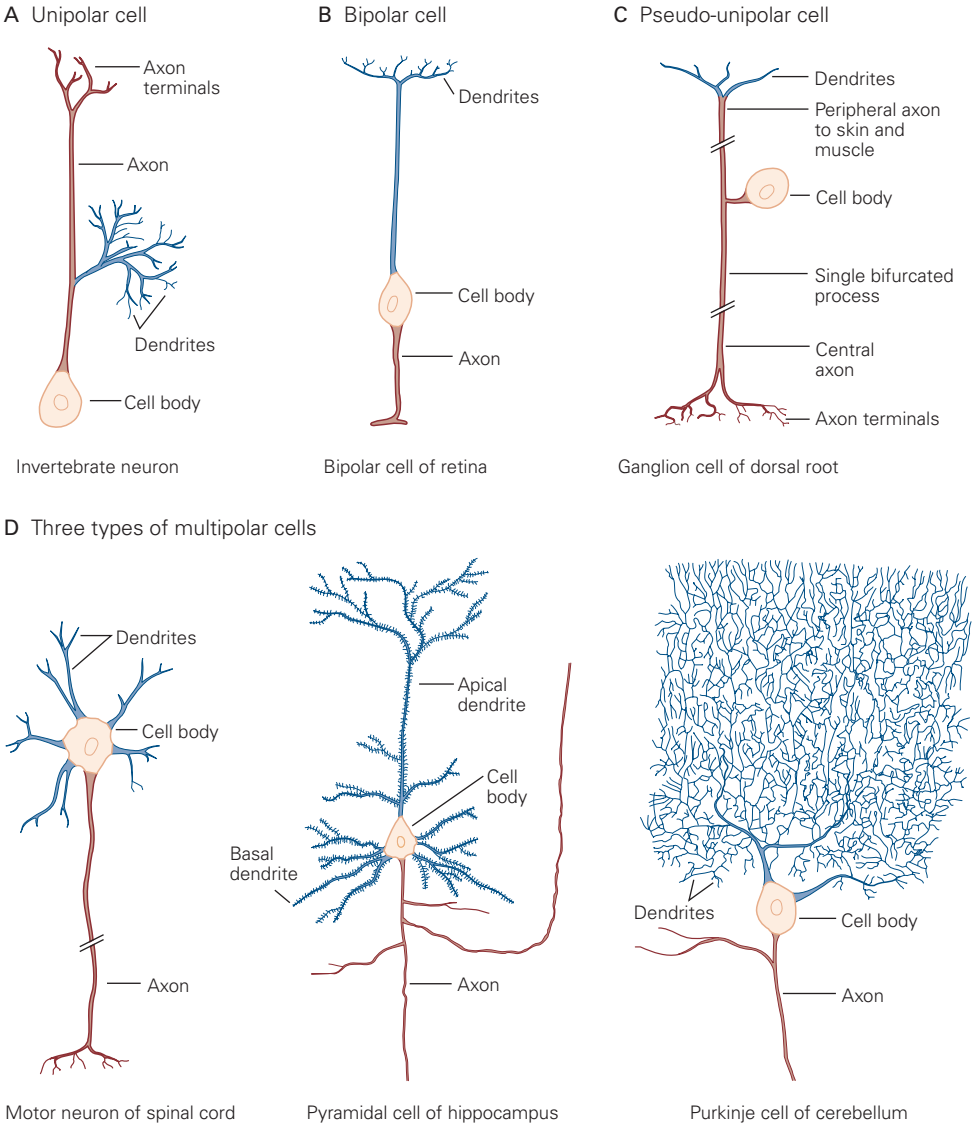

Figure 3–3 Neurons are classified as unipolar, bipolar, or multipolar according to the number of processes that originate from the cell body.

A. Unipolar cells have a single process emanating from the cell. Different segments serve as receptive surfaces or releasing terminals. Unipolar cells are characteristic of the invertebrate nervous system.

B. Bipolar cells have two types of processes that are functionally specialized. The dendrite receives electrical signals and the axon transmits signals to other cells.

C. Pseudo-unipolar cells, which are variants of bipolar cells, carry somatosensory information to the spinal cord. During development, the two processes of the embryonic bipolar cell fuse and emerge from the cell body as a single process that has two functionally distinct segments. Both segments function as axons; one extends to peripheral skin or muscle, the other to the central spinal cord. (Adapted, with permission, from Ramón y Cajal 1933.)

D. Multipolar cells have a single axon and many dendrites. They are the most common type of neuron in the mammalian nervous system. Three examples illustrate the large diversity of these cells. Spinal motor neurons innervate skeletal muscle fibers. Pyramidal cells have a roughly triangular cell body; dendrites emerge from both the apex (the apical dendrite) and the base (the basal dendrites). Pyramidal cells are found in the hippocampus and throughout the cerebral cortex. Purkinje cells of the cerebellum are characterized by a rich and extensive dendritic tree that accommodates an enormous number of synaptic inputs. (Adapted, with permission, from Ramón y Cajal 1933.)

neurons that do not innervate muscle directly but that command action indirectly. A useful characterization of motor and sensory neurons alike is their temporal fidelity to matters outside the nervous system. Their activity keeps up with changes in external stimuli and dynamical forces exerted by the body musculature. Sensory neurons supply the brain with data, whereas motor neurons convert ideation into praxis. Together they compose our interface with the world.

