Emotion and Social Cognition

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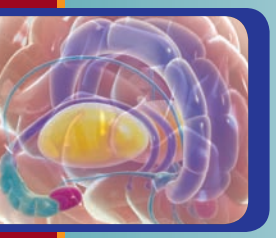


BY MARCH 30, 1981, James Brady had been serving for several months as the press secretary for U.S. President Ronald Reagan. Brady was known and liked by the White House press corps for his wit and energy. For example, during a lunch with reporters, he described a particular government bureaucrat as sleeping "in the closet hanging upside down with his wings over his eyes" (Bumiller, 1982). But March 30 was a terrible day for Jim Brady and all those who knew him. During a gunman's attempt to assassinate President Reagan, Brady took a bullet in the head.

The injury was very severe—in fact, at one point that evening, CBS anchorman Dan Rather mistakenly reported that Brady had died (Bumiller, 1982). Eventually surgeons were able to reduce the swelling and bleeding in his brain so that Brady survived. However, because of the bullet's trajectory, he suffered extensive brain damage to his right frontal lobe (Cytowic, 1981).

Many of the symptoms that Brady experienced are predictable from what we have already learned about the frontal lobes. Brady suffered paralysis of the left arm and leg, consistent with damage to the motor regions in the right frontal lobe. He also displayed cognitive symptoms of frontal lobe damage, such as difficulties with initiating action and a tendency to perseverate in his thought.

But the gunshot wound did not just affect Brady's cognitive functions—it affected his emotional regulation and emotional state as well. In an essay, his wife Sarah Brady wrote: "[S]trong feelings of any kind could bring on what we called a 'wail'—a very unnerving noise somewhere between crying and laughing. As his brain healed, he was increasingly able to control it, and in later years, he would wail only during extremely emotional moments—sad or happy—such as the singing of the national anthem. But in those early days, it happened all the time: He would start to say something, and suddenly his voice would just wail off." At other times, though, Brady was described as speaking in a "slow, measured cadence" (De Witt, 1990) that lacked the emotional inflections of normal speech.

After his injury, Brady also tended to be a bit more brutally honest than people in the political sphere are generally inclined to be. For example, he made highly unflattering remarks about some of his former colleagues in the White House, sometimes making those around him a bit uncomfortable (Bumiller, 1982). Although these tendencies may simply reflect the change in outlook that accompanies a brush with death, they may also reflect a failure of his damaged frontal lobes to inhibit socially inappropriate behavior.

Despite his tragic circumstances, Brady continued to maintain his trademark wit; about John Hinckley, the man whose bullet hit him but missed President Reagan, Brady said, "I think that guy was an awfully bad shot." He and Sarah Brady have dedicated themselves to advocating against gun violence and for the recognition of people with traumatic brain injuries. While James Brady can teach us about the emotional consequences of right frontal lobe damage, he also teaches us a lesson about emotional resilience in the face of tragedy.

IMAGINE A DAY without emotion. According to the psychologist and philosopher William James, living without emotion would require a person to "drag out an existence of merely cognitive or intellectual form." James thought this rather undesirable, arguing that "[s]uch an existence, although it seems to have been the ideal of the ancient sages, is too apathetic to be keenly sought after by those born after the revival of the worship of sensibility" (James, 1884, p. 194). Though emotions can be destructive, they also bring vitality to our lives; they permeate nearly all aspects of our thoughts, decisions, and interactions with other people.

This chapter surveys different components of emotion and the brain systems that are important in implementing them. First, though, we must be able to answer a basic question: What is an emotion? It has been said that everyone knows what an emotion is until they are asked to define it (LeDoux, 1996). The *American Heritage Dictionary of the American Language* defines *emotion* as "1. Agitation of the passions or sensibilities often involving physiological changes. 2. Any strong feeling, as of joy, sorrow, reverence, hate, or love, arising subjectively rather than through conscious mental effort." At first glance, this description seems to capture our everyday sense of the concept of emotion. However, when we examine this definition more closely, we begin to appreciate the complexity of trying to understand emotion.

This definition assumes some things about emotion that still arouse heated debate in the scientific literature. For instance, what does it mean for a feeling to arise "subjectively rather than through conscious mental effort"? Indeed, much of the processing that is associated with emotion seems to occur outside conscious awareness. We may know how to describe a feeling we

are experiencing, but we are often unaware of how that feeling was generated. Nevertheless, most of us would recognize that conscious mental effort can play a role in generating or maintaining emotions. Imagine, for example, the jealous lover who dwells on thoughts of his beloved in someone else's arms, making himself more miserable in the process.

Another contentious issue is whether physiological changes in the periphery of the body play an important role in emotion. Emotional experience is associated with changes in heart rate, blood pressure, skin temperature, and electrodermal response (the degree to which the skin conducts electricity depending on the amount of perspiration). For example, imagine the emotion of fear: you probably associate it with bodily sensations such as a racing heart and sweaty palms. But are these bodily changes just a side effect of consciously experiencing an emotion, or do they in fact bring about the conscious experience? This issue has been debated ever since William James posed the question more than 100 years ago (James, 1884).

