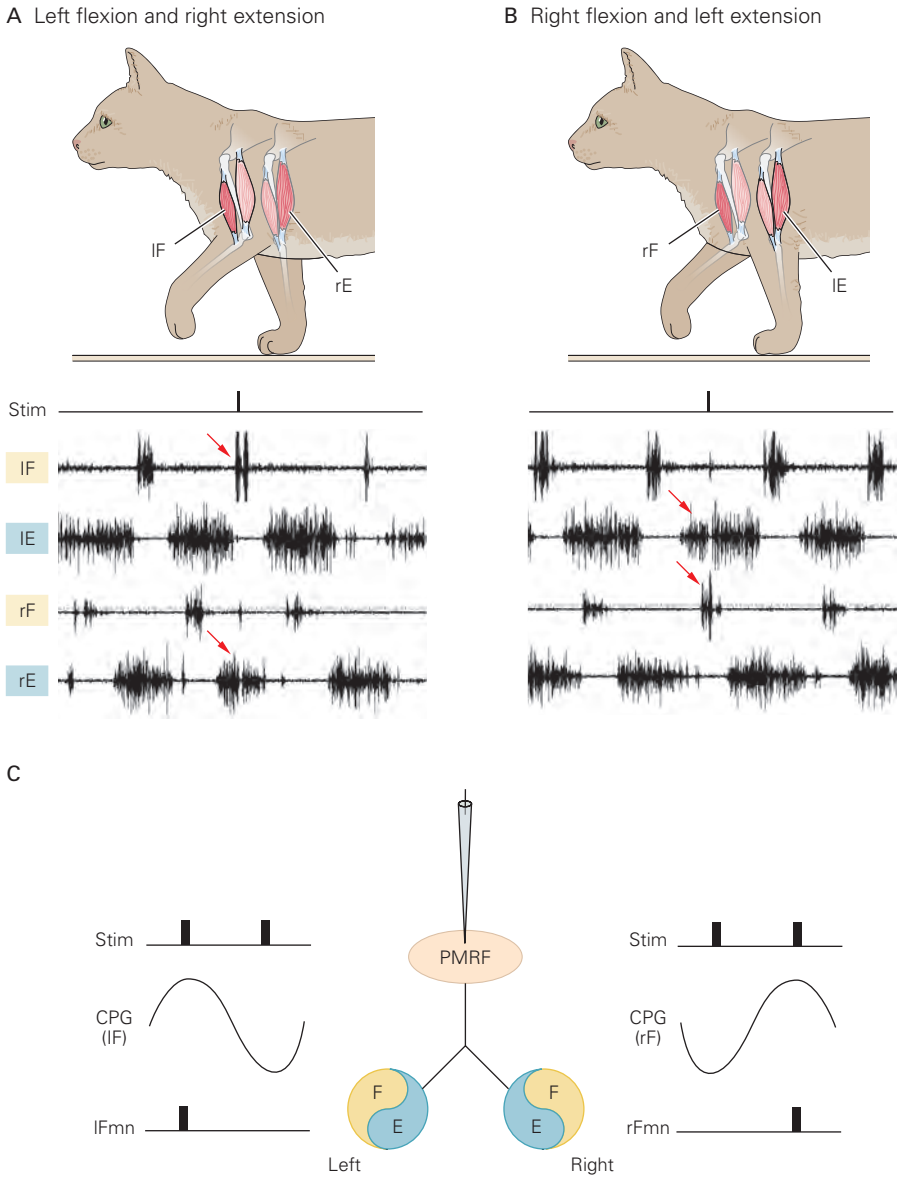

Figure 33–13 Microstimulation of the pontomedullary reticular formation (PMRF) produces phase-dependent responses in flexor and extensor muscles. (Data from T. Drew.)

A. Stimulation of the left PMRF during the swing phase of the left limb produces a transient increase in the electromyogram activity of the left flexor muscles (**IF**) and a simultaneous decrease in activity in the right extensor muscles (**rE**) (**red arrows**). There is little stimulus-evoked activity in the left extensor (**lE**) or right flexor (**rF**) muscles, which are inactive at this phase of the step cycle.

B. Stimulation at the same location in the PMRF during the swing phase of the right limb produces the inverse responses.

C. The phase-dependent nature of the responses is likely determined by the cyclical nature of the level of excitability in interneurons that are part of the locomotor central pattern generator (**CPG**). Responses are gated by activity in the flexor (**F**) and extensor (**E**) parts of the locomotor CPG. When the first stimulation arrives, flexor interneurons in the left CPG (**lF**) are active, whereas those in the right CPG (**rF**) are inactive. The stimulation therefore produces a response only in the left flexor motor neurons (**lFmn**). When the second stimulation arrives, flexor interneurons in the right CPG (**rF**) are active, whereas those on the left side are inactive, and therefore, the stimulation elicits a response only in the flexor motor neurons on the right (**rFmn**).

gait (see below). In a complementary manner, the widespread effects of the PMRF on multiple limbs allow it to produce the coordinated changes in postural activity that accompany gait modifications. The coordination between gait modifications and postural activity is assured by the strong connections from the motor cortex to the PMRF in the same manner as for discrete voluntary movements (Chapter 34). The PMRF also contributes to the compensatory changes in posture that occur as a consequence of perturbations. In this situation, it forms part of a spino-bulbo-spinal reflex that contributes to the widespread postural responses that follow the immediate spinal reflexes activated by a sudden perturbation.

