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Neural Bases of Postural Control

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The body posture during standing and walking is maintained due to the activity of a closed-loop control system. In the review, we consider different aspects of postural control: its functional organization, the distribution of postural functions in different parts of the central nervous system, and the activity of neuronal networks controlling posture.

Most terrestrial, aquatic, and flying animals maintain a definite orientation in the extra-subject space. Stabilization of the whole body orientation in relation to the gravity force or to the earth surface is often termed the postural and balance control. Maintenance of the basic body posture (upright in bipeds and dorsal side-up in quadrupeds and many aquatic animals) is a vital motor function. Maintenance of this posture is a nonvolitional activity based, to a large extent, on the in-born neural mechanisms (52, 74). Efficient control of the body posture is important both during standing and during walking (38, 47, 59). A well-stabilized body posture is necessary to provide support for voluntary limb, head, or trunk movements (4, 51, 53). Deficiency in sensory or motor mechanisms of the postural system dramatically affects postural stability and motor performance (18, 46, 50, 53, 67).

One can distinguish two main modes of postural control. In the feedback or reflex mode, the postural system responds to perturbations of orientation and causes corrections of posture (26, 38). In the anticipatory mode, the system causes changes of posture in advance to counteract the destabilizing effects of either expected external influences or expected voluntary limb movement initiated by the subject itself (13, 38). This review is devoted to the feedback mode of postural control and, specifically, to its neuronal mechanisms.

During the last decade, neuronal mechanisms for the control of body posture were investigated in "animal models" of different complexity: the mollusc *Clione* (e.g., Refs. 18–20), the lower vertebrate lamprey (14, 24, 61), and two quadrupedal mammals: the rabbit (6) and the cat (4, 64, 66). Comparative studies of postural mechanisms are based on the assumption that, despite differences in anatomy and behavior between species, a basic problem such as the nervous control of the antigravity behavior may have similar solutions in different species (26). 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 central nervous system. 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 (e.g., Refs. 18, 27, 62, 63) or spinal cord injury (46) have been investigated to better understand the postural mechanisms per se and the process of recovery of postural function, and to search means of promoting recovery. Specific aspects of the physiology and pathophysiology of postural control in humans are out of the scope of this paper (for review, see Ref. 38).

Postural Mechanisms in Simpler Animal Models

Clione and lamprey are aquatic animals; both actively maintain a specific body orientation due to gravitational reflexes driven by statocists (*Clione*) or by vestibular organs (lamprey). Under normal conditions, they actively stabilize a definite body orientation in space; however, specific environmental factors cause a change of stabilized orientation.

Postural networks in mollusc Clione

The marine mollusc *Clione* swims due to beating of two wings. *Clione* actively stabilizes its orientation in the gravity field. Usually it swims with the head up (FIGURE 1B). Deviation from this orientation evokes reflex bending of the tail in the opposite direction. Flexion of the tail causes rotation of the swimming animal toward the vertical. In this animal, some principles of postural control have been revealed. First, the postural network, responsible for stabilization of the body orientation in a particular plane, includes two chains of antagonistic postural reflexes driven by gravitational input from two statocysts and mediated by identified groups of interneurons and tail motoneurons (FIGURE 1A) (19, 20, 60). The system stabilizes the orientation at which the two reflexes compensate for each other [the equilibrium point of the control system (45)] (FIGURE 1B). Normally, this occurs at the vertical, head-up orientation (20). After removal of statocists, *Clione* is not able to maintain any definite

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orientation in space (60). Second, raising the water temperature causes reconfiguration of the postural network (FIGURE 1A), which results in a reversal of postural reflexes. This leads to a change of the equilibrium point in the system from head-up to head-down orientation, and this new orientation is stabilized (FIGURE 1C), which allows *Clione* to leave the warm water layers (19, 21). Third, the postural network can also gradually change the stabilized orientation by changing the gain in one of the reflex chains (22) (a similar phenomenon is illustrated for the lamprey in FIGURE 1F; see also explanations below).

