

## Control of Posture Is Task Dependent

The senses and muscles used to control posture vary, depending on task constraints and requirements. For example, when vestibular and somatosensory information is altered while working on a space station, vision is used to orient the body to tasks, and the goal of postural equilibrium changes from preventing falls due to gravity to preventing unintended collision with objects due to inertia. A healthy nervous system very quickly adapts to changing tasks, goals, and environments by modifying its relative dependence upon different sensory information and by using different sets of muscles to optimize achieving the goals of both posture control and voluntary movements.

### Task Requirements Determine the Role of Each Sensory System in Postural Equilibrium and Orientation

The postural control system must be able to change the weighting of different sensory modalities to accommodate changes in the environment and movement goals. Subjects standing on a firm stable surface tend to rely primarily on somatosensory information for postural orientation. When the support surface is unstable, subjects depend more on vestibular and visual information. However, even when the support surface is not stable, light touch with a fingertip on a stable object is more effective than vision in maintaining postural orientation and balance. Vestibular information is particularly critical when visual and somatosensory information is ambiguous or absent, such as when skiing downhill or walking below deck on a ship.

The changeable weighting of individual sensory modalities was demonstrated in an experiment in which subjects were blindfolded and asked to stand quietly on a surface with a tilt that slowly oscillated by varying amounts, up to  $8^\circ$  in magnitude. For tilts of less than  $2^\circ$ , all subjects sway with the platform, suggesting that they use somatosensory information to orient their body to the support surface (Figure 36–12B). At larger tilts, healthy subjects attenuate their sway and orient their posture more with respect to gravitational vertical than to the surface, as they rely more on vestibular information so they stop increasing body sway. Thus, relative sensory weighting changes in control subjects such that somatosensory weight is highest with a stable platform and vestibular weight is highest when standing on an unstable surface, such as with large surface tilts (Figure 36–12B2). In contrast, patients who have lost vestibular function persist in swaying along with the platform and subsequently fall

during large surface tilts. This behavior is consistent with the patients' inappropriate automatic postural response to platform tilts.

Studies such as these suggest that when people are standing on moving or unstable surfaces, the weighting of vestibular and visual information increases, whereas that of somatosensory information decreases. Any sensory modality may dominate at a particular time, depending on the conditions of postural support and the specific motor behavior to be performed.

