maintenance of the basic body posture – upright in bipeds and dorsal-side-up in quadrupeds and in many aquatic animals – is a vital motor function. Maintenance of this posture is a non-volitional activity based, in many species, on the in-born neural mechanisms [1]. Efficient control of the basic posture is equally important for standing and during walking [2–4], as well as for providing support for voluntary limb movements [5]. Deficiency in sensory or motor mechanisms of the postural system produces dramatic effects on postural stability and motor performance [5–8].

In the Department of Neuroscience of Karolinska Institutet, nervous mechanisms for the control of body posture are investigated on "animal models" of different complexity — mollusk, lamprey, rabbit, and cat. Comparative studies of postural mechanisms are based on the assumption that a basic problem such as the nervous control of the anti-gravity behavior has similar solutions in different species, and thus the results obtained on animal models may have significance for understanding the mechanisms of postural control in humans.

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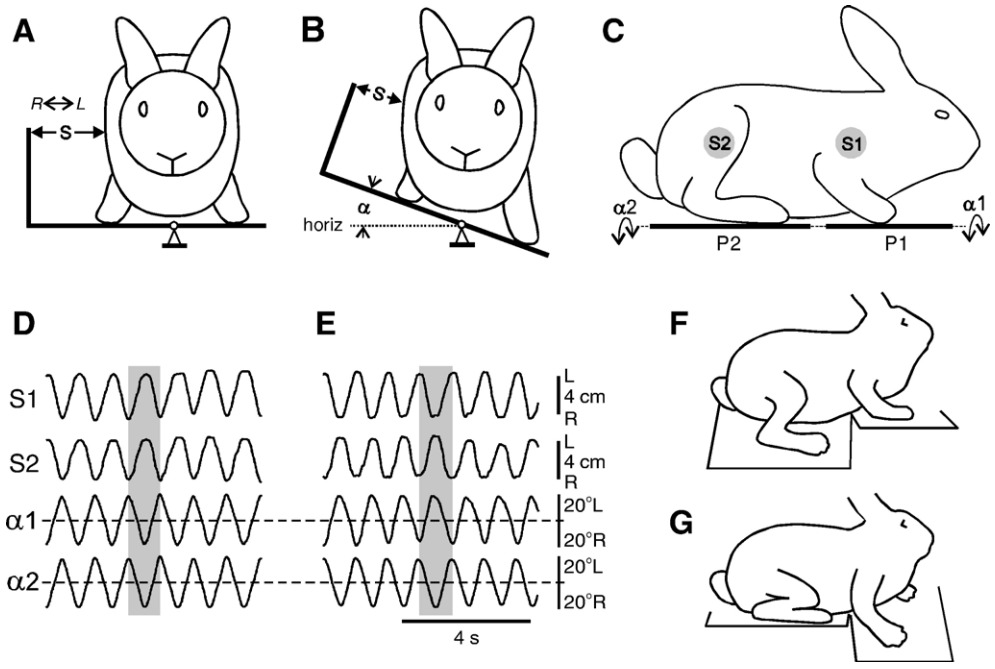

Fig. 1. Corrective motor responses in postural tasks of different complexity. A–C: The rabbit was maintaining balance on the two tilting platforms (P1 and P2) and produced postural corrections in response to tilts. D,E: Postural responses in the anterior (S1) and posterior (S2) areas of the trunk caused by in-phase (D) and anti-phase (E) tilts of the anterior (α1) and posterior (α2) platforms. F,G: The body outline for the two extreme positions (left and right) of the platforms tilted in anti-phase.

These studies have significantly expanded our knowledge of how the postural control system functions, how the stabilized body orientation can be changed, and how the postural functions are distributed within different parts of the CNS. For simpler animal models, the neuronal networks for the control of body posture have been analyzed in considerable detail, including identification of main cell types and their interactions. Also, alterations in the activity of postural mechanisms caused by the vestibular deficit are investigated to better understand the process of recovery of postural function, and to search for the means to promote the recovery.

2. Functional organization of the postural system

There are two principal modes of postural activity — the feedback mode, that is compensation for the deviation from the desired posture, and the feed-forward mode, that is anticipatory postural adjustments aimed at counteracting the destabilizing consequences of voluntary movements [2,9]. In this review we focused on the feedback mode of postural activity. There are two major concepts regarding functional organization of the postural system when it operates in the feedback mode.

