Biopsychology

TENTH EDITION

GLOBAL EDITION

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These signals excite the auditory nerve. Although cochlear implants can provide major benefits, they do not restore normal hearing. The sooner a person receives a cochlear implant after becoming deaf, the more likely he or she is to benefit, because disuse leads to alterations of the auditory neural pathways (see Kral & Sharma, 2012).

Scan Your Brain

Before we go on to discuss the other sensory systems, pause and test your knowledge of what you have learned in this chapter so far. The correct answers are provided at the end of the exercise. Before proceeding, review material related to your errors and omissions.

1. The _____ is the area of the sensory cortex that receives most of its input directly from the thalamic relay nuclei of the system.
2. _____ is the process of detecting the presence of stimuli.
3. Simultaneous analysis of a signal in different ways by the multiple pathways of a neural network is referred to _____.
4. _____ is the mathematical procedure for breaking down complex waves into their component sine waves.
5. _____ are also called sine wave vibrations.
6. The three _____ are malleus, incus, and the stapes.
7. The layout of the auditory system tends to be _____.
8. The axons of the auditory nerves synapse in the ipsilateral _____ nuclei.
9. One function of the superior olives is sound _____.
10. The _____ is made up of a fine sheet of neurons located just underneath the neocortex, toward the middle of the brain.
11. The _____ is the membrane that transfers vibrations from the ossicles to the fluid of the cochlea.
12. Many studies of auditory-visual interactions have focused on association cortex in the posterior _____ cortex.

Scan Your Brain answers: (1) primary sensory cortex, (2) Sensation, (3) parallel processing, (4) Fourier analysis, (5) Pure tones, (6) ossicles, (7) tonotopic, (8) cochlear, (9) localization, ((10) claustrum, (11) oval window, (12) parietal.

Somatosensory System: Touch and Pain

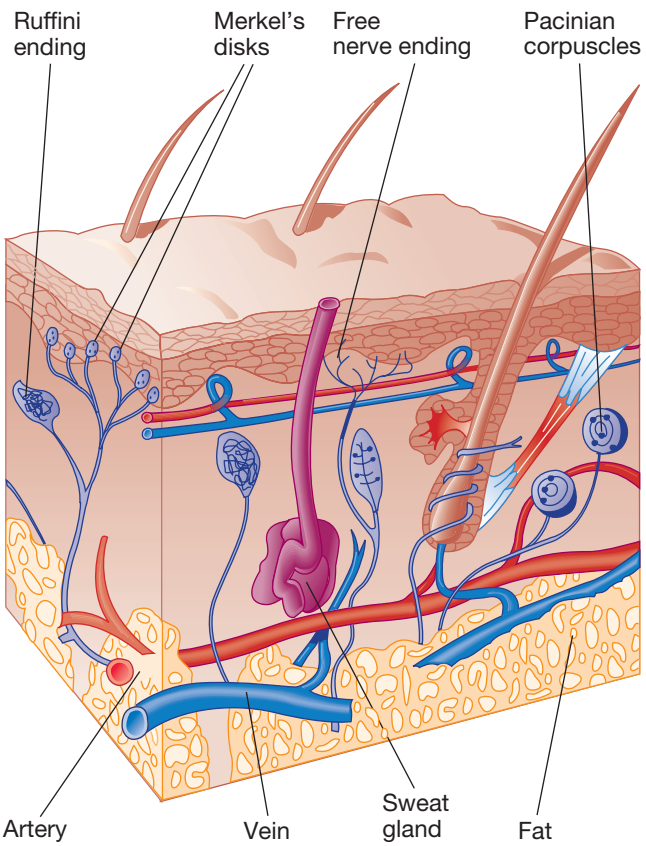
Sensations from your body are referred to as *somatosensations*. The system that mediates these bodily sensations—the *somatosensory system*—is three separate but interacting systems: (1) an *exteroceptive system*, which senses external stimuli that are applied to the skin; (2) a *proprioceptive system*, which monitors information about the position of the body that comes from receptors in the muscles, joints, and organs of balance; and (3) an *interoceptive system*, which provides general information about conditions within the body (e.g., temperature and blood pressure). This module deals almost exclusively with the exteroceptive system, which itself comprises three somewhat distinct divisions: a division for perceiving *mechanical stimuli* (touch), one for *thermal stimuli* (temperature), and one for *nociceptive stimuli* (pain).

Cutaneous Receptors

LO 7.9 Name some of the cutaneous receptors, and explain the functional significance of fast versus slow receptor adaptation.

There are many kinds of receptors in the skin (see Owens & Lumpkin, 2014; Zimmerman, Bai, & Ginty, 2014). Figure 7.9

Figure 7.9 Four cutaneous receptors that occur in human skin.



illustrates four of them. The simplest cutaneous receptors are the **free nerve endings** (neuron endings with no specialized structures on them), which are particularly sensitive to temperature change and pain. The largest and deepest cutaneous receptors are the onionlike **Pacinian corpuscles**; because they adapt rapidly, they respond to sudden displacements of the skin but not to constant pressure. In contrast, **Merkel's disks** and **Ruffini endings** both adapt slowly and respond to gradual skin indentation and skin stretch, respectively.