Postural networks in lamprey

In the swimming lamprey (a lower vertebrate, cyclostome), the body orientation in the transverse (roll) plane and in the sagittal (pitch) plane is stabilized by closed-loop control systems driven by vestibular input (14, 24, 61). Vestibular reflexes also contribute to stabilization of the direction of swimming in the hori-

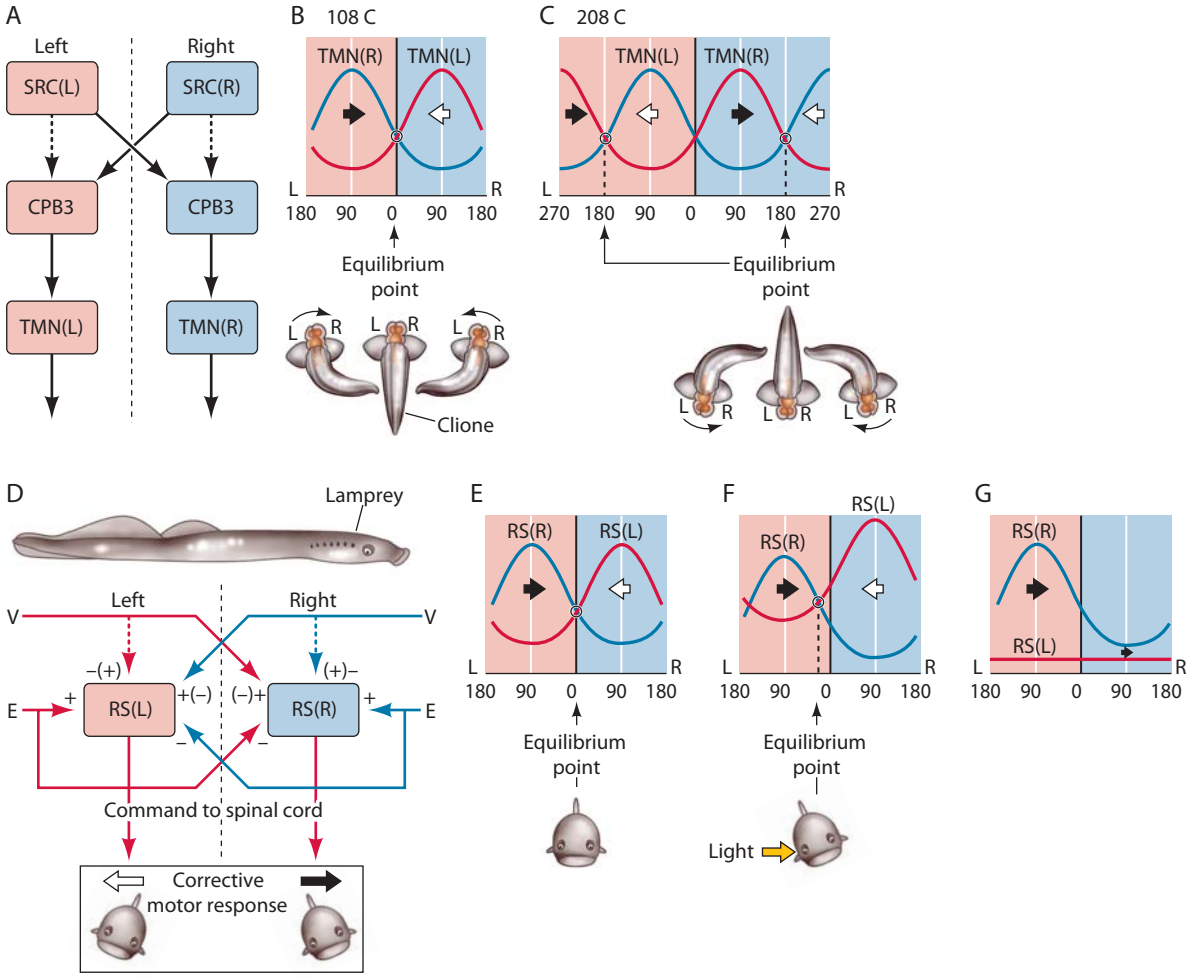


FIGURE 1. **Postural networks in *Clione* and lamprey**

A: neuronal network in the mollusc *Clione* for the control of body orientation in the frontal plane. Statocist receptor cells (SRC) exert an excitatory effect on CPB3 interneurons, which in turn excite tail motoneurons (TMN) that cause left (L) or right (R) tail flexion. Connections between SRC and CPB3 are not fixed but depend on temperature (solid lines, connections at lower temperature; interrupted lines, connections at higher temperature). *B* and *C*: operation of the network at lower temperature (*B*) and higher temperature (*C*). Due to input from statocists, the activity of left and right TMNs (ordinate) and their effect on tail flexion depend differently on lateral tilt. Directions of rotation of *Clione* caused by TMN(R) and TMN(L) are indicated by the black and white arrows, respectively. At lower temperature, the system has an equilibrium point at 0° (head-up orientation). At higher temperature, the system has an equilibrium point at 180° (head-down orientation). *D*: model of the roll-control system in the lamprey (body outline of the lamprey is shown at *top*). 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 indicate the minor effects. *E*: 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). *F*: 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. *G*: effect of the left labyrinthectomy (UL in *A*). The system has no equilibrium point, and the animal continuously rolls. *H*: the equilibrium point can be restored by left eye illumination.

zontal (yaw) plane (41). Any deviation from the stabilized orientation is reflected in vestibular signals, which cause a motor response (correction of orientation). In the pitch and yaw planes, the corrections occur due to the bending of the body in the corresponding plane (70, 71). In the roll plane, the corrections occur due to a change of the plane of locomotor body undulations (80). These different types of motor responses are caused by four motoneuron pools in each segment that innervate the dorsal and ventral muscles on the two sides (FIGURE 2A) (65, 75, 78).