Visually Guided Locomotion Involves the Motor Cortex

Walking is most often guided by vision, and the motor cortex is largely essential for visually guided movement, especially when gait must be modified to ensure precise control over limb trajectory and foot placement. In mammals, lesions of the motor cortex do not

prevent animals from walking on a smooth floor, but they severely impair "precision locomotion," which requires a high degree of visuomotor coordination, such as walking on the rungs of a horizontal ladder, stepping over a series of barriers, and stepping over single objects placed on a treadmill belt.

Experiments in intact cats trained to step over obstacles attached to a moving treadmill belt show that precision locomotion is associated with considerable modulation of the activity of numerous neurons in the motor cortex (Figure 33–14). Other neurons in the motor cortex show a more discrete pattern of activity and are activated sequentially during different parts of the swing phase. The activity of these cortical neurons correlates with the periods of modified muscle activity required to produce the gait modifications in a similar manner to what occurs during reaching (see Figure 34–21). Such subpopulations of neurons may serve to modify the activity of the groups of synergistic muscles required to produce flexible changes in limb trajectory.

Many of these cortical neurons project directly to the spinal cord (corticospinal neurons) and thus may regulate the activity of spinal interneurons, including those within the CPG, thereby adapting the timing and magnitude of motor activity to a specific locomotor task. Brief trains of electrical stimulation applied to either the motor cortex or the corticospinal tract in normal walking cats produce transient responses in the contralateral limb in a phase-dependent manner, similar to that produced by activity in various brain stem structures. However, in contrast to the situation

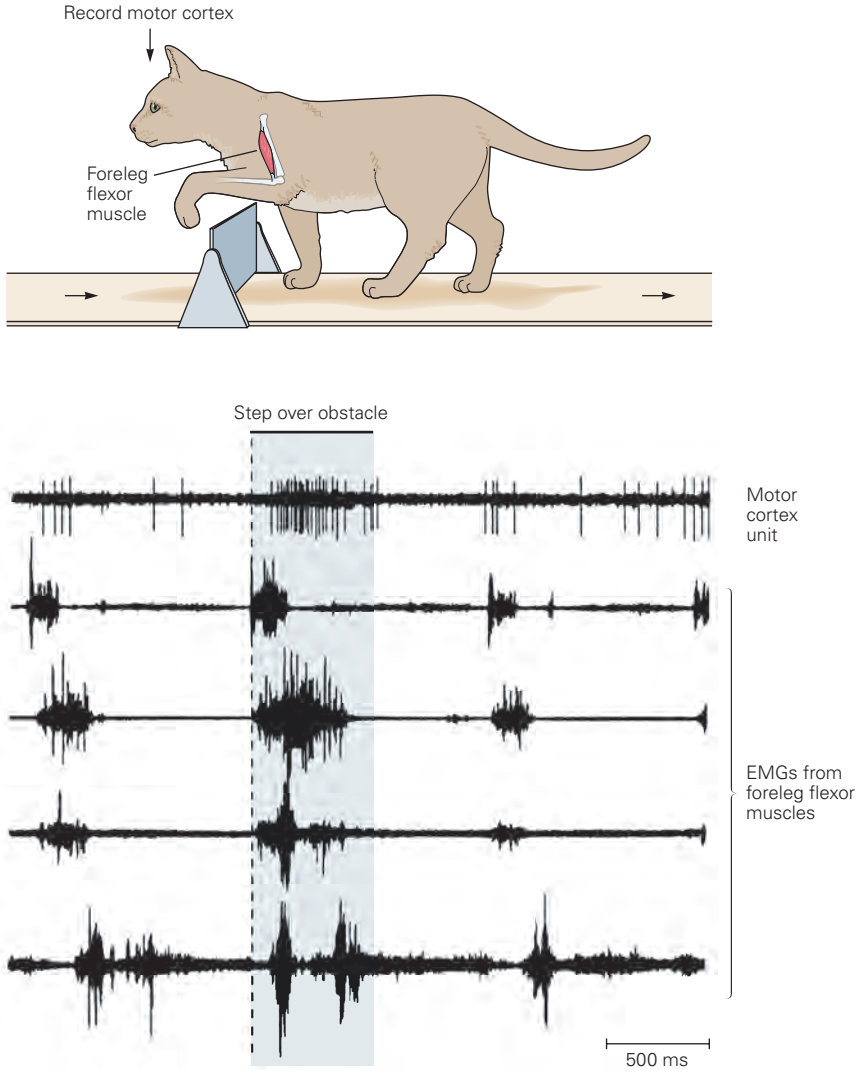

Figure 33–14 Stepping movements are adapted by the motor cortex in response to visual inputs. When a cat steps over a visible object fixed to a treadmill, neurons in the motor cortex increase in activity. This increase in cortical activity is associated with enhanced activity in foreleg muscles, as seen in the electromyograms (**EMG**). (Adapted, with permission, from Drew 1988.)