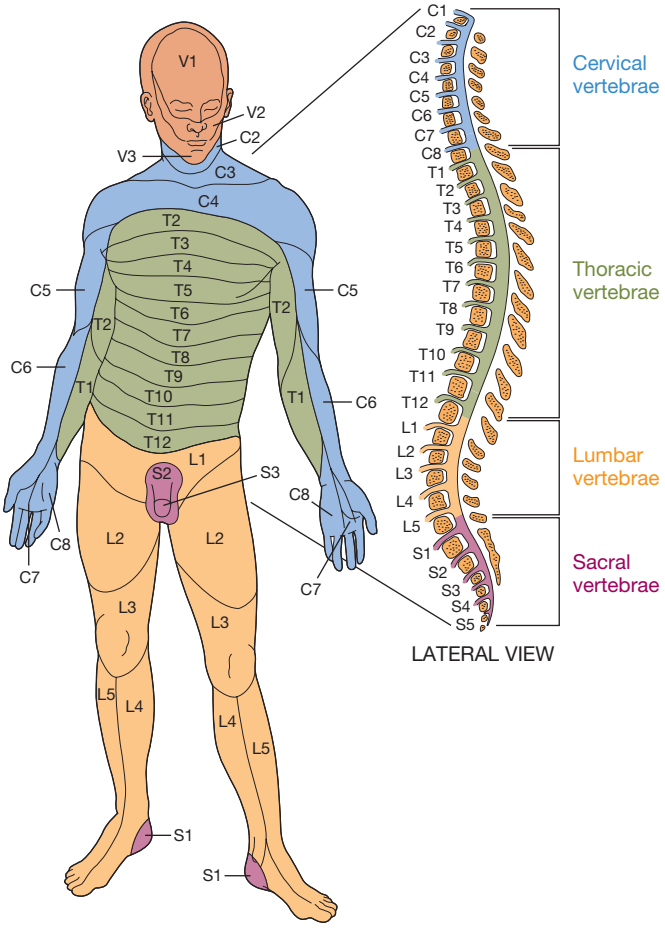
To appreciate the functional significance of fast and slow receptor adaptation, consider what happens when a constant pressure is applied to the skin. The pressure evokes a burst of firing in all receptors, which corresponds to the sensation of being touched; however, after a few hundred milliseconds, only the slowly adapting receptors remain active, and the quality of the sensation changes. In fact, you are often totally unaware of constant skin pressure; for example, you are usually unaware of the feeling of your clothes against your body until you focus attention on it. As a consequence, when you try to identify objects by touch, you manipulate them in your hands so that the pattern of stimulation continually changes. (The identification of objects by touch is called **stereognosis**.) Having some receptors that adapt quickly and some that adapt slowly provides information about both the dynamic and static qualities of tactual stimuli.

The structure and physiology of each type of somatosensory receptor seems to be specialized for a different function. However, in general, the various receptors tend to function in the same way: Stimuli applied to the skin deform or change the chemistry of the receptor, and this in turn changes the permeability of the receptor cell membrane to various ions (see Delmas, Hao, & Rodat-Despoix, 2011; Tsunozaki & Bautista, 2009). The result is a neural signal.

Initially, it was assumed that each type of receptor located in the skin (see Figure 7.9) mediates a different tactile sensation (e.g., touch, pain, heat), but this has not proven to be the case. Each tactile sensation appears to be produced by the interaction of multiple receptor mechanisms, and each receptor mechanism appears to contribute to multiple sensations (see Hollins, 2010; Lumpkin & Caterina, 2007; McGlone & Reilly, 2009). In addition, skin cells that surround particular receptors also seem to play a role in the quality of the sensations produced by that receptor (see Zimmerman, Bai, & Ginty, 2014). Indeed, new forms of tactile sensation are still being discovered (see McGlone, Wessberg, & Olausson, 2014).

DERMATOMES. The neural fibers that carry information from cutaneous receptors and other somatosensory receptors gather together in nerves and enter the spinal cord via the *dorsal roots*. The area of the body that is innervated by the left and right dorsal roots of a given segment of the spinal cord is called a **dermatome**. Figure 7.10 is a dermatomal map of the human body. Because there is considerable overlap between adjacent dermatomes, destruction of a single dorsal root typically produces little somatosensory loss.

Figure 7.10 The dermatomes of the human body. S, L, T, and C refer respectively to the sacral, lumbar, thoracic, and cervical regions of the spinal cord. V1, V2, and V3 stand for the three branches of the trigeminal nerve.



Two Major Somatosensory Pathways

LO 7.10 Describe the two major somatosensory pathways.

Somatosensory information ascends from each side of the body to the human cortex over several pathways, but there are two major ones: the dorsal-column medial-lemniscus system and the anterolateral system. The **dorsal-column medial-lemniscus system** tends to carry information about touch and proprioception, and the **anterolateral system** tends to carry information about pain and temperature. The key words in the preceding

sentence are "tends to": The separation of function in the two pathways is far from complete. Accordingly, lesions of the dorsal-column medial-lemniscus system do not eliminate touch perception or proprioception, and lesions of the anterolateral system do not eliminate perception of pain or temperature.