In the lamprey, commands for changing body orientation during swimming are transmitted from the brain stem to the spinal cord mainly by reticulospinal (RS) neurons (FIGURE 2A) (10, 12, 58), which constitute the only well-developed descending pathway in the lamprey. RS neurons, due to their vestibular inputs (FIGURE 2A), respond to rotation of the animal in the roll, pitch, and yaw planes (e.g., see FIGURE 2B) (14, 23, 24, 41, 61). Populations responding to rotation in different planes partly overlap (61, 81).

Like in *Clione*, operation of each control system, stabilizing orientation of the lamprey in a particular plane, is based on the interactions between two antagonistic vestibular reflexes, mediated by two groups of RS neurons causing rotation of the animal in the opposite directions. This is illustrated schematically for the roll control system in FIGURE 1D (16, 24, 77). Due to the vestibular input, the activity of each group of RS neurons depends on the orientation of the animal in the roll plane (FIGURE 1E). Each group, due to specific influences on the spinal mechanisms, elicits a motor response that counteracts a deviation from the stabilized orientation (Ref. 81; see also below). The system has an equilibrium point: it stabilizes the orientation at which the antagonistic vestibular reflexes compensate for each other. Normally, this occurs at the dorsal side-up orientation. Not only static but also dynamic compo-

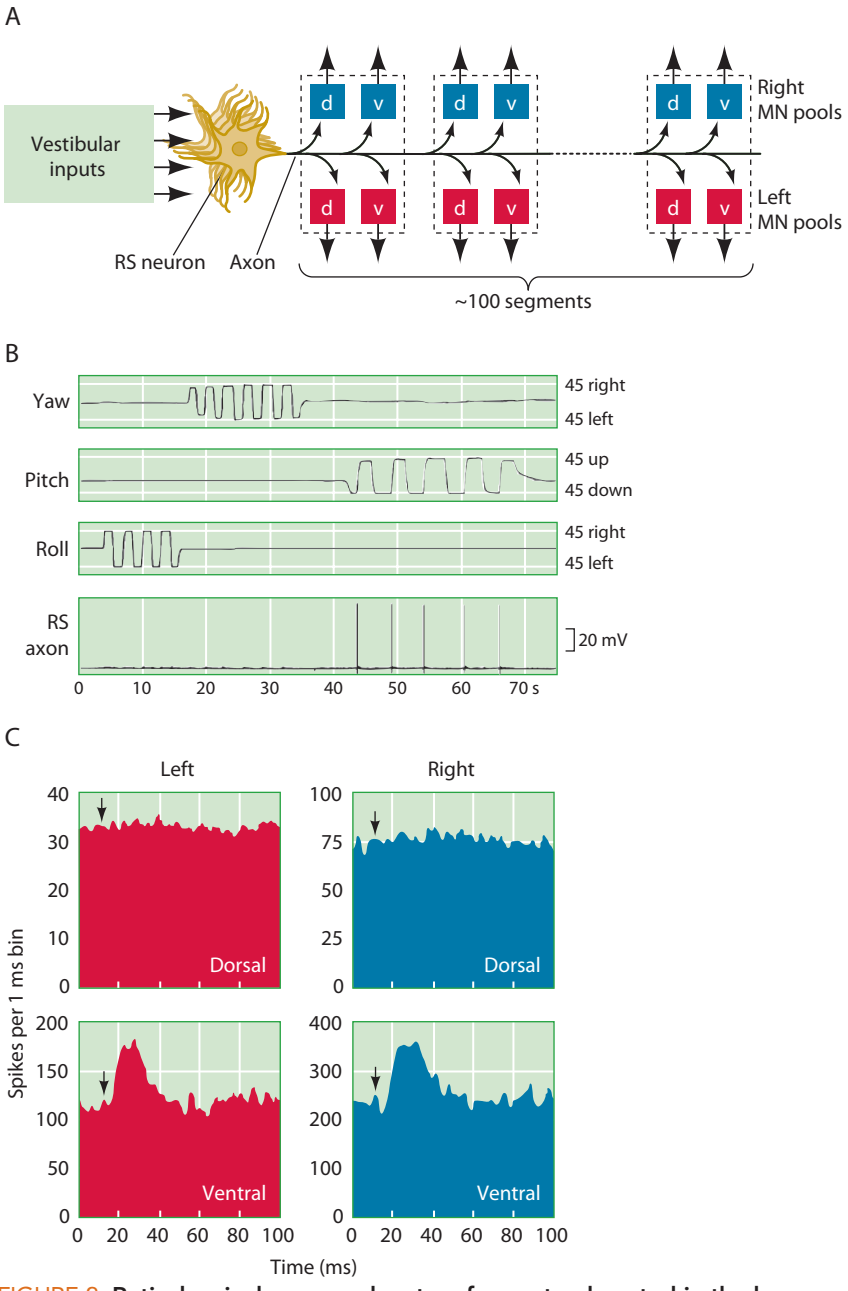


FIGURE 2. **Reticulospinal command system for postural control in the lamprey**
A: reticulospinal command system in the lamprey. Individual RS neurons send axons to the spinal cord; many of them reach the most caudal spinal segments. Segmental motor output is generated by four MN pools controlling the dorsal and ventral parts of a myotome on the two sides (*d* and *v* pools, respectively). The RS neurons may exert their effect on the pools directly or through interneurons. The RS neurons receive vestibular inputs. *B* and *C*: vestibular input and motor effect of one of the RS neurons. The neuron responded to the nose-up pitch tilt (*B*) and caused excitation of motoneurons projecting to the ventral muscles (*C*). Motor effect of the RS neuron spikes was determined by spike-triggered averaging of the activity of motoneuron pools projecting to the left dorsal, right dorsal, left ventral, and right ventral muscles, respectively. Arrows indicate the moment of arrival of RS spike to the recorded segment.

nents of vestibular responses (since they are position dependent) contribute to postural stabilization (77).