observed with brain stem structures, increasing the duration of the stimulation train applied to the motor cortex frequently results in a reset of the locomotor rhythm, characterized as an interruption of the ongoing step cycle and the initiation of a new step cycle. This suggests that in mammals the corticospinal tract has privileged access to the rhythm generator of the CPG.

Planning of Locomotion Involves the Posterior Parietal Cortex

When humans and animals approach an obstacle in their pathway, they must adjust their walking pattern to move around the obstacle or step over it. Planning of these adjustments begins two or three steps before the obstacle is reached. Recent experiments suggest that the posterior parietal cortex (PPC) is particularly involved in planning gait modifications. Lesions in this region cause walking cats to misplace the positioning of their paws as they approach an obstacle and increase the probability that one or more legs contact the obstacle as they step over it.

In contrast to what is observed in the motor cortex, recordings in PPC show that many neurons increase their activity in advance of the step over the obstacle. Moreover, many cells in the PPC discharge similarly regardless of which leg is first to step over the obstacle (Figure 33–15A,B). Such cells may provide an estimation of the position of the body with respect to objects in the environment (Figure 33–15B), allowing animals to modify gait as they approach the obstacle. The manner in which the PPC interacts with other cortical and subcortical structures generally considered to be involved in motor planning is unknown. However, recent work shows that the premotor cortex also makes an important contribution to planning visually guided gait modifications (Figure 33–15C) and may be implicated in the transformation of a global signal providing information concerning obstacle location to the muscle-based signal necessary for the execution of the step over the obstacle.

Visual information about the size and location of an obstacle is also stored in working memory, a form of short-term memory (Chapter 52). This information is used to ensure that gait modifications in the hindlimb are coordinated with those of the forelimb and is necessary because the obstacle is no longer within the visual field by the time the hindlimbs are stepping over it. The neurobiological mechanisms underlying this form of working memory remain to be established, but the persistence of the memory appears to depend, at least in part, on neuronal systems in the PPC. With bilateral lesions or cooling of the medial PPC, the memory is completely abolished (Figure 33–16A). Complementing this observation is the finding that the activity of some neurons in the PPC is elevated during a step over an obstacle, as well as throughout the time the animal straddles the obstacle (Figure 33–16B). This activity could represent the working memory of key features of the obstacle such as height.

The Cerebellum Regulates the Timing and Intensity of Descending Signals

Damage to the cerebellum results in marked abnormalities in locomotor movements, including the need for a widened base of support, impaired coordination of joints, and abnormal coupling between limbs during stepping. These symptoms, which are characteristic of *ataxia* (Chapter 37), indicate that the cerebellum contributes importantly to the regulation of locomotion.

A major function of the cerebellum is to correct movement based on a comparison of the motor signals sent to the spinal cord and the movement produced by that motor command (Chapter 37). In the context of locomotion, the motor signal is generated by neurons in the motor cortex and brain stem nuclei. Information about the movement comes from the ascending spinocerebellar pathways. For the hind legs of the cat, these are the dorsal and ventral spinocerebellar tracts. Neurons in the dorsal spinocerebellar tract (DSCT neurons) are strongly activated by numerous leg proprioceptors and thus provide the cerebellum with detailed information about the mechanical state of the hind legs. In contrast, neurons in the ventral tract (VSCT neurons) are activated primarily by interneurons in the CPG, thus providing the cerebellum with information about the state of the spinal locomotor network.

During locomotion, the motor command (the central efference copy), the movement (the afference copy, via the DSCT), and the state of the spinal networks (the spinal efference copy, via the VSCT) are integrated within the cerebellum and expressed as changes in the pattern of rhythmical discharge of Purkinje cells in the cerebellar cortex and neurons in the deep cerebellar nuclei. These signals from the deep cerebellar nuclei are then sent to the motor cortex and the various brain stem nuclei where they modulate descending signals to the spinal cord to correct any motor errors.

