Cutaneous Sensory Systems 4.3

Learning Objectives

- List examples of exteroreceptors and interoreceptors
- Define adequate stimulus, modality, and perception
- Distinguish between long receptors and short receptors
- Describe how the nervous system codes quality of sensation and intensity of sensation
- Define receptive field
- Describe adaptation
- List the five mechanoreceptors in the skin and distinguish them on the basis of their frequency response and rate of adaptation
- Describe hot and cold sensors
- Distinguish between first and second pain
- Draw the dorsal column pathway for most mechanoreceptors
- Explain somatotropic representation and how it originates
- Define dermatome, myotome, and sclerotome
- Describe the parts of the body surface projecting to the nucleus gracilis and nucleus cuneatus
- Draw the anterolateral tract for pain and temperature
- Describe the gate control theory of pain and explain how rubbing the body surface reduces the subjective experience of pain
- Indicate the somatosensory cortex in the brain and its somatotopic representation
- Describe the typical receptive field of somatosensory cortical neurons
- Explain how lateral inhibition sharpens spatial discrimination

SENSORS PROVIDE A WINDOW ONTO OUR WORLD

Sensory systems are the link between the central nervous system (CNS) and events that occur outside of it. They inform the CNS of what is happening in both the external world and the internal world. Sensors that convey information about the external environment are called **exteroreceptors**; sensors reporting on the internal environment are **interoreceptors**.

EXTERORECEPTORS INCLUDE THE FIVE CLASSICAL SENSES AND THE CUTANEOUS SENSES

The exteroreceptors include all those sensory systems that apprise the CNS of conditions in the external environment. These include the following:

- Eyes (vision)
- Cochlea (hearing)
- Vestibular apparatus (balance, rotation and linear acceleration, gravity)
- Olfactory epithelium (smell)
- Taste buds (taste)
- Touch receptors (touch)
- Temperature sensors
- Pressure receptors
- Nociceptors (pain)
- Skin stretch receptors.

INTERORECEPTORS REPORT ON THE CHEMICAL AND PHYSICAL STATE OF THE INTERIOR OF THE BODY

The interoreceptors also include a wide variety of receptors. They report on a variety of variables ranging from the stretch of skeletal muscles to the pH of the blood. They include the following:

- Stretch receptors (arteries, veins, atria, intestines, bladder, etc.)
- Chemosensors (CO_2, O_2, glucose)
- pH sensors (blood, intestinal lumen)
- Nociceptors (damage, pain)
- Muscle length sensors
- Muscle tension sensors
- Proprioceptors
- Temperature sensors
- Osmoreceptors.

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SENSORY SYSTEMS CONSIST OF THE SENSE ORGAN, THE SENSORY RECEPTORS, AND THE PATHWAYS TO THE CNS

Sensory systems include **receptors** that respond to what is called an **adequate stimulus**, which is the *kind* of stimulus to which receptors respond preferentially. A **sensory modality** is an identifiable class of sensation. Sensory receptors respond to the adequate stimulus with the lowest **threshold**, referring to the lowest stimulus intensity that elicits a response from the sensor. For example, vision is a sensory modality whose adequate stimulus is light within the narrow band of wavelengths that we can see. The receptors for vision are the rods and cones in the retina that actually respond to light. However, rods and cones will also respond to pressure on the eyeballs, causing us to "see" light, called **phosphenes**. The proper sensing of light requires not just the rods and cones but all of the accessory structures that enables us to see. Table 4.3.1 shows the various modalities, their receptor cells, and their sense organs.

PERCEPTION REFERS TO OUR AWARENESS OF A STIMULUS

Sensory systems bring information about the external or internal world into the CNS where it is processed to bring about an awareness or consciousness of the excitation. This awareness is called **perception**, and the process by which we become aware is called **sensation**. Both of these are distinct from what receptor cells do, which is to **transduce** an adequate stimulus into action potentials.

LONG AND SHORT RECEPTORS DIFFER IN THEIR PRODUCTION OF ACTION POTENTIALS

The body has two basic plans for sensory **transduction**: the long and short receptors as shown in Figure 4.3.1. **Long receptors** link sensory energy to action potentials. Those in skin and muscle have nerve endings at the periphery and their cell bodies lie in the dorsal root of the sensory nerve (see Figure 4.3.5). The single receptor cell transduces an adequate stimulus, such as mechanical deformation, into a **receptor potential**. The receptor potentials are **graded** responses whose intensity depends on the intensity of the stimulus. They are conducted electrotonically and so die out with distance away from the stimulus as well as with time after the stimulus. Such graded responses would die out before they could reach the end of the cell's long processes. Therefore, the sensory cell converts these receptor potentials into action potentials which can be conducted along the length of the receptor without decrement. The next cell to receive this information is a **second-order neuron**, located some distance away, in the spinal cord.

Short receptor cells have a short distance between the detection of the adequate stimulus and its transmission to the next cell, which is typically a **primary afferent neuron**. Here the sensory cells produce a receptor potential in response to the adequate stimulus. This receptor potential is linked to the transmission of excitation through the use of neurotransmitters, usually at the opposite end of the sensory cell. Fusion of neurotransmitters at the opposite end of the sensory cell requires a receptor potential but does not require an action potential. The released neurotransmitter then causes a **generator potential** in the primary afferent neuron. When the

TABLE 4.3.1 Sensory Modalities, Their Receptors, and Their Organs

Sensory Modality	Receptor Cells	Sense Organ
Vision	Rods or cones	Eye
Hearing	Hair cells	Ear (organ of Corti)
Rotational acceleration	Hair cells	Ear (semicircular canal)
Linear acceleration	Hair cells	Ear (utricle and saccule)
Smell	Olfactory neurons	Olfactory membrane
Taste	Taste receptor cells	Taste buds
Touch/pressure	Nerve endings	Skin
Heat	Nerve endings	Skin
Cold	Nerve endings	Skin
Pain	Nerve endings	Skin
Proprioception	Nerve endings	Joints, capsules, muscles
Muscle length	Nerve endings	Muscle spindles
Muscle tension	Nerve endings	Golgi tendon organ