Interneurons comprise the most numerous functional category and are subdivided into two classes: relay and local. Relay or projection interneurons have long axons and convey signals over considerable distances, from one brain region to another. Local interneurons have short axons because they form connections with nearby neurons in local circuits. Since almost every neuron can be regarded as an interneuron, the term is often used to distinguish between neurons that project to another neuron within a local circuit as opposed to neurons that project to a separate neural structure. The term is also sometimes used as shorthand for an inhibitory neuron, especially in studies of cortical circuits, but for clarity, the term *inhibitory interneuron* should be used when appropriate.

Each functional classification can be subdivided further. Sensory system interneurons can be classified according to the type of sensory stimuli to which they respond; these initial classifications can be broken down still further, according to location, density, and size as well as patterns of gene expression. Indeed, our view of neuronal complexity is rapidly evolving due to advances in mRNA sequence analysis that have enabled the molecular profiling of individual neurons. Such analyses have recently revealed a much greater heterogeneity of neuronal types than previously thought (Figure 3–4).

Glial Cells Support Nerve Cells

Glial cells greatly outnumber neurons—there are 2 to 10 times more glia than neurons in the vertebrate central nervous system. Although the name for these cells derives from the Greek for glue, glia do not commonly hold nerve cells together. Rather they surround the cell bodies, axons, and dendrites of neurons. Glia differ from neurons morphologically; they do not form dendrites and axons.

Glia also differ functionally. Although they arise from the same embryonic precursor cells, they do not have the same membrane properties as neurons and thus are not electrically excitable. Hence, they are not directly involved in electrical signaling, which is the function of nerve cells. Yet they play a role in allowing electrical signals to move quickly along the axons

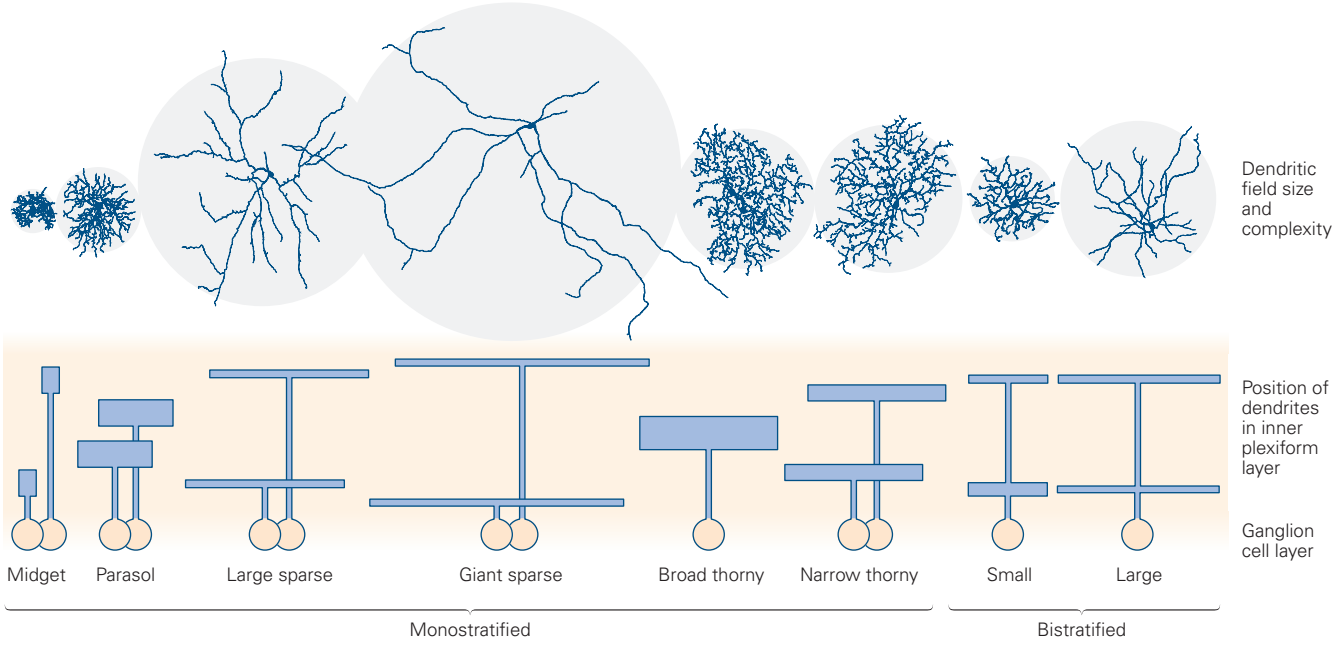

Figure 3–4 Sensory neurons can be subdivided into functionally distinct groups. For example, at least 13 types of retinal ganglion cells are distinguished based on the size and shape of their dendrites combined with the depth within the retina at which they receive their inputs. The inner plexiform layer contains the connections between interneurons of the retina (bipolar and amacrine cells) and the ganglion cells. (Reproduced, with permission, from Dacey et al. 2003. Copyright © 2003 Elsevier.)

of neurons, and they appear to play an important role in guiding connectivity during early development and stabilizing new or altered connections between neurons that occur through learning. Over the past decade, interest in the diverse functions of glia has accelerated, and their characterization has changed from support cells to functional partners of neurons (Chapter 7).

Each Nerve Cell Is Part of a Circuit That Mediates Specific Behaviors

Every behavior is mediated by specific sets of interconnected neurons, and every neuron's behavioral function is determined by its connections with other neurons. A simple behavior, the knee-jerk reflex, will illustrate this. The reflex is initiated when a transient imbalance of the body stretches the quadriceps extensor muscles of the leg. This stretching elicits sensory information that is conveyed to motor neurons, which in turn send commands to the extensor muscles to contract so that balance is restored.