Like in *Clione*, the stabilized orientation in the lamprey can be gradually changed under the effect of some environmental factors. Unilateral eye illumination affects the two antagonistic groups of RS neurons differently and causes a shift of the equilibrium point of the system, which results in a change of stabilized orientation (FIGURE 1F) (15, 24, 71, 73). In the pitch control system, the stabilized orientation also corresponds to the equal activity in the two antagonistic groups of RS neurons sensitive to the nose-up and nose-down tilt, respectively (14, 61). The stabilized angle can be changed by raising the water temperature, which affects the two groups differently and thus shifts the equilibrium point toward the nose-down orientation (61). The cellular basis of this phenomenon remains unclear.

The effects of signals transmitted from the brain stem to the spinal motor networks by a population of RS neurons depend on motor effects (functional projections) of individual neurons. To reveal these projections, responses of the spinal networks to the spikes in single RS axons were detected by employing the spike-triggered averaging technique (78, 79, 81). 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. A strong correlation was demonstrated between *1*) the vestibular inputs to individual RS neurons and *2*) the effects on efferent spinal output exerted by the same RS neurons (81). As a rule, if a neuron was activated by a turn in a given plane, its motor effect practically always caused a torque opposing the turn. For example, the neuron excited by the nose-up pitch tilts (FIGURE 2B) caused activation of both left and right ventral muscles (FIGURE 2C). These muscles will cause the nose-down body bending, i.e., the motor response that allows the animal to compensate for the initial postural perturbation (nose-up tilt). Correspondingly, the neurons responding to a turn in the roll or yaw plane caused a torque in the same plane but in the opposite direction. Some neurons responded to turns in more than one plane (61); they had functional projections that contributed to generation of the motor patterns causing torques in the corresponding planes (81). Thus the synergies for different postural corrections are formed by RS neurons.

To conclude, despite very different neuronal structure of postural networks in the two evolutionary remote species (*Clione* and lamprey), their function is based on the same principle: interaction between two antagonistic gravitational postural reflexes. Both animals stabilize the body orientation in a particular plane at which the antagonistic reflexes are equal to each other. Both animals are able to gradually change the stabilized orientation through the change of the gain in one of the reflex chains. In addition, *Clione* can maintain two distinct orientations due to the reconfiguration of connections between elements of the postural network.

