8 Cerebellum

External Appearance

See **Fig. 8.1**.

The cerebellum is composed of two cerebellar hemispheres joined by a median vermis. The *superior vermis* is confluent with the hemispheres, whereas the *inferior vermis* is a well-delineated structure that is located in a deep depression in the midline (the vallecula).
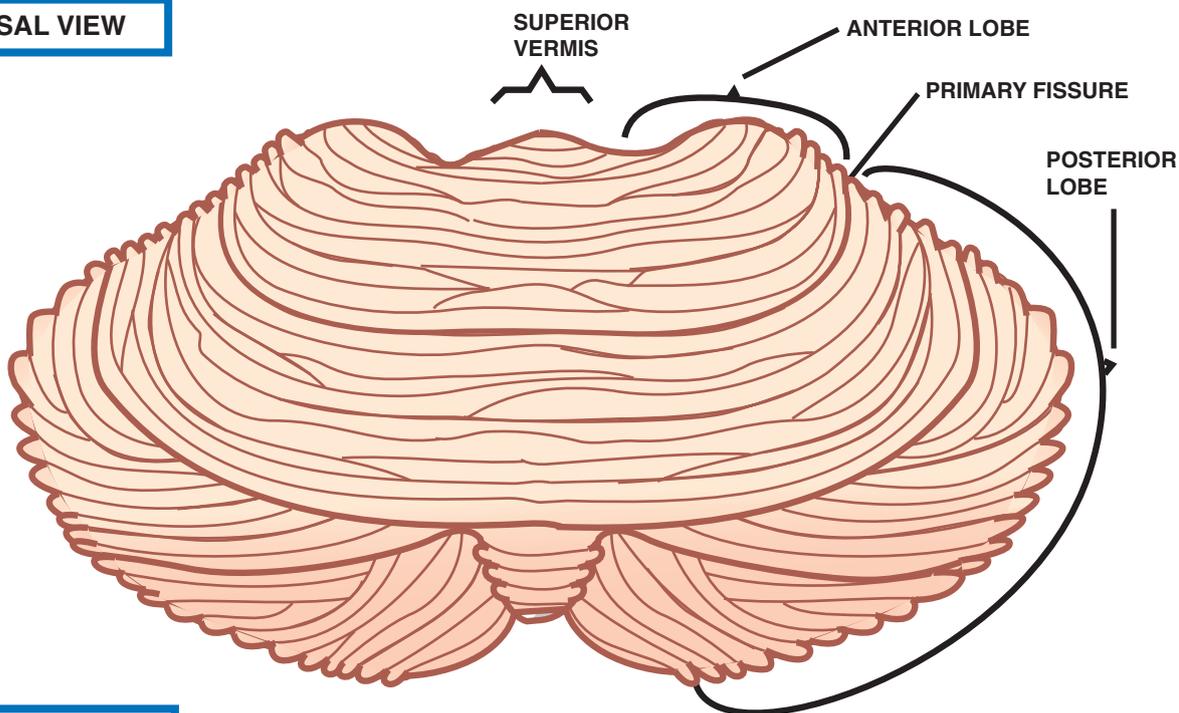
The cerebellum is divided into three lobes: the anterior lobe, the posterior lobe, and the flocculonodular lobe. The *anterior lobe* (paleocerebellum) constitutes the rostral portion of the rostral cerebellar surface; the *flocculonodular lobe* (archicerebellum) constitutes the rostral portion of the caudal cerebellar surface; and the *posterior lobe* (neocerebellum) makes up the remainder of the cerebellum on both surfaces.

Two major fissures of the cerebellum are identified: the *primary fissure* separates the anterior lobe from the posterior lobe on the superior surface, and the *dorsolateral fissure* separates the posterior lobe from the flocculonodular lobe on the inferior surface.
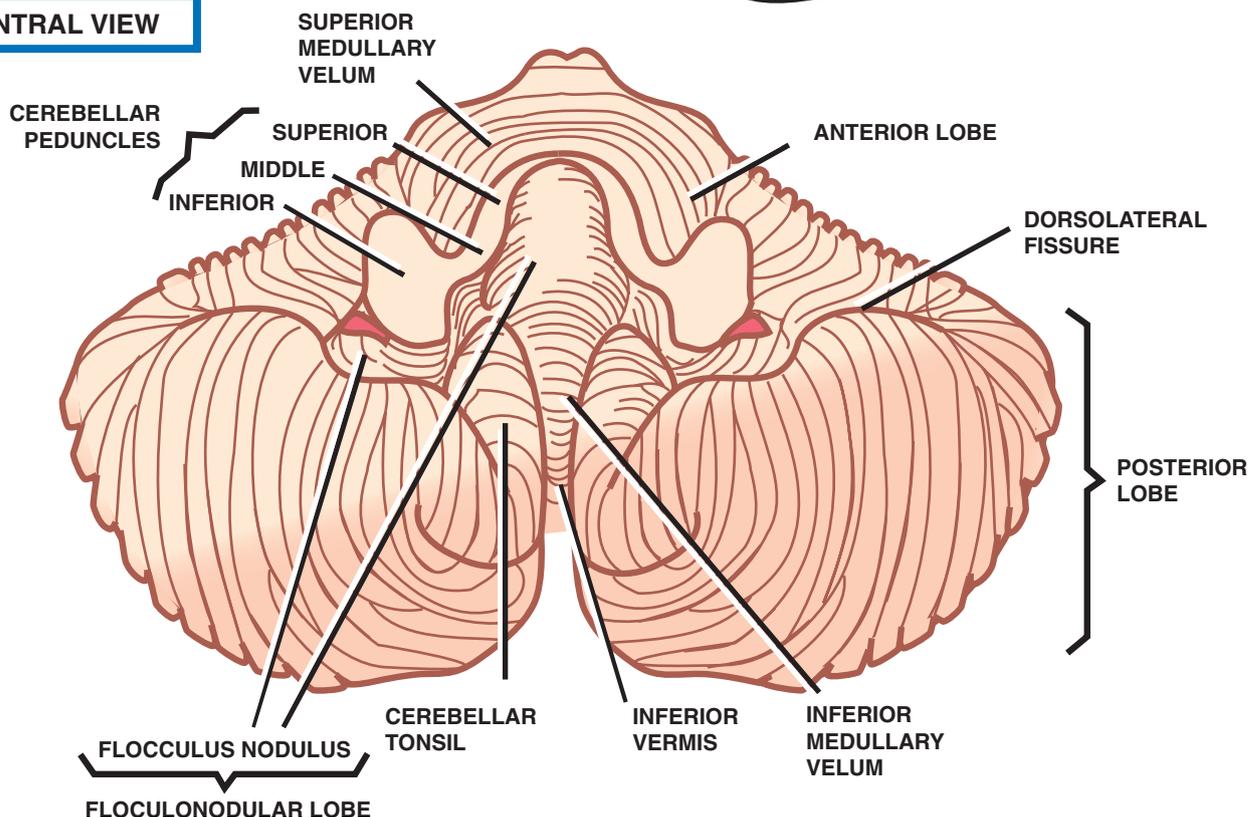
The rostral portion of the roof of the fourth ventricle is formed by the *superior cerebellar peduncles*. The caudal aspect of the roof of the fourth ventricle is formed by the *inferior medullary velum*, which frequently adheres to the inferior vermis. The foramen of Magendie is a median aperture, which constitutes an opening in the inferior medullary velum that connects the fourth ventricle with the cisterna magna. The three cerebellar peduncles are attached to the cerebellum in the interval between the anterior and the flocculonodular lobes.

Fig. 8.1 **External appearance of cerebellum.**

DORSAL VIEW

SUPERIOR
VERMIS

ANTERIOR LOBE

PRIMARY FISSURE

POSTERIOR
LOBE

VENTRAL VIEW

SUPERIOR
MEDULLARY
VELUM

CEREBELLAR
PEDUNCLES

SUPERIOR

MIDDLE

INFERIOR

ANTERIOR LOBE

DORSOLATERAL
FISSURE

POSTERIOR
LOBE

FLOCCULUS NODULUS

FLOCULONODULAR LOBE

CEREBELLAR
TONSIL

INFERIOR
VERMIS

INFERIOR
MEDULLARY
VELUM

Cerebellar Cortex

## Cortical Layers

See **Fig. 8.2**.

The cerebellar cortex is divided into three layers: a superficial *molecular layer*, a middle *Purkinje layer*, and a deep *granular layer*. These layers are collectively composed of five cell types: *stellate, basket, Purkinje, Golgi*, and *granule cells.*

The molecular layer is so-called because of its punctuated, sparsely populated appearance. It is largely a synaptic layer that contains dendrites of Purkinje cells and axons of granule cells. Scattered stellate and basket cells are also present. The Purkinje cell layer consists of a single row of Purkinje cell bodies; the granule cell layer consists of densely packed neurons that send axonal pro-jections into the molecular layer. The granule cell layer is composed of granule and Golgi cells.

An embryonic cerebellar layer, the external granule cell layer, is present during the prenatal and early post-natal periods but is completely gone by the first year of life. This layer is superficial to the molecular layer, and its cells are thought to be the cell of origin of medullo-blastomas.

Fig. 8.2 **Layers of the cerebellum.**
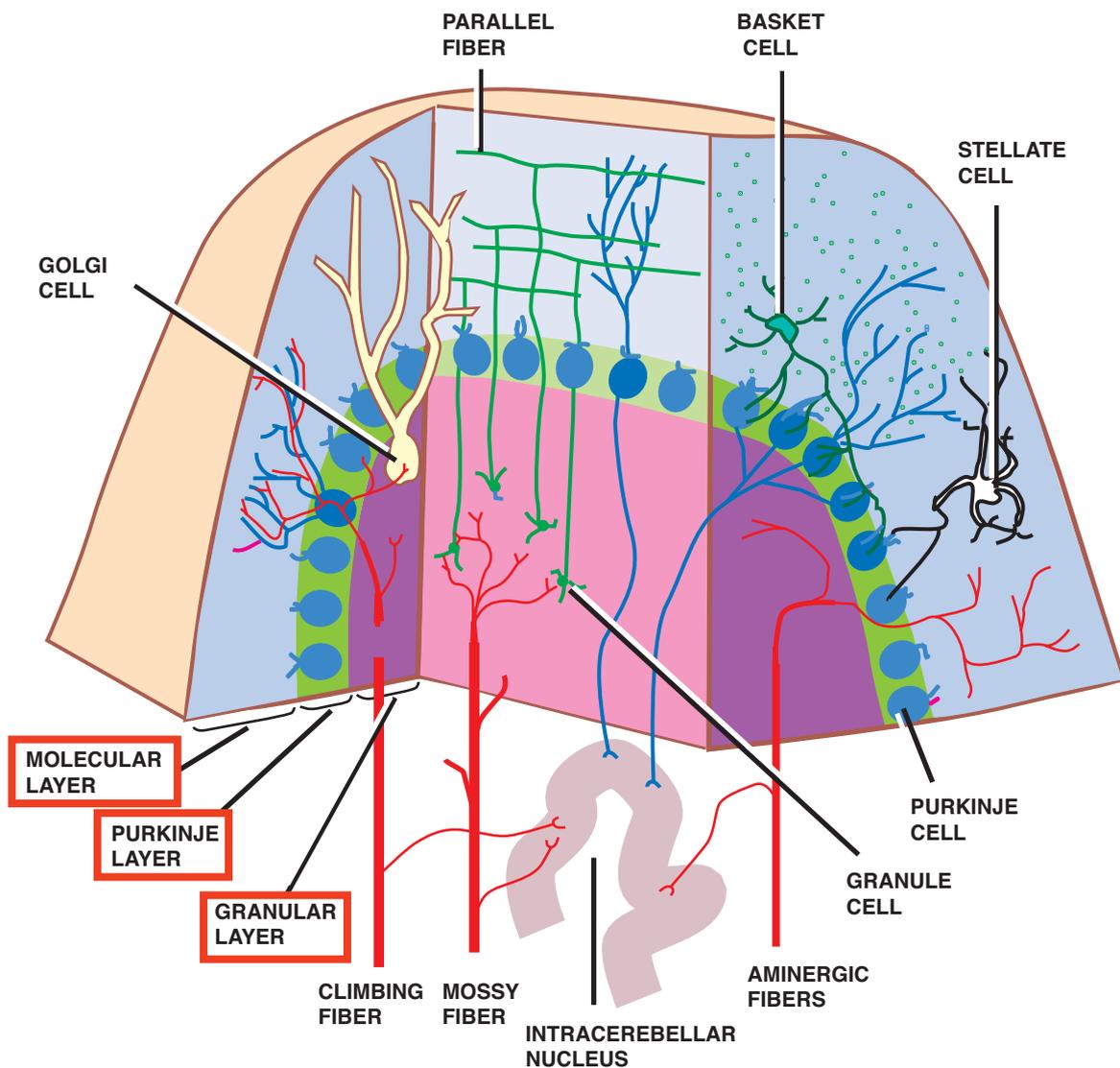
Intrinsic Circuitry

See **Fig. 8.3**.