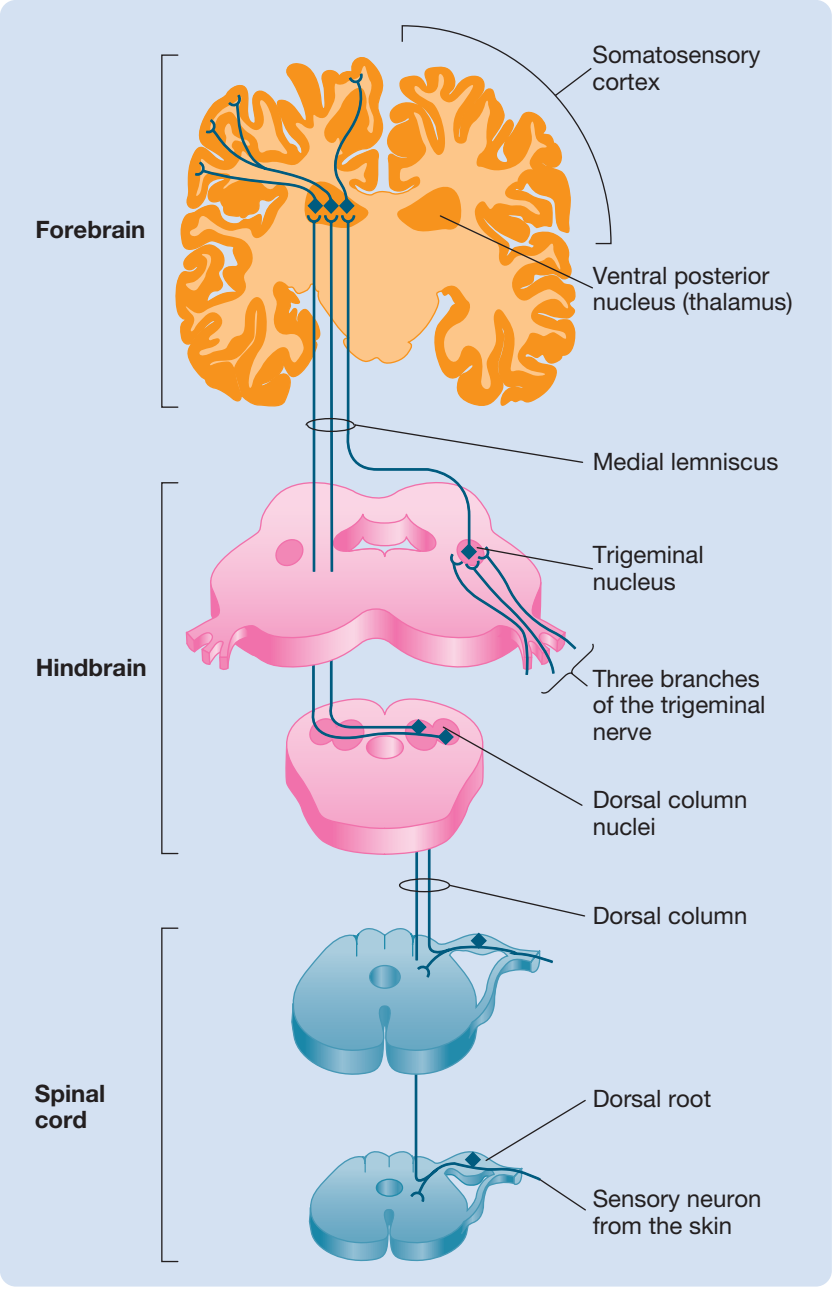
The dorsal-column medial-lemniscus system is illustrated in Figure 7.11. The sensory neurons of this system enter the spinal cord via a dorsal root, ascend ipsilaterally in the **dorsal columns**, and synapse in the *dorsal column nuclei* of the medulla. The axons of dorsal column nuclei neurons *decussate* (cross over to the other side of the brain) and then ascend in the **medial lemniscus** to the contralateral **ventral posterior nucleus** of the thalamus. The ventral posterior nuclei also receive input via the three branches of the trigeminal nerve, which carry somatosensory information from the contralateral areas of the face. Most neurons of the ventral posterior nucleus project to the *primary somatosensory cortex* (*SI*); others project to the *secondary somatosensory cortex* (*SII*) or the posterior parietal cortex. Neuroscience trivia buffs will almost certainly want to add to their collection the fact that the dorsal column neurons that originate in the toes are the longest neurons in the human body.

The anterolateral system is illustrated in Figure 7.12. Most dorsal root neurons of the anterolateral system synapse as soon as they enter the spinal cord. The axons of most of the second-order neurons decussate but then ascend to the brain in the contralateral anterolateral portion of the spinal cord; however, some do not decussate but ascend ipsilaterally. The anterolateral system comprises three different tracts: the *spinothalamic tract*, the *spinoreticular tract*, and the *spinotectal tract*. The three branches of the trigeminal nerve carry pain and temperature information from the face to the same thalamic sites. The pain and temperature information that reaches the thalamus is then distributed to somatosensory cortex and other parts of the brain.

If both ascending somatosensory paths are completely transected by a spinal injury, the patient can feel no body sensation from below the level of the cut. Clearly, when it comes to spinal injuries, lower is better.

Clinical Implications

Figure 7.11 The dorsal-column medial-lemniscus system. The pathways from only one side of the body are shown.