Postural Mechanisms in Mammals

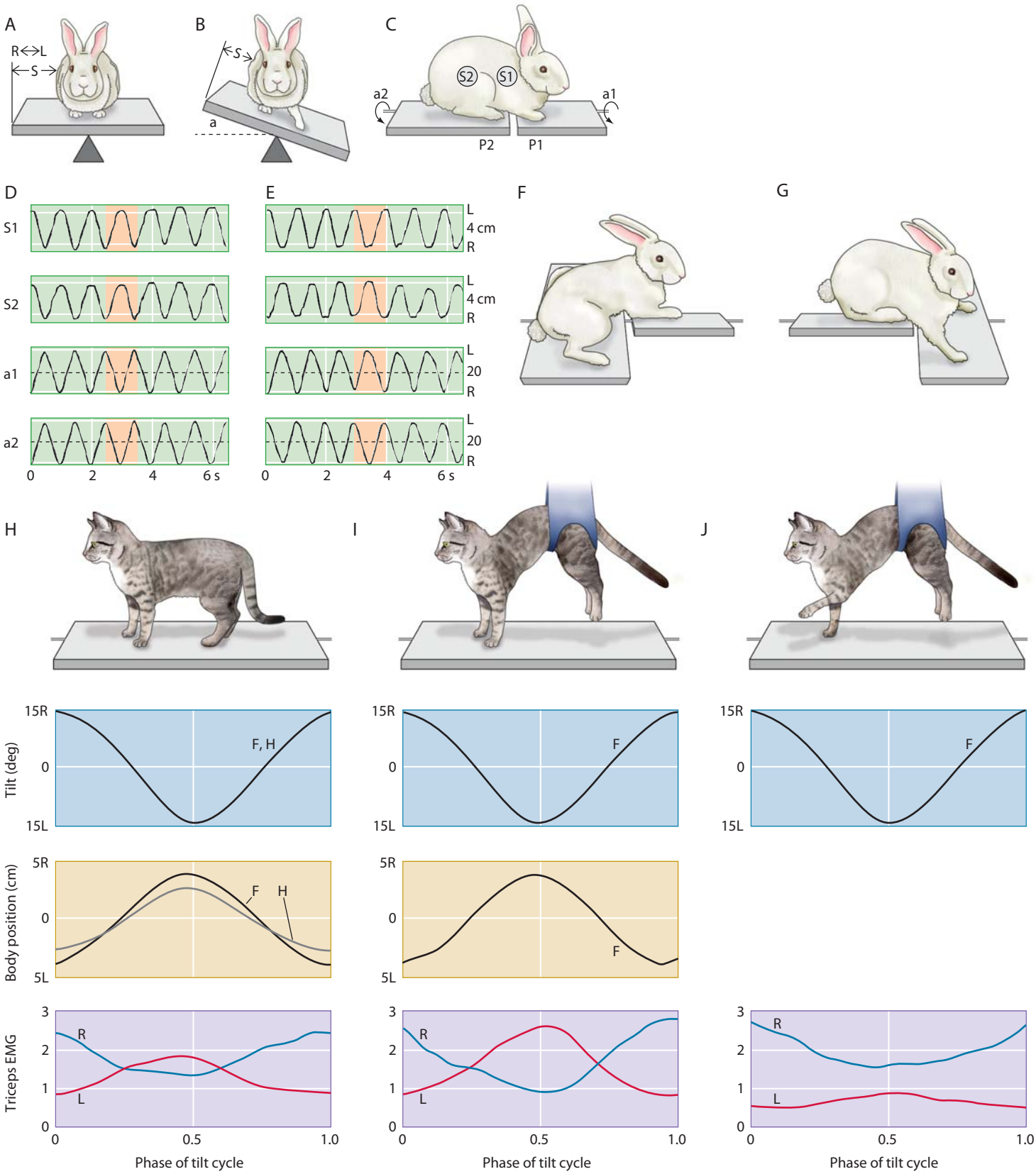
In contrast to *Clione* and lamprey, which are aquatic animals, the cat and rabbit are terrestrial quadrupeds. The main distinctions of their postural control as compared to the lamprey are *1*) an important role of somatosensory information in driving postural mechanisms (5, 25), *2*) existence of several descending pathways originating from the brain stem, *3*) existence of the forebrain motor centers, and the motor cortex in particular, which may supplement the basic (brain stem-spinal) postural mechanisms, and *4*) existence of several body segments with a large number of degrees of freedom.

Functional organization of postural system

The postural control system usually operates as a functional unit and stabilizes both head and trunk orientation. It was suggested that the regulated variable in the control system is the position of the center of mass or the orientation of the body and limb axes (33, 43, 52, 53). Under certain conditions, however, the system clearly dissociates into the subsystems independently controlling the head and the trunk (2, 4, 7, 8, 25). These subsystems are driven by sensory signals of different modalities: the head orientation is stabilized mainly on the basis of vestibular and visual information; for trunk stabilization, somatosensory inputs from limbs are most important (5, 25).

Studies on rabbits and cats 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 subsystems driven by somatosensory inputs from the corresponding limbs (5). In experiments with two platforms (FIGURE 3, A—C), the animal was able to stabilize its dorsal side-up orientation when the platforms were tilted not only in phase (simple postural task) but also in anti-phase (complex postural task). In the latter task, the compensatory movements in the fore and hindquarters were not linked with each other; they rather represented responses to tilts of the corresponding platform.