The intrinsic circuitry of the cerebellar cortex is primarily composed of two interrelated circuits, which receive input from two afferent nerve fibers, the climbing fibers and the mossy fibers. The input from these fiber systems is received by the Purkinje cells (directly and indirectly), which in turn project to the neurons in the deep cerebellar nuclei.

Climbing fibers, most of which originate in the inferior olive, make direct contacts with the dendrites of a limited number of Purkinje cells. By contrast, *mossy fibers*, which are derived from a variety of sources, indirectly influence a large number of Purkinje cells. To accomplish this, the mossy fibers branch extensively in the granule cell layer, where they establish synaptic contacts with the granule cells. Rosettes are the sites of synapses between mossy fibers and the clawlike terminals of granule cell dendrites. Mossy fiber rosettes also establish synapses with Golgi type II cell bodies. The granule cells project superficially to the molecular layer, where they bifurcate in a **T**-shaped manner to form the so-called parallel fibers. Each parallel fiber establishes contacts with a large number of Purkinje cell dendrites.

Thus, the climbing fibers exert a powerful influence on a few specific Purkinje cells, whereas the mossy fibers modulate a large number of Purkinje cells through a relay in the granular layer. In other words, a single climbing fiber may stimulate a Purkinje cell in an all-or-none phenomenon, whereas many mossy fiber discharges are required to stimulate a Purkinje cell.

A third category of afferent axons, the aminergic fibers, are distinct both from climbing and mossy fibers in that they contain biogenic amines. These fibers, which show a widespread distribution in the cerebellar cortex, are divided into two different types: serotonin-containing axons that originate in the raphe nuclei of the brainstem, and norepinephrine-containing axons that originate in the locus ceruleus.

These circuits involving climbing fibers, mossy fibers, and aminergic fibers are modified by intracortical circuits formed by three types of interneurons that appear to function as modulators of Purkinje cell activity. These are the *Golgi, basket*, and *stellate cells*. Like the Purkinje cells, these interneurons are inhibitory in nature, and they contain the neurotransmitter gamma-aminobutyric acid (GABA). Thus, four out of the five cells in the cerebellar cortex are GABA-containing inhibitory neurons. Only the granule cells and the afferent fiber system are excitatory.

The overall circuit in the cerebellar cortex may be summarized as follows. Excitatory input to the cerebellar cortex is primarily derived from the mossy fibers and the climbing fibers. This excitatory input is received by the Purkinje cells (directly and indirectly), which are responsible, in turn, for the entire (inhibitory) output of the cerebellar cortex. The excitatory input to the Purkinje cells is further modified by the inhibitory influences of the modulating interneurons. The Purkinje cells project into the deep cerebellar nuclei.

Fig. 8.3 **Intrinsic circuitry.**
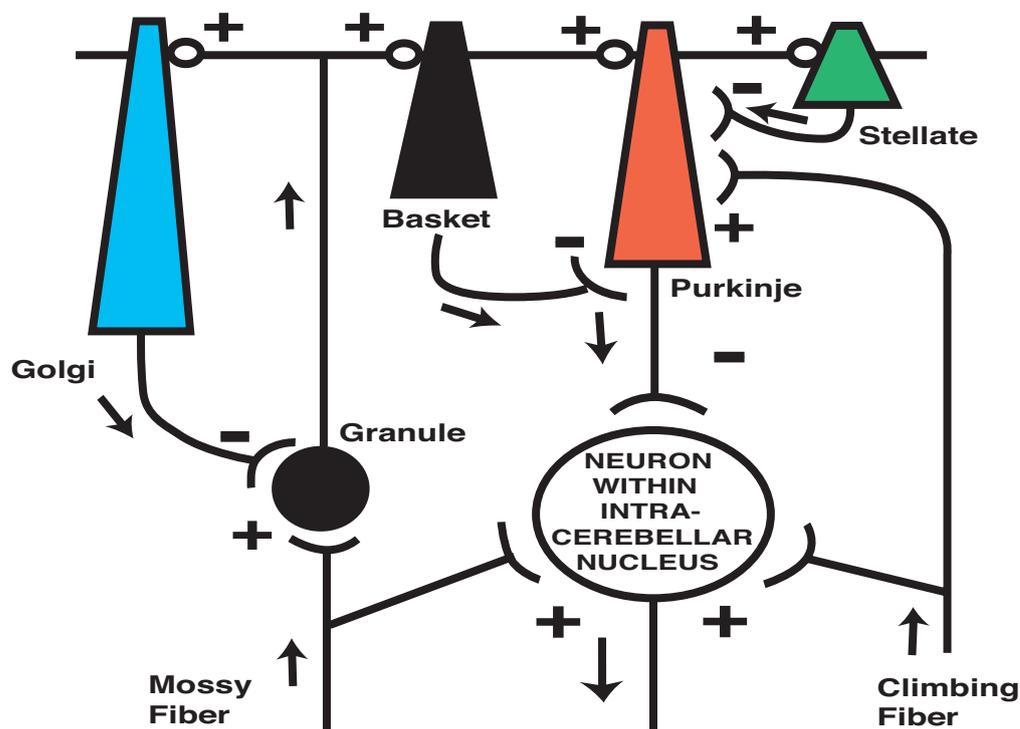
Deep Cerebellar Nuclei

See **Fig. 8.4**.

Four pairs of nuclei are located in the white matter of the cerebellum. From medial to lateral on each side of the midline, they are the *fastigial, globose, emboliform*, and *dentate nuclei*. The globose and the emboliform nuclei are collectively known as the interposed nuclei because they are interposed between the fastigial and dentate nuclei.

The input to the cerebellar nuclei is derived from two sources: (1) excitatory input is derived from fibers that originate in cells that lie outside the cerebellum, and (2) inhibitory input is derived from fibers that arise from the Purkinje cells of the cortex. Cells outside the cerebellum that send afferents directly to the cerebellar nuclei include pontocerebellar, spinocerebellar, and olivocerebellar fibers, most of which give collaterals to the cerebellar nuclei, then continue on to the cerebellar cortex.

The cells that constitute the intracerebellar nuclei act to modify muscular activity through the motor control areas of the brainstem and cerebral cortex. Efferents from the fastigial nucleus project to the brainstem through the inferior cerebellar peduncle, whereas efferents from the other nuclei are projected to the brainstem and cerebral cortex (with relays in the thalamus) through the superior cerebellar peduncle.

Fig. 8.4 Deep cerebellar nuclei.

FASTIGIAL NUCLEUS

GLOBOSE NUCLEUS

EMBOLIFORM NUCLEUS

DENTATE NUCLEUS
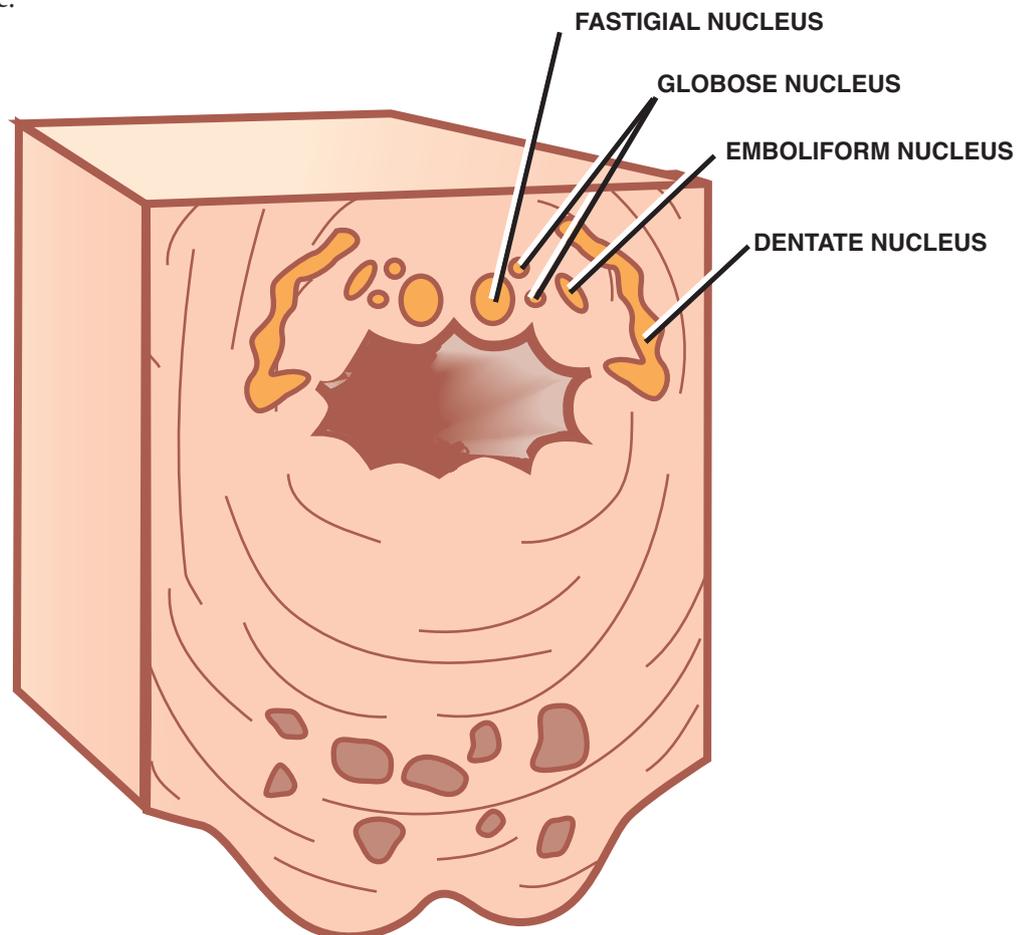
Cerebellar Connections

Afferent Connections

See **Fig. 8.5**.