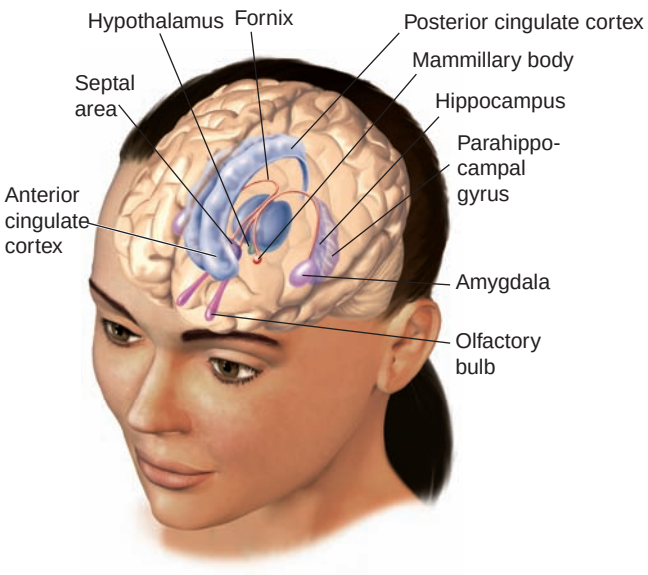
A final thorny problem involves the relationship between cognition and emotion; that is, between "thinking" and "feeling." People often assume that cognition and emotion are independent and even mutually exclusive—that rational thought runs counter to emotional impulses, or that more thinking involves less feeling and vice versa. But when we consider the many aspects of emotion and the neural systems that implement emotion, we will see that it is often difficult to draw sharp boundaries between cognition and emotion. For example, when we recognize a person's facial expression of happiness, are we using a cognitive system that decodes visual patterns, or are we using an emotional system that categorizes stimuli as pleasant or unpleasant? Does making adaptive choices in life depend upon elaborate rational thought, or upon instinctive understanding of the dangers and rewards of different choices? When a person's attention is captured by the sound of a scream in the distance, is that an emotional or a cognitive process? Although we will learn that certain brain systems are deeply involved in emotional functions, the brain does not divide neatly into two categories of "emotional" and "cognitive" regions, just as our psychological functions cannot be sharply divided into these two categories.

When you think of all the complexities subsumed in the term *emotion,* it should not surprise you to learn that there is no single brain region that serves as the emotion center. Rather, many different brain regions contribute to the experiences that we call emotion, which is sometimes also referred to as *affect* or *affective experience*. Some of these brain regions are concerned with specific emotions, such as fear or pleasure; others are concerned with specific processes, such as recognizing emotion in facial expressions or integrating emotion with cognitive processing. Our challenge is to work toward an understanding of how all these different brain regions work in concert to allow the full range of emotional experiences and abilities that we enjoy. This chapter focuses primarily on the emotions that most people feel every day, and it also lays the groundwork for Chapter 14, which examines disorders of emotion.

Subcortical Contributions to Emotion

Many emotions are uncomfortable to experience. However, their survival value is obvious. When a person is threatened, the body needs to mobilize its resources and take some kind of protective action: withdrawal (flight), perhaps, or aggression (fight). Furthermore, these responses often must be made quickly. As a result, they are often made before a person has time to perform any elaborate, conscious, cognitive assessments of the situation. In our survey of the brain regions involved in emotion, we begin by discussing the subcortical regions that implement these more automatic or subconscious aspects of emotion.

As long ago as 1937, James W. Papez (rhymes with "grapes") described a subcortical brain circuit involved in emotion that included the hypothalamus, hippocampus, anterior thalamus, and cingulate cortex. Paul MacLean (1949, 1952) later proposed that these structures are part of what was termed the **limbic system** (meaning "border" or "belt"), which consists of a series of structures that sit below the neocortex (● Figure 13.1). Although investigators agree that emotions depend on



● **FIGURE 13.1 The major structures of the limbic system.** The limbic system forms a belt sitting below the neocortex, consisting of a wide variety of structures that have been implicated in emotional processing. © 2010 Cengage Learning

the limbic system, scientists' ideas about exactly which structures constitute this system have changed over time (Brodal, 1998). For example, the hippocampus, once thought to be the hub of the limbic system, plays an important role in memory functions. In contrast, the amygdala, which in the past was not identified as a key component of the limbic system, has received a great deal of attention from neuroscientists who study emotion (LeDoux, 1996). In this section, we consider what is currently known about the role of subcortical structures in crucial emotional functions.