This reflex is used clinically to test the integrity of the nerves as well as the cerebrospinal control of the reflex amplitude (or gain). The underlying mechanism is important because it maintains normal tone in the quadriceps and prevents our knees from buckling when we stand or walk. The tendon of the quadriceps femoris, an extensor muscle that moves the lower leg, is attached to the tibia through the tendon of the patella (kneecap). Tapping this tendon just below the patella stretches the quadriceps femoris. This stretch initiates reflex contraction of the quadriceps muscle to produce the familiar knee jerk. By increasing the tension of a select group of muscles, the stretch reflex changes the position of the leg, suddenly extending it outward (Figure 3–5).

The cell bodies of the sensory neurons involved in the knee-jerk reflex are clustered near the spinal cord in the dorsal root ganglia. They are pseudo-unipolar cells; one branch of each cell's axon runs to the quadriceps muscle at the periphery, while the other runs centrally into the spinal cord. The branch that innervates the quadriceps makes contact with stretch-sensitive receptors (muscle spindles) and is excited when the muscle is stretched. The branch reaching the spinal cord forms excitatory connections with the motor neurons that innervate the quadriceps and control its contraction. This branch also contacts local interneurons that *inhibit* the motor neurons controlling the opposing flexor muscles (Figure 3–5). Although these local interneurons are not involved in producing the stretch reflex itself, they increase the stability of the reflex by coordinating the actions of opposing muscle groups.

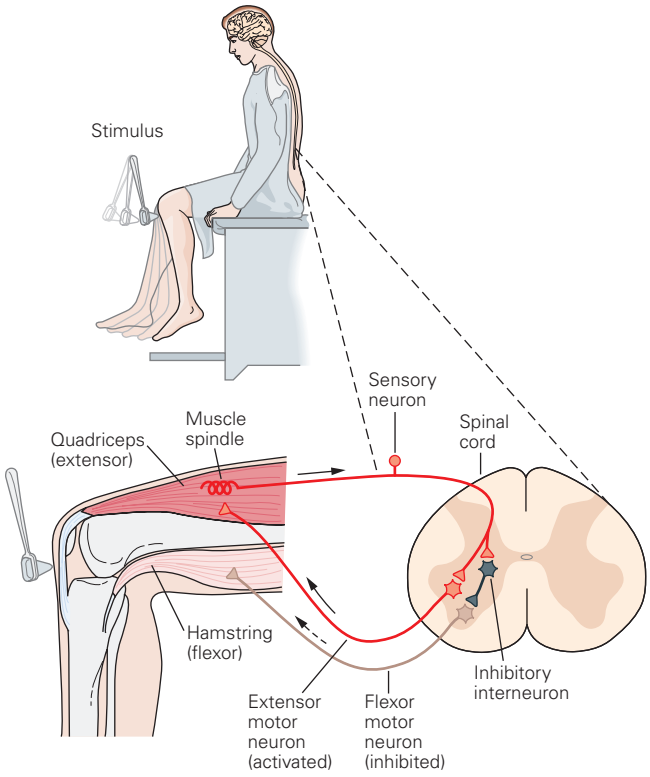

Figure 3–5 The knee-jerk reflex is controlled by a simple circuit of sensory and motor neurons. Tapping the kneecap with a reflex hammer pulls on the tendon of the quadriceps femoris, a muscle that extends the lower leg. When the muscle stretches in response to the pull of the tendon, information regarding this change in the muscle is conveyed to the central nervous system by sensory neurons. In the spinal cord, the sensory neurons form excitatory synapses with extensor motor neurons that contract the quadriceps, the muscle that was stretched. The sensory neurons act indirectly, through interneurons, to inhibit flexor motor neurons that would otherwise contract the opposing hamstring muscles. These actions combine to produce the reflex behavior. In the drawing, each extensor and flexor motor neuron represents a population of many cells.

Thus, the electrical signals that produce the stretch reflex carry four kinds of information:

1. Sensory information is conveyed to the central nervous system (the spinal cord) from muscle.
2. Motor commands from the central nervous system are issued to the muscles that carry out the knee jerk.
3. Inhibitory commands are issued to motor neurons that innervate opposing muscles.
4. Information about local neuronal activity related to the knee jerk is sent to higher centers of the central nervous system, permitting the brain to coordinate different behaviors simultaneously or in series.

In addition, the brain asserts context-dependent control of the reflex to adjust its gain. For example, when we run, the hamstring muscles flex the knee, thereby stretching the quadriceps. The brain and spinal cord suppress the stretch reflex to allow the quadriceps to relax. When these descending pathways are disrupted, as in some strokes, the reflex is exaggerated and the joint has stiffness.

The stretching of just one muscle, the quadriceps, activates several hundred sensory neurons, each of which makes direct contact with 45 to 50 motor neurons. This pattern of connection, in which one neuron activates many target cells, is called *divergence* (Figure 3–6A). It is especially common in the input stages of the nervous system; by distributing its signals to many target cells, a single neuron can exert wide and diverse influence. Conversely, a single motor cell in the knee-jerk circuit receives 200 to 450 input contacts from approximately 130 sensory cells. This pattern of connection is called *convergence* (Figure 3–6B). It is common at the output stages of the nervous system; a target motor cell that receives information from many sensory neurons is able to integrate information from many sources. Each sensory neuron input produces relatively weak excitation, so convergence also ensures that a motor neuron is activated only when a sufficient number of sensory neurons are activated together.