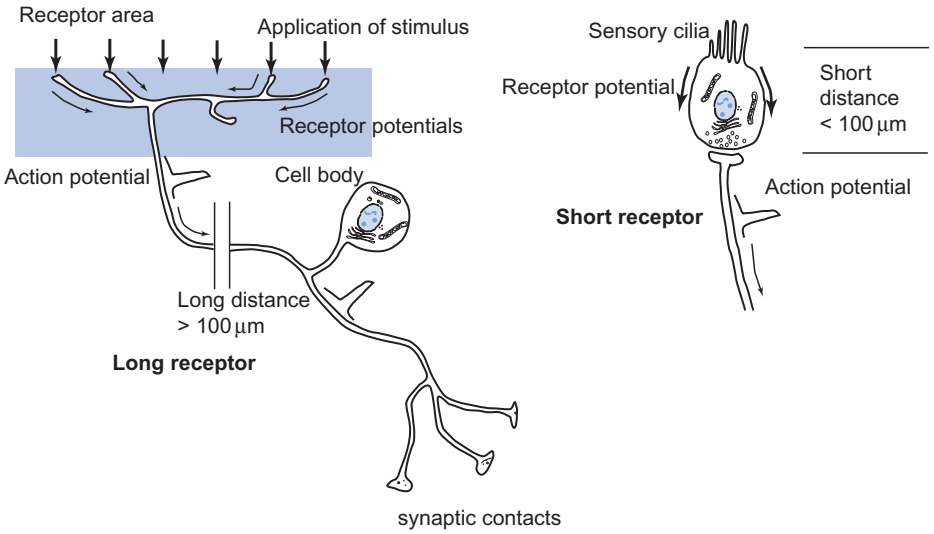

FIGURE 4.3.1 Long and short receptors. Long receptors initiate a receptor potential near the stimulus. If sufficiently strong, the receptor potential causes the cell to fire an action potential, which travels over a long fiber to reach a second-order neuron. Short receptors initiate a graded receptor potential in response to stimulation that causes release of neurotransmitters without an action potential. The graded release causes a graded generator potential which, if sufficiently strong, initiates an action potential in the sensory neuron.

generator potential reaches threshold, the primary afferent neuron fires an action potential.

ANATOMICAL CONNECTION DETERMINES THE QUALITY OF A SENSORY STIMULUS

How do we know that a stimulus is visual, or tactile, or pressure, or pain, or whatever modality it is? Each sensory modality conveys sensory information to the brain over distinct neural pathways with distinct connections in the brain. This method of encoding sensory information is called the **labeled line** of stimulus coding. Each sensory modality has its own pathway and its own destinations within the CNS, so action potentials carried along these lines have the "label" of that modality. Some people have the bizarre ability to experience one modality as another. This capacity is called **synesthesia**, which literally means "feeling together". About 1 person in 25,000 has some form of synesthesia. The most common form is colored hearing. People with this synesthesia say that they "see" music and speech in color. Curiously, the reverse is not true: they do not hear sounds when they see something. Why this happens is unknown, but it has some genetic component. Perhaps some auditory input lost its way in development and innervated the visual cortex. Other types of synesthesia also exist. Individuals have reported experiencing words that "taste"; others report experiencing shapes with different tastes.

THE INTENSITY OF SENSORY STIMULI IS ENCODED BY THE *FREQUENCY* OF SENSORY RECEPTOR FIRING AND THE *POPULATION* OF ACTIVE RECEPTORS

The nervous system encodes the quality of the sensory modality largely through labeled lines, but the intensity is encoded by the population of cells that respond and by the frequency of their response. Intense stimuli cause larger or longer lasting receptor potentials, which are converted into a train of action potentials on the sensory neuron. Thus, the first method of encoding the stimulus strength is through the frequency of action potentials on the sensory neuron. Repetitive firing of sensory neurons excites CNS interneurons by temporal summation. This is called **frequency coding**. Intense stimuli also excite more sensory neurons than do less intense stimuli. Thus, more intense stimulation recruits additional sensory input. The inputs of these sensory neurons travel to overlapping sets of interneurons in the CNS, so that these interneurons are excited by spatial summation. Thus, intensity can also be coded by the number of sensory cells that participate. This is **population coding**.

FREQUENCY CODING IS THE BASIS OF THE WEBER–FECHNER LAW OF PSYCHOLOGY

In 1846, Weber found that blindfolded subjects could discriminate between small increments of weights placed in their hands, but the sensitivity depended on how much weight was already there. As weight was added, a proportionately larger weight had to be added in order to discriminate between two weights. He found that the minimal detectable difference was about 3% of an object's weight. Fechner realized that this empirical observation implied a logarithmic relationship between stimulus and response.

Consider that we have two weights, W_1 and W_2, each of which gives rise to a sensation S_1 and S_2. We say that we can discriminate between the weights when we can detect their difference, $\Delta W = W_2 - W_1$; but what we really detect is the difference in their associated sensations, $\Delta S = S_2 - S_1$. Weber's observations suggest that the relationship between sensation and stimulus is approximately given as

$$\Delta S = k\frac{\Delta W}{W} \qquad [4.3.1]$$

Writing this in differential form and then integrating, we would expect the relationship between sensation and stimulus intensity to have the form

[4.3.2] $$S = k_1 \log W + k_2$$

The receptor potential is approximately related to the logarithm of stimulus intensity, and it appears that this receptor mechanism may be responsible for the Weber–Fechner psychological "law". However, the relationship fails at the extremes of ranges of sensory sensitivity. The exact relationship between stimulus intensity and sensation differs among the various modalities. A more general power law holds over a wider range of intensities and is given as

[4.3.3] $$\Psi = k\Phi^n$$

where Ψ is the psychological sensation, Φ is the stimulus intensity, and n is the exponent. The exponent n varies from 0.33 to 3.5 depending on the modality. This is called **Stevens' Power Law** and was first proposed in 1957. The logarithmic relationship between stimulus and response allows us to code a wide range of stimulus intensities. A three-fold change in frequency of firing on a logarithmic scale corresponds to a 1000-fold change in stimulus intensity.

ADAPTATION TO A STIMULUS ALLOWS SENSORY NEURONS TO SIGNAL POSITION, VELOCITY, AND ACCELERATION

Adaptation refers to the decrease in sensation that occurs upon continued stimulation. It results from processes within the receptors themselves, although central mechanisms can also play a part. The rate of adaptation depends on the sensory modality. Touch receptors adapt rapidly whereas sensors in the muscle spindle adapt slower and sensors for blood pressure do not adapt at all. Different rates of adaptation allow sensory systems to signal position, velocity, or acceleration as shown in Figure 4.3.2.