Behavioral experiments show that the cerebellum also plays an important role in the adaptation of gait. For example, when subjects walk on a split treadmill,

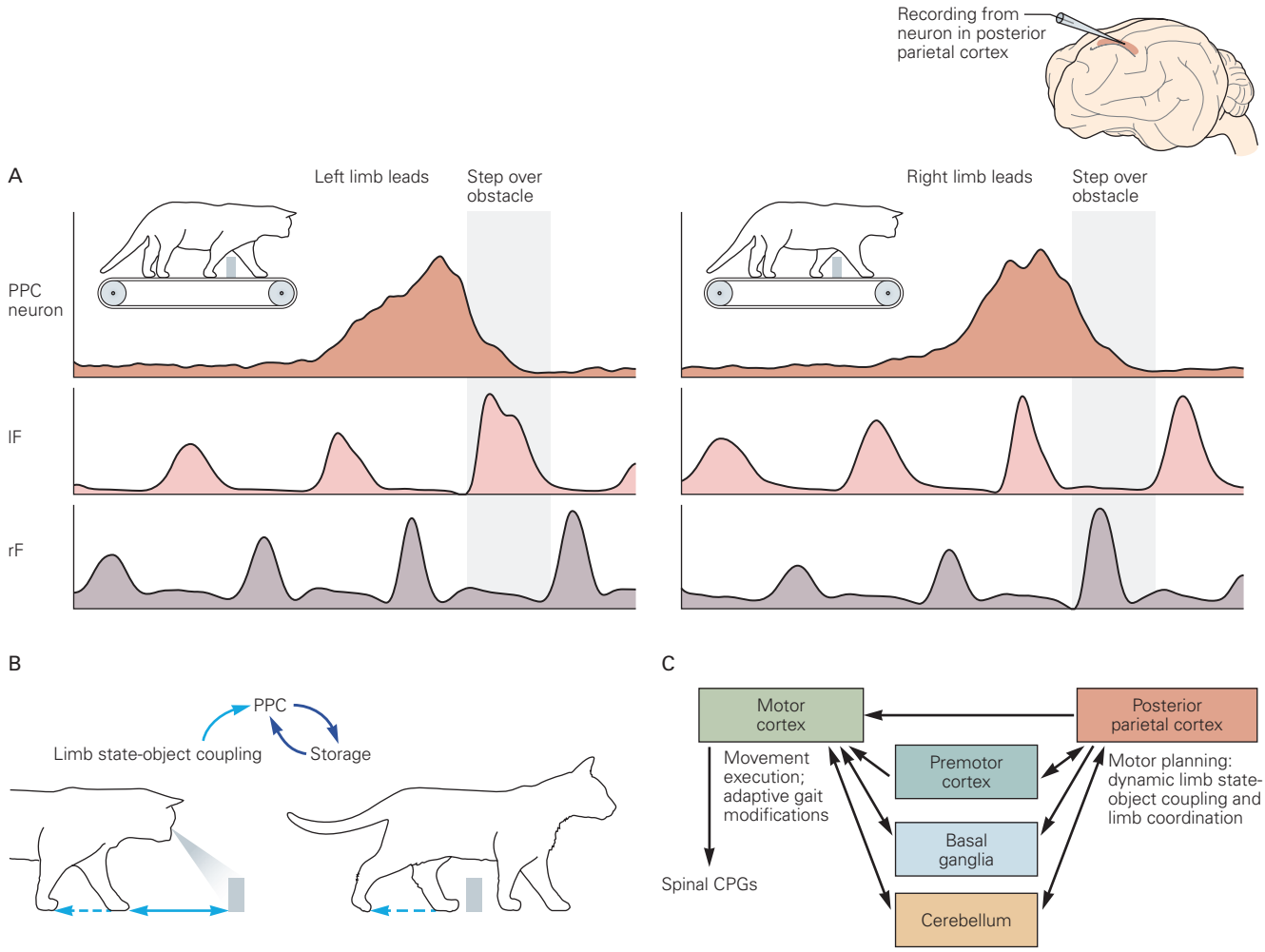

Figure 33–15 Neurons in the posterior parietal cortex (PPC) are involved in planning voluntary gait modifications.

A. Activity of a PPC neuron in the right cortex during a step over the obstacle when the left or right forelimb is the first to step over the obstacle. In each situation, the cell in the PPC discharges two to three steps in advance of the step over the obstacle.

B. The observation that PPC neurons discharge independent of which limb is the first to step over the obstacle suggests a global function of PPC in the planning of locomotion. In a general scheme, the PPC neurons are involved in the estimation of the relative location of an object with respect to the body (limb state–object coupling [**double arrow**]) and storage information in the PPC for later retrieval.

C. The PPC does not act alone in planning gait modifications. It is part of a cortical and subcortical network that includes, among other structures, the premotor cortex, the basal ganglia, and the cerebellum. Connections exist between each of these structures as well as between each of them and the motor cortex, which is responsible for the execution of the gait modification. (Abbreviation: **CPG**, central pattern generator.) (Adapted, with permission, from Drew and Marigold 2015.)

so that each leg walks at a different speed, they initially show a very asymmetric gait before adapting over time to a more asymmetric one. When the two treadmill belts are reset to the same speed, they again show an asymmetric gait, demonstrating that the experimental condition had produced adaptation (see Figure 30–13). Patients with damage to the cerebellum are not able to adapt to this condition.