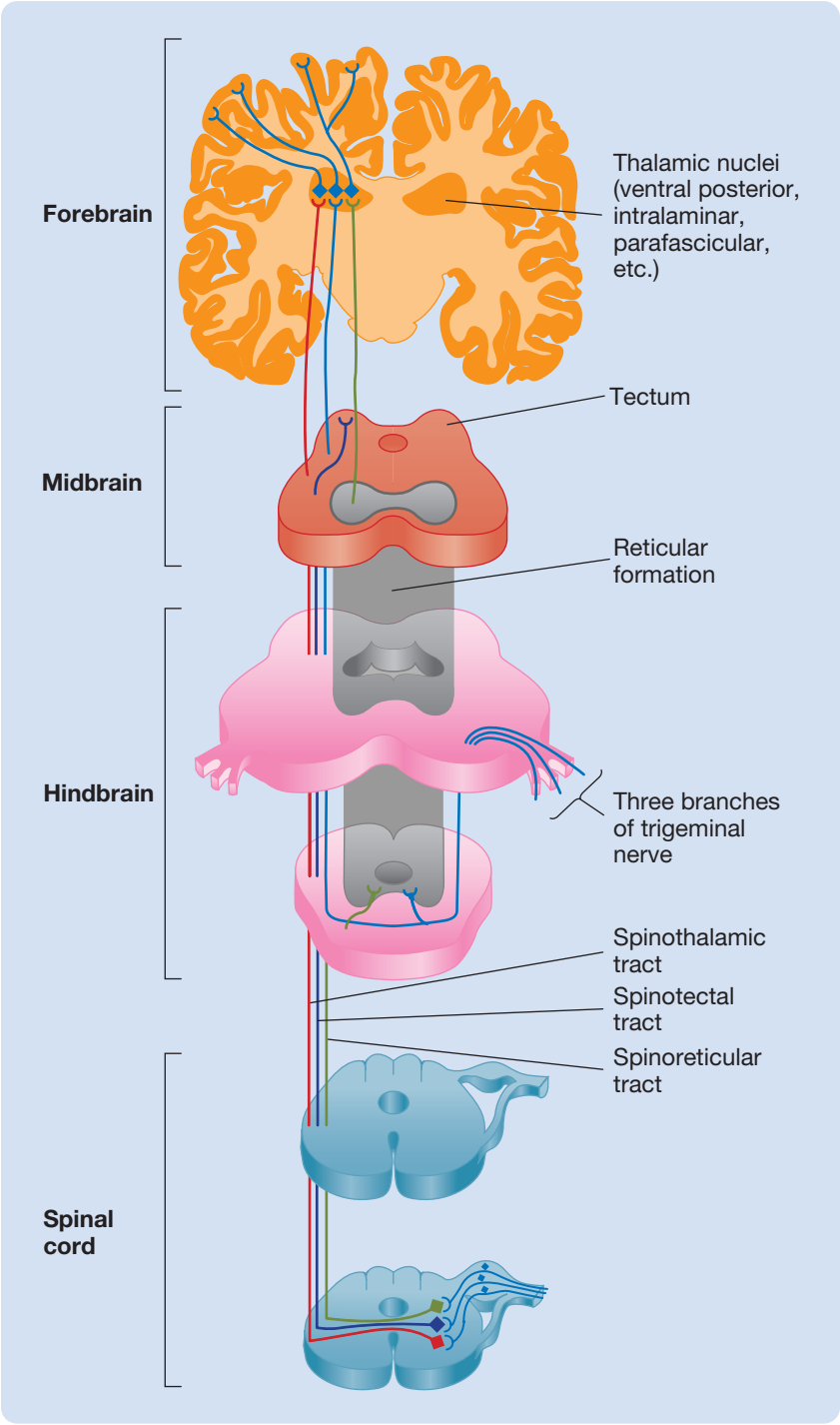


Cortical Areas of Somatosensation

LO 7.11 Describe the cortical somatosensory areas and their somatotopic layout.

In 1937, Penfield and his colleagues mapped the primary somatosensory cortex of patients during neurosurgery (see Figure 7.13). Penfield applied electrical stimulation to various sites on the cortical surface, and the patients, who were fully conscious under a local anesthetic, described what they felt. When stimulation was applied to the *postcentral gyrus*, the patients reported somatosensory sensations in

Figure 7.12 The anterolateral system. The pathways from only one side of the body are shown.



various parts of their bodies. When Penfield mapped the relation between each site of stimulation and the part of the body in which the sensation was felt, he discovered that the human primary somatosensory cortex (SI) is **somatotopic**—organized according to a map of the body surface (see Chen et al., 2015). This somatotopic map is commonly referred to as the **somatosensory homunculus** (*homunculus* means "little man").

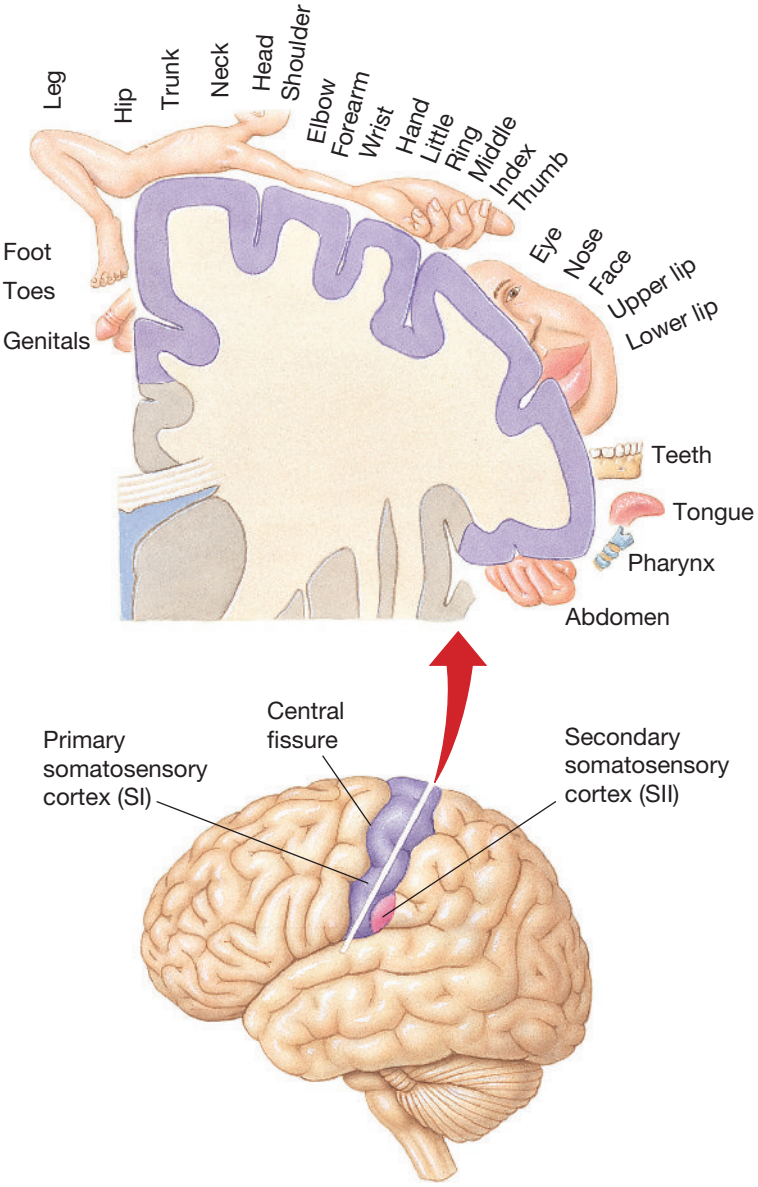
Notice in Figure 7.13 that the somatosensory homunculus is distorted; the greatest proportion of SI is dedicated to receiving input from the parts of the body we use to make tactile discriminations (e.g., hands, lips, and tongue). In contrast, only small areas of SI receive input from large areas of the body, such as the back, that are not usually used to make somatosensory discriminations. The Check It Out demonstration on page 180 allows you to experience the impact this organization has on your ability to perceive touches.