A widely accepted concept of postural stabilization is based on the notions of control theory [10]. It suggests that a body posture is characterized by a single "regulated variable" (*e.g.*, position of the center of mass, or orientation of the longitudinal body axis); a certain value of this variable is stabilized. According to this "regulated variable" concept of postural control, information about the head and body orientation is delivered by sensory inputs of different modalities (vestibular, visual, and somatosensory). This information is processed and integrated to obtain a generalized characteristic of body posture, *e.g.*, the position of the center of mass, or the orientation of the body axis. If this variable differs from its desired value, a command is sent to motor centers to elicit a corrective movement [2,5,11–13].

A different, "reflex" concept was formulated in the classical study by Magnus [7] who suggested that any particular stabilized posture of the animal results from interactions between numerous reflexes (driven by vestibular, visual, and somatosensory inputs), and that the reflexes either supplement or counteract each other. Experiments by von Holst [14] on the swimming fish presented additional evidence in favor of this idea. Our experiments on some other simpler systems — the mollusk *Clione* [15,16] and the lamprey (a lower vertebrate) [17–19] also support the reflex concept of postural control (see below).

The postural system normally operates as a functional unit and stabilizes both the head orientation and the trunk orientation. Under certain conditions, however, the system clearly dissociates into the sub-systems controlling independently the head and the trunk [20–23]. Our recent studies on the rabbit suggest that, with a complication of the postural task, lateral stability of the anterior and posterior parts of the body is maintained by two relatively independent sub-systems driven by their own somatosensory inputs [24]. These experiments are illustrated in Fig. 1. The rabbit was positioned on two platforms, one for the fore limbs and one for the hind limbs, subjected to periodical lateral tilts (P1 and P2 in Fig. 1C). Postural corrections, *i.e.*, lateral displacements of the anterior and posterior parts of the trunk in relation to the platforms, were recorded by mechanical sensors (S1 and S2 in Fig. 1A–C).

With in-phase tilts of the platforms, the animal stabilized its dorsal-side-up position by displacing the whole body in the direction opposite to tilt (Fig. 1B,D). These compensatory body movements were caused by simultaneous extension of the fore and

hind limbs on the side moving down, and flexion of the opposite limbs; the movements were presumably controlled together on the bases of integrated sensory information. With anti-phase tilts of the two platforms, the animal also maintained its dorsal-side-up position. In this case, however, the compensatory movements of the anterior and posterior body parts were in anti-phase (Fig. 1E). These movements were caused by anti-phase flexion/extension movements of the ipsilateral fore and hind limbs (Fig. 1F,G). The rabbit was also able to stabilize its dorsal-side-up orientation when the platforms were tilted at different frequencies, or when one platform was tilted and the other was not. These data are difficult to explain in the framework of the single regulated variable hypothesis. One can rather suggest that the anterior and posterior parts of the body have separate postural control mechanisms driven by the corresponding somatosensory inputs and, under certain conditions, these mechanisms operate independently of each other.

Such a functional organization is similar to that of the locomotor system in quadrupeds, where the shoulder and hip girdles have their own control mechanisms, and even individual limbs have relatively autonomous controllers that generate stepping movements and interact with each other to secure the inter-limb coordination [4]. It was suggested that a control system consisting of semi-autonomous sub-systems better adapts to complicated environmental conditions [25].

3. Localization of postural functions in the mammalian CNS

As was shown in earlier studies, chronic decerebrate animals can sit, stand, and walk; when positioned on its side, the animal exhibits a set of righting reflexes and rapidly assumes the normal, dorsal-side-up posture [26]. These findings indicate that an essential part of the nervous mechanisms responsible for the control of the basic posture in quadrupeds is located below the decerebration level, that is, in the brain stem, cerebellum, and spinal cord.

The involvement of the brainstem and cerebellum in postural control has been confirmed in two lines of experiments. First, it was found that electrical stimulation of specific sites in the brain stem (dorsal and ventral tegmental field) and in the cerebellum (hook bundle) strongly affected the tone of anti-gravity (extensor) muscles [27–29]. These effects are mediated by reticulospinal and vestibulospinal pathways. Second, single neuron recordings in the intact cat walking on the tilted treadmill demonstrated that brain stem neurons giving rise to descending tracts, vestibulospinal and reticulospinal, considerably changed their activity with a change of tilt angle [30]. It remains unclear, however, if the tilt-related activity of these neurons is responsible for the generation of postural corrections, or only for modulation of postural responses generated by the spinal mechanisms (see below).