The Basal Ganglia Modify Cortical and Brain Stem Circuits

The basal ganglia are found in all vertebrates from the oldest vertebrates to primates and probably contribute to the selection of different motor patterns. The importance of the basal ganglia to the control of locomotion is clearly demonstrated by the deficits in

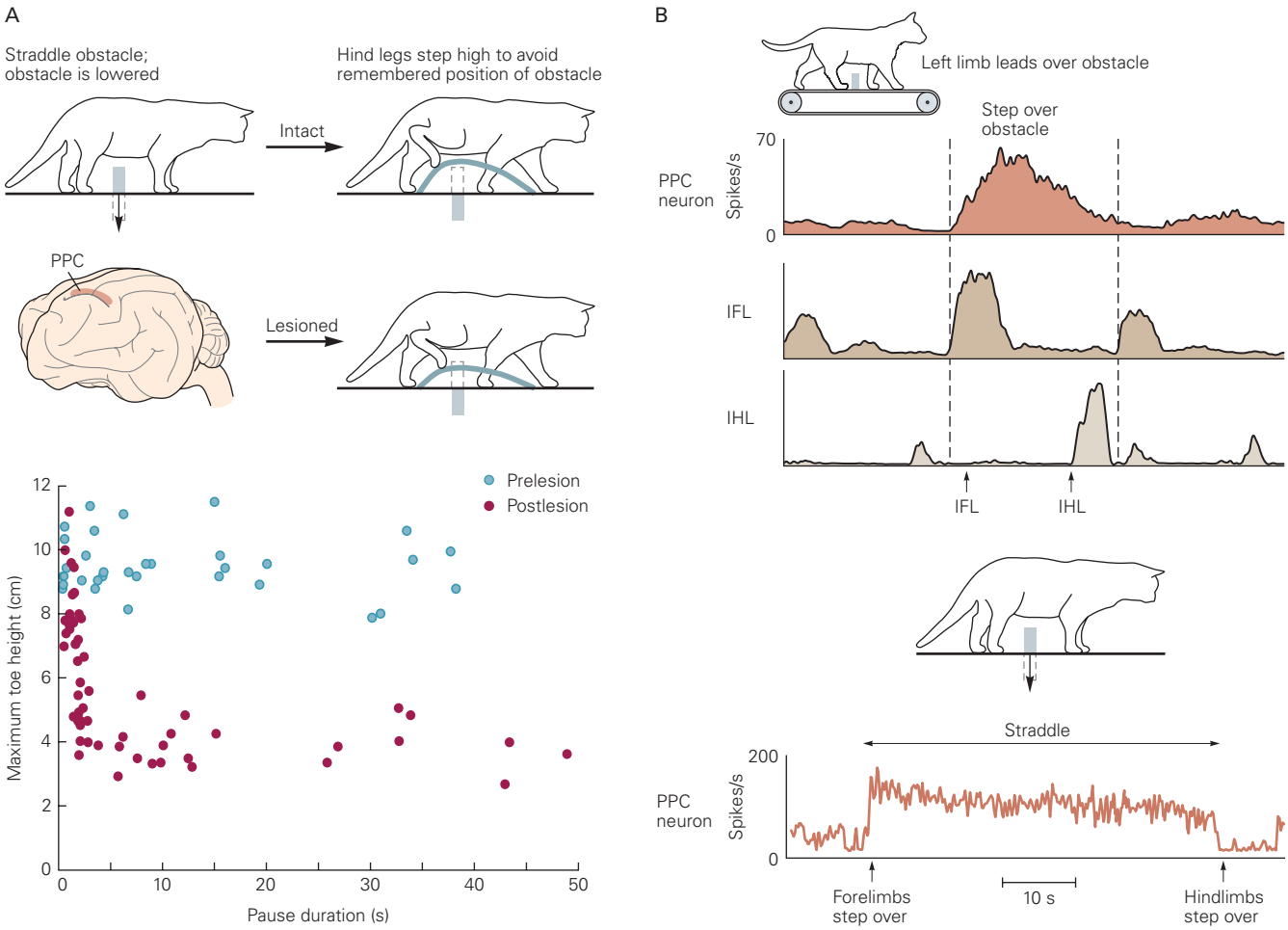

Figure 33–16 The posterior parietal cortex (PPC) is involved in maintaining an estimate of an obstacle in working memory during locomotion.

A. **Upper figure:** Normal animals were trained to walk forward, step over an obstacle, and then pause. While the animal paused, the obstacle was removed. When walking resumed, the hind legs stepped high to avoid the remembered obstacle. This memory lasted for more than 30 seconds. The trajectory of the hindlimbs was scaled appropriately for the height of the obstacle and for the relative position of the hind paws. Bilateral lesions of the PPC led to an impairment in the memory, making it impossible for the animal to pass the obstacle without hitting it. **Lower figure:** Following the lesion, animals stored the memory for only 1 to 2 seconds, and the maximum height of the toe was insufficient to clear the obstacle and was significantly lower than in the prelesion condition. (Adapted from McVea and Pearson 2009.)