Fight-or-Flight Response

As we have already discussed, emotional experiences often include bodily changes, such as an increased heart rate or sweaty palms. The body's fight-or-flight response depends upon the autonomic nervous system (● Figure 13.2), which consists of nerves that contact body organs such as the heart, the lungs, and the sweat glands. The hypothalamus governs the level of activity in the autonomic system, determining the extent to which the fight-or-flight response is activated. Activation of the sympathetic branch of the autonomic system causes increases in heart rate, blood pressure, respiration, and sweat secretion.

The hypothalamus also controls the hormonal systems of the body. For example, through its interactions with the pituitary gland, the hypothalamus influences the level of stress hormones in the body (● Figure 13.3). When stimulated by the hypothalamus, the pituitary gland releases hormones into the bloodstream. These pituitary hormones can affect target organs such as the adrenal glands, which in turn produce stress hormones like adrenaline and cortisol. Therefore, because the hypothalamus governs both the autonomic and hormonal systems of the body, it serves as an important gateway through which the brain can influence the state of the body.

So, how does the hypothalamus know when to kick the body's fight-or-flight response into high gear? How does it determine when a threatening event is present, for example? Such decisions appear to be determined by the amygdala, another subcortical limbic region that sends its outputs to the hypothalamus. We consider the role of the amygdala in fear and other emotions in the next section.