RECEPTIVE FIELDS REFER TO THE PHYSICAL AREAS AT WHICH A STIMULUS WILL EXCITE A RECEPTOR

For cutaneous sensory receptors, **receptive fields** are the areas of the body surface that, when stimulated, excite the sensory neuron. Stimulation within only a small area of the finger tips excites touch receptors there, and they have small receptive fields. Stimulation of a person's back over a much larger area excites cutaneous receptors there. Because of the small receptive fields on the finger tips, we can easily distinguish between two closely placed stimuli. But on the back we cannot distinguish between two closely placed stimuli. The ability to distinguish between two stimuli on the body surface is called **two-point discrimination**. The receptive field can be defined for the sensory neurons or for higher order interneurons that receive inputs from a number of sensory neurons or other interneurons. More integrative neurons involved in processing of sensory information have more complicated receptive fields.

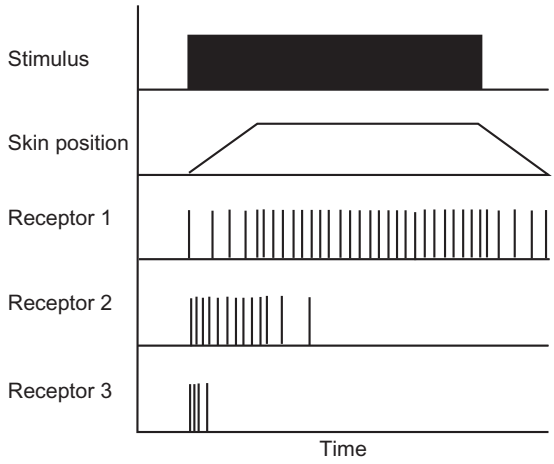

FIGURE 4.3.2 How adaptation can be used to signal position, velocity, or acceleration. Application of a square wave pressure pulse to the skin (top) results in a displacement of the skin (skin position, second from top). Sensors that are sensitive to position (stretch of the receptors) respond with a train of action potentials, the frequency of which is related to position. Lack of adaptation of these sensors allows their action potentials to signal skin position (Receptor 1). Rapidly adapting receptors respond only when there is movement (Receptor 2) and so they signal the velocity of skin movement. Still more rapidly adapting sensors (bottom, Receptor 3) inform the CNS of the acceleration of the skin.

CUTANEOUS RECEPTORS INCLUDE *MECHANORECEPTORS, THERMORECEPTORS,* AND *NOCICEPTORS*

THE SURFACE OF THE BODY CONTAINS A VARIETY OF MECHANORECEPTORS

The skin contains a variety of mechanoreceptors including Pacinian corpuscles, Meissner's corpuscles, Ruffini's corpuscles, Merkel's disks, and free nerve endings in the skin and surrounding hair follicles. All of these are long receptors, in which the receptor or generator potential is developed within the sensory cell and it fires action potentials based on this receptor potential without an additional connection to a sensory cell. Their axons are all myelinated, so that the CNS is informed quickly whenever any of these receptors detects a stimulus (see Figure 4.3.3).

The **Pacinian corpuscles** consist of a free nerve ending enclosed by a layered capsule, much like an onion. They lie in the subcutaneous layers of the skin, are rapidly adapting, and respond best to vibration. This response is due to the mechanical properties of the capsule, as the frequency response is removed when the capsule is removed.

Meissner's corpuscles reside in the dermis just below the epidermis. These also rapidly adapt and are thought to respond to fluttering types of stimuli. That is, Meissner's corpuscles also respond to vibration but at a lower frequency than Pacinian corpuscles.

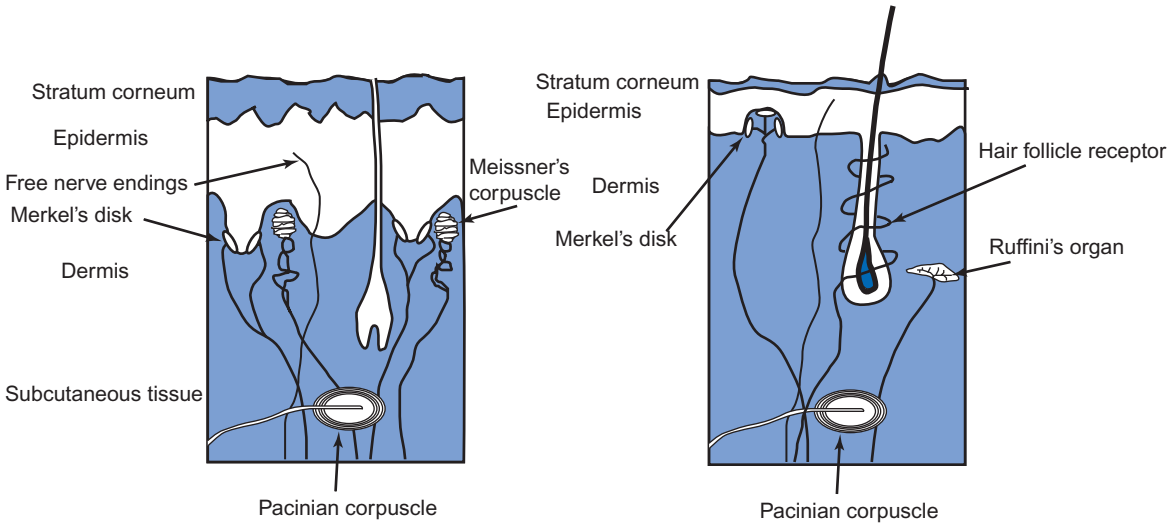

FIGURE 4.3.3 Highly diagrammatic representation of the different mechanoreceptors in the skin. The glabrous or nonhairy skin, shown at left, contains a different set of mechanoreceptors from the hairy skin, as shown at the right. Both contain a variety of receptors.

Merkel's disks are located in the dermis and have small receptive fields. They are slowly adapting and respond to steady touch-pressure on the skin.

Ruffini's corpuscles are located in the dermis and are slowly adapting. They have much larger receptive fields and so they may participate both in touch-pressure sense and in proprioceptive sense by detecting the push or pull of skin from one segment of the body on another.

All of the encapsulated nerve endings listed above are present in both the hairy and nonhairy (**glabrous**) skin. **Free nerve endings** are present in the epidermis in glabrous skin. They contribute to the tactile sense. They also wrap around hair follicles in the hairy skin, where they detect movement of the hair.