Cerebellar afferents are derived from three main sources: the cerebral cortex, the spinal cord, and the vestibular nerve. A small number of afferents originate in the red nucleus and the tectum.

Fig. 8.5 Afferent cerebellar connections.
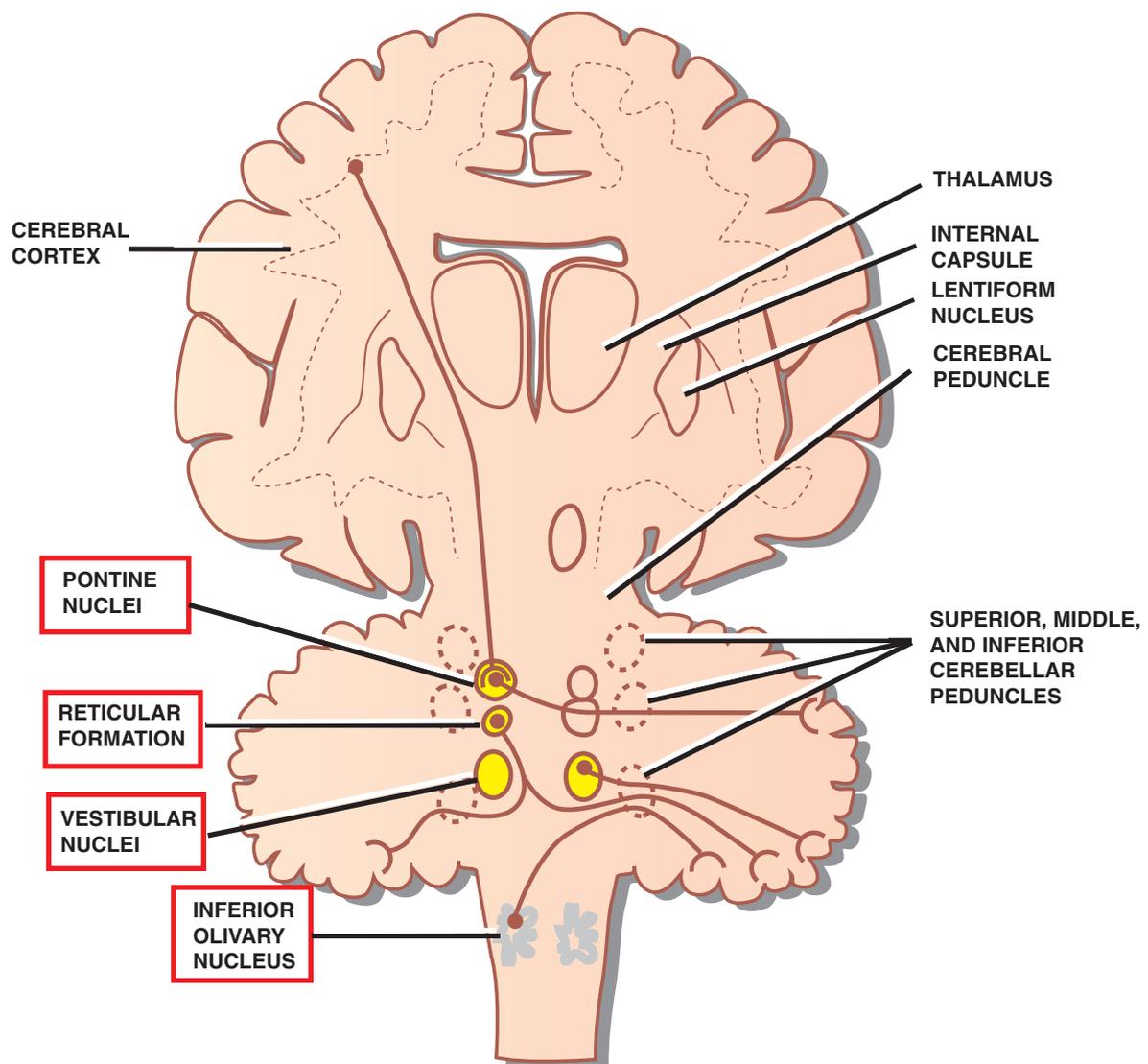
Cerebral Cortex

See **Fig. 8.6**.

Before reaching the cerebellum, cortical projections establish synaptic contacts with three brainstem structures: (1) the *pontine nuclei*, (2) the *inferior olivary nucleus*, and (3) the *reticular formation*.

The *corticopontocerebellar pathway* originates from a large area of the cerebral cortex, descends through the corona radiata and the internal capsule, and terminates in the pontine nuclei. The cells of the pontine nuclei give rise to mossy fibers that cross the midline to reach the opposite cerebellar hemisphere via the middle cerebellar peduncle.

The *cortico-olivocerebellar pathway* also originates from a large area of the cerebral cortex, descends through the corona radiata and the internal capsule, and terminates bilaterally in the inferior olivary nuclei. The cells of the inferior olivary nuclei give rise to climbing fibers that cross the midline to enter the opposite cerebellar hemisphere via the inferior cerebellar peduncle.

The *corticoreticulocerebellar pathway* originates from a large area of the cerebral cortex, descends through the corona radiata and the internal capsule, and terminates bilaterally in the reticular formation of the pons and medulla. The cells of the reticular formation give rise to mossy fibers that enter the ipsilateral cerebellar hemisphere via the inferior and the middle cerebellar peduncles.
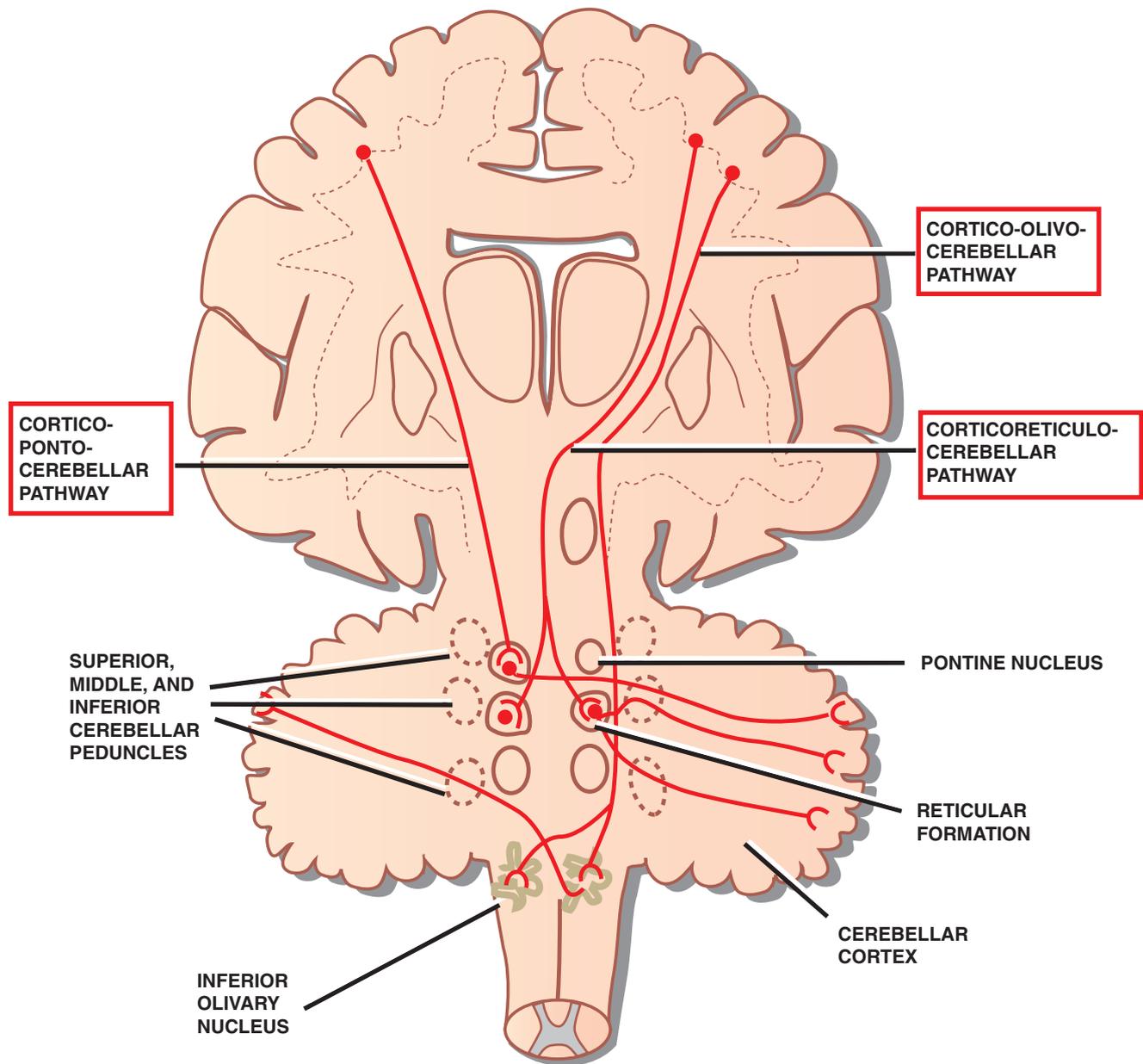
Spinal Cord

See **Fig. 8.7**, p. 288.

Spinal cord projections to the cerebellum are carried in three spinocerebellar pathways: (1) the *ventral spinocerebellar tract*, (2) the *dorsal spinocerebellar tract*, and (3) the *cuneocerebellar tract.*

- The ventral spinocerebellar tract originates in the ventral and intermediate gray matter of the spinal cord. Most of its fibers cross the midline to enter the ventral spinocerebellar tract on the opposite side, although a small number of fibers are uncrossed. The tract ascends bilaterally in the dorsolateral region of the lateral funiculus. After ascending the spinal cord, the ventral spinocerebellar tract enters the cerebellum via the superior cerebellar peduncle, crosses the midline for a second time, and terminates as mossy fibers in the cerebellar cortex. Functionally, this tract carries sensory information (mainly proprioceptive) from one side of the body (lower limbs) to the same side of the cerebellum.
- The dorsal spinocerebellar tract originates in the nucleus dorsalis (Clark's column). Most of its fibers are uncrossed. The tract ascends bilaterally in the ventrolateral region of the lateral funiculus. After ascending the spinal cord, the dorsal spinocerebellar tract enters the cerebellum via the inferior cerebellar peduncle and terminates as mossy fibers in the intermediate zone of the cerebellar cortex. Functionally, this tract carries sensory information (mainly proprioceptive) from one side of the body (trunk and lower limbs) to the cerebellum ipsilaterally.
- The cuneocerebellar tract originates in the accessory cuneate nucleus of the medulla. It is the upper extremity equivalent of the dorsal spinocerebellar tract. It enters the cerebellar hemisphere on the ipsilateral side through the inferior cerebellar peduncle. Functionally, this tract transmits sensory (mainly proprioceptive) information from the upper limb and upper part of the thorax.