B. **Upper figure:** Neurons recorded in an intact animal in the PPC on the right side discharged in the period between the passage of the left forelimb (**IFL**) and hindlimb (**IHL**) over an obstacle (represented by electromyogram activity from representative flexor muscles in each limb). This discharge may be used to coordinate the movement of the hindlimb with that of the forelimb during the visually guided gait modification. **Lower figure:** When the cat steps over an obstacle and pauses, as in part **A**, cells in the PPC show a maintained discharge that could provide the neural representation of the working memory. (Adapted, with permission, from Lajoie et al. 2010.)

locomotion observed in patients with Parkinson disease, which disrupts the normal functioning of the basal ganglia due to degradation of their dopaminergic inputs from the substantia nigra (Chapter 38).

Such patients show a characteristic slow, shuffling gait and, in later stages of the disease, can also show "freezing" of gait. Patients with Parkinson disease also show problems with balance during locomotion and with the anticipatory postural adjustments that occur at the initiation of a gait pattern. These deficits suggest that the basal ganglia contribute to the initiation, regulation, and modification of gait patterns. This regulation is mediated by the two major projections of the basal ganglia to the brain stem pathways and cortical structures.

The basal ganglia influence brain stem activity through their projections to the PPN. The PPN receives

inhibitory inputs from GABAergic inhibitory neurons in the substantia nigra pars reticulata (SNr) as well as from the globus pallidus pars interna (GPi); it also receives glutamatergic input from neurons in the subthalamic nucleus (STN). Decreased inhibitory input and increased glutamatergic input to PPN from the basal ganglia are thought to promote activity in PPN and favor exploratory locomotion. The STN and GPi are major targets of deep brain stimulation for improvement of motor symptoms such as rigidity and reduced mobility in patients with Parkinson disease.

The basal ganglia influence cortical activity by means of its connections via the thalamus to different parts of the frontal cortex, including the supplementary motor regions. These connections allow the basal ganglia to exert a modulatory effect on visually guided locomotion, possibly by selecting the appropriate motor patterns required in different behavioral situations.

Computational Neuroscience Provides Insights Into Locomotor Circuits

While functional studies have revealed much about the organization of the locomotor networks, their overall complexity makes it difficult to capture the integrative function of synaptic and cellular properties of the circuit. Computational network modeling, however, allows one to simulate the circuit activity and to investigate the dynamic interactions between the circuit elements. Computational models can be developed at many levels: to study the ionic basis of neural activity within a given circuit, to study the connectivity between different groups of neurons in a particular circuit, or to better understand the interactions between different structures in the locomotor network. Computational models at each of these levels have been developed to study rhythm and pattern generation in both invertebrates and vertebrates and in the latter, ranging from the lamprey to mammals. As in other domains, approaches combining experimental manipulation and computational modeling are likely to increase in the coming years and have the potential to advance our understanding of the complex systems and interconnections between structures that are required to produce the full locomotor repertoire.

Neuronal Control of Human Locomotion Is Similar to That of Quadrupeds

By necessity, most of our understanding of the neural mechanisms underlying the control of locomotion comes from experiments on quadrupedal animals. Nonetheless, the available evidence suggests that all the major principles concerning the origin and regulation of walking in quadrupeds also pertain to locomotion in humans. Although the issue of whether CPGs exist in humans remains contentious, several observations are compatible with the view that CPGs are important for human locomotion.

For example, observations of some patients with spinal cord injury parallel the findings from studies of spinal cats. Striking cases of patients with nearly complete transection of the spinal cord have shown uncontrollable, spontaneous, rhythmic movements of the legs when the hips were extended. This behavior closely parallels the rhythmic stepping movements in chronic spinal cats. Moreover, tonic electrical stimulation of the spinal cord below the injury can evoke locomotor-like activity, as in other mammals.

Parallels between human and quadrupedal walking have also been found in patients trained after spinal cord injury. Daily training combined with drug treatments restores stepping in spinal cats and improves stepping in patients with chronic spinal injuries. People with severe spinal cord injury who have been exposed to both treadmill-induced stepping and drug treatments similar to those that have been shown to activate the CPG in cats have demonstrated dramatic improvements in the ability to produce locomotion (Box 33–4). These results suggest that CPGs are present in humans and share functional similarities with CPGs found in other vertebrates.

Compelling evidence for the existence of spinal CPGs in humans also comes from studies in human infants who make rhythmic stepping movements immediately after birth if held upright and moved over a horizontal surface. This strongly suggests that some of the basic neuronal circuits for locomotion are innate and present at birth when descending control systems are not well developed. Because stepping can also occur in infants who lack cerebral hemispheres (*anencephaly*), these circuits must be located at or below the brain stem, perhaps entirely within the spinal cord.