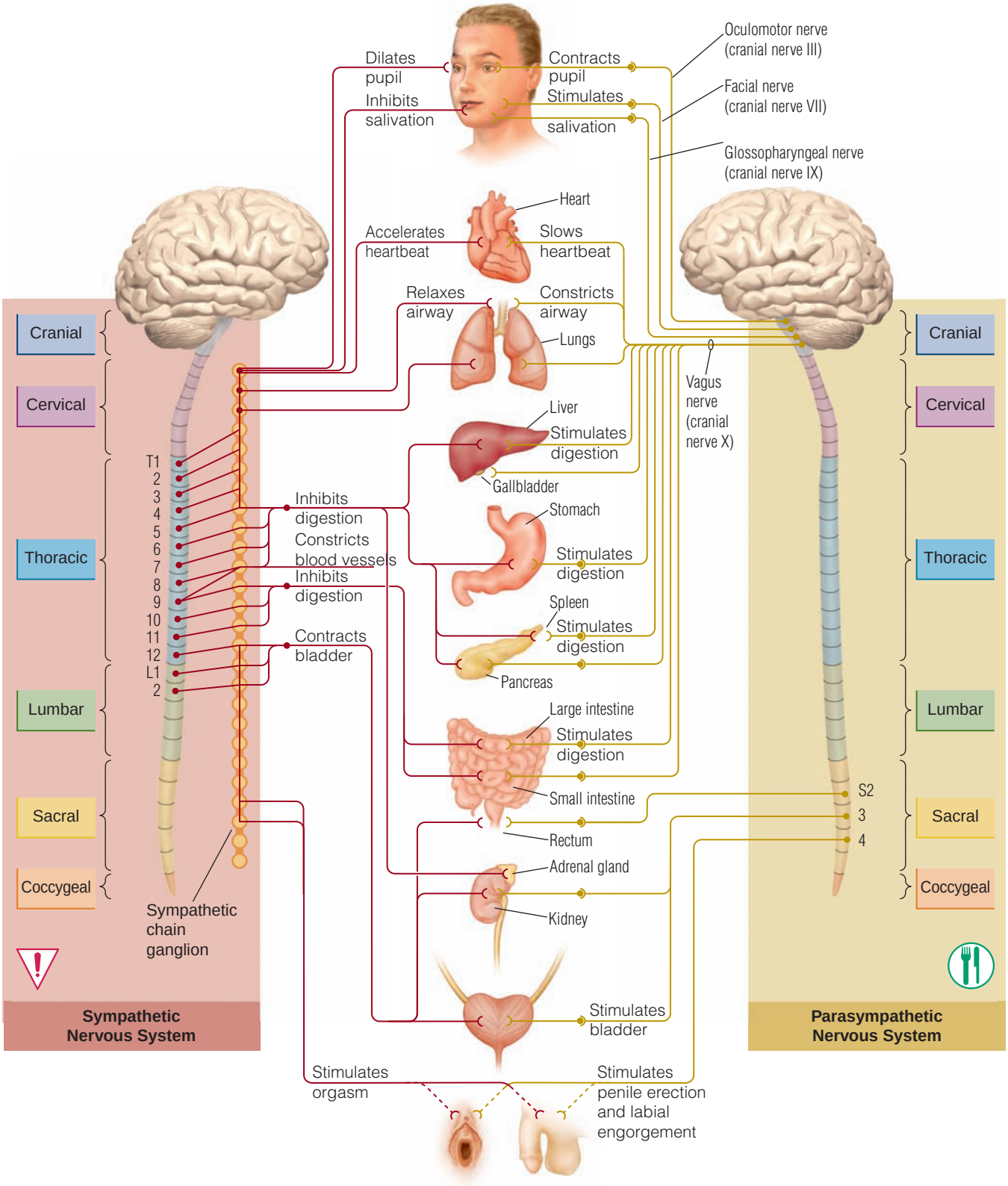
Fear and Emotional Learning

The amygdala plays an important role in early detection of emotional information and in learning the emotional significance of information. ● Figure 13.4 shows the location and subdivisions of the amygdala. Although it is a small structure, the amygdala consists of several identifiable and interacting nuclei. Some researchers refer to this region as the *amygdalar complex*, a phrase intended to capture the complicated nature of the region. A somewhat simplified description of this region notes that the basolateral nuclei project to the hippocampus and prefrontal cortex, as well as brain regions involved in reward and punishment, allowing the amygdala to influence learning and memory. The central nucleus and corticomedial nuclei connect to the hypothalamus and other brain regions involved in autonomic and hormonal responses, enabling emotional modulation of these responses (see Freese & Amaral, 2009, for more detailed anatomy).

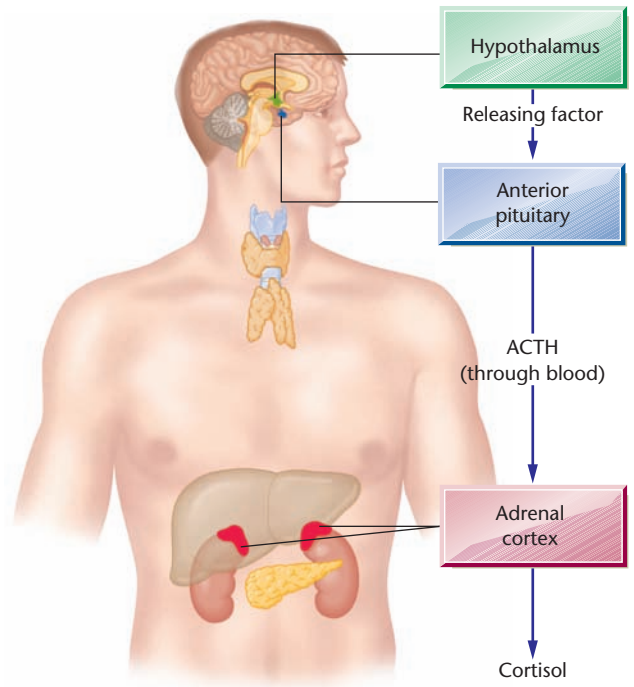
Scientists first became aware of the role of the amygdala when it was discovered that large temporal-lobe lesions in monkeys resulted in a set of behavioral changes known as *Klüver-Bucy syndrome*. These monkeys showed extremely abnormal reactions to the environment. They stopped being afraid of things they had feared in the past, attempted to engage in sexual behaviors with other species, and tried to ingest objects indiscriminately, including feces and rocks. Klüver and Bucy (1937) used the term **psychic blindness** to describe the disconnection between the animals' ability to process the sensory properties of objects and their understanding of the affective properties of these same objects. These initial studies involved the removal of the entire temporal lobes, including both the cortex and the subcortical areas such as the amygdala, but subsequent research found that amygdala damage alone could produce many of these behavioral changes (e.g., Emery, Capitanio, Mason, Machado, Mendoza, & Amaral, 2001; Machado, Kazama, & Bachevalier, 2009).

Lesions of the amygdala in humans also interfere with the processing of emotional information, though the effects are not as dramatic as with Klüver and Bucy's monkeys. Case studies of people with amygdala damage indicate that they lose the ability to detect aversive emotional cues embedded in visual and auditory stimuli. They have difficulty identifying fearful facial expressions as well as fearful or angry sounds (Aggleton & Young, 2000); they even have trouble recognizing scary music (Gosselin, Peretz, Johnsen, & Adolphs, 2007). When such patients are asked to judge faces for trustworthiness and approachability, they rate unfamiliar photographs as more trustworthy and approachable than neurologically intact individuals do (Adolphs, Tranel, & Damasio, 1998).

Neuroimaging studies provide converging evidence about the amygdala's role in responding to emotionally salient information. Activity in the human amygdala is increased in response to fearful compared to neutral faces (e.g., Dolan & Morris, 2000). Not surprisingly, the amygdala is also activated in people with phobias when they are exposed to their feared object (e.g., spiders or snakes) (Larson, Schaefer, Siegle, Jackson, Anderle, & Davidson, 2006; Phan, Fitzgerald, Nathan, & Tancer, 2006). Currently there is debate about whether the amygdala responds to fearful images even when those images are presented outside of conscious awareness. Some researchers have found that the amygdala does respond to subconsciously presented images (Whalen,



● **FIGURE 13.2 The autonomic nervous system.** Activation of the sympathetic branch of the autonomic nervous system is important in many bodily expressions of emotion, such as changes in heart rate, respiration, and sweat secretion. In contrast, the parasympathetic branch is activated under resting conditions. © 2010 Cengage Learning