Fig. 8.6 **Afferent cerebellar connections from cortex.**

CORTICO-OLIVO-CEREBELLAR PATHWAY

CORTICO-PONTO-CEREBELLAR PATHWAY

CORTICORETICULO-CEREBELLAR PATHWAY

SUPERIOR, MIDDLE, AND INFERIOR CEREBELLAR PEDUNCLES

PONTINE NUCLEUS

RETICULAR FORMATION

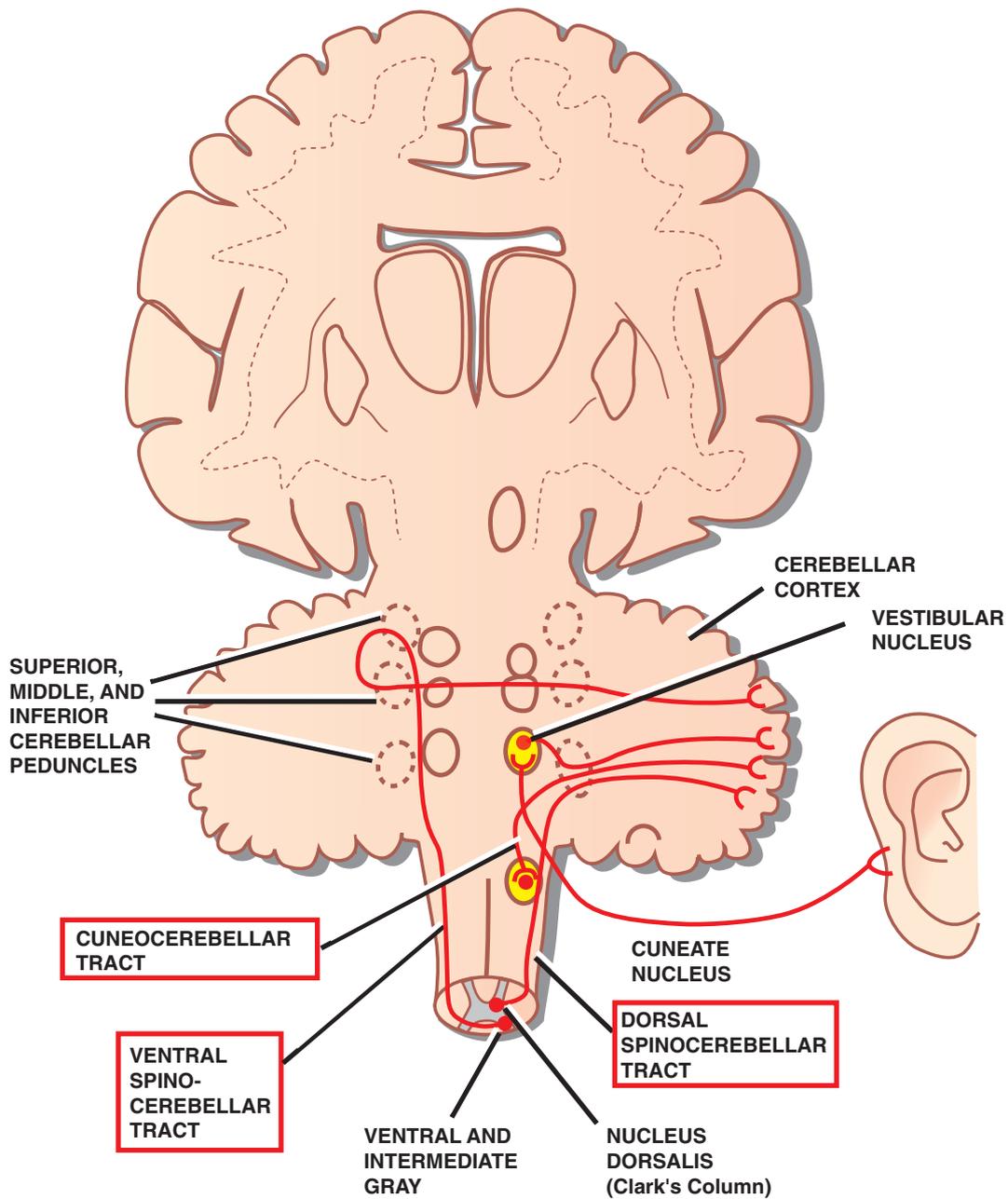CEREBELLAR CORTEX

INFERIOR OLIVARY NUCLEUS

Vestibular Nerve

See **Fig. 8.7**.

The vestibular nerve gives rise to afferent fibers that terminate in the *vestibular nuclei* of the brainstem. The neurons of the vestibular nuclei in turn give rise to mossy fibers that pass through the inferior cerebellar peduncle to enter the ipsilateral flocculonodular lobe on the same side.

Fig. 8.7 **Afferent cerebellar connections from brainstem and spinal cord.**

Efferent Connections

The entire output of the cerebellar cortex is transmitted by the inhibitory Purkinje cells, most of which terminate on the deep cerebellar nuclei (a few Purkinje cell axons continue past the cerebellar nuclei to synapse on the lateral vestibular nucleus in the medulla). The cells of the cerebellar nuclei constitute the entire efferent outflow system of the cerebellum. These cells leave the cerebellum through the superior and inferior cerebellar peduncles to terminate in the following four destinations: (1) the red nucleus, (2) the thalamus, (3) the vestibular complex, and (4) the reticular formation.
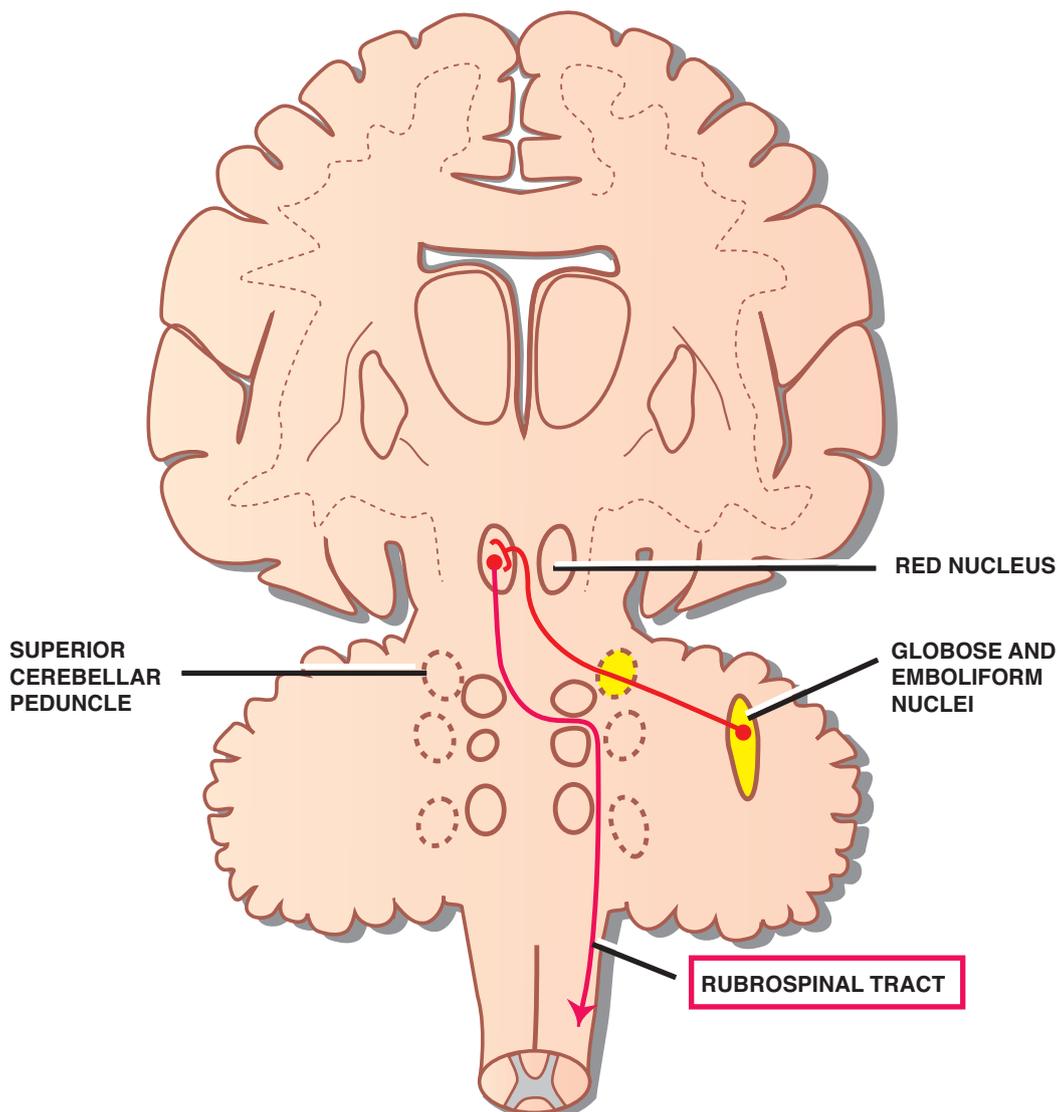
The superior cerebellar peduncle transmits those fibers that ascend to the red nucleus and the thalamus. These constitute the majority of the fibers. The inferior cerebellar peduncle transmits those fibers that descend to the vestibular and the reticular formation.

Red Nucleus

See **Fig. 8.8**.

The axons of neurons in the *globose* and *emboliform nuclei* pass out of the cerebellum through the *superior cerebellar peduncle* and cross the midline. These axons ascend and synapse in the contralateral *red nucleus*, which in turn projects fibers in the crossed *rubrospinal tract*. Thus, projections from the globose and emboliform nuclei, which cross twice before reaching their final destination, influence motor body activity ipsilaterally. The rubrospinal tract influences flexor activity of the extremities. Therefore, the globose and emboliform nuclei are involved with tone.

*Fig. 8.8* **Efferent cerebellar connections to red nucleus.**

Thalamus

See **Fig. 8.9**.

The axons of neurons in the *dentate nucleus* (and some from the globose and emboliform nuclei) exit the cerebellum through the *superior cerebellar peduncle* and cross the midline in the same decussation. These axons ascend to synapse in the contralateral ventrolateral, ventroposterolateral, and centrolateral nuclei of the thalamus, which in turn project axons through the internal capsule and the corona radiata to terminate in the primary motor cortex. The dentate nucleus thus influences the motor neurons of the cerebral cortex on the contralateral side. The motor cortex, however, projects descending fibers in the corticospinal tract, which cross the midline in the decussation of the pyramids. Thus, the neurons in the dentate nucleus influence motor activity on the same side of the body. Therefore, the dentate mainly influences coordination of the ipsilateral body.