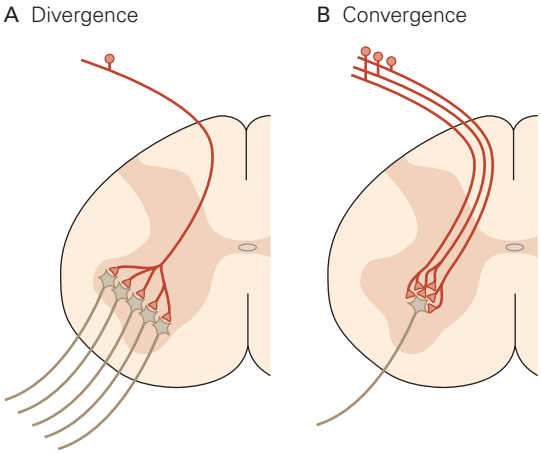

Figure 3–6 Diverging and converging neuronal connections are a key organizational feature of the brain.

A. In the sensory systems, each receptor neuron usually contacts several neurons that represent the second stage of processing. At subsequent processing stages, the incoming connections diverge even more. This allows sensory information from a single site to be distributed more widely in the spinal cord and brain.

B. By contrast, motor neurons are the targets of progressively converging connections. With this arrangement, input from many presynaptic cells is required to activate the motor neuron.

A stretch reflex such as the knee-jerk reflex is a simple behavior produced by two classes of neurons connecting at excitatory synapses. But not all important signals in the brain are excitatory. Many neurons produce inhibitory signals that reduce the likelihood of firing. Even in the simple knee-jerk reflex, the sensory neurons make both excitatory and inhibitory connections. Excitatory connections in the leg's extensor muscles cause these muscles to contract, whereas connections with inhibitory interneurons prevent the antagonist flexor muscles from contracting. This feature of the circuit is an example of *feedforward inhibition* (Figure 3–7A). In the knee-jerk reflex, feedforward

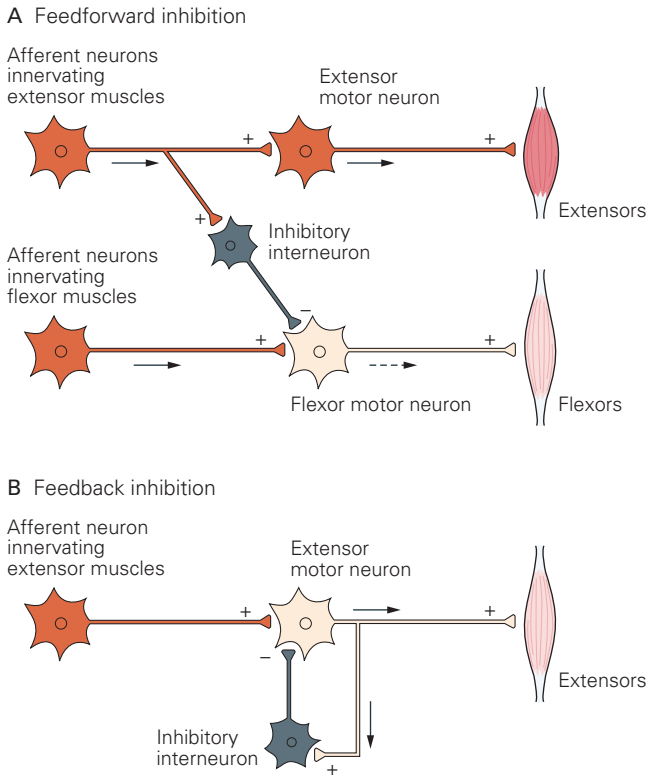

Figure 3–7 Inhibitory interneurons can produce either feedforward or feedback inhibition.

A. Feedforward inhibition enhances the effect of the active pathway by suppressing the activity of pathways mediating opposing actions. Feedforward inhibition is common in monosynaptic reflex systems. For example, in the knee-jerk reflex circuit (**Figure 3–5**) afferent neurons from extensor muscles excite not only the extensor motor neurons but also inhibitory interneurons that prevent the firing of the motor cells innervating the opposing flexor muscles.

B. Feedback inhibition is a self-regulating mechanism. Here extensor motor neurons act on inhibitory interneurons that in turn act on the extensor motor neurons themselves and thus reduce their probability of firing. The effect is to dampen activity within the stimulated pathway and prevent it from exceeding a certain critical level.

inhibition is *reciprocal*, ensuring that the flexor and extensor pathways always inhibit each other so that only muscles appropriate for the movement and not those opposed to it are recruited.

Some circuits provide *feedback inhibition*. For example, a motor neuron may have excitatory connections with both a muscle and an inhibitory interneuron that itself forms a connection with the motor neuron. When the inhibitory interneuron is excited by the motor neuron, the interneuron is able to limit the ability of the motor neuron to excite the muscle (Figure 3–7B). We will encounter many examples of feedforward and feedback inhibition when we examine more complex behaviors in later chapters.

Signaling Is Organized in the Same Way in All Nerve Cells

To produce a behavior, a stretch reflex for example, each participating sensory and motor nerve cell must generate four different signals in sequence, each at a different site within the cell. Despite variations in cell size and shape, transmitter biochemistry, or behavioral function, almost all neurons can be described by a model neuron that has four functional components that generate the four types of signals: a receptive component for producing graded input signals, a summing or integrative component that produces a trigger signal, a conducting long-range signaling component that produces all-or-none conducting signals, and a synaptic component that produces output signals to the next neuron in line or to muscle or gland cells (Figure 3–8).