A second somatotopically organized area, SII, lies just ventral to SI in the postcentral gyrus, and much of it extends into the lateral fissure. SII receives most of its input from SI and is thus regarded as secondary somatosensory cortex. In contrast to SI, whose input is largely contralateral, SII receives substantial input from both sides of the body. Much of the output of SI and SII goes to the association cortex of the *posterior parietal lobe* (see McGlone & Reilly, 2010).

Studies of the responses of single neurons in primary somatosensory cortex found evidence for columnar organization—similar to what you have already seen in visual and auditory cortex. Each neuron in a particular column of primary somatosensory cortex had a receptive field on the same part of the body and responded most robustly to the same type of tactile stimuli (e.g., light touch or heat). Moreover, single-neuron recordings suggested that primary somatosensory cortex is composed of four functional strips, each with a similar, but separate, somatotopic organization. Each strip of primary somatosensory cortex is most sensitive to a different kind of somatosensory input (e.g., to light touch or pressure). Thus, if one were to record from neurons across the four strips, one would find neurons that "preferred" four different kinds of tactile stimulation, all to the same part of the body.

Reminiscent of the developments in the study of visual and auditory cortex, it has been proposed that two streams of analysis proceed from SI: a dorsal stream that projects to posterior parietal cortex and participates in multisensory integration and direction of attention and a ventral stream that projects to SII and

Figure 7.13 The locations of human primary somatosensory cortex (SI) and one area of secondary somatosensory cortex (SII) with the conventional portrayal of the somatosensory homunculus. Something has always confused us about this portrayal of the somatosensory homunculus: The body is upside down, while the face is right side up. It now appears that this conventional portrayal is wrong. The results of an fMRI study suggest that the face representation is also inverted. (Based on Servos et al., 1999.)



participates in the perception of objects' shapes (Yau, Connor, & Hsiao, 2013).

Check It Out

Touching a Back

Because only a small portion of human primary somatosensory cortex receives input from the entire back, people have difficulty recognizing objects that touch their backs. You may not have noticed this tactile deficiency—unless, of course, you often try to identify objects by feeling them with your back. You will need one thing to demonstrate the recognition deficiencies of the human back: a friend. Touch your friend on the back with one, two, or three fingers, and ask your friend how many fingers he or she feels. When using two or three fingers, be sure they touch the back simultaneously because temporal cues invalidate this test of tactile discrimination. Repeat the test many times, adjusting the distance between the touches on each trial. Record the results. What you should begin to notice is that the back is incapable of discriminating between separate touches unless the distance between the touches is considerable. In contrast, fingertips can distinguish the number of simultaneous touches even when the touches are very close.

EFFECTS OF DAMAGE TO THE PRIMARY SOMATOSENSORY CORTEX. Like the effects of damage to the primary auditory cortex, the effects of damage to the primary somatosensory cortex are often remarkably mild—presumably because, like the auditory system, the somatosensory system features numerous parallel pathways. Corkin, Milner, and Rasmussen (1970) assessed the somatosensory abilities of epileptic patients before and after a unilateral excision that included SI. Following the surgery, the patients displayed two minor contralateral deficits: a reduced ability to detect light touch and a reduced ability to identify objects by touch (i.e., a deficit in stereognosis). These deficits were bilateral only in those cases in which the unilateral lesion encroached on SII.

Somatosensory System and Association Cortex

LO 7.12 Name the areas of association cortex that somatosensory signals are sent to, and describe the functional properties of one of those areas.

Somatosensory signals are ultimately conducted to the highest level of the sensory hierarchy, to areas of association cortex in prefrontal and posterior parietal cortex.

Posterior parietal cortex contains *bimodal neurons* (neurons that respond to activation of two different sensory systems) that respond to both somatosensory and visual stimuli (see Rosenblum, 2013). The visual and somatosensory receptive fields of each neuron are spatially related; for example, if a neuron has a somatosensory receptive field centered in the left hand, its visual field is adjacent to the left hand. Remarkably, as the left hand moves, the visual receptive field of the neuron moves with it. The existence of these bimodal neurons motivated the following interesting case study by Schendel and Robertson (2004).

The Case of W.M., Who Reduced His Scotoma with His Hand

W.M. suffered a stroke in his right posterior cerebral artery. The stroke affected a large area of his right occipital and parietal lobes and left him with severe left *hemianopsia* (a condition in which a scotoma covers half the visual field). When tested with his left hand in his lap, W.M. detected 97.8 percent of the stimuli presented in his right visual field and only 13.6 percent of those presented in his left visual field. However, when he was tested with his left hand extended into his left visual field, his ability to detect stimuli in his left visual field improved significantly. Further analysis showed that this general improvement resulted from W.M.'s greatly improved ability to see those objects in the left visual field that were near his left hand. Remarkably, this area of improved performance around his left hand was expanded even further when he held a tennis racket in his extended left hand.