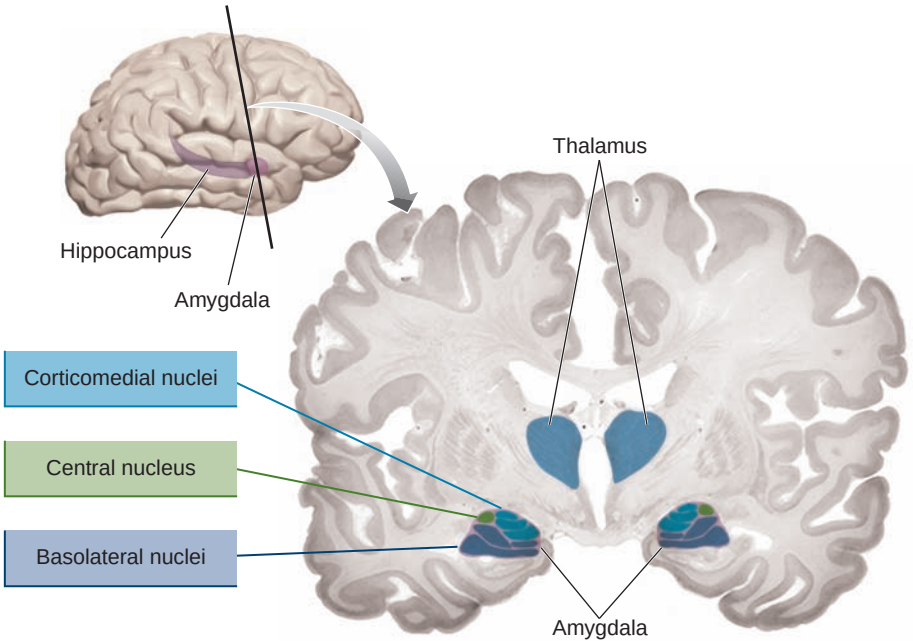


● **FIGURE 13.3 The HPA axis.** The brain controls the body's stress response through a loop that connects the hypothalamus, pituitary gland, and adrenal glands. When stimulated by the hypothalamus, the pituitary gland secretes adrenocorticotropic hormone (ACTH) into the bloodstream, and this hormone stimulates the adrenal gland to produce the stress hormone cortisol. © 2009 Cengage Learning

Rauch, Etcoff, McInerney, Lee, & Jenike, 1998; Whalen et al., 2004), but others question such claims (Pessoa, Japee, Sturman, & Ungerleider, 2006).

The amygdala is especially involved in emotional learning, as demonstrated repeatedly in studies of fear conditioning. As discussed in Chapter 10, in fear conditioning paradigms a neutral stimulus develops a negative emotional connotation by virtue of its association with an aversive stimulus (● Figure 13.5). After pairing a neutral image with a very unpleasant noise, for example, people will eventually respond to the previously neutral image as if it were inherently aversive. This emotional response is reflected in physiological responses such as heart rate, skin conductance, and the *startle response,* which is a blink that occurs when a puff of air is blown into a person's eye.

Damage to the amygdala is known to disrupt fear conditioning in humans, as well as in other mammalian species. In one study, neurologically intact people, a patient with bilateral amygdala damage, and a patient with hippocampal damage were shown repeated pairings of a specific color slide with an unpleasant noise (Bechara, Tranel, Damasio, Adolphs, Rockland, & Damasio, 1995). After conditioning, neurologically intact people reacted to the slide by showing increased skin conductance. Although the patient with amygdala damage was able to remember the pairing explicitly



● **FIGURE 13.4 The amygdaloid complex.** The amygdala is made up of several interrelated nuclei. The basolateral nuclei project to the hippocampus and prefrontal cortex, as well as regions involved in reward and punishment (including the caudate nucleus and nucleus accumbens), allowing the amygdala to influence learning and memory. The basolateral nuclei also project to the central nucleus and corticomedial nuclei, which have a different pattern of connections. They connect to the hypothalamus and other brain regions involved in autonomic and hormonal responses, allowing the amygdala to perform emotional modulation of these responses. © 2010 Cengage Learning

(e.g., "I know that the blue slide is the one with the shock"), she did not show the expected autonomic conditioned response. In contrast, the patient with hippocampal damage showed normal conditioned skin-conductance responses, but was unable to explicitly remember that the blue slide led to the shock! This is an example of a classic double dissociation, linking the amygdala with acquired fear responses and the hippocampus with explicit memory.