Interactions between the spinal and supraspinal levels of the trunk stabilization system are rather complex; they are schematically shown in Fig. 2A. There are two closed-loop nervous mechanisms (loops L1 and L2). *One of the mechanisms* (loop L1) resides in the spinal cord. This mechanism is driven by input from limb mechanoreceptors, and compensates for postural disturbances by generating corrective motor responses. These responses can be considered as a manifestation of the spinal postural reflexes, uncrossed and crossed [7,26].

The other mechanism (loop L2) resides, to a large extent, in the brain stem and motor cortex [27,28,31,32]. This mechanism is also driven by signals from limb mechanoreceptors but, in addition, it receives information about head orientation from visual and vestibular systems. Output of this mechanism is a phasic corrective command (Com. 1 in Fig. 2A) addressed to the spinal mechanisms *via* different descending pathways (reticulospinal, corticospinal, *etc.*). This command will elicit a correction of posture [2].

There are two sources of supraspinal *tonic drive* addressed to the spinal postural mechanisms. One of them is the command system for activation of spinal postural mechanisms. Its output is designated as Com. 2 in Fig. 2A. In the resting animal, the tone in anti-gravity (extensor) muscles and the postural reflexes are absent. They appear with activation of postural/locomotor mechanisms due to the excitatory drive from the brain stem, most likely through the reticulospinal pathways [27,28,33].

The other source of tonic drive to the spinal networks is the command system causing modifications of the stabilized body configuration (Com. 3 in Fig. 2A). It is known that animals are able to keep balance at different body configurations, that is with different inter-limb distance, different orientation of their body axis, different degree of limbs extension, *etc.* [24,32,34]. To maintain these different configurations, the tonic activity of limb should be correspondingly modified. The supraspinal mechanisms providing these tonic commands seem to be located in the brain stem [27,28,35].

The relative importance of the two functions of the spinal cord – mediating the supraspinal commands for postural corrections and generation of spinal postural reflexes – is not clear, however. It is well established that animals with a complete transection of the spinal cord in a lower thoracic region exhibit poor postural responses and, as a rule, are not able to maintain the dorsal-side-up orientation of their hindquarters [36,37], though a reduced postural control may remain [38] and can be improved by training [39–41]. These results may have two different interpretations. First, they can be considered as evidence for the minor role that is played by spinal postural reflexes for maintenance of body posture [2]. The second, alternative hypothesis is that the transection of the spinal cord deprives the spinal postural networks of the necessary supraspinal tonic drive (Com. 2 in Fig. 2A), which results in a reduction in the activity of spinal postural reflex mechanisms [21]. Indirect evidence for this hypothesis was obtained in lesion experiments. Animals subjected to a lateral hemisection of the spinal cord, after a short period of recovery, were able to maintain equilibrium during locomotion and standing [42,43] and generate postural corrections in response to lateral tilts [35]. Since the hemisection causes dramatic changes both in ascending signals and in descending commands, one can suggest that the persistence of postural control after this lesion is due to the activity of spinal postural mechanisms.

Until recently, participation of the forebrain in feedback postural control was hypothesized [2] mainly on the basis of clinical and lesion studies [see *e.g.*, [44,45]]. Participation of the motor cortex in the control of basic posture was directly demonstrated in our experiments by recording the activity of motor cortex neurons in the rabbit during postural corrections [31]. It was found that most corticofugal neurons of layer 5 (that give rise to the corticospinal tract) were modulated during postural