In our laboratory's recent studies (27a), the cat performed postural corrections on the tilting platform when different parts of the body were suspended. It was found that a single girdle (shoulder or hip) compensates for tilts as efficiently as in control, i.e., when the other girdle also participates (FIGURE 3, H AND I). When only one limb supported the girdle, the timing of the muscular pattern in both limbs of that girdle (FIGURE 3J) was similar to that in control (FIGURE 3H). It seems likely that reflex mechanism of an individual limb generates



only a part of postural corrective response; another part is produced on the basis of crossed influences.

These results suggest that the anterior and posterior parts of the body have separate postural control mechanisms driven by the corresponding somatosensory inputs and that, 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 interlimb coordination (59). It was suggested that a control system consisting of semi-autonomous subsystems better adapts to complicated environmental conditions (32).

Role of spinal and supraspinal postural mechanisms

As 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 posture (3, 50). Thus an essential part of nervous mechanisms for the control of basic posture in quadrupeds is located below the decerebration level, i.e., in the brain stem, cerebellum, and spinal cord.

The involvement of the brain stem 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 (1, 56, 57, 69). These effects are likely to be mediated by RS 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 (54, 55). It remains unclear, however, whether the activity of these neurons is responsible for the generation of postural corrections or only for modulation of postural responses generated by spinal mechanisms.

FIGURE 3. **Functional organization of postural control in quadrupeds**
A—*G*: corrective motor responses in the rabbit observed in postural tasks of different complexity. *A*—*C*: the rabbit was positioned on two platforms, one for the fore limbs and one for the hind limbs (P1 and P2), subjected to periodical lateral tilts. 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). *D* and *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. 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 (*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. With anti-phase tilts of the two platforms (*E*), 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. *F* and *G*: the body outline for the two extreme positions (left and right) of the platforms tilted in anti-phase to show that compensatory body movements were caused by anti-phase flexion/extension movements of the limbs on each side. *H*—*J*: postural responses in the cat to lateral tilts of the platform under different conditions: four-limb support (*H*), two-limb support (*I*), and one-limb support (*J*). Different values are shown as a function of the phase of the tilt cycle: tilt angle (*top* graphs), postural corrections (*middle* graphs) in the fore (F) and hind (H) quarters, and electromyograms of left (L) and right (R) triceps (*bottom* graphs).

Interactions between the spinal and supraspinal levels of the trunk stabilization system are rather complex; they are schematically shown in FIGURE 4, A AND B, for the hindquarters. There are two closed-loop nervous mechanisms (*loops L1* and *L2* in FIGURE 4A). One of them (*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. The other mechanism (*loop L2*) includes the brain stem and motor cortex (6, 4, 56, 57). 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 command addressed to the spinal cord via different descending pathways (reticulospinal, corticospinal, etc.). This command, together with spinal reflexes, contributes to corrections of posture.

There are two sources of supraspinal tonic drive addressed to the spinal postural mechanisms (FIGURE 4B). One of them is the command system for activation of the spinal networks. In the resting animal, the tone in anti-gravity (extensor) muscles and the postural reflexes are absent. They appear with activation of postural/locomotor networks due to the excitatory drive from the brain stem, most likely through the RS pathways (34, 56, 57, 69). Experiments with different damages of the spinal cord have shown that ventral pathways (presumably vestibulospinal and reticulospinal) are crucially important for postural control (11, 46). In their turn, the brain stem postural mechanisms are controlled by input from basal ganglia (69).

The other source of tonic drive to the spinal networks is the command system causing modifications of the stabilized body configuration. It is known that animals are able to keep balance at different body configurations, e.g., with different interlimb distance, different orientation of their body axis, different degree of limbs extension, etc. (5, 4, 43). To maintain these different configurations, the tonic activity of limb muscles and the pattern of spinal postural reflexes should be correspondingly modified. Damage to different spinal pathways causes specific distortions of basic body configuration, which are poorly compensated with time (46). Supraspinal mechanisms providing tonic commands to postural mechanisms seem to be located in the brain stem (46, 56, 57).