Fig. 8.9 **Efferent cerebellar connections to thalamus.**
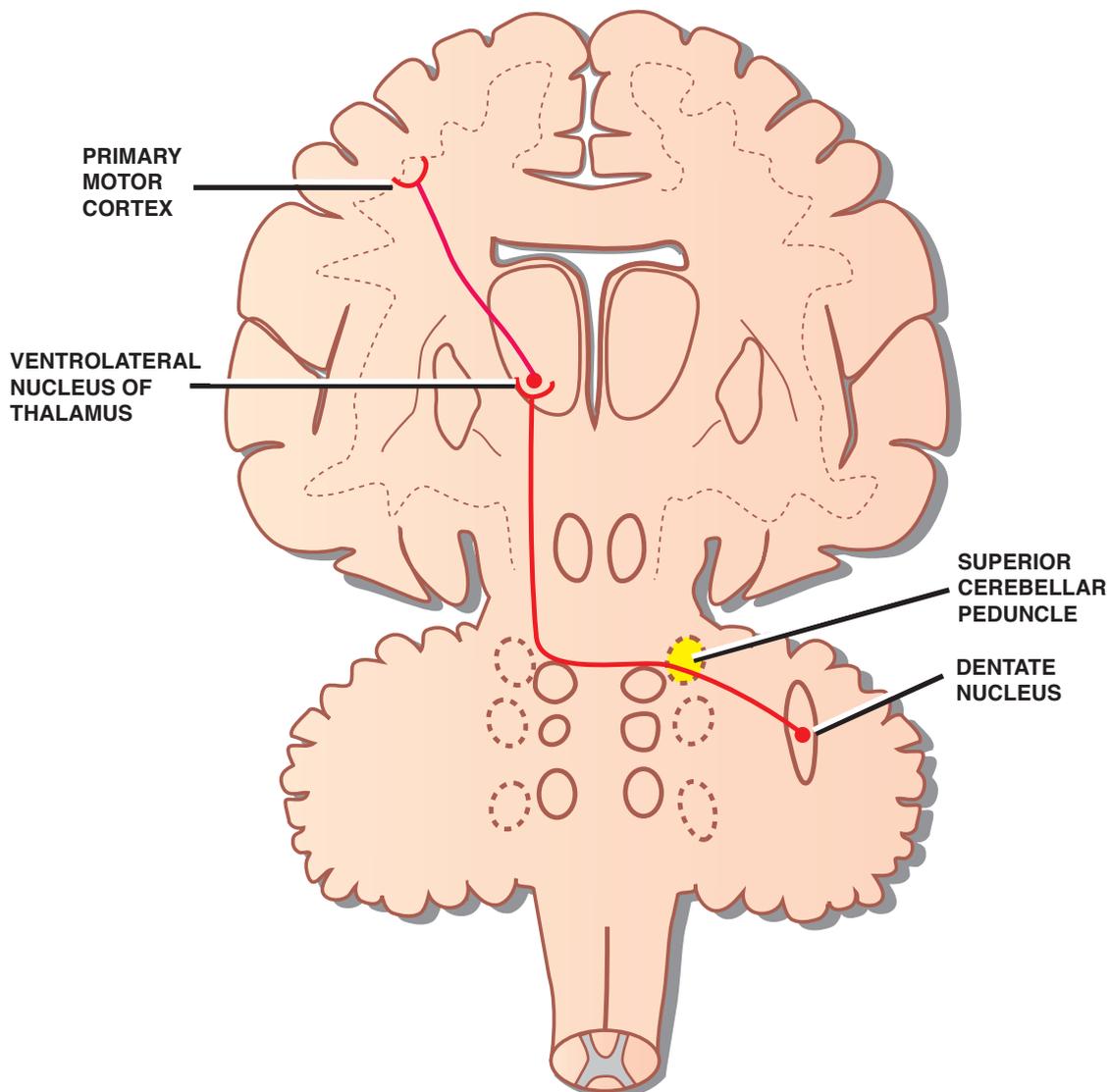
Vestibular Complex

See **Fig. 8.10**.

The axons of the neurons in the fastigial nucleus pass out of the cerebellum through the inferior cerebellar peduncle. These axons descend to terminate on the lateral vestibular nucleus on both sides. As already stated, a few Purkinje cell axons pass the deep cerebellar nuclei and project directly on the lateral vestibular nucleus. The neurons of the lateral vestibular nucleus form the uncrossed descending vestibulospinal tract. Thus, the neurons in the fastigial nucleus influence motor activity (facilitate extensor muscle tone) on the same side of the body. Fibers also synapse on the superior and medial vestibular nuclei. Therefore, the coordination of extensor muscles is also influenced by the cerebellum.

Reticular Formation

The axons of the neurons in the fastigial nucleus pass out of the cerebellum through the inferior cerebellar peduncle. Some of these axons descend to synapse with cells in the reticular formation on both sides. The reticular formation in turn gives rise to the descending reticulospinal tract, which projects both ipsilaterally and bilaterally to the spinal gray matter. The axons of the reticulospinal tract end on interneurons and influence motor neurons indirectly through synaptic relays within the spinal cord.

Fig. 8.10 **Efferent cerebellar connections to vestibular complex.**
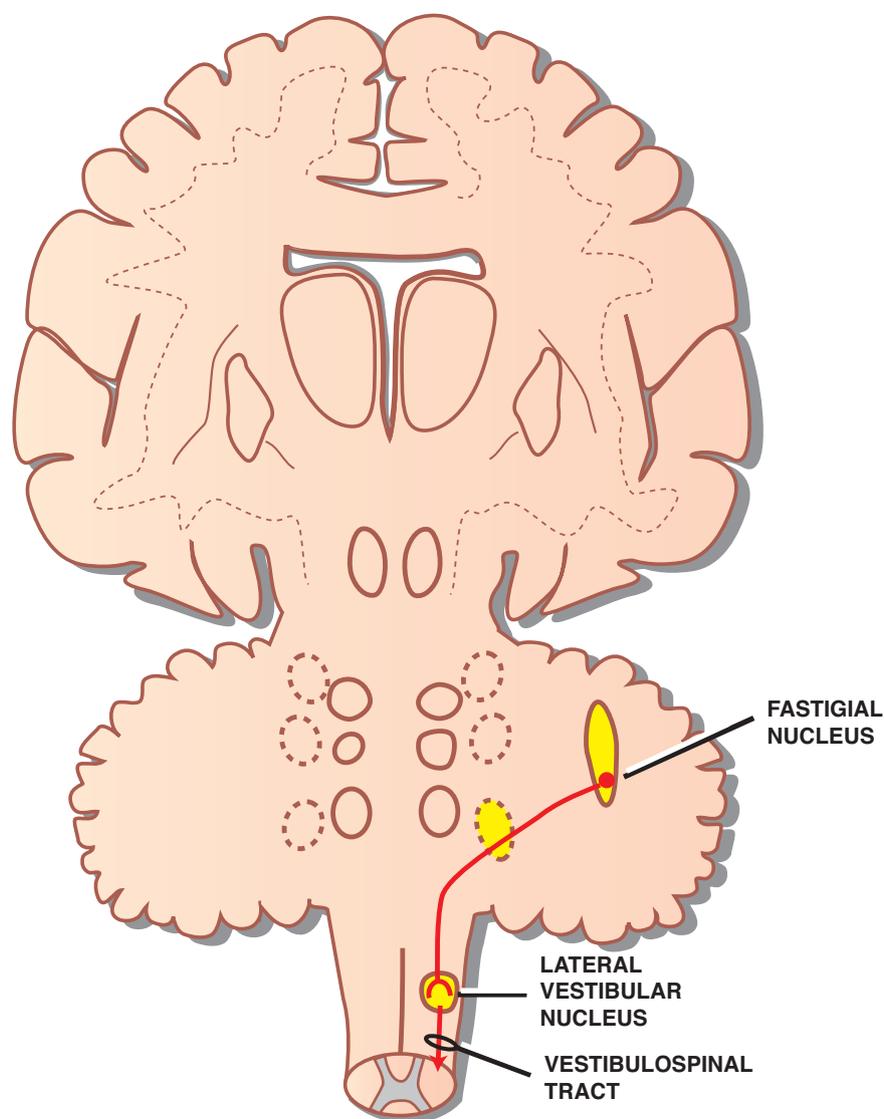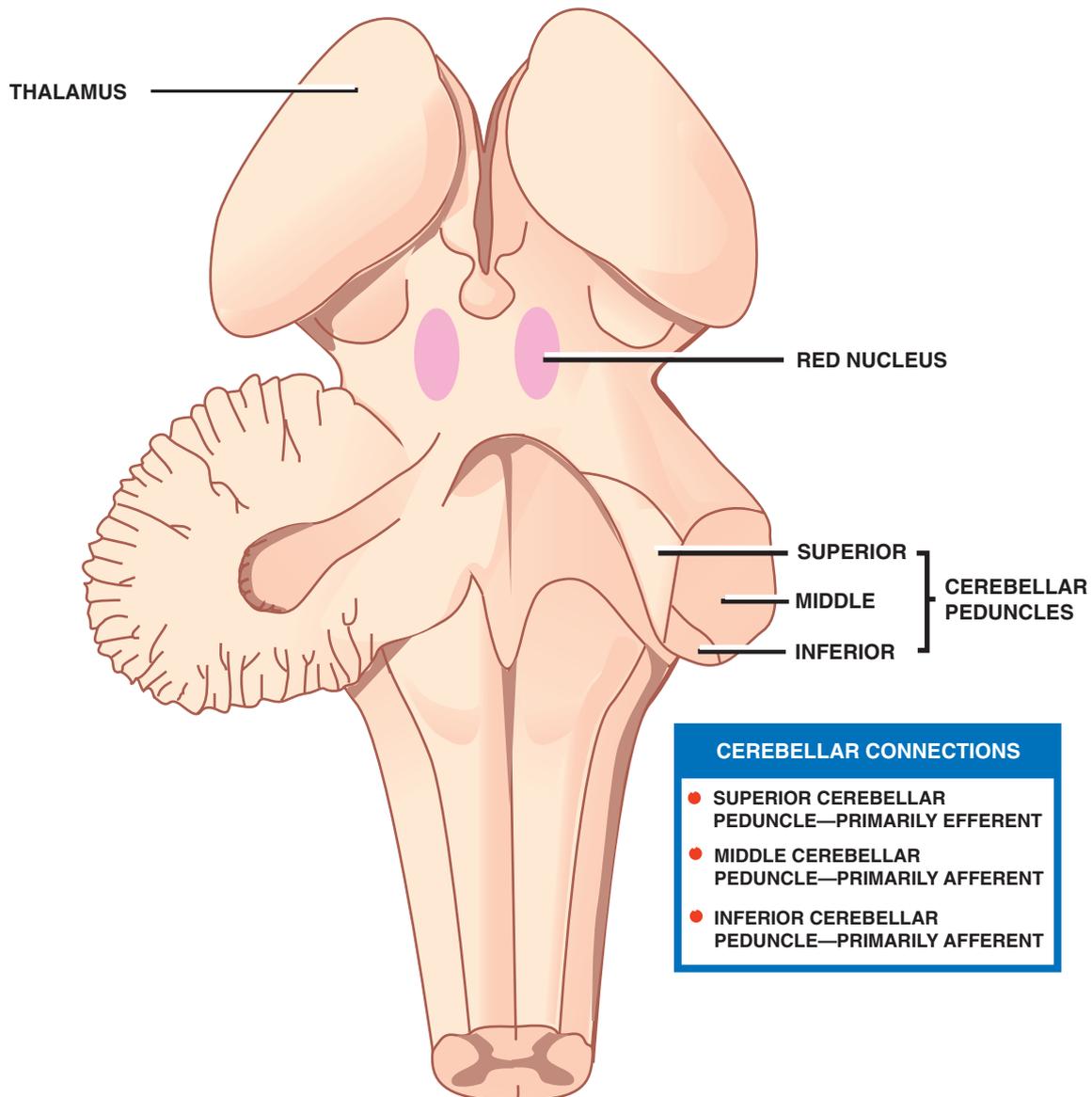
Cerebellar Peduncles

See **Fig. 8.11**.

All the efferent and afferent fibers of the cerebellum reside in the three cerebellar peduncles. The *superior cerebellar peduncle* consists primarily of efferent fibers to the globose, emboliform, and dentate nuclei. A smaller number of afferent fibers, including the ventral spinocerebellar tract, are also present.

The *middle cerebellar peduncle* is the largest of the three peduncles. It contains the afferent pontocerebellar fibers that arise from the contralateral side.