The different types of signals generated in a neuron are determined in part by the electrical properties of the cell membrane. Every cell, including a neuron, maintains a certain difference in the electrical potential on either side of the plasma membrane when the cell is at rest. This is called the *resting membrane potential*. In a typical resting neuron, the voltage of the inside of the cell is about 65 mV more negative than the voltage outside the cell. Because the voltage outside the membrane is defined as zero, we say the resting membrane potential is –65 mV. The resting potential in different nerve cells ranges from –40 to –80 mV; in muscle cells, it is greater still, about –90 mV. As described in detail in Chapter 9, the resting membrane potential results from two factors: the unequal distribution of electrically charged ions, in particular the positively charged Na^+ and K^+ ions, and the selective permeability of the membrane.

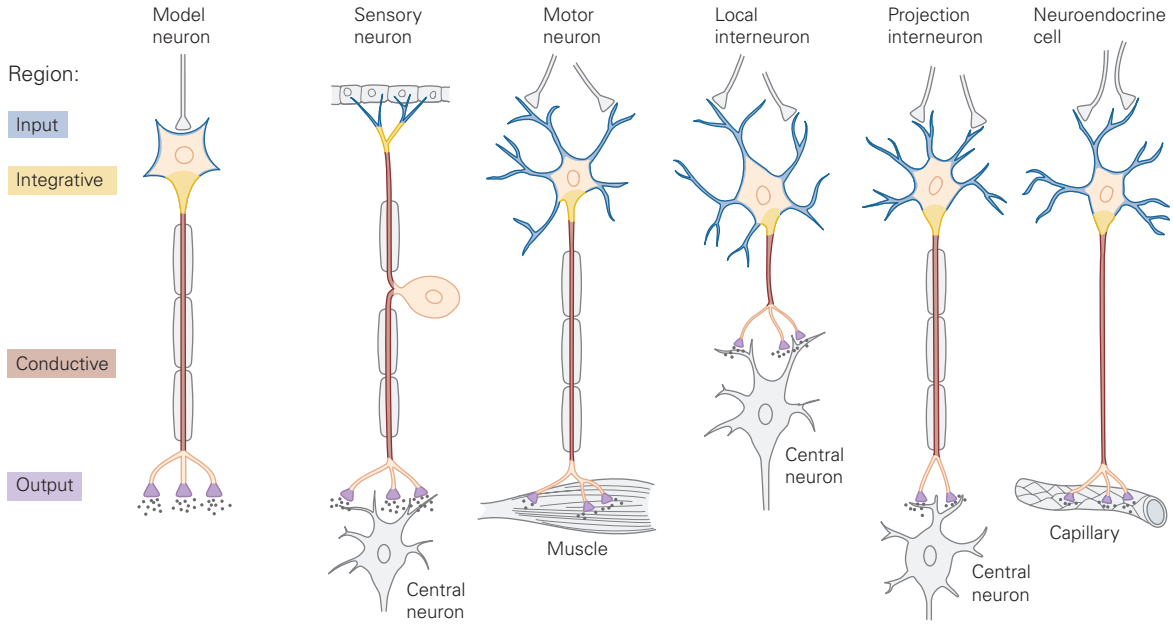

Figure 3–8 **Most neurons have four functional regions in which different types of signals are generated.** Thus, the functional organization of most neurons, regardless of type, can be represented schematically by a model neuron. This model neuron is the physiological expression of Ramón y Cajal's principle of dynamic polarization. The input, integrative, and conductive signals are all electrical and integral to the cell, whereas the output signal is a chemical substance ejected by the cell into the synaptic cleft. Not all neurons share all of these features; for example, some local interneurons lack a conductive component.

The unequal distribution of positively charged ions on either side of the cell membrane is maintained by two main mechanisms. Intracellular Na^+ and K^+ concentrations are largely controlled by a membrane protein that actively pumps Na^+ out of the cell and K^+ back into it. This *Na^+-K^+ pump*, about which we shall learn more in Chapter 9, keeps the Na^+ concentration in the cell low (about one-tenth the concentration outside the cell) and the K^+ concentration high (about 20 times the concentration outside). The extracellular concentrations of Na^+ and K^+ are maintained by the kidneys and the astroglial cells, also known as astrocytes.

The otherwise impermeable cell membrane contains proteins that form pores called *ion channels*. The channels that are active when the cell is at rest are highly permeable to K^+ but considerably less permeable to Na^+. The K^+ ions tend to leak out of these open channels, down the ion's concentration gradient. As K^+ ions exit the cell, they leave behind a cloud of unneutralized negative charge on the inner surface of the membrane, so that the net charge inside the membrane is more negative than that outside. With this state of affairs, the membrane potential is typically maintained at around –65 mV relative to outside of the neuron, and the neuron is said to be at rest.

The resting state is perturbed when the cell begins to take up Na^+ (or Ca^{2+}), which are at a higher concentration outside the cell. The inward movement of these positively charged ions (*inward current*) partially neutralizes the negative voltage inside the cell. We will say more about these events below. What happens next, however, holds the key to understanding what it is about neurons that makes signaling suitable for conveying information.