The relative contribution of spinal and supraspinal mechanisms to generation of postural corrections at present is not clear. Understanding their roles is important for a choice of appropriate rehabilitation strategies in spinal patients. It is known 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 (48, 49), although

a reduced postural control may remain (31) and some of its aspects (weight support) can be improved by training (28–30, 35). 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 (39). The alternative hypothesis (25, 26) is that the transection of the spinal cord deprives the spinal postural networks of the necessary supraspinal tonic drive (FIGURE 4B), which results in a reduction of activity of the spinal postural mechanisms. Indirect evidence for this hypothesis was obtained in lesion experiments. Animals subjected to a lateral or dorsal hemisection of the spinal cord, after a short period of recovery, were able to maintain equilibrium during locomotion and standing (36, 40, 42) and generate postural corrections in response to lateral tilts (46). 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 (38) mainly on the basis of clinical and lesion studies (e.g., see Refs. 37, 39) as well as studies with transcranial magnetic stimulation during postural tasks (44, 68). Participation of the motor cortex in the control of basic posture was directly demonstrated by recording the activity of motor cortex neurons in the rabbit during postural corrections (6). It was found that most corticofugal neurons of *layer 5* (that give rise to the corticospinal tract) were modulated during postural corrections caused by lateral tilts. Similarly, a tilt-related modulation of the activity was found in the pyramidal tract neurons of the cat maintaining balance on the tilting platform (FIGURE 4, C—E) (4). 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 (3). Cortical contribution might be important for acquiring new postural synergies (39) or for compensation of motor and postural disorders caused by damage to postural networks (9).

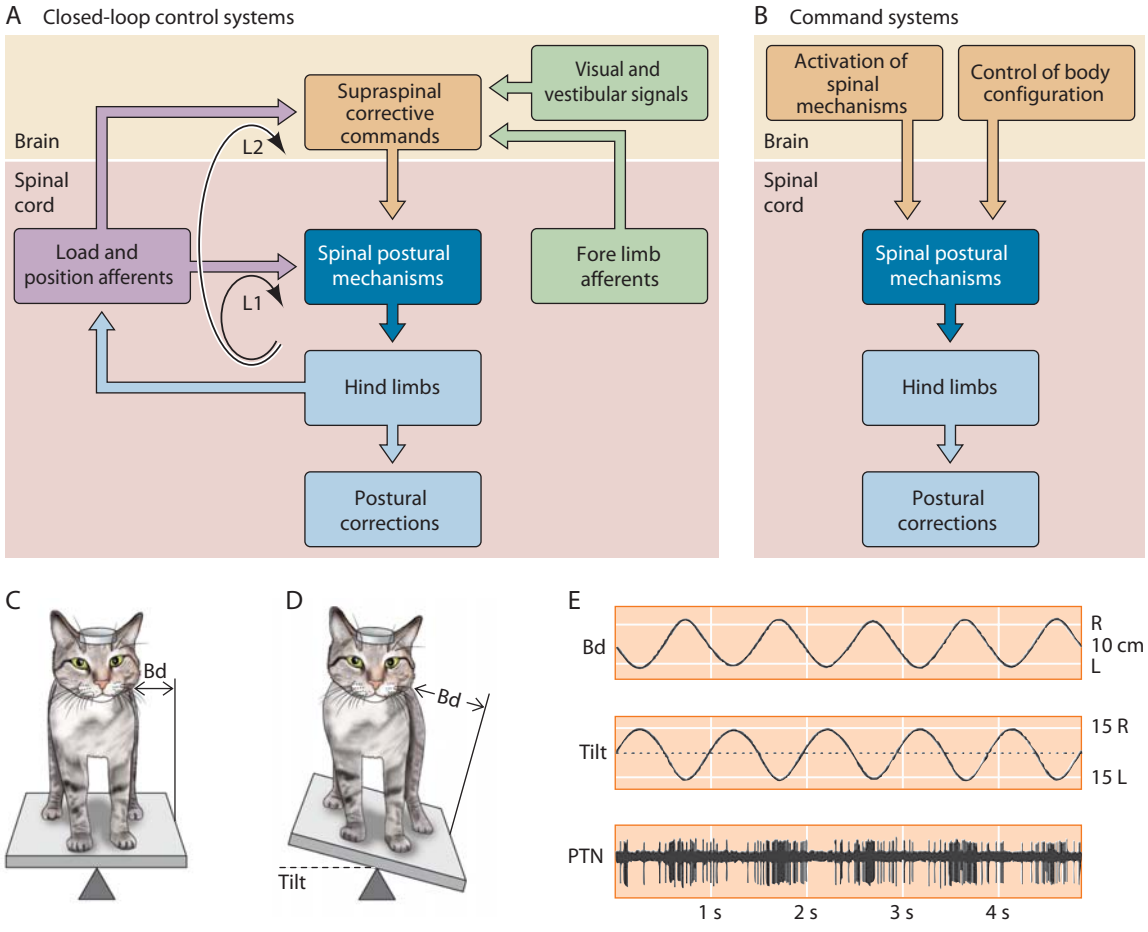


FIGURE 4. **Spinal and supraspinal mechanisms for postural control in quadrupeds**
A and *B*: presumed basic mechanisms of postural control in hindquarters. *A*: functional organization of the feedback mode of postural control. Two closed-loop control systems (*loops L1* and *L2*) stabilize the body orientation. *B*: two supraspinal command systems are responsible for the activation of spinal postural mechanisms and for the control of postural body configuration. *C—E*: motor cortex participates in postural control. *C* and *D*: the cat was maintaining balance on the tilting platform and produced postural corrections (Bd) in response to tilts. *E*: the activity of a pyramidal tract neuron (PTN) from the forelimb representation of the motor cortex was modulated in relation to both tilts and postural corrections.