## Control of Posture Is Distributed in the Nervous System

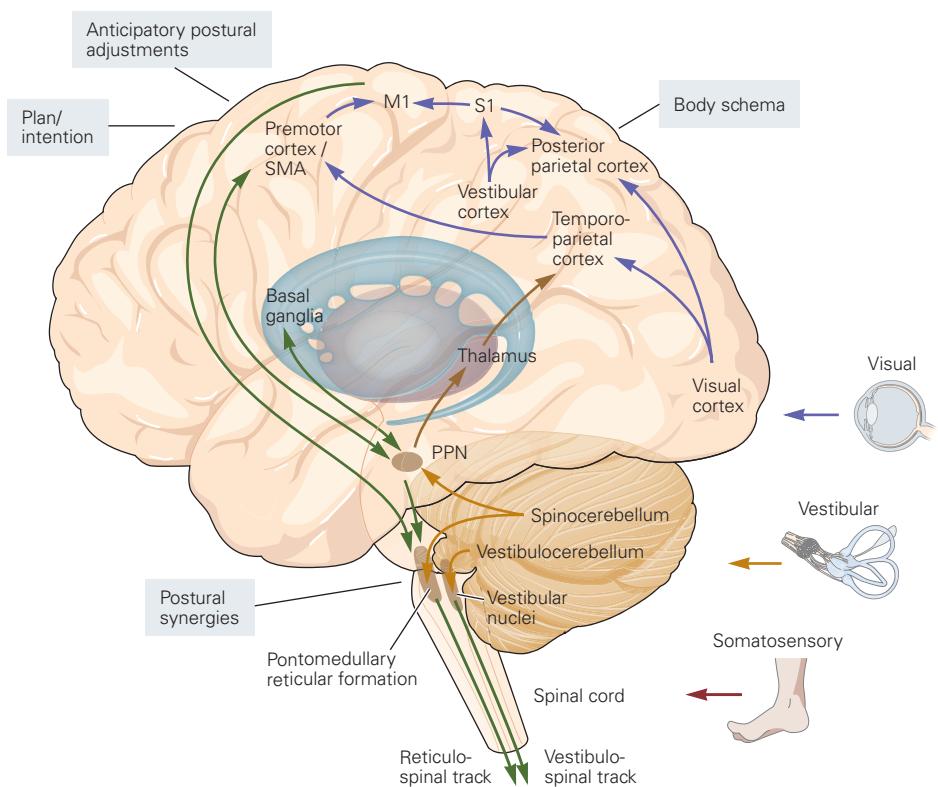
Postural orientation and balance are achieved through the dynamic and context-dependent interplay among all levels of the central nervous system, from the spinal cord to cerebral cortex. The major areas of the brain involved in postural control are shown in Figure 36–13. Signals from specific areas in all lobes of the cerebral cortex converge and are integrated to determine appropriate outputs from motor cortical areas to subcortical structures. The basal ganglia, cerebellum, and pedunculopontine nucleus then send outputs to the brain stem. Ultimately, inputs from these varied sources result in activation of the reticulospinal and vestibulospinal pathways, which descend to the spinal cord where they contact interneurons and spinal motor neurons for postural control.

Afferent inputs from visual, vestibular, and somatosensory sources are integrated along the neuraxis, including the vestibular nuclei and right parietal cortex, to inform the internal model of body orientation and balance. This internal model is continually updated by the cerebellum based on error signals between expected and actual sensory feedback following motor commands.

### Spinal Cord Circuits Are Sufficient for Maintaining Antigravity Support but Not Balance

Adult cats with complete spinal transection at the thoracic level can, with experience, support the weight of their hindquarters with fairly normal hind limb and trunk postural orientation, but they have little control of balance. These animals do not exhibit normal postural responses in their hind limbs when the support surface moves. Their response to horizontal motion consists of small, random, and highly variable bursts of activity in extensor muscles, and postural activity in flexor muscles is completely absent. Active balance is absent despite the fact that extensors and flexors can

**Figure 36–13** Many parts of the nervous system control posture. Areas of frontal, parietal, temporal, and occipital cortex, as well as the basal ganglia, cerebellum, and pendunculopontine nucleus (PPN), provide inputs to the reticulospinal and vestibulospinal pathways descending to spinal motor neurons. Afferent inputs from the visual, vestibular, and somatosensory systems are integrated in the brainstem and cortex to update the body schema and inform future postural commands. (Abbreviations: M1, primary motor cortex; S1, primary somatosensory cortex; SMA, supplementary motor area.) (Adapted from Beristain 2016.)



be recruited for other movements such as stepping on a treadmill, suggesting that unlike locomotion, postural muscle activation requires supraspinal control.

An adult cat with a spinal transection can stand independently for only short periods of time and within a narrow range of stability; head turns in particular cause the animal to lose balance. What stability there is likely results from the broad base of support afforded by quadrupedal stance, the stiffness of the tonically contracting hind limb extensors that support the weight of the hindquarters, and active compensation by forelimbs that continue to produce postural responses. Humans with spinal cord injuries have various amounts of antigravity muscle tonus but lack automatic postural responses below the level of the lesion. These results emphasize that antigravity support and balance control are distinct mechanisms and that the control of balance requires the involvement of supraspinal circuits.

### The Brain Stem and Cerebellum Integrate Sensory Signals for Posture

If spinal circuits alone are not capable of producing automatic postural responses, what supraspinal centers are responsible for these responses? Although the

answer to this question remains unknown, good candidates include the brain stem and cerebellum, which are highly interconnected and work together to modulate the descending commands to spinal motor centers of the limbs and trunk. These regions have the input-output structure that would be expected of centers for postural control.