A cell, such as nerve and muscle, is said to be excitable when its membrane potential can be quickly and significantly altered. In many neurons, a 10-mV change in membrane potential (from –65 to –55 mV) makes the membrane much more permeable to Na^+ than to K^+. The resultant influx of Na^+ further neutralizes the negative charge inside the cell, leading to even more permeability to Na^+. The result is a brief and explosive change in membrane potential to +40 mV, the *action potential*. This potential is actively conducted down the cell's axon to the axon's terminal, where it initiates an elaborate chemical interaction with postsynaptic neurons or muscle cells. Since the action potential is actively propagated, its amplitude does not diminish by the time it reaches the axon terminal. An action potential typically lasts approximately 1 ms, after which the membrane returns to its resting state, with its normal separation of charges and higher permeability to K^+ than to Na^+.

The mechanisms underlying the resting potential and action potential are discussed in detail in Chapters 9 and 10. In addition to the long-distance signals represented by the action potential, nerve cells also produce local signals—receptor potentials and synaptic potentials—that are not actively propagated and that typically decay within just a few millimeters (see next section).

Changes in membrane potential that generate long-range and local signals can be either a decrease or an increase from the resting potential. That is, the resting membrane potential is the baseline from which all signaling occurs. A reduction in membrane potential, called *depolarization*, enhances a cell's ability to generate an action potential and is thus excitatory. In contrast, an increase in membrane potential, called *hyperpolarization*, makes a cell less likely to generate an action potential and is therefore inhibitory.

The Input Component Produces Graded Local Signals

In most neurons at rest, no current flows from one part of the cell to another, so the resting potential is the same throughout. In sensory neurons, current flow is typically initiated by a physical stimulus, which activates specialized receptor proteins at the neuron's receptive surface. In our example of the knee-jerk reflex, stretching of the muscle activates specific ion channels that open in response to stretch of the sensory neuron membrane, as we shall learn in Chapter 18. The opening of these channels when the cell is stretched permits the rapid influx of Na^+ ions into the sensory cell. This ionic current changes the membrane potential, producing a local signal called the *receptor potential*.

The amplitude and duration of a receptor potential depend on the intensity of the muscle stretch: The larger or longer-lasting the stretch, the larger or longer-lasting is the resulting receptor potential (Figure 3–9A). That is, receptor potentials are graded, unlike the all-or-none action potential. Most receptor potentials are depolarizing (excitatory); hyperpolarizing (inhibitory) receptor potentials are found in the retina.

The receptor potential is the first representation of stretch to be coded in the nervous system. However, because this depolarization spreads passively from the stretch receptor, it does not travel far. The distance is longer if the diameter of the axon is bigger, shorter if the diameter is smaller. Also, the distance is shorter if current can pass easily through the membrane, and longer if the membrane is insulated by myelin. The receptor potential from the stretch receptor therefore travels only 1 to 2 mm. In fact, just 1 mm away, the