The amygdala is also important in learning fear through words rather than just through direct experience of an aversive consequence. For example, if you brought your hand very close to an electrical socket, you would probably show an elevated skin-conductance response, indicating activation of the sympathetic nervous system. To learn that response, you didn't need to actually stick your finger in the socket and experience the shock. Instead, you probably developed the response through verbal learning: when you were young, your parents told you not to stick your fingers in sockets. Recently, studies have demonstrated that this kind of verbal learning depends on the amygdala. In one experiment, participants were shown different colored squares and simply told that one specific color could be associated with a shock (although no shock actually occurred). When participants viewed that specific color, the left amygdala became activated (Phelps, O'Connor, Gatenby, Gore, Grillon, & Davis, 2001). Another study found that damage to the left (but not the right) amygdala disrupted verbal learning of fear (Olsson & Phelps, 2007). These studies indicate that the left amygdala is especially important in verbal learning of fear responses, which fits in well with other evidence of the left hemisphere's involvement in language.

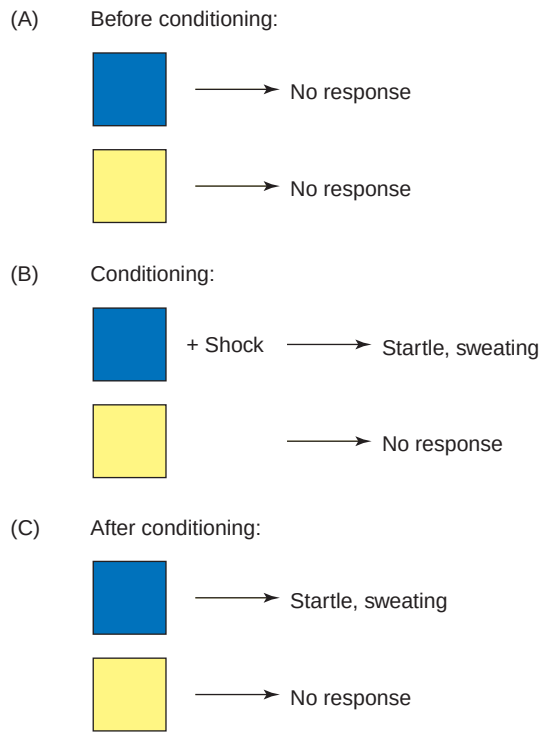


FIGURE 13.5 An example of fear conditioning. (A) Prior to conditioning, an emotionally neutral item, such as a blue or yellow slide, does not lead to a fear response. (B) During conditioning, one neutral item, such as the blue slide, is repeatedly paired with a shock, which produces a fear response including a startle reaction and increase in skin sweating. (C) After conditioning, the blue slide presented alone will produce a conditioned fear response (namely, the startle and skin sweating). Damage to the amygdala impairs the ability to acquire conditioned fears.

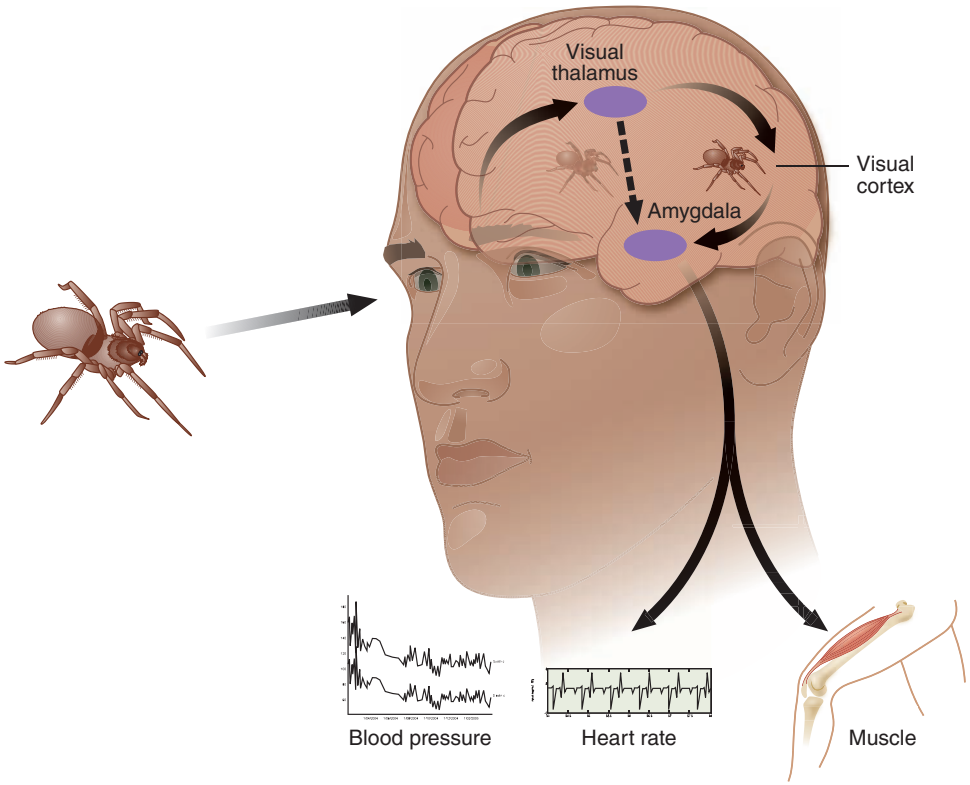
Because fear learning is so easily studied in many species, for some time scientists tended to focus on fear learning as a model for understanding emotion more generally. However, subsequent research has found that damage to the amygdala disrupts not only fear learning, but also certain types of reward-based learning in rodents and primates (Baxter & Murray, 2002; Murray, 2007). Several neuroimaging studies have also found that the amygdala is more responsive to happy faces than to neutral faces, indicating that positive emotional images can activate this structure as well (e.g., Breiter et al., 1996; Williams, Morris, McGlone, Abbott, & Mattingley, 2004). However, the response of the amygdala to positive stimuli may be somewhat less reliable than its response to negative stimuli (see Zald, 2003, for a review).