Muscle synergies for automatic postural responses may be organized in the brain stem, perhaps the reticular formation. However, adaptation of postural synergies to changes in the environment and task demands may require the cerebellum.

Two regions of the cerebellum influence orientation and balance: the vestibulocerebellum (nodulus, uvula, and fastigial nucleus) and the spinocerebellum (anterior lobe and interpositus nucleus). These regions are interconnected with the vestibular nuclei and reticular formation of the pons and medulla (see Figure 37–4). Lesions of the brain stem and vestibulocerebellum produce a variety of deficits in head and trunk control including a tendency to tilt from vertical, even with eyes open, suggesting a deficit in the internal representation of postural orientation. Lesions of the spinocerebellum result in excessive postural sway that is worse with the eyes closed, ataxia during walking, and hypermetric postural responses, suggesting

deficits in balance corrections. Certain regions in the pons and medulla facilitate or depress extensor tonus and could be involved in antigravity support.

The brain stem and cerebellum are sites of integration of sensory inputs, perhaps generating the internal model of body orientation and balance. Vestibular and visual inputs are distributed to brain stem centers (Chapters 25 and 27) and the vestibulocerebellum. The spinocerebellum receives signals from rapidly conducting proprioceptive and cutaneous fibers. More slowly conducting somatosensory fibers project to the vestibular nuclei and reticular formation.

Two major descending systems carry signals from the brain stem and cerebellum to the spinal cord and could trigger automatic postural responses for balance and orientation. The medial and lateral vestibulospinal tracts originate from the vestibular nuclei, and the medial and lateral reticulospinal tracts originate from the reticular formation of the pons and medulla (see Figure 37–5). Lesions of these tracts result in profound ataxia and postural instability. In contrast, lesions of the corticospinal and rubrospinal tracts have minimal effect on balance even though they produce profound disturbance of voluntary limb movements.

### **The Spinocerebellum and Basal Ganglia Are Important in Adaptation of Posture**

Patients with spinocerebellar disorders such as alcoholic anterior-lobe syndrome and basal ganglia deficits such as Parkinson disease experience postural difficulties. Studies suggest that the spinocerebellum and basal ganglia play complementary roles in adapting postural responses to changing conditions.

The spinocerebellum is where the amplitude of postural responses is adapted based on experience. The basal ganglia are important for quickly adjusting the postural set when conditions suddenly change. Both the spinocerebellum and the basal ganglia regulate muscle tone and force for voluntary postural adjustments. They are not necessary, however, for triggering or constructing the basic postural patterns.

Patients with disorders of the spinocerebellum have difficulty modifying the magnitude of balance adjustments with practice, over the course of repeated trials, but can readily adapt postural responses immediately after a change in conditions based on sensory feedback. For example, a patient standing on a movable platform scales the size of postural responses appropriately when platform velocity is increased with each trial. These postural adjustments rely on velocity information, which is encoded by somatosensory inputs at the beginning of platform movement.