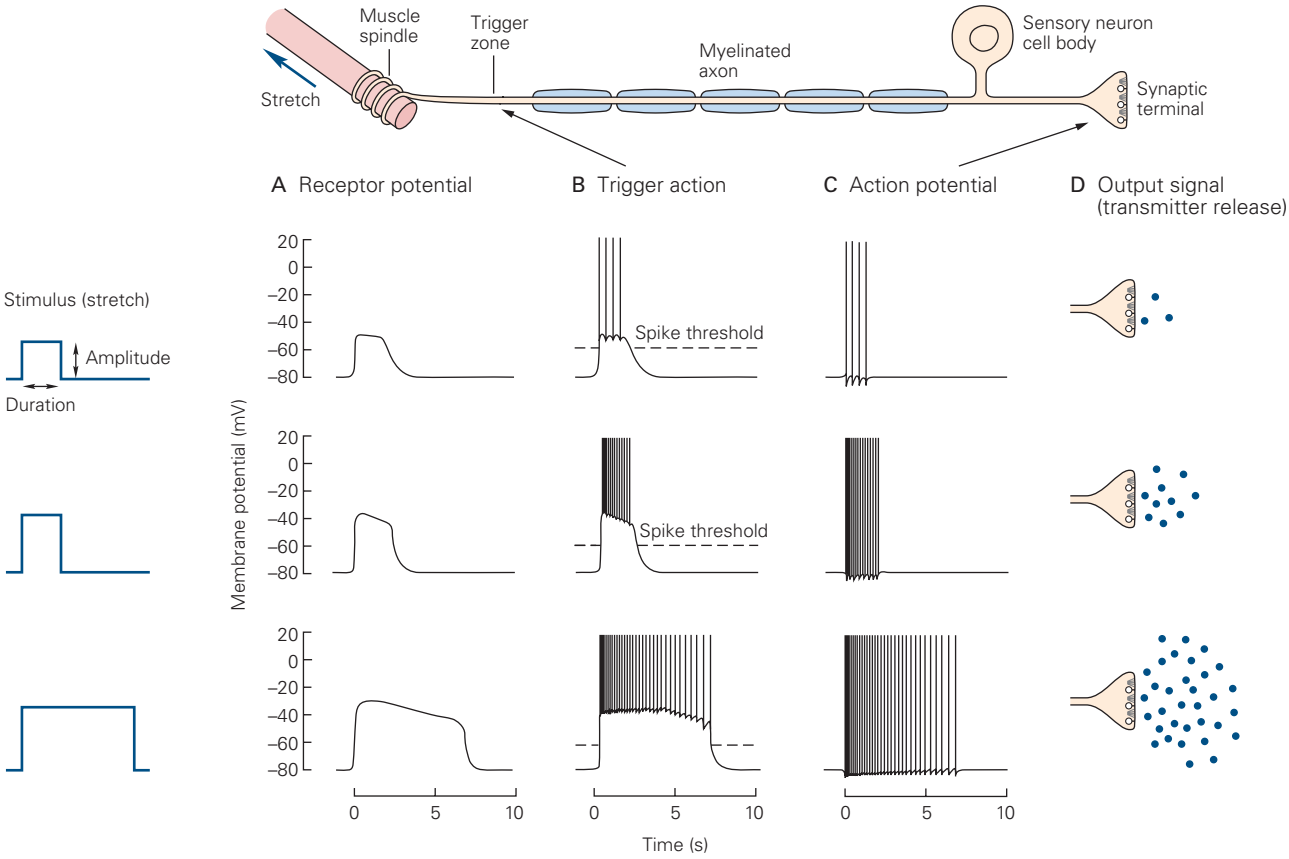

Figure 3–9 Each of the neuron's four signaling components produces a characteristic signal. The figure shows a sensory neuron activated by stretching of a muscle, which the neuron senses through a specialized receptor, the muscle spindle.

A. The input signal, called a receptor potential, is graded in amplitude and duration, proportional to the amplitude and duration of the stimulus.

B. The trigger zone sums the depolarization generated by the receptor potential. An action potential is generated only if the receptor potential exceeds a certain voltage threshold. Once this threshold is surpassed, any further increase in amplitude of the receptor potential can only increase the frequency with which the action potentials are generated, because action potentials have a constant amplitude. The duration of the receptor potential determines the duration of the train of action potentials. Thus, the graded amplitude and duration of the receptor potential are translated into a frequency code in the action potentials generated at the trigger zone. All action potentials produced are propagated faithfully along the axon.

C. Action potentials are all-or-none. Because all action potentials have a similar amplitude and duration, the frequency and duration of firing encodes the information carried by the signal.

D. When the action potential reaches the synaptic terminal, it initiates the release of a neurotransmitter, the chemical substance that serves as the output signal. The frequency of action potentials in the presynaptic cell determines how much neurotransmitter is released by the cell.

amplitude of the signal is only about one-third what it was at the site of generation. To be carried successfully to the spinal cord, the local signal must be amplified—it must generate an action potential. In the knee-jerk reflex, if the receptor potential in the sensory neuron reaches the first node of Ranvier in the axon and is large enough, it will trigger an action potential (Figure 3–9B), which then propagates without failure to the axon terminals in the spinal cord (Figure 3–9C). At the synapse between the sensory neuron and a motor neuron, the action potential produces a chain of events that results in an input signal to the motor neuron.

In the knee-jerk reflex, the action potential in the presynaptic terminal of the sensory neuron initiates the release of a chemical substance, or neurotransmitter, into the synaptic cleft (Figure 3–9D). After diffusing across the cleft, the transmitter binds to receptor proteins in the postsynaptic membrane of the motor neuron, thereby directly or indirectly opening ion channels. The ensuing flow of current briefly alters the membrane potential of the motor cell, a change called the *synaptic potential*.

Like the receptor potential, the synaptic potential is graded; its amplitude depends on how much transmitter is released. In the same cell, the synaptic

Table 3–1 Comparison of Local (Passive) and Propagated Signals

Signal type	Amplitude (mV)	Duration	Summation	Effect of signal	Type of propagation
Local (passive) signals					
Receptor potentials	Small (0.1–10)	Brief (5–100 ms)	Graded	Hyperpolarizing or depolarizing	Passive
Synaptic potentials	Small (0.1–10)	Brief to long (5 ms–20 min)	Graded	Hyperpolarizing or depolarizing	Passive
Propagated (active) signals					
Action potentials	Large (70–110)	Brief (1–10 ms)	All-or-none	Depolarizing	Active

potential can be either depolarizing or hyperpolarizing depending on the type of receptor molecule that is activated. Synaptic potentials, like receptor potentials, spread passively. Thus, the change in potential will remain local unless the signal reaches beyond the axon's initial segment where it can give rise to an action potential. Some dendrites are not entirely passive but contain specializations that boost the synaptic potential, thereby increasing its efficacy to produce an action potential (Chapter 13). The features of receptor and synaptic potentials are summarized in Table 3–1.

The Trigger Zone Makes the Decision to Generate an Action Potential

Sherrington first pointed out that the function of the nervous system is to weigh the consequences of different types of information and then decide on appropriate responses. This *integrative* function of the nervous system is clearly seen in events at the trigger zone of the neuron, the initial segment of the axon.

Action potentials are generated by a sudden influx of Na^+ through channels in the cell membrane that open and close in response to changes in membrane potential. When an input signal (a receptor potential or synaptic potential) depolarizes an area of membrane, the local change in membrane potential opens local Na^+ channels that allow Na^+ to flow down its concentration gradient, from outside the cell where the Na^+ concentration is high to inside where it is low.

Because the initial segment of the axon has the highest density of voltage-sensitive Na^+ channels and therefore the lowest threshold for generating an action potential, an input signal spreading passively along the cell membrane is more likely to give rise to an action potential at the initial segment of the axon than at other sites in the cell. This part of the axon is therefore known as the *trigger zone*. It is here that the activity of all receptor (or synaptic) potentials is summed and where, if the sum of the input signals reaches threshold, the neuron generates an action potential.