Clinical Implications

Neuroplasticity

Somatosensory Agnosias

LO 7.13 Describe the two major types of somatosensory agnosia.

There are two major types of somatosensory agnosia. One is **astereognosia**—the inability to recognize objects by touch. Cases of pure astereognosia—those that occur in the absence of simple sensory deficits—are rare (Corkin, Milner, & Rasmussen, 1970). The other type of somatosensory agnosia is **asomatognosia**—the failure to recognize parts of one's own body. Asomatognosia is usually unilateral, affecting only the left side of the body, and it is usually associated with extensive damage to the right temporal and posterior parietal lobe (Feinberg et al., 2010). The case of Aunt Betty (Klawans, 1990) is an example.

The Case of Aunt Betty, Who Lost Half of Her Body*

Aunt Betty was my patient. She wasn't really my aunt, she was my mother's best friend.

As we walked to her hospital room, one of the medical students described the case. "Left hemiplegia [left-side paralysis], following a right-hemisphere stroke." I was told.

Aunt Betty was lying on her back with her head and eyes turned to the right. "Betty," I called out.

I approached her bed from the left, but Aunt Betty did not turn her head or even her eyes to look toward me.

"Hal," she called out. "Where are you?"

I turned her head gently toward me, and we talked. It was clear that she had no speech problems, no memory loss, and no confusion. She was as sharp as ever. But her eyes still looked to the right, as if the left side of her world did not exist.

I held her right hand in front of her eyes. "What's this?" I asked.

"My hand, of course," she said with an intonation that suggested what she thought of my question.

"Well then, what's this?" I said, as I held up her limp left hand where she could see it.

"A hand."

"Whose hand?"

"Your hand, I guess," she replied. She seemed puzzled. I placed her hand back on the bed.

"Why have you come to the hospital?" I asked.

"To see you," she replied hesitantly. I could tell that she didn't know why.

Aunt Betty was in trouble.

As in the case of Aunt Betty, asomatognosia is often accompanied by **anosognosia**—the failure of neuropsychological patients to recognize their own symptoms. Indeed, anosognosia is a common, but curious, symptom of many neurological disorders—many neurological patients with severe behavioral problems think that they are doing quite well.

Asomatognosia is commonly a component of **contralateral neglect**—the tendency not to respond to stimuli that are contralateral to a right-hemisphere injury. You will learn more about contralateral neglect in Chapter 8.

Rubber-Hand Illusion

LO 7.14 Describe the rubber-hand illusion and its neural mechanisms.

We perceive ownership of our own body parts. Somesthetic sensation is so fundamental that it is taken for granted.

*Based on NEWTON'S MADNESS by Harold Klawans (Harper & Row 1990).

This is why exceptions to it, such as asomatognosia, are so remarkable. In the past decade, another exception—one that is in some respects the opposite of asomatognosia—has been a focus of research. This exception is the **rubber-hand illusion** (the feeling that an extraneous object, in this case a rubber hand, is actually part of one's own body).

The rubber-hand illusion can be generated in a variety of ways, but it is usually induced in the following manner (see Kilteni et al., 2015; Moseley, Gallace, & Spence, 2012). A healthy volunteer's hand is hidden from view by a screen, and a rubber hand is placed next to the hidden hand but in clear sight. Then the experimenter repeatedly strokes the hidden hand and the rubber hand synchronously—see Figure 7.14. In less than a minute, many volunteers begin to feel that the rubber hand is part of their own body (see Blanke, Slater, & Serino, 2015). Interestingly, when this happens, the temperature in the hidden hand drops (Moseley et al., 2008).

Although the neural mechanisms for the rubber-hand illusion are unknown, functional imaging studies have suggested that association cortex in the posterior parietal and frontal lobes plays a role in its induction (see Limanowski & Blankenburg, 2015; Tsakiris et al., 2007). It has been suggested that those frontal and parietal *bimodal neurons* with both visual and somatosensory fields play a critical role (see Kilteni et al., 2015).

Schaefer and colleagues (Schaefer et al., 2007; Schaefer, Heinze, & Rotte, 2009) adapted the rubber-hand technique to induce two particularly interesting somatosensory illusions. In one, volunteers felt that one of their arms had been stretched, and in the other they felt that they had three arms.

Figure 7.14 Induction method for the rubber-hand illusion. The participant's hand is hidden from view by a screen, and a rubber hand is placed next to their hidden hand but in clear sight. Then the experimenter repeatedly strokes the hidden hand and the rubber hand synchronously.

Perception of Pain

LO 7.15 Explain why the perception of pain is said to be paradoxical.

A paradox is a logical contradiction. The perception of pain is paradoxical in three important respects, which are explained in the following three subsections.

ADAPTIVENESS OF PAIN. One paradox of pain is that an experience that seems in every respect to be so bad is in fact extremely important for our survival. There is no special stimulus for pain; it is a response to potentially harmful stimulation of any type. It warns us to stop engaging in potentially harmful activities or to seek treatment (see Navratilova & Porreca, 2014).

Evolutionary Perspective

The value of pain is best illustrated by the cases of people, like Miss C., who experience no pain (Melzack & Wall, 1982).

The Case of Miss C., the Woman Who Felt No Pain

Miss C., a university student, was very intelligent, and she was normal in every way except that she never felt pain. Her condition is now referred to as *congenital insensitivity to pain*.

She felt no pain when subjected to strong electric shock, burning hot water, or an ice bath. Equally astonishing was the fact that she showed no changes in blood pressure, heart rate, or respiration when these stimuli were presented. Furthermore, she did not sneeze, cough, or display corneal reflexes (blinking to protect the eyes). As a child, she had bitten off the tip of her tongue while chewing food and had suffered severe burns after kneeling on a radiator.