THERMORECEPTORS CONSIST OF COLD RECEPTORS AND WARM RECEPTORS

Cold receptors are free nerve endings with thin myelinated fibers, whereas the warm receptors are free nerve endings with unmyelinated axons with low conduction speeds. They differ from the mechanoreceptors in that they exhibit tonic level of activity at most temperatures. They respond to temperature changes with a phasic component followed by a tonic component that depends on the temperature. This is why when you get into a hot bath it feels hot for a while and then it feels warm. The phasic component indicates the change in temperature upon immersion in the hot water and the tonic component indicates that it is still warm. Much of this perception is due to central processing of the peripheral thermoreceptor input (see Figure 4.3.4).

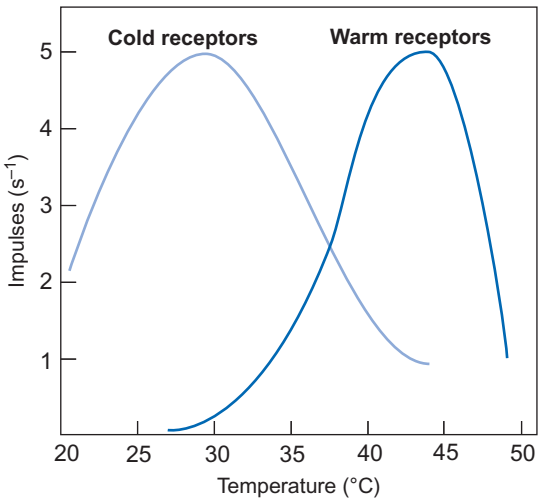

FIGURE 4.3.4 Response of thermoreceptors to skin temperature. The skin temperature was held constant at the indicated temperature, while the frequency of action potentials was recorded from fibers representing each of the thermoreceptor types.

NOCICEPTORS PRODUCE FIRST AND SECOND PAIN

Free nerve endings called **nociceptors** in the skin have a high threshold for mechanical, chemical, or thermal stimuli and respond only when the intensity of these stimuli is high enough to damage tissue. We perceive the input from these receptors as pain. Superficial pain that arises from the skin has two components. The onset of an intense stimuli is sensed by an immediate, sharp, and highly localized pain called **first pain** or **initial pain**. After a delay of about 1 s or so, we are aware of a more diffuse, dull, and aching sensation which is **second pain** or **delayed pain**. First pain is carried over Aδ fibers, which are myelinated fibers that conduct action potentials relatively fast. Second pain is carried over small unmyelinated C fibers that conduct more slowly. This explains the difference in the onset of the sensations, but their subjective experience must be explained by their CNS connections, according to the labeled line theory of sensation (Table 4.3.2).

TABLE 4.3.2 Summary of Cutaneous Receptors

Type of Receptor	Location	Sensation	Fiber Type	Conduction Velocity ($m\ s^{-1}$)	Adaptation
Pacinian corpuscle	Subcutaneous	Vibration	Aβ, large myelinated	30–70	Rapid
Meissner's corpuscle	Dermis of nonhairy skin	Flutter, tapping	Aβ, large myelinated	30–70	Rapid
Merkel's disks	Dermis	Touch, pressure	Aδ, small myelinated	12–30	Slow
Ruffini's corpuscles	Dermis	Touch, pressure, propioception	Aβ, large myelinated	30–70	Slow
Nerve ending	Hair follicle	Touch	Aβ, large myelinated	30–70	Rapid
Cold receptor	Dermis	Cold	Aδ, small myelinated	12–30	Phasic and tonic components
Warm receptor	Dermis	Warmth	C, small unmyelinated	0.5–2	Phasic and tonic components
Nociceptors	Epidermis	First pain	Aδ, small myelinated	12–30	Slow
Nociceptors	Epidermis	Second pain	C, small unmyelinated	0.5–2	Slow

SOMATOSENSORY INFORMATION IS TRANSMITTED TO THE BRAIN THROUGH THE *DORSAL COLUMN PATHWAY*

All of the cutaneous receptors we have discussed so far have a nerve ending in or near the skin and a cell body that resides in the dorsal root of the afferent or sensory nerve leading to the spinal cord (see Figure 4.3.5). The primary afferent neuron is a first-order neuron, being the first neuron to be affected by environmental stimuli. In many cases, the axon from the sensory neuron enters the spinal cord and turns upward and travels to the brainstem in tracts of axons located in the dorsal part of the spinal cord. Accordingly, these tracts form part of the **dorsal columns**, which consist of the **fasciculus cuneatus** and the **fasciculus gracilis**. These parts derive from their destination: the **nucleus cuneatus** and the **nucleus gracilis**, both located in the **medulla**. Neurons in the nucleus gracilis serve the lower parts of the body, whereas neurons in the nucleus cuneatus serve upper parts of the body. These neurons are second-order neurons, being the second stage in the communication between cutaneous sensory receptors and the sensory cortex.

The second-order neurons located in the nucleus gracilis and nucleus cuneatus send axons to the opposite side of the medulla and upward toward the thalamus. They send collaterals to an area of the brainstem called the **reticular activating system (RAS)** that functions in alertness. Using this pathway, somatic stimuli can wake us from a deep sleep. Neurons in the ventral posterolateral thalamus form third-order neurons that relay sensory information up to the cerebral cortex. The fourth-order neurons reside in the **somatosensory cortex**.

Our basic body plan, an evolutionary legacy from our ancient segmented ancestors, is based on body segments which contain a skin element **(dermatome)**, a muscle element **(myotome)**, and a bone element **(sclerotome)**. The vertebrae in each segment comprise part of the sclerotome. Each spinal segment corresponds to a body segment and the sensory nerves that enter the cord serve the corresponding dermatome. The efferent motor nerves at each segment serve the corresponding myotome. When the sensory nerves enter the cord, their input is added to the previous input, from parts lower down the cord, in a layered fashion. Thus, the nerves traveling up the cord are well-organized into the two main tracts and within the tracts preserve a topology. Their mapping is preserved all the way to the cortex, where there is a **somatotopic representation** of the body on the cortex. "Somatotopic" means that adjacent areas of the body have sensory inputs into adjacent areas of the cortex, so that the body surface maps onto the neural surface.