The Conductive Component Propagates an All-or-None Action Potential

The action potential is all-or-none: Stimuli below the threshold do not produce a signal, but stimuli above the threshold all produce signals of the same amplitude. Regardless of variation in intensity or duration of stimuli, the amplitude and duration of each action potential are pretty much the same, and this holds for each regenerated action potential at a node of Ranvier along a myelinated axon. In addition, unlike receptor and synaptic potentials, which spread passively and decrease in amplitude, the action potential, as we have seen, does not decay as it travels along the axon to its target—a distance that can be as great as 1 m—because it is periodically regenerated. This conducted signal can travel at rates as fast as 100 m/s. Indeed, the remarkable feature of action potentials is that they are highly stereotyped, varying only subtly (but in some cases importantly) from one nerve cell to another. This feature was demonstrated in the 1920s by Edgar Adrian, one of the first to study the nervous system at the cellular level. Adrian found that all action potentials have a similar shape or waveform (see Figure 3–2). The action potentials carried into the nervous system by a sensory axon often are indistinguishable from those carried out of the nervous system to the muscles by a motor axon.

Only two features of the conducting signal convey information: the number of action potentials and the time intervals between them (Figure 3–9C). As Adrian put it in 1928, summarizing his work on sensory fibers: "all impulses are very much alike, whether the message is destined to arouse the sensation of light, of touch, or of pain; if they are crowded together the sensation is intense, if they are separated by long intervals the sensation is correspondingly feeble." Thus, what determines

the intensity of sensation or speed of movement is the frequency of the action potentials. Likewise, the duration of a sensation or movement is determined by the period over which action potentials are generated.

In addition to the frequency of the action potentials, the pattern of action potentials also conveys important information. For example, some neurons are spontaneously active in the absence of stimulation. Some spontaneously active nerve cells (beating neurons) fire action potentials regularly; others (bursting neurons) fire in brief bursts of action potentials. These diverse cells respond differently to the same excitatory synaptic input. An excitatory synaptic potential may initiate one or more action potentials in a cell that is not spontaneously active, whereas that same input to spontaneously active cells will simply increase the existing rate of firing.

An even more dramatic difference is seen when the input signal is inhibitory. Inhibitory inputs have little information value in a silent cell. By contrast, in spontaneously active cells, inhibition can have a powerful *sculpting* role. By establishing periods of silence in otherwise ongoing activity, inhibition can produce a complex pattern of alternating firing and silence where none existed. Such subtle differences in firing patterns may have important functional consequences for the information transfer between neurons. Mathematical modelers of neuronal networks have attempted to delineate neural codes in which information is also carried by the fine-grained pattern of firing—the exact timing of each action potential.

If signals are stereotyped and reflect only the most elementary properties of the stimulus, how can they carry the rich variety of information needed for complex behavior? How is a message that carries visual information about a bee distinguished from one that carries pain information about the bee's sting, and how are these sensory signals distinguished from motor signals for voluntary movement? The answer is simple and yet is one of the most important organizational principles of the nervous system: Interconnected neurons form anatomically and functionally distinct pathways—labeled lines—and it is these pathways of connected neurons, these labeled lines, not individual neurons, that convey information. The neural pathways activated by receptor cells in the retina that respond to light are completely distinct from the pathways activated by sensory cells in the skin that respond to touch.

The Output Component Releases Neurotransmitter

When an action potential reaches a neuron's terminal, it stimulates the release of chemical substances from the cell. These substances, called *neurotransmitters*, can be small organic molecules, such as L-glutamate and acetylcholine, or peptides like substance P or LHRH (luteinizing hormone–releasing hormone).

Neurotransmitter molecules are held in subcellular organelles called *synaptic vesicles*, which accumulate in the terminals of the axon at specialized release sites called *active zones*. To eject their transmitter substance into the synaptic cleft, the vesicles move up to and fuse with the neuron's plasma membrane, then burst open to release the transmitter into the synaptic cleft (the extracellular space between the pre- and postsynaptic cell) by a process known as *exocytosis*. The molecular machinery of neurotransmitter release is described in Chapters 14 and 15.

The released neurotransmitter molecules are the neuron's output signal. The output signal is thus graded according to the amount of transmitter released, which is determined by the number and frequency of the action potentials that reach the presynaptic terminals (Figure 3–9C,D). After release, the transmitter molecules diffuse across the synaptic cleft and bind to receptors on the postsynaptic neuron. This binding causes the postsynaptic cell to generate a synaptic potential. Whether the synaptic potential has an excitatory or inhibitory effect depends on the type of receptor in the postsynaptic cell, not on the particular chemical neurotransmitter. The same transmitter substance can have different effects at different receptors.

The Transformation of the Neural Signal From Sensory to Motor Is Illustrated by the Stretch-Reflex Pathway

As we have seen, the properties of a signal are transformed as the signal moves from one component of a neuron to another or between neurons. In the stretch reflex, when a muscle is stretched, the amplitude and duration of the stimulus are reflected in the amplitude and duration of the receptor potential generated in the sensory neuron (Figure 3–10A). If the receptor potential exceeds the threshold for an action potential in that cell, the graded signal is transformed at the trigger zone into an action potential. Although individual action potentials are all-or-none signals, the more the receptor potential exceeds threshold, the greater the depolarization and consequently the greater the frequency of action potentials in the axon. The duration of the input signal also determines the duration of the train of action potentials.

The information encoded by the frequency and duration of firing is faithfully conveyed along the axon to its terminals, where the firing of action potentials determines the amount of transmitter released.