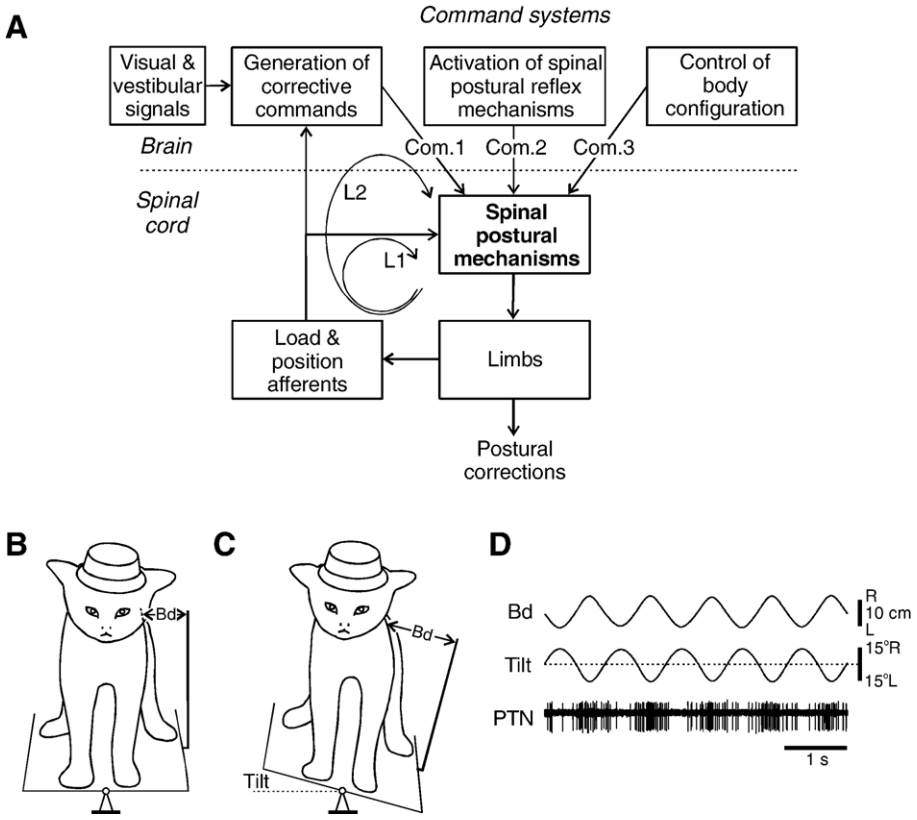

Fig. 2. Distribution of postural functions in the CNS (hypothesis). A: Relationships between spinal and supraspinal levels of postural control for the fore- and hindquarters. Operation of the system is based on the activity of two closed-loop mechanisms, the spinal (loop L1) and supraspinal (loop L2) ones. Supraspinal influences include phasic corrective commands (Com. 1) and two tonic drives — Com. 2 that activates the spinal postural mechanisms, and Com. 3 that determines the postural body configuration. B–D: Motor cortex participates in postural control. B,C: The cat was maintaining balance on the tilting platform and produced postural corrections (Bd) in response to tilts. D: The activity of a pyramidal tract neuron (PTN) from the forelimb representation of the motor cortex was modulated in relation to tilts and postural corrections.

corrections caused by lateral tilts of the supporting platform. Similarly, a tilt-related modulation of the activity was found in the pyramidal tract neurons of the cat maintaining balance on the tilting platform (Fig. 2B–D) [32]. Functional significance of these corticofugal signals is not clear, however, since integrity of the motor cortex is not necessary for stabilization of the basic body posture [26]. One can suggest that cortical contribution is important for acquiring new postural synergies [45] or for compensation of postural disorders caused by damage to the postural networks.

4. Postural networks

An analysis of postural neuronal networks has been performed for two simpler animal models, the marine mollusk *Clione* and the lamprey. These aquatic animals actively stabilize their orientation in the gravity field: *Clione* usually swims with its head up, the lamprey — with the back up. For both models, similar experimental approaches were used. First, the principal elements of the postural network and their interconnections were revealed. Second, the corrective motor commands under open-loop conditions, when these commands did not affect the body orientation, were characterized. Third, by employing a method of an artificially closed feedback loop [15,46], the activity of the network during stabilization of a particular body orientation was examined. It was found that in both animals the operation of the postural systems is based on interaction of vestibular reflexes.

The postural network in *Clione*, responsible for stabilization of the body orientation in a particular plane, includes two chains of antagonistic tail reflexes driven by gravitational input from two statocysts [15,16]. The system stabilizes the orientation at which the two reflexes compensate for each other (the equilibrium point). Normally this occurs at the vertical, head-up orientation [16]. Raising the water temperature causes a dramatic reconfiguration of the network and a reversal of postural reflexes. This leads to a change of the equilibrium point in the system from the head-up orientation to the head-down orientation [15,47]. The system is also able to modify gradually the stabilized orientation by changing the gain in one of the reflex chains [48].