Impairment of Postural Control by Vestibular Deficit

In rabbits and cats, normally the vestibular input signaling head position and movement contributes significantly to head stabilization but is not necessary for trunk stabilization. However, when phasic vestibular signals caused by platform tilts were artificially amplified (by preventing the head of the rabbit from compensatory postural responses to tilts), they evoked strong postural responses in the limbs and trunk (5). These findings suggest that phasic vestibular signals do not play any significant role for trunk stabilization under ordinary conditions, but vestibular input becomes important when ordinary postural corrections (elicited by somatosensory input) are not effective, and there is a danger of losing balance (as was also demonstrated for falling cats; Ref. 76).

It was found that large asymmetry in tonic vestibular signals in the rabbit (caused by galvanic stimulation of the labyrinths) did not affect the magnitude of corrective responses to lateral tilts but changed the stabilized trunk orientation. It seems likely that asymmetry in tonic vestibular drive affects the set point of trunk stabilizing subsystems (5).

In all classes of vertebrates, dysfunction of 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, whereas others remain noticeable for many months and even years (67). The process of recovery after UL is termed "vestibular compensation." The mechanisms involved in plastic changes underlying vestibular compensation are not quite clear so far (67, 73).

The analysis of postural control after UL was done in the lamprey at the network level. In this animal, because its postural control system is driven primarily by vestibular input, the effect of UL is most dramatic. After UL, swimming lampreys completely lose equilibrium and continuously roll toward the damaged labyrinth (16, 17). During the process of vestibular compensation (a few weeks), UL lampreys gradually recover their capacity to maintain equilibrium (16).

The model of the roll-control system based on two antagonistic vestibular reflexes (FIGURE 1D) 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 main (contralateral) vestibular input is inactivated and cannot counteract the antagonistic group that elicits continuous rolling (FIGURE 1G). Thus UL results in disappearance of the equilibrium point in the roll-control system. Experiments with recording vestibular responses in the same individual RS neurons before and soon after UL confirmed this suggestion (27, 62).

In the rolling UL lamprey, the equilibrium can be temporarily restored by several means, such as asymmetrical visual input, tonic electrical stimulation of the central part of the transected vestibular nerve, etc. (17). Illumination of the eye contralateral to UL restores the equilibrium point of the roll-control system through restoration of vestibular responses in deafferented RS neurons (FIGURE 1H). This is due to an increase of excitability of deafferented neurons, which become able to respond to a weak vestibular input from the ipsilateral labyrinth (27). One can therefore suggest that, during natural vestibular compensation, the nonactive (deafferented) group of RS neurons restores its activity due to plastic changes in the postural network. This process may lead to an increase in the excitability of deafferented neurons and to enhancement of the existing but weak input from the intact (ipsilateral) labyrinth. This suggestion was confirmed experimentally (63).

The idea of elimination of the UL-induced postural deficit by compensation of the central vestibular asymmetry (and restoration of the equilibrium point of the system), formulated initially for the lamprey, was then successfully tested in mammals (rats). Electrical stimulation of the vestibular nerve on the UL side, by substituting a tonic vestibular drive to the vestibulospinal system, restored a normal postural function (18). This result suggests a similarity in some essential principles of postural control in simpler animals and in mammals. In particular, symmetrical tonic vestibular input to brain stem mechanisms is of crucial importance for normal activity of postural networks.

To conclude, during the last decade, due to a use of different animal models and novel techniques, considerable progress has been made in understanding the functional organization of postural mechanisms and in the analysis of the corresponding neuronal networks. In simpler animals, striking similarities in the functioning of postural mechanisms (such as interaction of antagonistic postural reflexes) were found between species as remote as vertebrates and invertebrates. When the mammals and the lower species were compared, considerable distinctions in the design of postural mechanisms were found, such as separate control of different parts of the body as well as a multi-level structure of the control mechanisms in mammals. A common feature of postural mechanisms in all investigated species was the ability to gradually change the stabilized posture by changing a set point in the control system. ■

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