The *inferior cerebellar peduncle* consists primarily of afferent fibers, including (1) the dorsal spinocerebellar tract, (2) the cuneocerebellar tract, (3) the olivocerebellar tract, (4) the reticulocerebellar tract, and (5) the vestibulocerebellar tract. A smaller group of efferent fibers include those that originate in the flocculonodular lobe and the fastigial nucleus, to project on the vestibular nuclei and the central group of reticular nuclei of the medulla and the pons.

Fig. 8.11 **Cerebellar peduncles.**

THALAMUS

RED NUCLEUS

SUPERIOR

MIDDLE — CEREBELLAR PEDUNCLES

INFERIOR

CEREBELLAR CONNECTIONS

- SUPERIOR CEREBELLAR PEDUNCLE—PRIMARILY EFFERENT
- MIDDLE CEREBELLAR PEDUNCLE—PRIMARILY AFFERENT
- INFERIOR CEREBELLAR PEDUNCLE—PRIMARILY AFFERENT
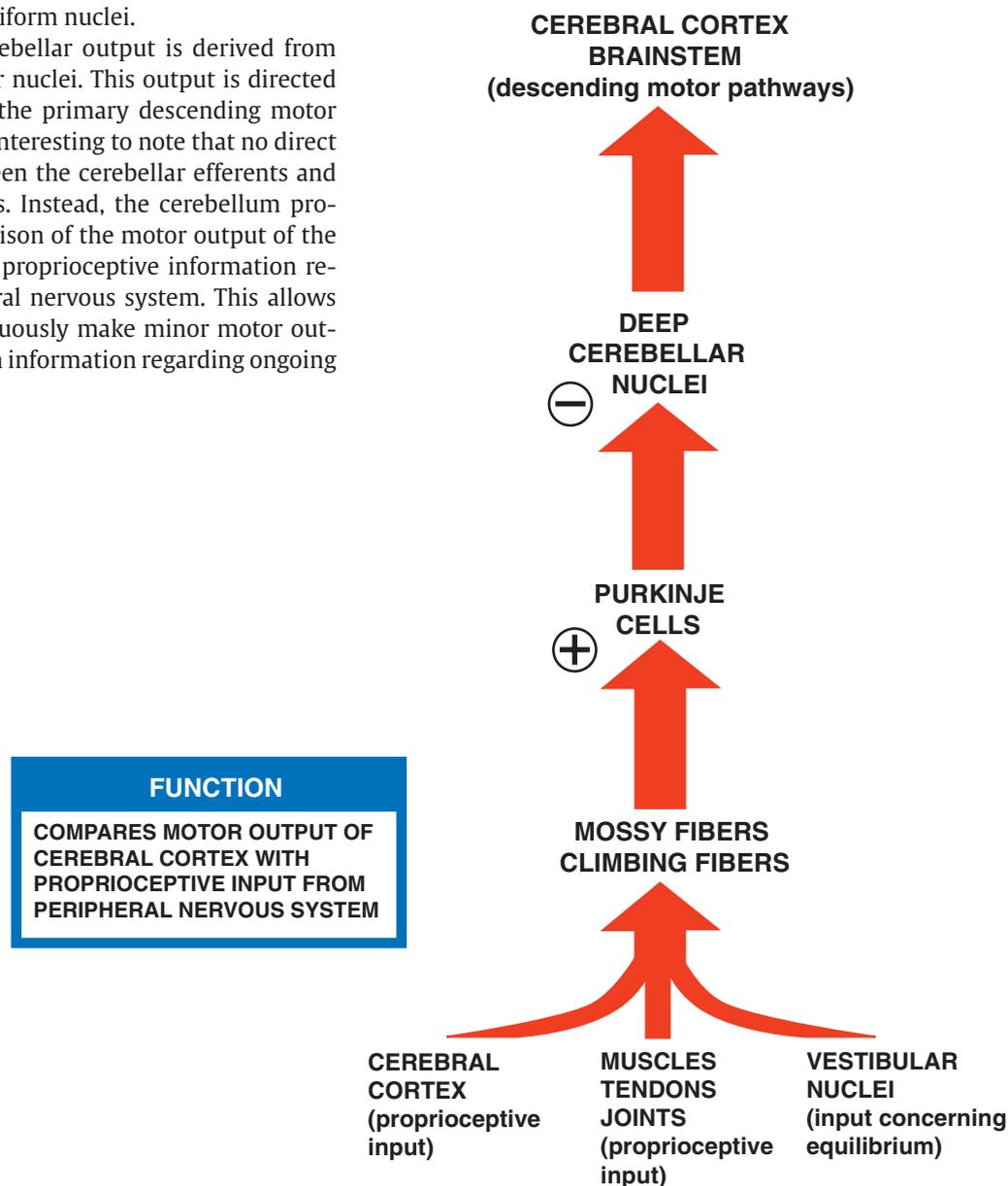
Cerebellar Function

See **Fig. 8.12**.

The cerebellum influences motor activity through its connections with the brainstem and cerebral cortex. It receives information on the activity of the muscles via proprioceptive input from the cerebral cortex, the muscles, the tendons, and the joints. It also receives input concerning equilibrium from the vestibular nuclei.

This afferent input is transmitted to the cerebellar cortex by the excitatory *climbing* and *mossy fibers*. These fibers establish direct or indirect synaptic contacts with the *Purkinje cells*, which in turn exert an inhibitory influence on the *deep cerebellar nuclei* and the lateral vestibular nuclei of the brainstem. Purkinje cell fibers located in the lateral cerebellar hemispheres project to the dentate nuclei; those located in the cerebellar vermis project to the fastigial nuclei; and those located in between project to the globose and emboliform nuclei.

Almost the entire cerebellar output is derived from the cells of the cerebellar nuclei. This output is directed to the sites of origin of the primary descending motor pathways, although it is interesting to note that no direct contacts are made between the cerebellar efferents and the alpha motor neurons. Instead, the cerebellum provides an ongoing comparison of the motor output of the cerebral cortex with the proprioceptive information received from the peripheral nervous system. This allows the cerebellum to continuously make minor motor output adjustments based on information regarding ongoing muscle activity.

Fig. 8.12 **Cerebellar function.**

Functional Anatomic Organization of Cerebellum

The cerebellum may be loosely divided into three separate parts that are distinguished by their input and their major functional activities. These are the vestibulocerebellum, the spinocerebellum, and the pontocerebellum.

Vestibulocerebellum

See **Fig. 8.13**.

The vestibulocerebellum primarily consists of the flocculonodular lobe. It receives mossy fibers from the ipsilateral *vestibular nuclei* and the *vestibular ganglion* via the inferior cerebellar peduncles. It also receives visual information from the lateral geniculate nucleus, superior colliculi, and striate cortex.

Purkinje cell axons of the vestibulocerebellum primarily project to the *fastigial nucleus*. Axons of the cells in the fastigial nucleus leave the cerebellum via the inferior cerebellar peduncles to terminate in the vestibular nuclei.

The vestibulocerebellum influences primary motor activity through its contacts with the vestibulospinal tract. It is concerned with the adjustment of axial muscle tone and the maintenance of equilibrium. It also plays a role in eye movements, control, and the coordination of head and eye movements.

Fig. 8.13 **Vestibulocerebellum.**
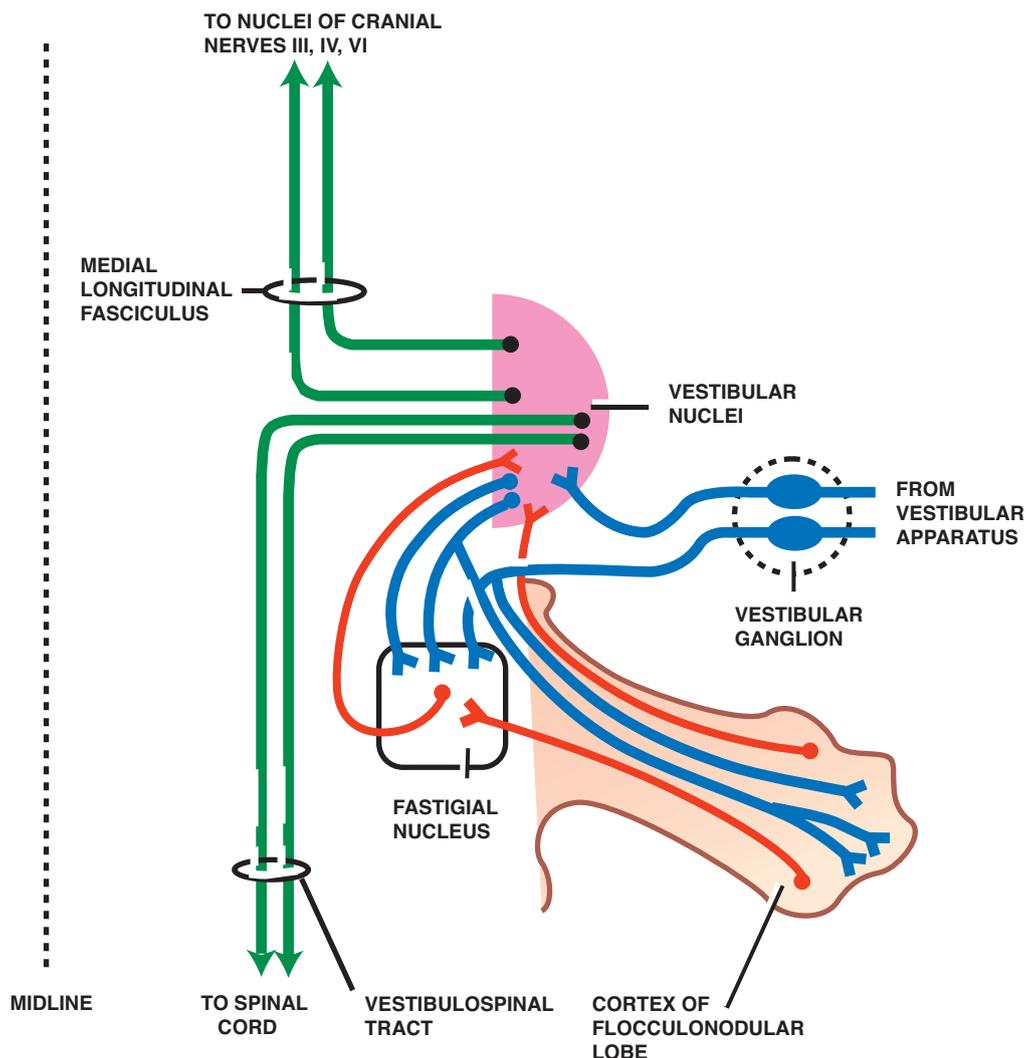
Spinocerebellum

See **Fig. 8.14**.

The spinocerebellum primarily consists of the vermis and the intermediate part of the cerebellar hemispheres. Its primary source of input is somatosensory information from the *dorsal* and *ventral spinocerebellar tracts* of the spinal cord. It also receives information from the auditory, visual, and vestibular systems.

The two parts of the spinocerebellum are composed of two separate output pathways. Purkinje cells in the vermis of the cerebellum send axons to the fastigial nucleus. Axons from this nucleus then project to the brainstem reticular formation, the lateral vestibular nuclei, and the primary motor cortex (via relays in the ventrolateral thalamus). This portion of the spinocerebellum is responsible for control of the medial descending systems, which regulate axial and proximal musculature.