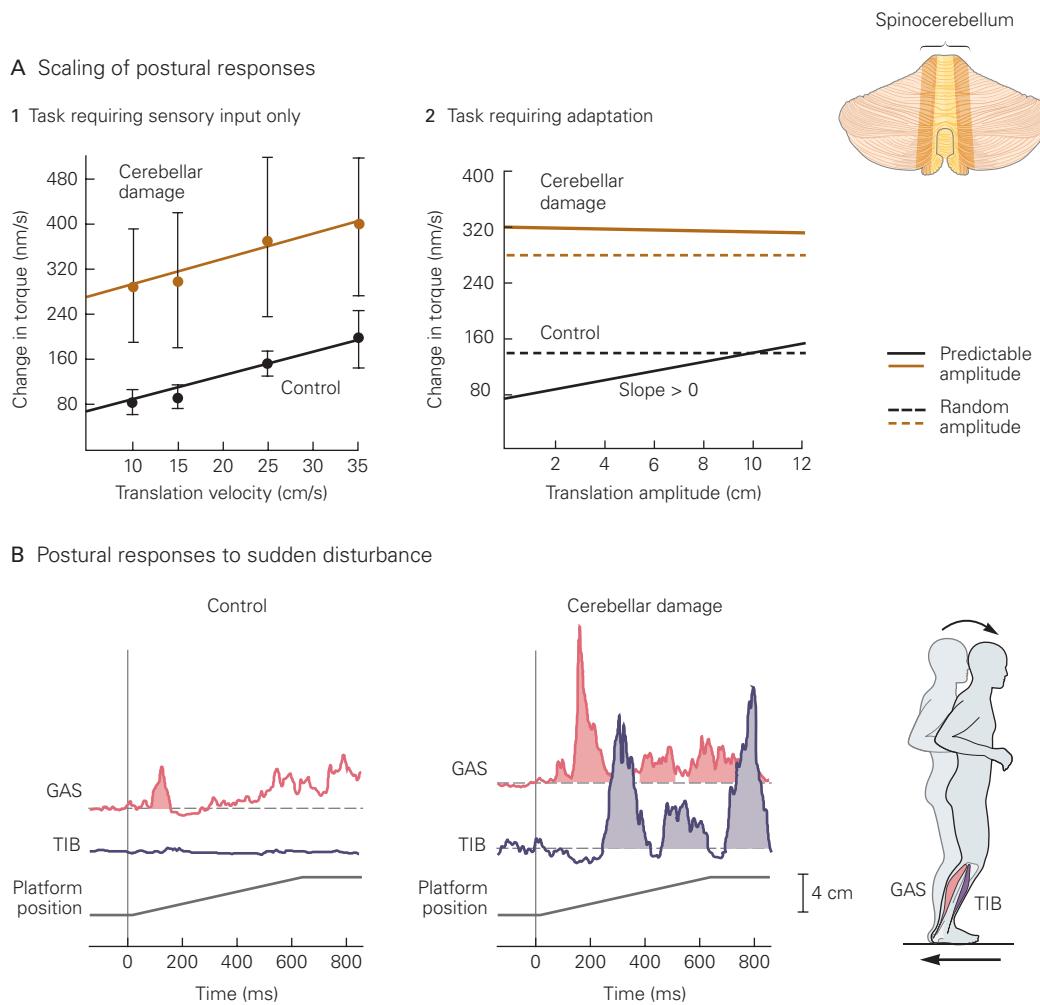
In contrast, patients with cerebellar disorders cannot scale the size of postural responses based on feedforward control using the anticipated amplitude of postural displacements. Because the amplitude of platform movement is not known until the platform has stopped moving, well after the initial postural response is complete, a subject cannot use feedback from the trial at hand to guide the response but must instead use his experience from previous trials to inform his response in a subsequent trial of the same amplitude. Whereas a healthy subject does this quite readily, a patient with spinocerebellar disorders is unable to efficiently adapt his postural responses based on recent experience (Figure 36–14A).

A healthy subject standing on a moveable platform is able to scale muscle activity during sudden backward motion of the platform to counteract the forward sway induced by the perturbation. A subject with spinocerebellar disease always overresponds, although the timing of muscle activation is normal (Figure 36–14B). As a result, this individual returns beyond the upright position and oscillates back and forth. Reminiscent of the hypermetria observed immediately after labyrinthectomy, cerebellar hypermetria may also result from loss of Purkinje cell inhibition on spinal motor centers.