In the lamprey, the postural control system can be subdivided into the roll and pitch control systems stabilizing body orientation in the transverse and sagittal planes, respectively. Operation of each system is based on the interactions between two antagonistic vestibular postural reflexes, mediated by two groups of reticulospinal (RS) neurons causing rotation of the animal in the opposite directions. This is illustrated for the roll control system in Fig. 3A [17,46,49]. Due to vestibular input, the activity of RS neurons depends on the orientation of the animal in the transverse plane (Fig. 3B). The roll control system stabilizes the orientation at which the antagonistic reflexes compensate for each other (the equilibrium point).

Normally, this occurs at the dorsal-side-up orientation of the animal. The stabilized orientation can be changed by unilateral eye

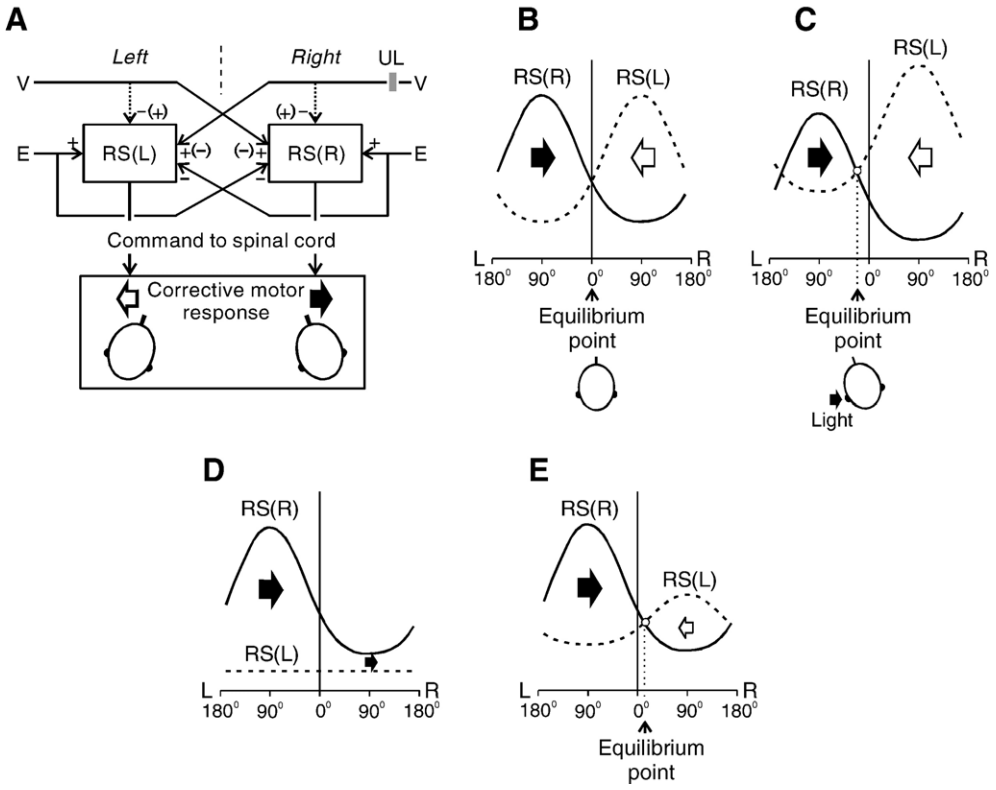

Fig. 3. A: Conceptual model of the roll control system in the lamprey. Two groups of RS neurons, RS(R) and RS(L), receive inputs from the labyrinths (V) and eyes (E); they affect the spinal networks to evoke rolling of the lamprey. The signs (+ and −) indicate the major effects on RS neurons produced by sensory inputs, the signs in brackets — the minor effects. B: Operation of the system when driven only by vestibular inputs. The curves represent activity in RS(R) and RS(L) as a function of roll angle (L — left tilt, R — right tilt). Vestibular input causes activation of RS(R) and RS(L) with the contralateral tilt. Directions of rolling caused by RS(R) and RS(L) are indicated by the black and white arrows, respectively. The system has an equilibrium point at 0° (dorsal-side-up orientation). C: Operation of the system when the left eye is illuminated. This visual input causes a shift of the equilibrium point to the left and the corresponding tilt of the animal. D: Effect of the left labyrinthectomy (UL in A). The system has no equilibrium point, and the animal continuously rolls. E: In the compensated animal, the equilibrium point re-appears.

illumination, which affects differently the two groups of RS neurons and causes a shift of the equilibrium point (Fig. 3C). In the pitch control system, the stabilized orientation also corresponds to the equal activity in the two antagonistic groups of RS neurons. The stabilized pitch angle can be changed by raising the water temperature, which affects differently the activity of the two groups [19].