By contrast, Purkinje cells in the intermediate part of the cerebellar hemispheres send axons to interposed nuclei, which in turn project to the rubrospinal and lateral corticospinal tracts. A smaller number of projections from the interposed nuclei relay in the ventrolateral thalamus and continue on to the primary motor cortex. The hemispheric part of the spinocerebellum is responsible for control of the lateral descending systems, which regulate the distal limb muscles.

Efferent fibers from the intermediate zone of the cerebellar hemispheres leave the cerebellum through the superior cerebellar peduncles to reach the rubrospinal and lateral corticospinal tracts. Along the way, these fibers cross in the decussation of the superior cerebellar peduncles. Because the rubrospinal and lateral corticospinal tract fibers also cross the midline before they terminate in the spinal cord, destructive lesions in the intermediate zone of the cerebellar hemispheres result in neurologic deficits on the ispilateral side of the body: the spinocerebellum influences the skeletal musculature on the ipsilateral side of the body.

The spinocerebellum controls the execution of movement and regulates muscle tone. It carries out these functions by continuously comparing information about the intended motor commands of the primary motor cortex with feedback about ongoing movement that is received from the spinal cord and periphery. This organization allows the spinocerebellum to correct for deviations in intended movement.

Fig. 8.14 **Spinocerebellum.**
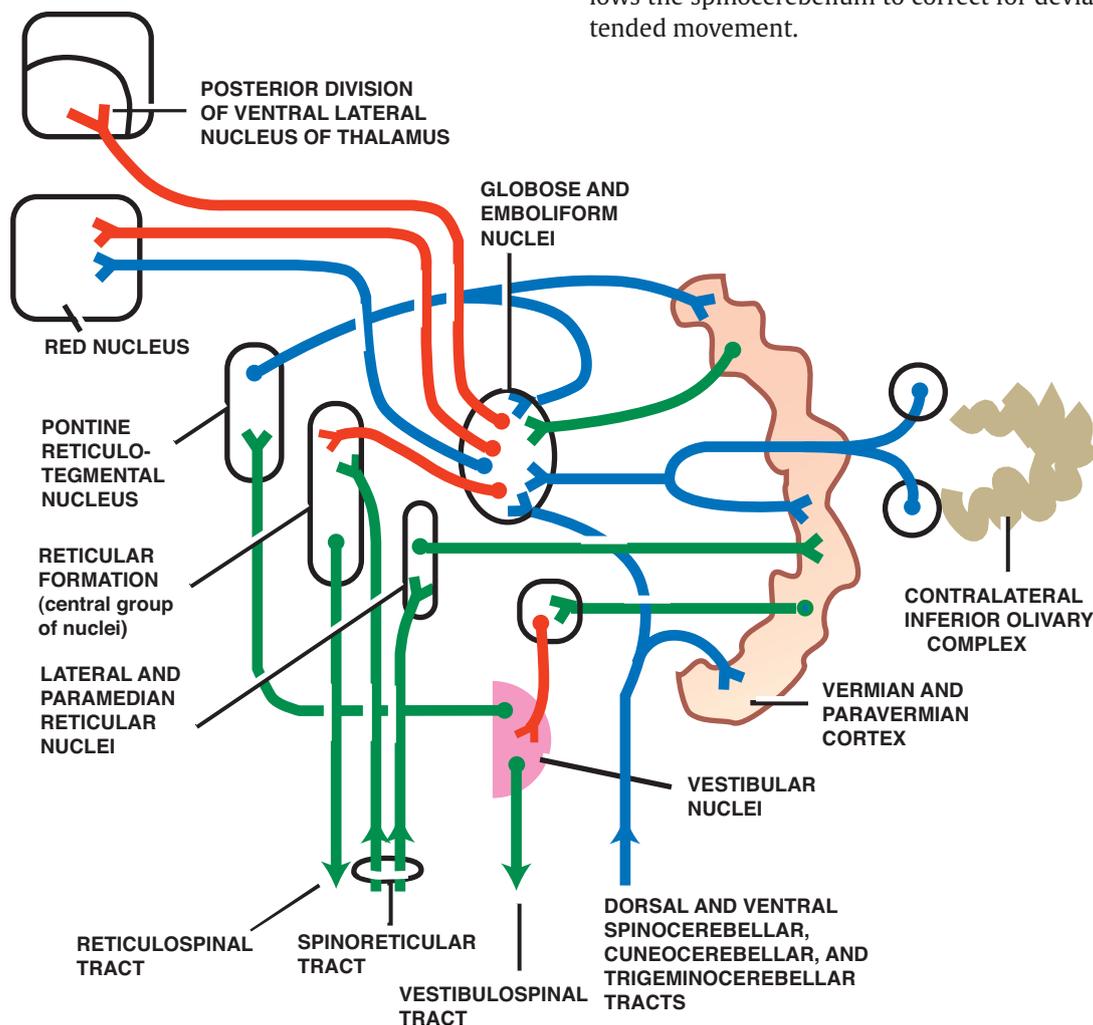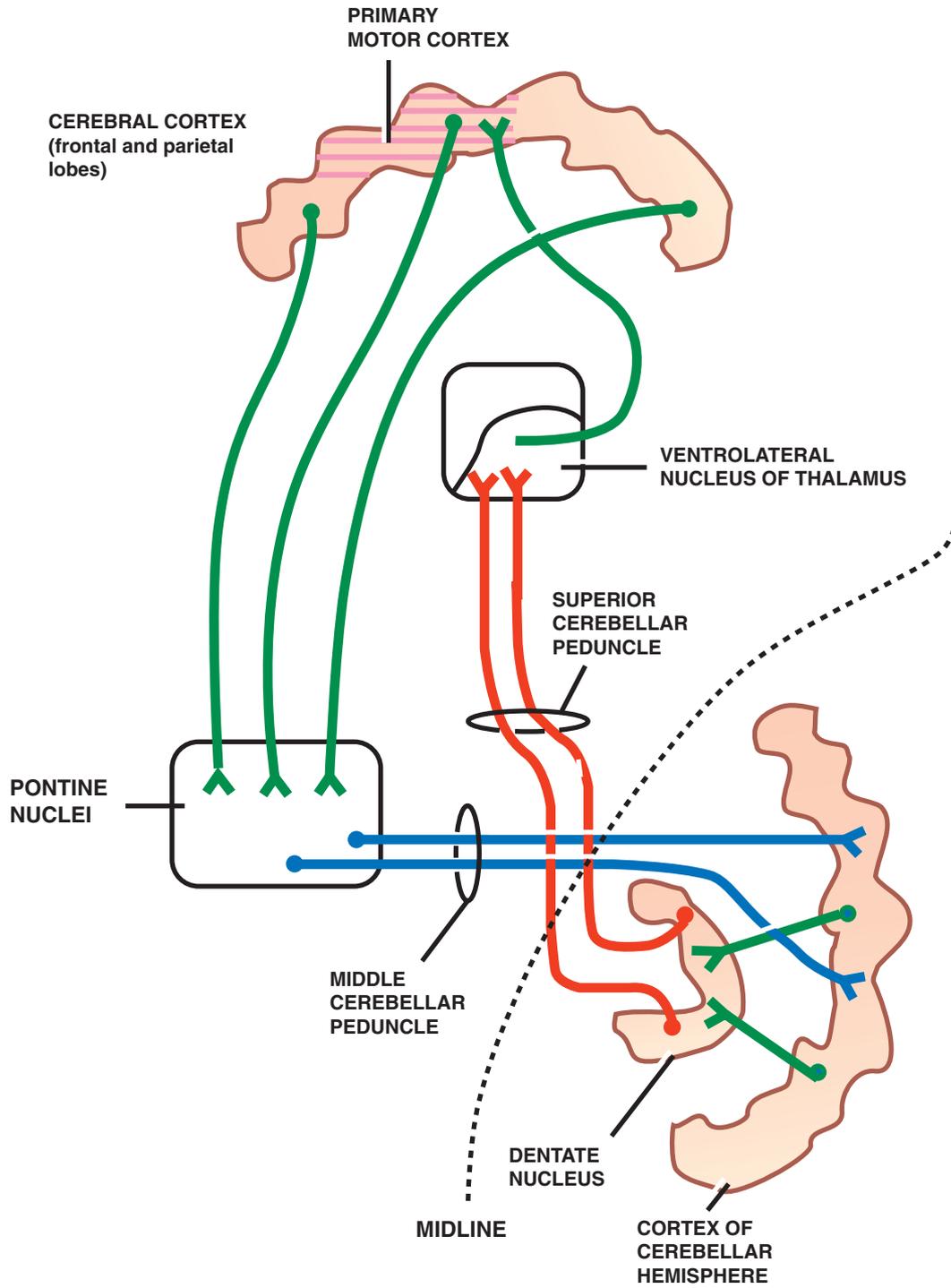
Pontocerebellum

See **Fig. 8.15**.

The pontocerebellum primarily consists of the large lateral regions of the cerebellar hemispheres. Its primary source of input is from large areas of the contralateral *cerebral cortex* (especially that of the frontal and parietal lobes). These corticopontine fibers relay in the *pontine nuclei* and then enter the cerebellum through the middle cerebellar peduncles, of which they are the sole constituents. The information that is transmitted from the cerebral cortex to the cerebellum concerns volitional movements that are ongoing or are about to happen.

Purkinje cell axons of the pontocerebellar cortex project to the *dentate nucleus*, which in turn projects fibers via the superior cerebellar peduncles to the *ventrolateral nucleus of the thalamus*. Pontocerebellar afferents make up most of the fibers in the superior cerebellar peduncles. Axons from the nucleus of the ventrolateral thalamus project to the *primary motor cortex*, thus completing the corticopontine–thalamic–cortical loop.

Because of decussations in both the superior cerebellar peduncles and the corticospinal tract and other descending pathways, the pontocerebellum exerts its influence on the ipsilateral side of the body.

The pontocerebellum is concerned with precision in the control of rapid limb movements and with tasks requiring fine dexterity. It ensures a smooth and orderly sequence in muscle contractions and regulates the force, direction, and extent of volitional movement. It functions in these capacities by modulating activity in the primary motor cortex, a role that is also performed by the premotor cortical areas. Destructive lesions in the pontocerebellum may lead to various movement disorders, such as delays in the initiation or termination of movement, or involuntary tremor at the end of a movement.

Fig. 8.15 **Pontocerebellum.**

PRIMARY
MOTOR CORTEX

CEREBRAL CORTEX
(frontal and parietal
lobes)

VENTROLATERAL
NUCLEUS OF THALAMUS

SUPERIOR
CEREBELLAR
PEDUNCLE

PONTINE
NUCLEI

MIDDLE
CEREBELLAR
PEDUNCLE

DENTATE
NUCLEUS

MIDLINE

CORTEX OF
CEREBELLAR
HEMISPHERE

Clinical Manifestations of Cerebellar Disease

The clinical manifestations of cerebellar disease may be divided into three categories: (1) the symptoms of cerebellar disease, (2) the signs of midline cerebellar disease, and (3) the signs of lateral (hemispheric) cerebellar disease. Although the functional anatomy of the cerebellum comprises three separate zones—a midline, an intermediate, and a lateral (hemispheric) zone—only the midline and lateral zones are associated with distinct abnormalities; no abnormalities that are specifically associated with the intermediate zone have been identified. Furthermore, many of the signs that are observed with midline lesions may also be associated with lateral lesions, and vice versa.