The sensory pathways for cutaneous senses cross over from the left side of the body onto the right side of the brain and from the right side of the body to the left brain. This crossover occurs in the medulla, as shown in Figure 4.3.5, in the dorsal column pathway, but it is a recurrent theme in neurophysiology. Sensory input from the right side of the body travels to the left hemisphere of the brain, and the left side of the body is felt in the right hemisphere of the brain. En route, however, the sensory inputs branch to a variety of areas for

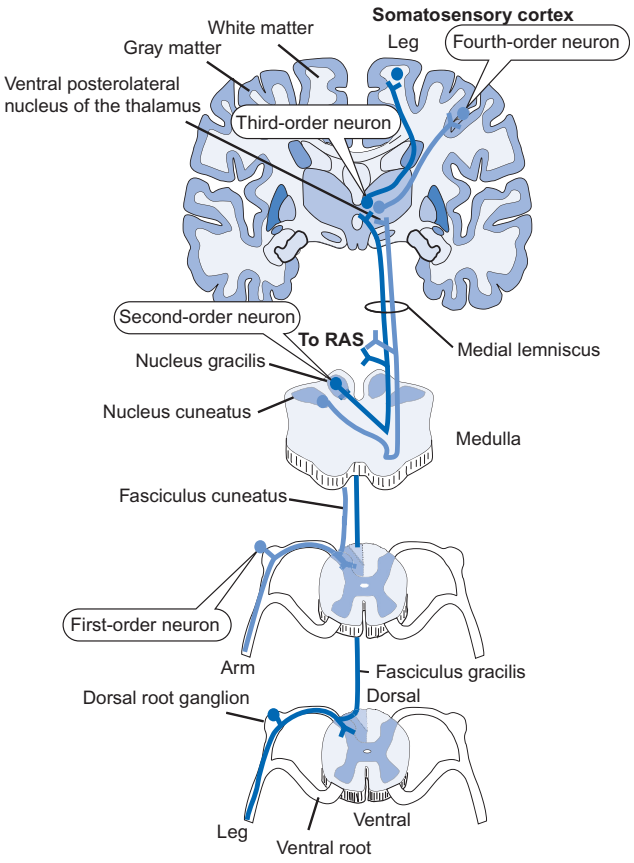

FIGURE 4.3.5 Dorsal column pathway for somatic senses. Primary afferent sensory neurons have cell bodies in the dorsal root ganglia and long axons that terminate in receptors at the skin. Graded receptor potentials are produced in the skin approximately in proportion to the logarithm of the stimulus intensity; those that reach threshold produce an action potential that is conducted toward the cord. The primary afferent fibers make synapses in the cord but also send a long fiber up the cord toward the brainstem. The sensory fibers travel in tracts, the fasciculus gracilis and the fasciculus cuneatus, in the dorsal part of the spinal cord. Because sensory inputs are arranged according to body segments, or dermatomes, there is a regular arrangement of fibers traveling in these fasciculi. These fibers make synapses with second-order neurons in the nucleus gracilis and nucleus fasciculatus. The axons from these second-order neurons cross over to the opposite side of the medulla, sending collaterals to the RAS and to the ventral posterolateral thalamus. These relay sensory information onto the cerebral cortex. The area of the cerebral cortex that receives sensory input is called the primary somatosensory cortex.

defined reasons. Sensory information entering the cord branches immediately to join the dorsal column tracts and to make synapses with interneurons within the spinal cord. These synapses allow for the spinal reflexes that we will discuss in Chapter 4.4. In addition, collaterals synapse onto the RAS to alert us.

THE CUTANEOUS SENSES MAP ONTO THE SENSORY CORTEX

The primary somatosensory cortex is called S1. This area of the cerebral cortex receives sensory information from the somatic senses, plus proprioceptive senses and some visceral senses. It is located on the postcentral gyrus of

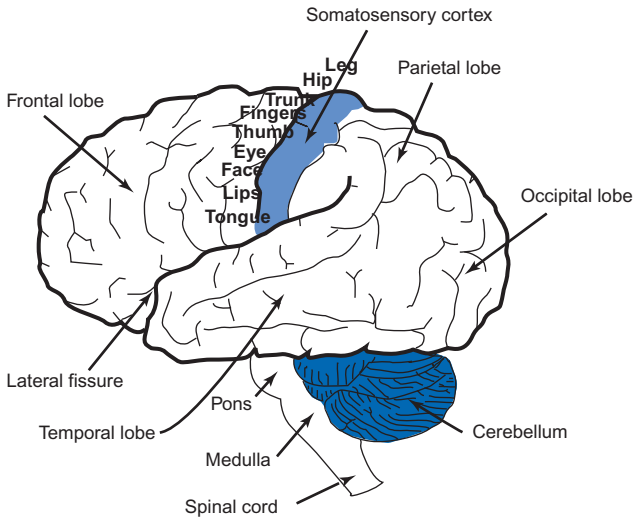

FIGURE 4.3.6 The somatosensory cortex. Sensory inputs reach the postcentral gyrus after having been relayed there by the ventral posterolateral thalamus. The projections of sensory neurons form a kind of neural map of the body, with adjacent areas of the cortex receiving sensory input from adjacent areas of the body. The inputs from the foot and toes are on the postcentral gyrus adjacent to the longitudinal fissure, out of view in this diagram.

the parietal lobe, as shown in Figure 4.3.6. The topological arrangement of the somatic senses is preserved as they enter the spinal cord, travel up the dorsal column tracts, to the nucleus gracilis or nucleus cuneatus, and is preserved through the thalamus to eventually map onto the cortex. Thus the surface of the body maps onto the surface of the brain.

PAIN AND TEMPERATURE INFORMATION TRAVEL IN THE ANTEROLATERAL TRACT

Nociceptors and thermoreceptors do not send axons up the dorsal columns. Instead, these receptors synapse on interneurons within the spinal cord that immediately send axons across the cord to the opposite side, where they ascend in the **anterolateral tract**, or the **ventrolateral tract**. These are also called the **ventral spinothalamic tract** and the **lateral spinothalamic tract**. The neurons making these tracts are second-order neurons whose processes ascend to the thalamus. There, third-order neurons project to the cerebral cortex. Cells in the ventral spinothalamic tract give off collateral branches in the medulla, whereas some cells terminate there (see Figure 4.3.7).