A patient with Parkinson disease can, with sufficient practice, gradually modify his postural responses but has difficulty changing responses when conditions change suddenly. Such postural inflexibility is seen when initial posture changes. For example, when a normal subject switches from standing upright to sitting on a stool on a movable platform, the pattern of his automatic postural response to backward movement of the platform changes immediately. Because leg muscle activity is no longer necessary after the switch from standing to sitting, this component ceases to be recruited.

In contrast, a patient with Parkinson disease employs the same muscle activation pattern for both sitting and standing (Figure 36–15). L-DOPA replacement therapy does not improve the patient's ability to switch postural set. With repetition of trials in the seated posture, however, the leg muscle activity eventually disappears, showing that enough experience permits adaptation of postural responses. A patient with Parkinson disease also has difficulty when instructed to increase or decrease the magnitude of a postural response, a difficulty that is consistent with the inability to change cognitive sets quickly.

Patients with Parkinson disease have problems with postural tone and force generation in addition to

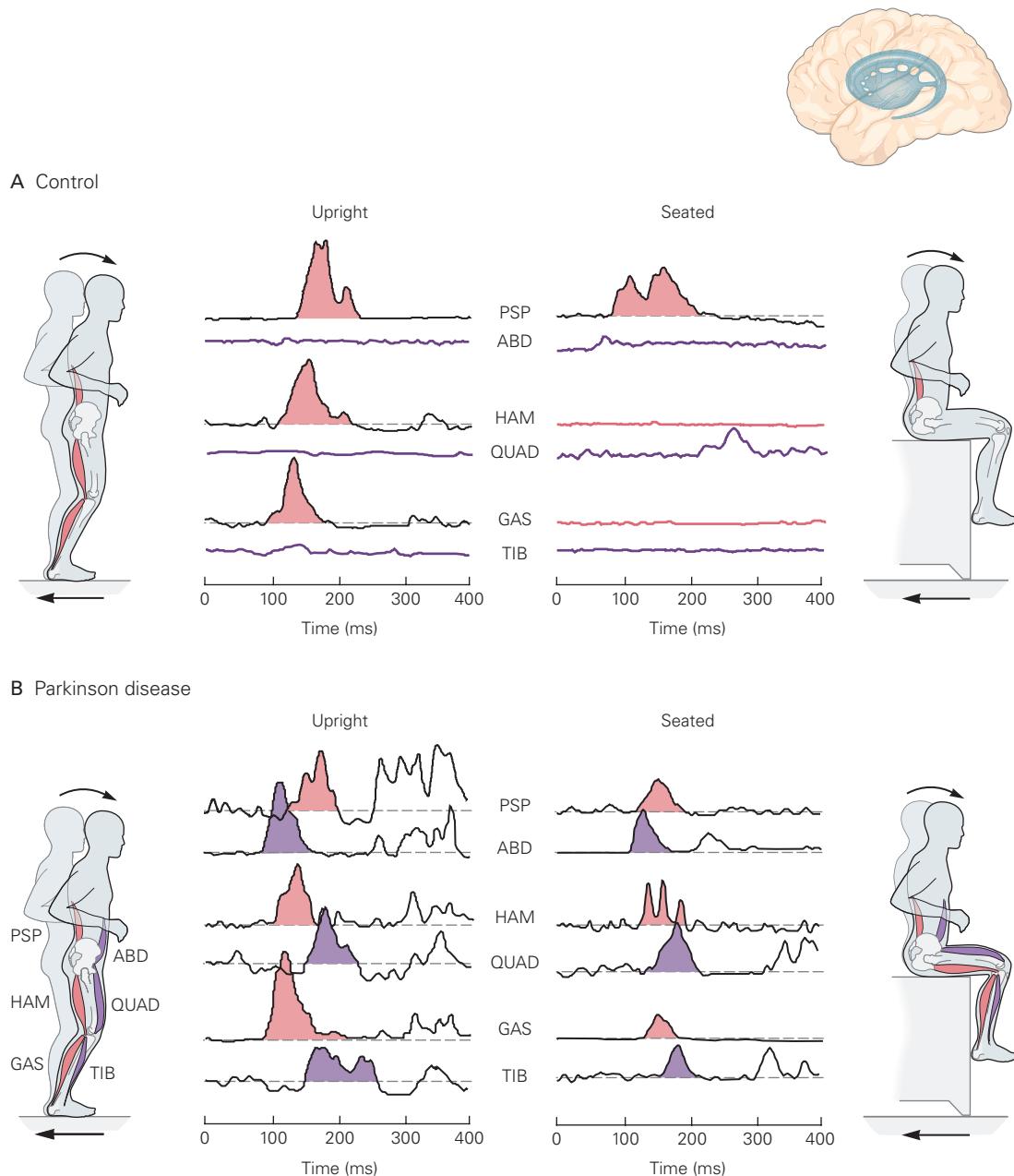


**Figure 36–14** The spinocerebellum has a role in adapting postural responses to changing conditions and in scaling postural responses to anticipated postural disturbances. The spinocerebellum is important for adapting postural responses based on experience. Patients with a spinocerebellar disorder are able to use immediate sensory input, but not experience, to adjust automatic postural responses. (Adapted, with permission, from Horak and Diener 1994.)

**A. 1.** In this experiment, subjects stand on a platform that is moved horizontally; the velocity is increased on each trial. Maintaining balance requires scaling responses to the velocity of the platform using sensory feedback. The adjustments in a subject with a spinocerebellar disorder have the same regression coefficient (slope) as those of a control subject, even though in each trial the responses are larger and more variable than those of the control subject. **2.