During the first year of life, as automatic stepping is transformed into functional walking, these basic circuits are thought to be brought under supraspinal control. In particular, the stepping pattern gradually develops from a more primitive flexion-extension pattern that generates little effective forward movement to the mature pattern of complex movements. It is plausible, based on studies of cats, that this adaptation reflects maturation of descending systems that originate in the motor cortex and brain stem nuclei and are modulated by the cerebellum.

Box 33–4 Rehabilitative Training Improves Walking After Spinal Cord Injury in Humans

According to the World Health Organization, between 250,000 and 500,000 people worldwide incur spinal cords injuries annually. For many, this results in permanent loss of sensation, movement, and autonomic function. The devastating loss of functional abilities, together with the enormous cost of treatment and care, creates an urgent need for effective methods to repair the injured spinal cord and to facilitate functional recovery.

Over the past decades, progress has been made in animal research aimed at preventing secondary damage after injury, repairing the axons of lesioned neurons in the spinal cord, and promoting the regeneration of severed axons through and beyond the site of injury. In many instances, the regeneration of axons has been associated with modest recovery of locomotor function. However, none of the regeneration strategies has reached the point where they can be confidently used in humans with spinal cord injury.

Thus, rehabilitative training is the preferred treatment for people with spinal cord injury. One especially successful technique for enhancing walking in patients with partial damage to the spinal cord is repetitive, weight-supported stepping on a treadmill (Figure 33–17). This technique is based on the observation that spinal cats and rodents can be trained to step with their hind legs on a moving treadmill.

For humans, partial support of the body weight through a harness system is critical to the success of training; presumably, it facilitates the training of spinal cord circuits by reducing the requirements for supraspinal control of posture and balance.

Although the neural basis for the improvement in locomotor function with treadmill training has not been established, it is thought to depend on synaptic plasticity in local spinal circuits as well as successful transmission of at least some command signals from the brain through preserved descending pathways if the spinal cord injury is only partial.

Locomotor training is sometimes combined with other treatments. These include different types of medication designed to reduce spasticity, seen as involuntary muscle contractions, and facilitation of activity in spinal circuits by electrical transcutaneous activation of spinal circuits and/or activation of corticospinal pathways by transcranial magnetic stimulation.

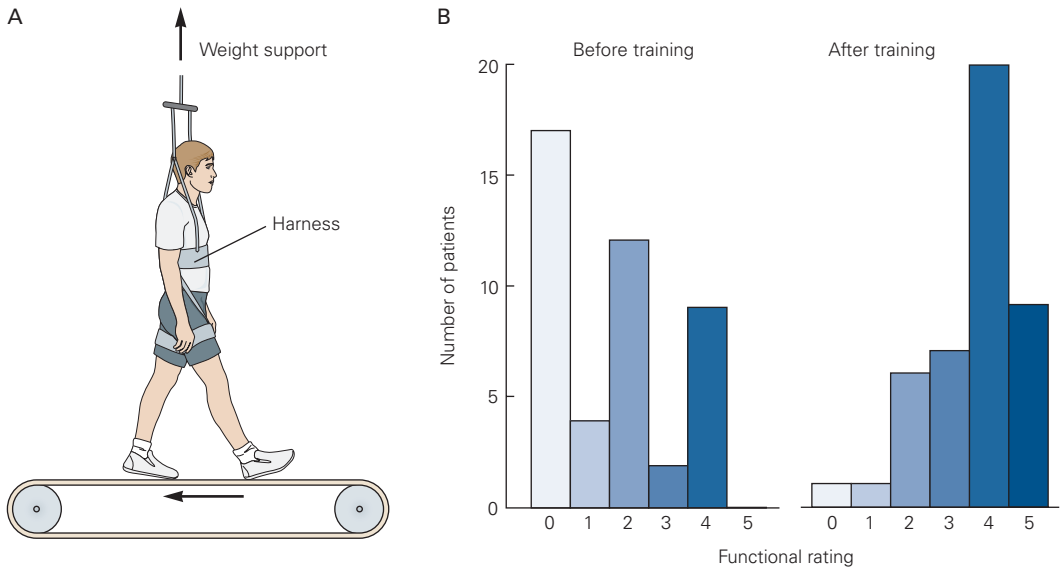

Figure 33–17 Treadmill training improves locomotor function in patients with partial spinal cord injury.

A. The patient is partially supported on a moving treadmill by a harness, and stepping movements are assisted by therapists.

B. Locomotor function improvement in 44 patients with chronic spinal cord injury after daily training lasting from 3 to 20 weeks. The functional rating ranges from 0 (unable to stand or walk) to 5 (walking without devices for more than five steps). (Adapted, with permission, from Wernig et al. 1995.)