One factor driving this difference in response to positive and negative stimuli is the arousal level of the stimuli. Negative stimuli, such as pictures of angry faces, snakes, or spiders, tend to be rated as more highly arousing than positive stimuli, such as pictures of happy faces or puppies. Studies using olfactory stimuli (pleasant and unpleasant odors) found that amygdala activity increased as the intensity level of the stimulus increased, regardless of whether it was pleasant or unpleasant (Anderson et al., 2003; see also Small, Gregory, Mak, Gitelman, Mesulam, & Parrish, 2003, for similar results with pleasant and unpleasant tastes). However, researchers are still debating whether the amygdala's response is better explained by the valence of the stimulus—how pleasant or unpleasant it is—or by its emotional intensity level. For example, one study using pleasant and unpleasant pictures and sounds found that valence was a stronger determinant of amygdala response than arousal level (Anders, Eippert, Weiskopf, & Veit, 2008), inconsistent with the results from studies of odors and tastes. In another study, patients with damage to the amygdala rated the arousal level of negative pictures lower than did control participants, although they did not differ from controls in arousal ratings of positive pictures (Berntson, Bechara, Damasio, Tranel, & Cacioppo, 2007). While there is still much to be learned about the dimensions of emotional meaning that the amygdala encodes, it is clear that both

emotional valence and intensity level are important factors in driving its response.

Given the amygdala's role in emotional learning, the brain must have some way in which sensory information from the outside world can be sent to the amygdala to enable such learning. In fact, there are two distinct pathways that convey sensory information to the amygdala (● Figure 13.6) (Armony & LeDoux, 2000). One pathway, which is important for quick, instinctive emotional responses, projects straight from the anterior thalamus to the amygdala. For example, this pathway allows a jogger to leap away from a shape on the road before the conscious mind has time to think, "That might be a snake." Another pathway connects the sensory areas of the neocortex to the amygdala. This pathway provides a more comprehensive context for processing emotional information. For example, after leaping to safety, the jogger might study the shape more carefully and realize that it is only a stick, not something to be feared. Thus, the amygdala appears to receive a progressively more complete image of the same information, much like a fade-in shot in the movies that becomes progressively clearer and more focused with time. The thalamo-amygdaloid pathway carries a crude, preliminary sketch of some basic properties of the stimulus—not enough to clearly identify the object, but enough, perhaps, to ready or initiate a response. In contrast, the cortico-amygdaloid pathway, which is slower because it involves more synapses, delivers enough information to give rise to an affective reaction that takes into account the complexity and details of the situation.

This model emphasizes how incoming sensory information can influence the amygdala. When the amygdala registers something fearful or frightening, though, it is also important for that information to be taken into account by other brain regions. This is accomplished by additional connections running in the opposite direction from the amygdala to the cortex. These back-projecting fibers are thought to allow the amygdala to influence how attention is directed to different aspects of sensory information as they are processed by the cortex. Once the amygdala identifies an image as threatening or otherwise emotionally urgent, it can tell the cortex to pay more attention to that image. In neurologically intact people, attention tends to be captured by emotional stimuli (for reviews, see Compton, 2003;



● **FIGURE 13.6 Two pathways by which sensory information reaches the amygdala.** One pathway from the thalamus to the amygdala provides basic sensory information very quickly, whereas the longer route involving the cortex provides more highly processed and detailed information. Output from the amygdala can then influence autonomic and hormonal responses. Although not depicted here, the amygdala can also influence how incoming sensory information is processed through backward projections from the amygdala to cortical regions. *Source:* LeDoux, J. (2002) Emotion, memory, and the brain. Scientific American, special issue on The Hidden Mind.

Vuilleumier, 2005), but patients with amygdala damage do not show such attentional effects (Anderson & Phelps, 2001).