** When subjects are required to anticipate and adapt to platform translation, the postural

adjustments in the spinocerebellar subject are compromised. When translation amplitude is random, responses are large, as if the subject expects a large translation. When trials with the same amplitude are repeated, a control subject learns to predict the amplitude of the disturbance and adjust his response. In contrast, a spinocerebellar subject shows no improvement in performance; he cannot use his experience in one trial to adjust his responses in subsequent trials. All responses are large, as if the subject always expects the large translation.

**B.** In this experiment, subjects stand on a platform that is moved backward (6 cm amplitude at 10 cm/s). In a control subject, the onset of movement evokes a small burst of activity in the gastrocnemius (GAS), an ankle extensor. In a subject with damage to the anterior lobe of the cerebellum, the muscle responses are overly large, with bursts of activity alternating between the gastrocnemius and its antagonist, the tibialis anterior (TIB).



**Figure 36-15** The basal ganglia are important for adapting postural responses to a sudden change in initial conditions. (Adapted, with permission, from Horak, Nutt, and Nashner 1992.)

**A.** When a normal subject switches from upright stance to sitting, he immediately modifies his response to backward movement of the support platform. The postural response to movement while seated does not involve the leg muscles—the gastrocnemius (**GAS**) and hamstrings (**HAM**)—but does

activate the paraspinal muscles (**PSP**) and with shorter latency than in the response to movement while standing. (Abbreviations: **ABD**, abdominals; **QUAD**, quadriceps; **TIB**, tibialis anterior.)

**B.** A patient with Parkinson disease does not suppress the leg muscle response in the first trial after switching from standing to sitting. The postural response of this subject is similar for both initial positions: Antagonist muscles (**purple**) are activated along with agonists (**pink**).

an inability to adapt to changing conditions. The disease's bradykinesia (slowness of movement) is reflected in slow development of force in postural responses, and its rigidity is manifested in co-contraction. L-DOPA replacement greatly improves a patient's ability to generate not only forceful voluntary movements but also the accompanying postural adjustments, such as rising onto the toes and gait. However, neither the automatic postural response to an unexpected disturbance nor postural adaptation is improved by L-DOPA, suggesting that these functions involve the nondopaminergic pathways affected by Parkinson disease.