Clinical Implications

Miss C. exhibited pathological changes in her knees, hip, and spine because of the lack of protection to joints provided by pain sensation. She apparently failed to shift her weight when standing, to turn over in her sleep, or to avoid harmful postures.

Miss C. died at the age of 29 of massive infections and extensive skin and bone trauma.

Clinical Implications Cases of congenital insensitivity to pain illustrate something important about the adaptive value of pain. Based on this case study, can you specify what that adaptive value might be?

Cox and colleagues (2006) studied six cases of congenital insensitivity to pain among members of a family from Pakistan. They were able to identify the gene abnormality underlying the disorder in these six individuals: a gene that influences the synthesis of sodium ion channels. Indeed, knockout mice that

Clinical Implications

are missing this sodium ion channel gene show a comparable indifference to pain (Gingras et al., 2014). Other genetic disorders of painlessness have been identified—each involve a different genetic alteration (see Nahorski, Chen, & Woods, 2015).

LACK OF CLEAR CORTICAL REPRESENTATION OF PAIN. The second paradox of pain is that it has no obvious cortical representation (Rainville, 2002). Painful stimuli activate many areas of cortex including the thalamus, SI and SII, the insula, and the anterior cingulate cortex (see Figure 7.15)—see Navratilova and Porreca (2014). However, none of those areas seems necessary for the perception of pain. For example, painful stimuli usually elicit responses in SI and SII (see Zhuo, 2008). However, removal of SI and SII in humans is not associated with any change in the threshold for pain. Indeed, *hemispherectomized* patients (those with one cerebral hemisphere removed) can still perceive pain from both sides of their bodies.

The cortical area that has been most frequently linked to pain is the **anterior cingulate cortex** (see Figure 7.15). However, the anterior cingulate cortex appears to be involved in the expectation of pain, the emotional reaction to pain, and adaptive responses to minimize pain—rather than to the perception of pain itself (Shackman et al., 2011).

DESCENDING PAIN CONTROL. The third paradox of pain is that this most compelling of all sensory experiences can be so effectively suppressed by cognitive and emotional factors (see Bushnell, Čeko, & Low, 2013; Senkowski, Höfle, & Engel, 2014). For example, men participating in a certain religious ceremony suspend objects from hooks embedded in their backs with little evidence of pain (see Figure 7.16); severe wounds suffered by soldiers in battle are often associated with little pain; and people injured in life-threatening situations frequently feel no pain until the threat is over.

Thinking Creatively

Figure 7.15 Location of the anterior cingulate cortex in the cingulate gyrus.