DISORDERS OF SENSATION CAN PINPOINT DAMAGE

Because pain and temperature signals cross the spinal cord at their level of entry and the other cutaneous senses cross in the brainstem, a relatively small lesion affecting only one side of the spinal cord could affect the sensation of pain and temperature on the **contralateral** side (the opposite site of the lesion) while it affects

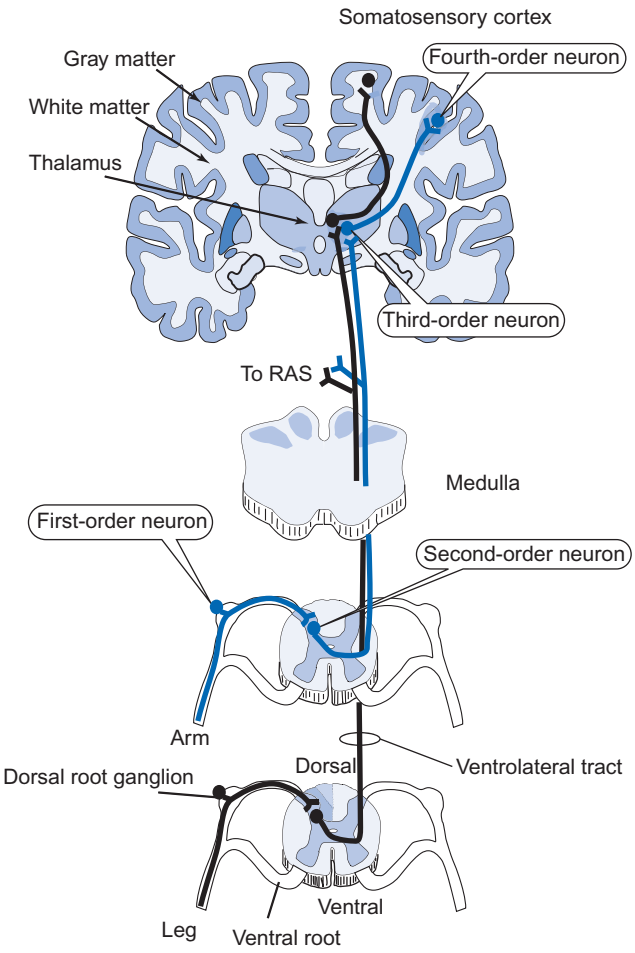

FIGURE 4.3.7 Spinothalamic tracts for pain and temperature sensation. Primary afferent sensory neurons synapse on interneurons within the spinal cord at the level of the primary afferent. These second-order neurons send an axon across the midline that ascends in the antero or ventral spinothalamic tract or the lateral spinothalamic tract. These fibers connect to third-order neurons in the thalamus, which then project to the cerebral cortex. Some cells in the ventral spinothalamic tract give off collaterals in the medulla and some terminate in the reticular formation as part of the spinoreticular tract.

other sensations on the **ipsilateral** side (the same side of the lesion). Both senses project to the contralateral cerebral cortex. In addition, the vertical location of sensory damage can be assessed from the part of the skin that shows sensory loss because each dermatome enters the spinal cord at known places.

PAIN SENSATION CAN BE REDUCED BY SOMATOSENSORY INPUT

Our subjective experience shows that perceived pain can be reduced by gently stroking the skin over the affected area. The explanation for this forms the **gate control theory** of pain. Large diameter somatosensory Aβ fibers make excitatory connections with interneurons in the dorsal horn of the spinal cord. These interneurons also receive inhibitory inputs from small diameter C afferents. When these interneurons are excited, they inhibit type C primary afferents by presynaptic inhibition, thereby acting as a gate to control transmission from type C primary afferents. The activity of the interneuron is determined by the balance between activity on the small nociceptive fibers and the larger somatic cutaneous receptors. When activity on C fibers dominates, the interneuron is inhibited and the gate is opened; when the skin is stroked, Aβ activity may dominate and the interneuron is activated, closing the gate (see Figure 4.3.8).

THE RECEPTIVE FIELD OF SOMATOSENSORY CORTICAL NEURONS IS OFTEN ON-CENTER, OFF-SURROUND

The receptive field of primary cutaneous sensory neurons is the area of the skin where stimulation produces excitation. The receptive field of neurons in the somatosensory cortex is more complex than this because the output of many different cells converges on these neurons. Many cortical neurons exhibit an **inhibitory**

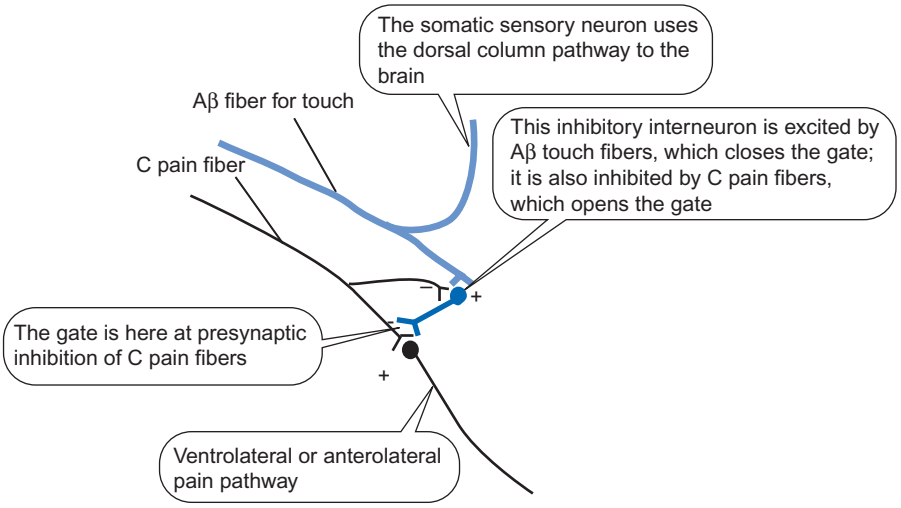

FIGURE 4.3.8 The gate theory of pain modulation by sensory fibers. Pain information enters the spinal cord over small unmyelinated C fibers. Normally these sensory fibers excite a second-order neuron that crosses over to the ventrolateral or spinothalamic tracts and ascends to the medulla. Sensory Aβ fibers send processes up the dorsal columns to synapse with second-order neurons in the medulla. They also synapse with interneurons in the dorsal horn of the gray matter of the spinal cord. These neurons are excited by somatosensory input, and they have inhibitory connections to the second-order neurons of the pain pathway. Thus, stimulation of the somatic sensory neurons inhibits the activity of neurons that signal pain.

surround. Stimulation of a central region on the skin stimulates the neuron, whereas stimulation of the area around that central region inhibits the neuron (see Figure 4.3.9). This phenomenon has its origins in **lateral inhibition**, which refers to the inhibition of a higher order neuron by primary sensory afferents serving the areas around an excitatory area. Figure 4.3.10 shows a hypothetical wiring diagram that would produce lateral inhibition.