### Cerebral Cortex Centers Contribute to Postural Control

Several areas of cerebral cortex influence postural orientation and equilibrium, including both anticipatory and automatic postural responses. Most voluntary movements, which are initiated in the cerebral cortex, require postural adjustments that must be integrated with the primary goal of the movement in both timing and amplitude. Where this integration occurs is not clear.

The cerebral cortex is more involved in anticipatory postural adjustments than in automatic postural reactions. However, recent electroencephalographic studies show that areas of cerebral cortex are activated by anticipation of a postural disturbance before an automatic postural response is initiated. This finding is consistent with the idea that the cortex optimizes balance control as part of motor planning.

The supplementary motor area and temporoparietal cortex have both been implicated in postural control. The supplementary motor area, anterior to the motor cortex, is likely involved with anticipatory postural adjustments that accompany voluntary movements. The temporoparietal cortex appears to integrate sensory information and may comprise internal models for perception of body verticality. Lesions of insular cortex can impair perception of the visual vertical, whereas lesions of superior parietal cortex impair perception of the postural vertical, and either of these defects may impair balance when standing on an unstable support.

Sensorimotor cortex receives somatosensory inputs signaling balance disturbances and postural responses. However, this region is not essential for automatic postural adjustments. Lesioning the motor cortex in cats impairs lifting of the forelimb in response to a light touch during stance but does not abolish the accompanying postural adjustment in the contralateral forelimb. Although the sensorimotor cortex is not

responsible for postural adjustments, it may have a role in the process.

Behavioral studies, too, have implicated cortical processes in postural control. Control of posture, like control of voluntary movement, requires attention. When subjects must press a button following a visual or auditory cue while also maintaining balance, their reaction time increases with the difficulty of the task (balancing on one foot versus sitting, for example). Moreover, when subjects try to perform a cognitive task while actively maintaining posture, the performance of either or both can degrade. For example, when a subject is asked to count backward by threes while standing on one foot, both the cognitive task and postural adjustment deteriorate. The timing of automatic postural responses to unexpected disturbances is little affected by cognitive interference.

Balance control is also influenced by emotional state, thus implicating the limbic system in posture control. Fear of falling, for example, can increase postural tone and stiffness, reduce sway area, increase sway velocity, and alter balancing strategies in response to disturbances.

Finally, balance control is also influenced by attentional ability and demands, thus implicating the frontoparietal attention network. There is evidence of competition for central processing resources in dual task conditions, where a person must maintain balance and perform a concurrent cognitive task. Both postural control and cognitive performance may be impaired in dual-task conditions as compared to single-task conditions assessing either postural or cognitive performance in isolation. As cognitive demands increase, responses to postural perturbations are smaller in amplitude and occur at longer latency. However, when necessary, healthy individuals prioritize postural control over the cognitive task and demonstrate decreasing cognitive performance as postural demands increase. In contrast, individuals with nervous system disorders such as Parkinson disease may not prioritize postural control in dual-task situations and may be at increased risk for falls in dual-task situations.

Although the roles of specific areas of cerebral cortex in postural control are largely undefined, there is no doubt that the cortex is important for learning new, complex postural strategies. The cortex must be involved in the amazing improvement in balance and postural orientation of athletes and dancers who use cognitive information and advice from coaches. In fact, the cerebral cortex is involved in postural control each time we consciously maintain our balance while walking across a slippery floor, standing on a moving bus, or waiting tables on a rocking ship.