At the cortical level, stroke involving the motor cortex or damage to the corticospinal tracts leads to deficits in locomotion, as in cats. However, the deficits in humans are much stronger than in cats or even nonhuman primates, suggesting that the motor cortex in humans plays a more important role in locomotion than in other mammals. Studies using transcranial magnetic stimulation (TMS) to modulate motor cortical activity also show that the motor cortex contributes importantly to the control of human locomotion. TMS parameters that result in cortical inactivation, for example, produce a decrease in the level of muscle activity during locomotion. In contrast, TMS parameters that activate the motor cortex improve the recovery of locomotion following incomplete spinal cord injury.

Imaging studies, together with high-resolution electroencephalogram recordings, show changes in the activity of several cortical regions, including the motor cortex, premotor cortex, and PPC, during locomotion and particularly during imagined locomotion over obstacles. Imaging studies have also shown increased activity during locomotion in those parts of the midbrain shown to be important for the initiation and speed control of locomotion in animals. Similarly, neurons in the pedunculopontine nucleus can be affected in Parkinson disease, contributing to the severe gait disturbances seen in the late phase of the disease.

Highlights

1. Locomotion is a highly conserved behavior that is essential for the survival of the species. Our understanding of the neuronal mechanisms involved in the generation and control of locomotion came initially from the study of phylogenetically older animals, such as the lamprey and the tadpole. More recently, in mammals, with their more complex nervous systems, the organization of the different neural pathways involved in the generation and regulation of locomotion has also been determined in significant detail.
2. The spinal cord, in isolation from descending and rhythmical peripheral afferent inputs, can generate a complex locomotor pattern that contains elements of the rhythms and patterns observed in intact animals. The circuits responsible for producing this activity are referred to as central pattern generators (CPGs). Activity in spinal circuits can be modified by experience.
3. The basic components of CPGs controlling swimming are excitatory rhythm-generating neurons together with commissural inhibitory neurons responsible for left–right alternation. This organizational principle is also found in CPGs controlling limbed movements with the addition of flexor-extensor pattern-generating circuits and additional commissural neuronal networks. The circuits in the locomotor networks have a modular organization with distinct transmitter and molecular codes for the constituent neurons. Descending command signals act on these circuit elements to produce the diverse aspects of locomotor behavior.
4. Ionic membrane properties in interneurons and motor neurons contribute to rhythm and pattern generation. Cell-specific manipulation of these properties will enable a precise understanding of their relative contributions to locomotor production.
5. Peripheral afferent inputs modulate the function of spinal locomotor circuits. Proprioceptive sensors are used to stabilize phase transitions between stance and swing (and vice versa), whereas input from exteroceptors is used to modify limb activity in response to unexpected perturbations.
6. Circuits that are involved in initiating locomotion, controlling speed of locomotion, and selecting gaits are localized in the midbrain and encompass excitatory neurons in the pedunculopontine and cuneiform nuclei. These excitatory nuclei serve diverse roles in controlling either slow explorative locomotion or the full range of speeds and gaits including fast escape locomotion. Molecular-genetically driven cell-specific approaches allow unparalleled access to the organization of these pathways in the brain stem and how they integrate with spinal locomotor networks.
7. Activity in the three main structures in the brain stem with axons that descend to the spinal cord (the pontomedullary reticular formation, the lateral vestibular nucleus, and the red nucleus) contributes to the control of posture and interlimb coordination. Signals from these structures modify the level of muscle activity in a structure-specific manner.
8. The motor cortex provides precise control of muscle activity patterns to allow animals to make visually guided anticipatory adjustments of their gait. The signal from the motor cortex is integrated into the ongoing rhythm.
9. The posterior parietal cortex (PPC) is part of a network that contributes to the advanced planning

of gait based on visual information. PPC neurons estimate the relative location of objects with respect to the body and retain information in working memory to facilitate coordination of the limbs. The contribution of other cortical and subcortical areas to locomotor planning remains little studied.

10. Inputs from the cerebellum and the basal ganglia are used to correct motor errors and select the appropriate patterns of motor activity. The contribution of the basal ganglia to the control of locomotion is complex and is only now being determined.
11. The available evidence suggests that the neural control mechanisms determined from experiments in animals are also used to control locomotion in humans, including the existence of a CPG. Major advances remain to be made in understanding the mechanisms of spinal and supraspinal influences on human locomotor control.
12. Recent technological advances now give us an unparalleled opportunity to investigate the control mechanisms underlying locomotion. Molecular and genetic advances provide the ability to manipulate behavior at both the cellular and systems level and allow detailed study of the contributions of brain stem and spinal circuits to the initiation and regulation of locomotion. Advances in multineuronal recording techniques in animals, as well as the development of high-resolution recordings of human brain activity, will facilitate our understanding of the contribution of cortical structures to the control of locomotion.

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Ole Kiehn

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