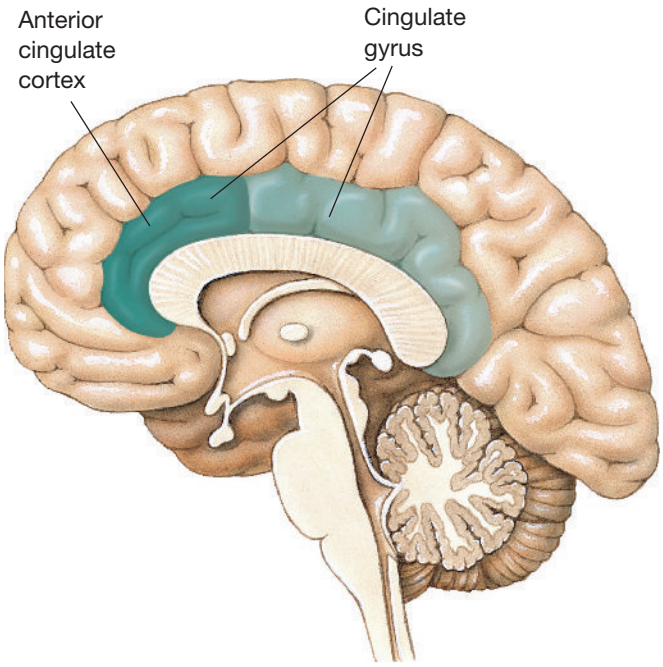


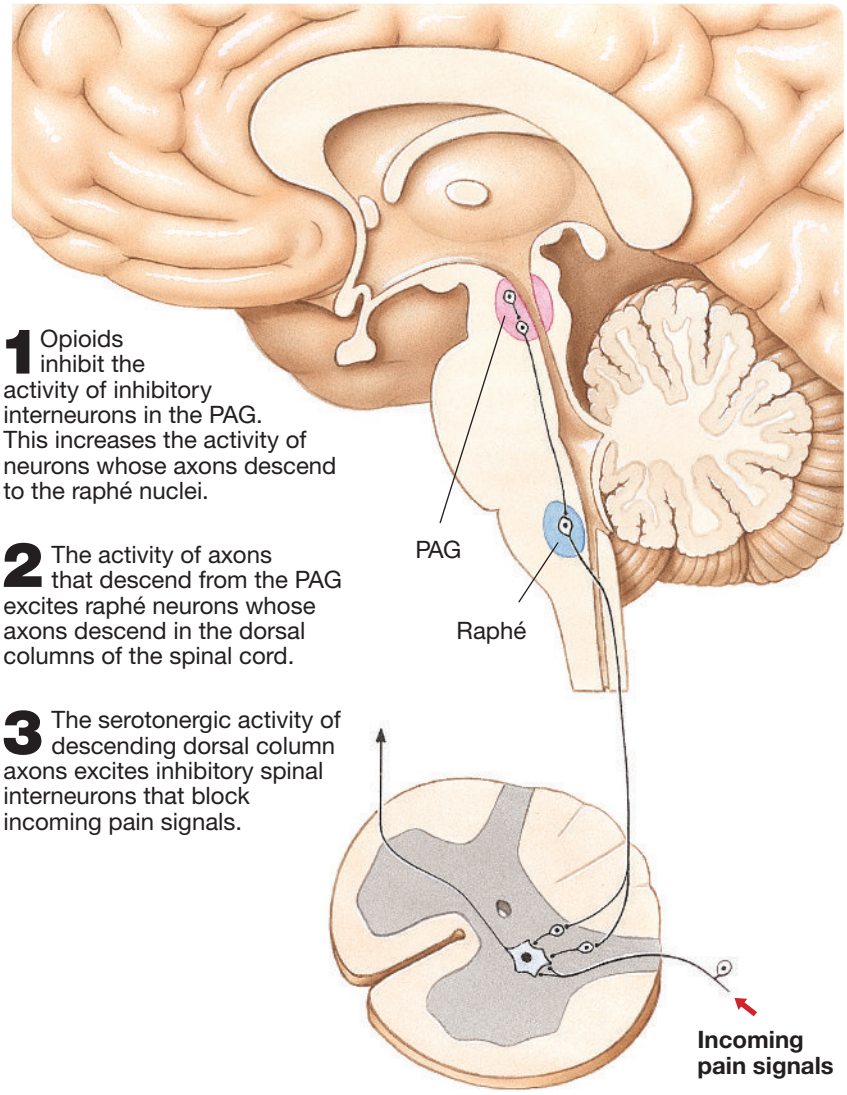
Figure 7.16 When experienced as part of a ritual, normally excruciating conditions often produce little pain.

Three discoveries led to the identification of a descending pain-control circuit. First was the discovery that electrical stimulation of the **periaqueductal gray (PAG)** has analgesic (pain-blocking) effects: Reynolds (1969) was able to perform surgery on rats with no analgesia other than that provided by PAG stimulation. Second was the discovery that the PAG and other areas of the brain contain specialized receptors for opioid analgesic drugs such as morphine. And third was the isolation of several endogenous (internally produced) opioid analgesics, the **endorphins**, which you learned about in Chapter 4. These three findings together suggested that analgesic drugs and psychological factors might block pain through an endorphin-sensitive circuit that descends from the PAG.

Figure 7.17 illustrates the descending analgesia circuit first hypothesized by Basbaum and Fields (1978). They proposed that the output of the PAG excites the serotonergic neurons of the *raphé nuclei* (a cluster of serotonergic nuclei in the core of the medulla), which in turn project down the dorsal columns of the spinal cord and excite interneurons that block incoming pain signals in the dorsal horn.

Descending analgesia pathways have been the subject of intensive investigation since the first model was proposed by Basbaum and Fields in 1978. In order to incorporate the mass of accumulated data, models of the descending analgesia circuits have grown much more complex (see Lau & Vaughan, 2014). Still, a descending component involving endogenous opioid activity in the PAG and serotonergic activity in the raphé nuclei remains a key part of most models (see Mason, 2012).

Figure 7.17 Basbaum and Field's (1978) model of the descending analgesia circuit.



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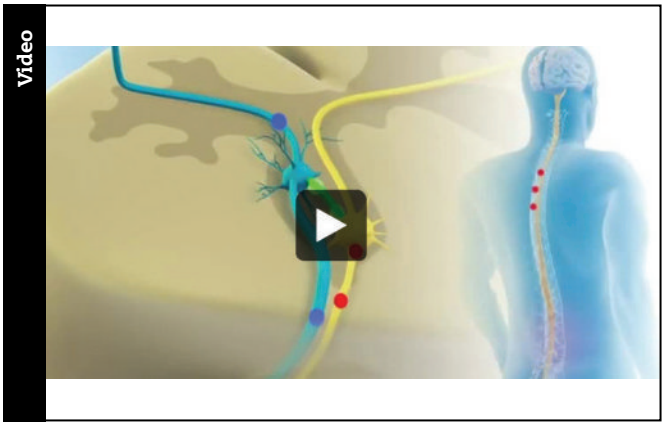
MANAGING PAIN



Neuropathic Pain

LO 7.16 Define neuropathic pain and describe some of its putative neural mechanisms.

In most cases, plasticity of the human nervous system helps it function more effectively. In the case of neuropathic pain, just the opposite is true (see Luo, Kuner, & Kuner, 2014). **Neuropathic pain** is severe chronic pain in the absence of a recognizable pain stimulus. A typical case of neuropathic pain develops after an injury: The injury heals and there seems to be no reason for further pain, but the patient experiences chronic excruciating pain. In many cases, neuropathic pain can be triggered by an innocuous stimulus, such as a gentle touch.

Although the exact mechanisms of neuropathic pain are unknown, it is somehow caused by pathological changes in the nervous system induced by the original injury (see Elman & Borsook, 2016). Recent research has implicated signals from aberrant microglia in neuropathic pain; these signals are thought to trigger hyperactivity in neural pain pathways (Beggs & Salter, 2010; Tsuda et al., 2013).

Neuroplasticity

Although the neuropathic pain may be perceived to be in a limb—even in an amputated limb (see Chapter 10)—it is caused by abnormal activity in the CNS. Thus, cutting nerves from the perceived location of the pain often brings little or no comfort. And, unfortunately, medications that have been developed to treat the pain associated with injury are usually ineffective against neuropathic pain.

Chemical Senses: Smell and Taste

Olfaction (smell) and *gustation* (taste) are referred to as the chemical senses because their function is to monitor the chemical content of the environment. Smell is the response of the olfactory system to airborne chemicals that are drawn by inhalation over receptors in the nasal passages, and taste is the response of the gustatory system to chemicals in solution in the oral cavity.