Symptoms of Cerebellar Disease

The symptoms of cerebellar disease are nonspecific. They include **headache, nausea and vomiting, gait difficulty**, and **vertigo**.

Signs of Midline Cerebellar Disease

See **Fig. 8.16**.

The midline of the cerebellum comprises the anterior and posterior vermis, the flocculonodular lobe, and the fastigial nuclei. Functionally, this zone is responsible for equilibrium required during ambulation, the maintenance of truncal posture, the position of the head in relation to the trunk, and the control of extraocular eye movements. As a result, lesions in this area tend to produce **gait difficulty, truncal imbalance, abnormal head postures**, and **ocular motor dysfunction**.

Disorders of Stance and Gait

Truncal instability may be manifested during walking by a tendency to fall to the right, left, forward, or backward. While sitting, the patient may lean or fall to one side. These tendencies tend to be toward the side of the lesion.

Several abnormalities of gait, which are collectively described as gait ataxia, include a wider than normal base, unsteadiness and irregularity of steps, and lateral veering. The steps may be uncertain, some shorter and some longer than intended, and there may be a tendency to stagger or lurch to one side. There is little localizing value in cerebellar gait disorders to distinguish between midline and lateral cerebellar lesions, although lateral lesions tend to cause the patient to veer toward the side of the lesion.

Mild cerebellar gait disorders may be exacerbated by asking the patient to tandem walk. The Romberg's sign, in which the patient loses balance after closing the eyes, is a result of disordered position sensation secondary to posterior column disease and is not related to cerebellar dysfunction.

Abnormal Postures of the Head

Abnormal postures of the head may be due to midline or lateral cerebellar lesions. They may present as a head tilt (i.e., lateral deviation of the head) or a rotated posture of the head.

Ocular Motor Dysfunction

Like the clinical signs already described, ocular motor dysfunction may occur in association with midline cerebellar lesions but is also associated with lesions in other parts of the cerebellum.

The cerebellum and brainstem are involved in computing the location of targets in space, deriving temporal information from spatial information for appropriate muscle innervation, and adjusting the gain on saccadic eye movements to minimize target overshoot and undershoot. As a result, the primary disorders of extraocular movements in midline cerebellar disease are nystagmus and ocular dysmetria.

Nystagmus consists of rhythmic oscillatory movements of one or both eyes, occurring with the eyes in the primary position or with ocular deviation. The most common types of nystagmus observed in association with midline cerebellar lesions are (1) *gaze-evoked nystagmus*, (2) *rebound nystagmus*, and (3) *optokinetic nystagmus*.

Gaze-evoked nystagmus occurs when an individual cannot maintain conjugate eye deviation away from the midposition. Conjugate lateral gaze is accompanied by a slow, involuntary drift of the eyes back to midposition, followed by a rapid corrective return of the eyes to the laterally located target. The result is a to-and-fro oscillation of the eyes involving a fast component in the direction of gaze and a slow component away.

Rebound nystagmus is specific for cerebellar disease, although it is poorly localized within the cerebellum. It essentially represents a type of gaze-evoked nystagmus that changes direction after sustained lateral gaze or after refixation to the primary position.

Optokinetic nystagmus is a nonpathological nystagmus that develops normally when an individual attempts to count the stripes on a rotating drum or a moving cloth strip. In the presence of cerebellar disease, optokinetic nystagmus may become exaggerated, producing unusually large amplitudes of both the fast and the slow components.

Ocular dysmetria is defined as the conjugate overshoot of a target with voluntary saccades. The eyes appear to jerk back and forth because of repeated inaccuracies in saccadic movements intended to bring the target to the fovea.

Fig. 8.16 **Signs of midline cerebellar disease.**

OCULAR MOTOR DYSFUNCTION

GAZE-EVOKED NYSTAGMUS

QUICK MOVEMENT TO LATERALLY LOCATED TARGET
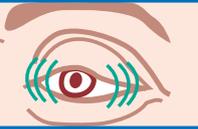
SLOW MOVEMENT BACK TO MIDPOSITION

REBOUND NYSTAGMUS

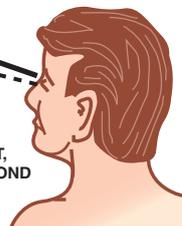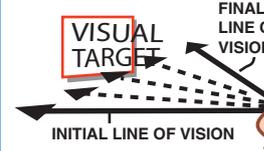LIKE GAZE-EVOKED NYSTAGMUS BUT WITH INTERMITTENT CHANGE OF DIRECTION

OPTOKINETIC NYSTAGMUS
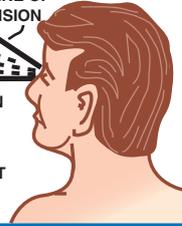
AS DRUM ROTATES, EYES FOLLOW DRUM TO A POINT, THEN TURN BACK TO BEYOND START WITH INCREASED AMPLITUDE

OCULAR DYSMETRIA

VISUAL TARGET
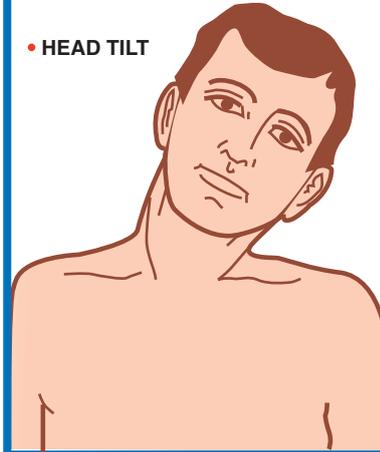
FINAL LINE OF VISION

INITIAL LINE OF VISION

PATIENT ATTEMPTING TO FIXATE ON VISUAL TARGET (NOTE OVERSHOOT)

DISORDERS OF STANCE AND GAIT

- WIDE-BASED, IRREGULAR STEPS
- VEERS TO ONE SIDE

ABNORMAL POSTURES OF THE HEAD

- HEAD TILT

Signs of Lateral (Hemispheric) Cerebellar Disease

The lateral (hemispheric) zone of the cerebellum comprises the cerebellar hemisphere and the dentate and interposed nuclei of each side. The intermediate zone is also included here because it does not appear to be associated with distinct abnormalities. Compared with the midline zone, the lateral zone is involved in a greater variety of clinical disorders. These include (1) **hypotonia**, (2) **dysarthria**, (3) **limb ataxia**, (4) **intention tremor**, (5) **impaired check**, and (6) **oculomotor disorders**.

Hypotonia

Hypotonia, a decrease in resistance to passive movement of the limbs, is associated with lateral cerebellar lesions. It is best demonstrated by grasping the patient's forearms and shaking the relaxed wrists. Typically, the hypotonic limb is identified as the limb with more of a flail hand. In addition, examination of the patellar reflex in a hypotonic lower limb may demonstrate an increased duration and amplitude of swing. This pendular cerebellar reflex should be distinguished from clonus, due to corticospinal tract disease, which occurs at a more rapid rate than the pendular reflex.

Dysarthria

The dysarthria of cerebellar disease is characterized by slow, labored, slurred, or garbled speech that may be mistaken, like cerebellar ataxia, as a manifestation of alcohol intoxication. Comprehension and grammar remain intact.

Limb Ataxia

Limb ataxia, like gait ataxia, comprises a combination of disturbances in voluntary movement, the most important of which are dysmetria and the decomposition of movement.

Dysmetria consists of an error in trajectory and speed of movement. It is most easily demonstrated in the upper extremity, where it is tested by asking patients to touch the nose with the finger. Frequently, patients with lateral cerebellar disease undershoot or overshoot (past-pointing) the target. As the finger approaches the nose, it may oscillate around the nose and strike the cheek (overshoot) or stop short of touching the nose (undershoot). To test the lower extremity, ask the patient, in the supine position, to raise the heel of one foot over the knee of the other and to slide the heel smoothly down the shin. Frequent deviations to one side or the other are indicative of dysmetria in that limb.

Decomposition of movement involves errors in the sequence and speed of the component parts of a movement. An affected limb is unable to execute a movement, such as reaching out to grasp a glass of water, in a smooth and fluid manner. Instead, the movement is halting, imprecise, and jerky. Decomposition of movement may be tested by the same finger-to-nose test just described. Alternatively, decomposition of movement may be brought out in examination by asking the patient to perform a series of rapidly alternating or fine repetitive movements, such as a sequence of pronation and supination movements of the hand. The presence of decomposition of movement during this maneuver is referred to as dysdiadochokinesis.

Intention Tremor

Intention tremor is an irregular, more or less rhythmic, interruption of a voluntary movement that begins and increases as the patient approaches a target. It may be tested, like ataxia, by asking the patient to perform a finger-to-nose maneuver. Intention tremor is distinguished from the rest tremor of parkinsonism, which only occurs at rest, and the action tremor of familial or essential origin, which characteristically occurs during a sustained posture and from the beginning to the end of a movement.

Impaired Check

An impaired check response is characterized by the wide excursion of an affected limb following an involuntary displacement of the limb by an examiner. It may be tested by tapping the wrists of a patient with outstretched, pronated arms. With the patient's eyes closed, normally there is a small displacement of the arm that has been tapped, followed by a rapid, accurate return to the original position. In a patient with cerebellar disease, the displaced arm demonstrates an unusually wide excursion, followed by an overshoot of the original position. The original position is finally reached only after considerable oscillation about it.

Another method that may be used to test for an impaired check response is to provide resistance against a patient's flexed arm. On abrupt release of an affected arm, the arm will continue unchecked in the direction of its force and will strike the patient's chest.

Oculomotor Disorders

See **Fig. 8.17**.

Many oculomotor disturbances are associated with both lateral and midline cerebellar disorders but defy more specific localization. Several of these disturbances have already been described, such as gaze-evoked nystagmus, rebound nystagmus, ocular dysmetria, and optokinetic nystagmus. Other oculomotor disorders are frequently associated with widespread cerebellar disease or with disease involving both the cerebellum and the brainstem, including the following:

- *Opsoclonus* This disorder is characterized by constant, random, conjugate saccades of unequal amplitudes in all directions. Frequently, they are most marked immediately before and after a fixation.
- *Ocular flutter* Ocular flutter is defined as rapid to-and-fro oscillations of the eyes. These abnormal movements may develop abruptly, last for only seconds, and disturb vision for the duration of the episode.

- *Ocular bobbing* Ocular bobbing comprises intermittent (abrupt) downward displacement of the eyes, followed by a slow, synchronous return to the primary position. The relatively quick downward displacement is slower than the fast component of nystagmus; this disorder should therefore be distinguished from downbeat nystagmus, which is associated with lesions in the cervicomedullary junction. Horizontal eye movements are typically paralyzed.
- *Ocular myoclonus* Ocular myoclonus is defined as a rhythmic, pendular oscillation of the eyes that is associated with synchronous oscillation of the plate (palatal myoclonus).

Fig. 8.17 **Signs of lateral cerebellar disease (oculomotor disorders).**

OPSOCLONUS
CONSTANT RANDOM CONJUGATE SACCADES OF UNEQUAL AMPLITUDES IN ALL DIRECTIONS

OCULAR BOBBING
ABRUPT DOWNWARD DISPLACEMENT OF EYES IS FOLLOWED BY SLOW RETURN TO PRIMARY POSITION

OCULAR FLUTTER
RAPID TO-AND-FRO OSCILLATIONS OF THE EYES

OCULAR MYOCLONUS
RHYTHMIC, PENDULAR OSCILLATION OF THE EYES