## Highlights

1. The two goals of posture are balance and orientation. Balance control maintains the body in stable equilibrium to avoid falls. Postural orientation aligns the body segments with respect to each other and to the world, such as maintaining the head vertical.
2. A sudden displacement of the body center of mass while standing triggers ankle, hip, and/or stepping strategies to return the center of mass within the base of foot support.
3. Postural responses are fast and automatic, but adapt quickly to changes in environmental context, intention, and conditions. Postural responses can also be improved with practice.
4. Activation of centrally organized muscle synergies is used to control balance. This synergy organization simplifies the neural control so only a few central commands are required, instead of a separate command for each muscle, while allowing flexibility and adaptability for postural control.
5. Somatosensory, vestibular, and visual sensory modalities are integrated to form an internal representation of the body that the nervous system uses for postural orientation and balance control. Somatosensory signals trigger the fastest, largest postural responses and are most critical for control of postural sway in standing. Vestibular signals are particularly critical when standing on an unstable surface, when it is difficult to use somatosensory information for postural orientation. Visual inputs provide spatial orientation and motion information.
6. The body center of mass is often outside of the base of foot support during walking and running so balance is provided by adjusting foot placement and lateral trunk stability to control the center of mass with respect to the changing base of support.
7. The vestibulocerebellum and the spinocerebellum are interconnected with the vestibular nuclei and reticular formation of the brainstem for control of balance and postural orientation.
8. The basal ganglia are important for control of axial postural tone, adapting postural response strategies based on initial conditions, and anticipatory postural control. The cerebellum is important for adapting the magnitude of balance responses with practice, over the course of repeated trials, and for scaling the size of postural responses.
9. Posture control involves many brain areas from the brainstem to the frontal cortex, but the specific circuits involved in different types of posture

control (automatic postural responses, anticipatory postural adjustments, body sway in stance, sensory integration for a body schema and verticality) have yet to be determined.

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# The Cerebellum

## **Damage of the Cerebellum Causes Distinctive Symptoms and Signs**

Damage Results in Characteristic Abnormalities of Movement and Posture

Damage Affects Specific Sensory and Cognitive Abilities

## **The Cerebellum Indirectly Controls Movement Through Other Brain Structures**

The Cerebellum Is a Large Subcortical Brain Structure

The Cerebellum Connects With the Cerebral Cortex Through Recurrent Loops

Different Movements Are Controlled by Functional Longitudinal Zones

## **The Cerebellar Cortex Comprises Repeating Functional Units Having the Same Basic Microcircuit**

The Cerebellar Cortex Is Organized Into Three Functionally Specialized Layers

The Climbing-Fiber and Mossy-Fiber Afferent Systems Encode and Process Information Differently

The Cerebellar Microcircuit Architecture Suggests a Canonical Computation

## **The Cerebellum Is Hypothesized to Perform Several General Computational Functions**

The Cerebellum Contributes to Feedforward Sensorimotor Control

The Cerebellum incorporates an Internal Model of the Motor Apparatus

The Cerebellum Integrates Sensory Inputs and Corollary Discharge

The Cerebellum Contributes to Timing Control

## **The Cerebellum Participates in Motor Skill Learning**

Climbing-Fiber Activity Changes the Synaptic Efficacy of Parallel Fibers

The Cerebellum Is Necessary for Motor Learning in Several Different Movement Systems

Learning Occurs at Several Sites in the Cerebellum

## **Highlights**

**T**HE CEREBELLUM CONSTITUTES ONLY 10% of the total volume of the brain but contains more than one-half of its neurons. The cerebellar cortex comprises a series of highly regular, repeating units, each of which contains the same basic microcircuit. Different regions of the cerebellum receive projections from distinct brain and spinal structures and then project back to the brain. The similarity of the architecture and physiology in all regions of the cerebellum implies that different regions of the cerebellum perform similar computational operations on different inputs.

The symptoms of cerebellar damage in humans and experimental animals provide compelling evidence that the cerebellum participates in the control of movement. The symptoms, in addition to being diagnostic for clinicians, thus help define the possible roles of the cerebellum in controlling behavior.

Several fundamental principles define our understanding of the physiological function of the cerebellum. First, the cerebellum acts in advance of sensory feedback arising from movement, thus providing feed-forward control of muscular contractions. Second, to achieve such control, the cerebellum relies on internal models of the body to process and compare sensory inputs with copies of motor commands. Third, the cerebellum plays a special role in motor and perceptual timing. Fourth, the cerebellum is critical for adapting