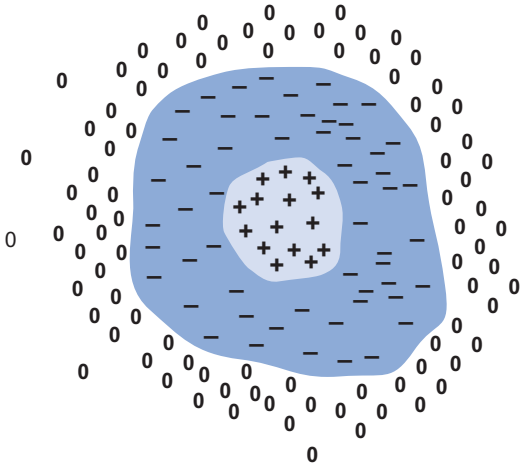

FIGURE 4.3.9 Receptive field of a somatosensory cortical neuron. Stimulation of the skin in a certain area causes increased frequency of firing of the cortical cell as indicated by "+" in the figure. Stimulation of the surrounding area decreases the excitation of the cell, as indicated by a "−" in the figure. Stimulation of an area still further removed results in no effect on the cell, as indicated by a "0" in the figure. The receptive fields are not necessarily circular but may take on complex shapes.

SUMMARY

Sensory systems apprise the body of the conditions of the external environment (exteroreceptors) and the internal environment (interoreceptors). Long receptors have sensory processes in the periphery, but their cell bodies are in the dorsal root of the sensory nerve just outside the spinal cord. These receptors must convert sensory energy into action potentials. Short receptors typically do not fire action potentials; they generate receptor potentials in response to stimuli which causes them to release neurotransmitters onto primary afferent neurons. This release of neurotransmitter is graded

Clinical Applications: Neuropathic Pain and Transcutaneous Electrical Neural Stimulation

Subjective experience tells us that pain differs from other sensations such as cold, warmth, touch, and sound. Pain is a complex experience derived from nociception, somewhat like vision is a complex experience derived from light sensation. But pain carries with it distressful affect (from the latin *affectus*, meaning "state of mind") that the other modalities ordinarily lack. Under some circumstances pain becomes emotionally distressful. The mechanism by which this occurs is not fully understood.

Clinicians recognize two broad classes of pain: nociceptive or physiological pain, and neuropathic or intractable pain. Physiological pain arises from stimulation of nociceptors and is carried by Aδ and C fibers to the CNS. Stimulation of Aδ fibers causes a sharp and highly localized pain that is usually short lived. Stimulation of C fibers produces a dull, aching, or burning pain that persists even when the stimulation is removed. These two types of pain are often discriminated by the adjectives **epicritic** (from the Greek *epi*, meaning "on" and *krino* meaning "separate" or "judge"), referring to the sharp pain that is easily localized, and **protopathic** (from the Greek *protos* meaning "first" and *pathos* meaning "suffering") that describes the suffering of second pain. Physiological pain can be increased by peripheral sensitization or central sensitization. Peripheral sensitization results from changes in the nociceptors brought about by inflammation. Central sensitization is caused by changes in neuronal activity in the spinal cord, usually interneurons. Many of these neurons have NMDA receptors that respond to glutamic acid. These receptors at rest typically bind Mg^{2+} ions, and the bound Mg^{2+} inhibits depolarizing Ca^{2+} influx. When the cells are depolarized further, Mg^{2+} dissociates from the receptor and the cell becomes hypersensitive. Although both peripheral and central sensitization can last for minutes to hours after removal of the stimulus, it does not cause permanent changes in the CNS.

Neuropathic pain, on the other hand, results from injury to the CNS that permanently changes CNS connections. For example, injury can break axons, and the somas will regenerate new axons. But recovery is incomplete, and small fibers are less likely to regenerate than larger ones. Injury to a nerve will lose C and Aδ fibers more than the large Aβ fibers. In attempts to make new synaptic connections, the Aβ fibers can make new synapses on neurons abandoned by the C fibers. Thus, previously innocuous stimuli become severely painful. This change from innocuous to noxious perception is called **allodynia**. Persons with amputations sometimes report this type of pain that appears to them to originate from the removed limb. Such pain is called **phantom pain**.

Neuropathic or intractable pain is severely distressing and affected people seek relief from the misery. Surgical remedies include sectioning of the dorsal root (**rhizotomy)** or of the anterolateral tract (**tractotomy**). These procedures often provide immediate relief, but the pain almost always returns when the severed axons once again form inappropriate synapses.

Transcutaneous electrical neural stimulation (TENS) sometimes provides relief from intractable pain. This less invasive procedure uses gate control theory. Electrodes are placed on the skin over a peripheral nerve. The large fibers have a lower threshold for stimulation and are preferentially activated by TENS. Preferential activation of the large fibers closes the "gate" by which pain enters the CNS. TENS devices successfully reduce some types of intractable pain.