The effects of signals transmitted from the brain stem to the spinal motor networks by a population of RS neurons are determined by specific functional projections of each individual neuron. To reveal these projections, responses of the spinal networks to the spikes in single RS axons were detected by employing the spike-triggered averaging [50]. It was found that individual RS neurons exert a uniform effect on the segmental motor output along the whole extent of their axons. Twenty different patterns of effect, that is combinations of influences on the segmental motoneuron pools, were found. The widespread projections and heterogeneity of the population of RS neurons present a basis for formation of different motor synergies responsible for postural corrections.

5. Impairment of postural control caused by vestibular deficit

Dysfunction of the vestibular organ results in numerous pathological symptoms. The most dramatic symptoms follow a complete removal of the vestibular organ (unilateral labyrinthectomy, UL) and include loss of postural stability, asymmetry in the head and trunk posture, *etc*. During the recovery from UL, all symptoms gradually become less pronounced and some of them disappear almost completely, while others remain noticeable for many months and even years [8]. The process of recovery after UL is termed "vestibular compensation". The mechanisms involved in vestibular compensation are still not clear [8,51].

In the lamprey, because its postural control system is driven primarily by vestibular input, the effect of UL is most dramatic. After UL, lampreys completely lose equilibrium and continuously roll during swimming toward the damaged labyrinth [49]. In the rolling lamprey, however, the equilibrium can be temporarily restored by creating an asymmetry in visual input, *e.g.*, by illuminating the eye contralateral to UL [52]. During the process of vestibular compensation, which lasts a few weeks, the UL lampreys gradually recover their capacity to maintain equilibrium [49].

The model of the roll control system based on two antagonistic vestibular reflexes (Fig. 3A) well explains the origin of the main postural deficit (continuous rolling) observed after UL. The model suggests that the group of RS neurons deprived of the contralateral vestibular input is inactivated and cannot counteract the antagonistic group which elicits continuous rolling (Fig. 3D). Our experiments, with recording vestibular responses in individual RS neurons before and soon after UL, confirmed this suggestion [18]. It was also found that illumination of the eye contralateral to UL

(which causes recovery of equilibrium in non-compensated UL animals) restores vestibular responses in the deafferented RS neurons [18]. It was suggested that, during vestibular compensation, the non-active (deafferented) group of RS neurons restores its activity due to plastic changes in the postural network that enhance input to RS neurons from the intact (ipsilateral) labyrinth (Fig. 3E) [18]. This suggestion was also confirmed experimentally [53].

6. Concluding remarks

Information about the organization and operation of postural networks is necessary both for a deeper understanding of the postural system *per se*, and as a prerequisite for any attempt to understand the pathological cases, *e.g.*, impairment and recovery of postural control after damage to different parts of the brain or to sensory organs. Our analysis of postural networks in simpler animal models (*Clione* and lamprey) revealed remarkable similarities in their organization and operation, despite a great evolutionary distance between the two species. In both animals, stabilization of the body orientation in a particular plane is based on the interaction of two antagonistic postural reflexes, and the animal stabilizes the orientation at which the reflexes are equal to each other. This knowledge on the organization of postural networks allowed us to explain the origin of postural dysfunctions (loss of equilibrium) caused by vestibular deficit in lampreys, and to develop the means for restoration of postural functions.

Studies on higher vertebrates have revealed three sub-systems responsible for the stabilization of different parts of the body (head, anterior and posterior parts of the trunk) in the transversal plane. At present we address the questions: (i) Where are these mechanisms located? (ii) How do the corresponding neuronal networks operate? Answering these questions is important for developing the methods of restoration of postural functions in subjects with sensory and motor deficits.

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