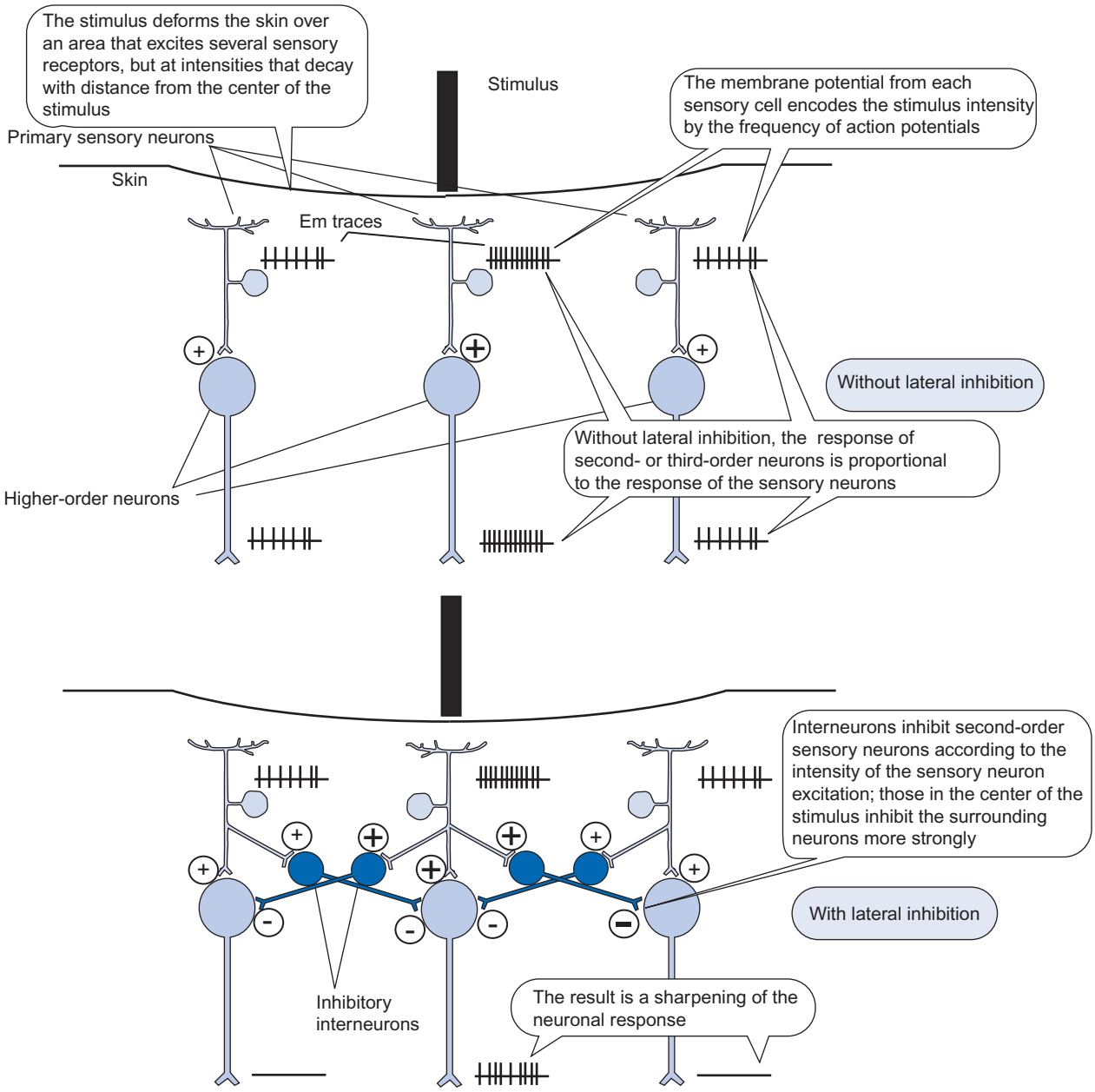

FIGURE 4.3.10 How lateral inhibition sharpens spatial discrimination. Without lateral inhibition, a mechanical stimulation of the skin produces a response from three sensory neurons which is passed forward up to the somatosensory cortex. Lateral inhibition by interneurons inhibits the cells responding to the periphery of the stimulus more than at the center. This sharpens the spatial discrimination of the stimulus and also produces the on-center and off-surround receptive field. That is, a stimulus localized to the periphery inhibits the response at the center. The sharpening of the response enhances two-point discrimination for the cutaneous senses. The effects shown here are exaggerated and are the net effect of events at several layers in the sensory pathway and not at the level of the primary sensory afferents.

according to the strength of the stimulus, and the primary afferent neuron responds with a train of action potentials.

The quality of the stimulus (light, touch, sound, etc.) is conveyed to the CNS by labeled lines: the connectivity of the neurons determines its interpreted quality. The intensity is encoded by the frequency of sensory neuron action potentials (the frequency code) and by the number of sensory neurons that are excited (the population code). The frequency of action potential firing is roughly proportional to the logarithm of the stimulus intensity, so that a three-fold change in frequency of action potentials encodes a 1000-fold change in stimulus intensity.

The dynamics of sensory receptor response allows sensation of position, velocity, and acceleration. The physical location of the receptor means that it will respond only to stimuli within its receptive field. Receptive fields can also be defined for higher order neurons within the CNS. Regions of high spatial sensitivity are characterized by neurons with small receptive fields.

Cutaneous sensory cells bring information into the CNS by the spinal cord. Somatic sensory neurons for touch and pressure enter the cord through the dorsal root and send fibers up to two dorsal columns, the fasciculus gracilis and the fasciculus cuneatus. The fasciculus gracilis serves the lower body and the fasciculus cuneatus serves the upper torso. The axons are laid down at each level of the cord so that there is a topological representation of the body surface within the cord. This topology is preserved all the way to the cortex, where the somatic senses map onto the somatosensory cortex immediately posterior to the central sulcus. The area of the body surface served by each level of the spinal cord is called a dermatome. The dorsal columns make contact with second-order neurons in the medulla, whose axons cross over and project onto third-order neurons in the thalamus. These third-order cells then send axons up to the somatosensory cortex. Thus, sensory information crosses over at the level of the medulla. Somatic senses of pain and temperature, however, make a synapse in the spinal cord at the level of the spinal root. The second-order neuron crosses over at that level and sends fibers up the spinothalamic tracts. Both somatic sensory neurons and pain and temperature sensors send collaterals to the reticular activating system in the medulla that will awake you if you are sleeping, and otherwise contributes to alertness.

Pain reception can be reduced by somatic senses due to interneurons that are excited by touch but inhibit pain fibers. This is the basis by which pain is lessened by movement or by mechanical stimulation of the skin.

REVIEW QUESTIONS

1. What is a sensory modality? What is the adequate stimulus? How is quality of sensory information encoded?
2. How is intensity of sensory information encoded?
3. What is one way to distinguish between position, velocity, and acceleration?
4. What is lateral inhibition and how does it sharpen spatial discrimination? How does this give rise to an on-center, off-surround receptive field? What is meant by "receptive field"?
5. What is the dorsal column pathway? What sensory modalities use it? What is the anterolateral pathway, and what sensory modalities use it?
6. What is the somatosensory cortex? What is somatotopic mapping? Why does it occur?
7. What is a fasciculus? What is the fasciculus gracilis, and what part of the body does it serve? What is the fasciculus cuneatus, and what part of the body does it serve?