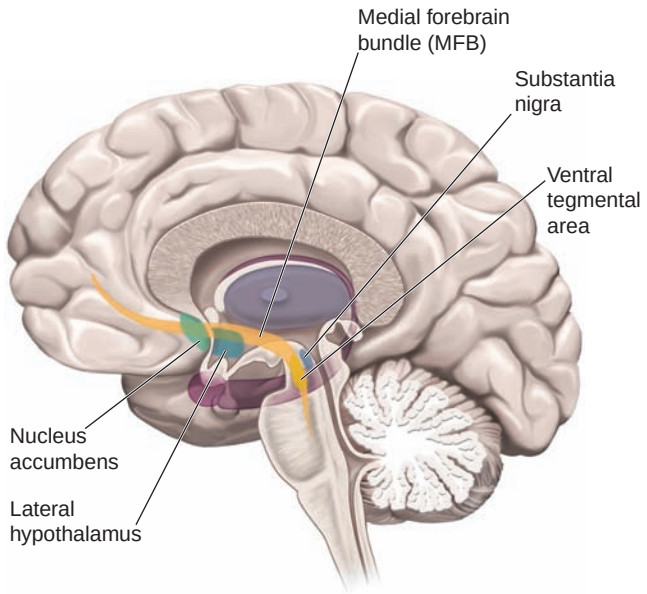
The amygdala also interacts very closely with another important subcortical structure, the hippocampus. As we reviewed in Chapter 10, the hippocampus is crucial in encoding new information into long-term memory storage and in consolidating that information in memory over time. Close bidirectional interactions between the hippocampus and the amygdala allow them to influence one another's activity in several ways (see Phelps, 2004, for a review). For example, input from the amygdala to the hippocampus can allow the emotional meaning of a stimulus (coded by the amygdala) to influence the encoding and subsequent consolidation of that information by the hippocampus.

Indeed, the amygdala plays an important role in remembering events that are emotionally charged (LaBar & Cabeza, 2006). Normally, the greater the emotional intensity associated with an event or experience, the better it is remembered, a phenomenon known as the *memory enhancement effect.* Amygdala damage interferes with this memory enhancement effect (Adolphs, Cahill, Schul, & Babinsky, 1997; Cahill, Babinsky, Markowitsch, & McGaugh, 1995). Using PET to investigate this pattern further, researchers found that better memory for emotional versus neutral film clips was correlated with higher glucose metabolism in the right amygdala (Cahill et al., 1996; see also Dolcos, LaBar, & Cabeza, 2004). Furthermore, a pharmacological manipulation that reduced connectivity between the amygdala and hippocampus led to a reduction in the memory enhancement effect (Alkire, Gruver, Miller, McReynolds, Hahn, & Cahill, 2008).

In sum, while it is clear that the amygdala plays an important role in responding to salient emotional events and in emotional learning, we also know that it does not work in isolation. Rather, the amygdala's unique role in emotional functions comes about by virtue of its interactions with interconnected brain regions. These brain regions include higher-level areas involved in perception and memory, such as the sensory cortices and hippocampus, as well as lower-level areas that implement the fight-or-flight response, such as the hypothalamus.

Reward and Motivation

Although positive emotions have sometimes been neglected by scientists interested in emotion, there is a research tradition focused on pleasure and its close cousin, motivation for rewards. In the 1950s, Olds and Milner carried out experiments demonstrating that electrical stimulation to certain parts of the brain was "rewarding" for a rat. But what do we mean when we say the rats found the stimulation "rewarding"? Olds and Milner (1954) found that the rats would press a lever hundreds and hundreds of times to activate a current in certain brain regions. Because the rats would work so hard for this stimulation, the researchers inferred that it was rewarding. The areas where stimulation is most rewarding are the dopaminergic pathways stretching from the ventral tegmental area of the midbrain to a cluster of cells in the basal forebrain known as the **nucleus accumbens** (● Figure 13.7). This region is also referred to as the *ventral striatum,* because it is the ventral part of the basal ganglia (striatum).



● **FIGURE 13.7 The location of the nucleus accumbens.** The nucleus accumbens receives ascending dopaminergic input from the ventral tegmental area. © 2010 Cengage Learning

It is tempting to refer to the reward pathway as the "pleasure center" of the brain, but caution is required here. Just because an animal presses a lever repeatedly for stimulation, does that mean the animal gets pleasure from it? Researchers have argued that "wanting" and "liking" can be dissociated. An analogy might be a cocaine addict who goes to great lengths to obtain the drug (i.e., wanting), but no longer experiences pleasure once she takes it (i.e., liking). Some researchers have proposed that the dopaminergic path leading to the core of the nucleus accumbens is not responsible for pleasure itself, but for the "wanting" aspects of reward-related behavior—those aspects that propel an animal toward desired goals (Berridge & Robinson, 1998, 2003). In contrast, only a certain part of the nucleus accumbens—specifically, a layer of cells surrounding the accumbens and referred to as the *nucleus accumbens shell*—is thought to underlie the sensation of consummatory pleasure upon achieving a desired goal; that is, the "liking" (● Figure 13.8) (Berridge, 2003).

Many studies of the reward pathways have focused on nonhuman animals, but neuroimaging studies have also examined the conditions under which the nucleus accumbens is activated in humans. (Note that because

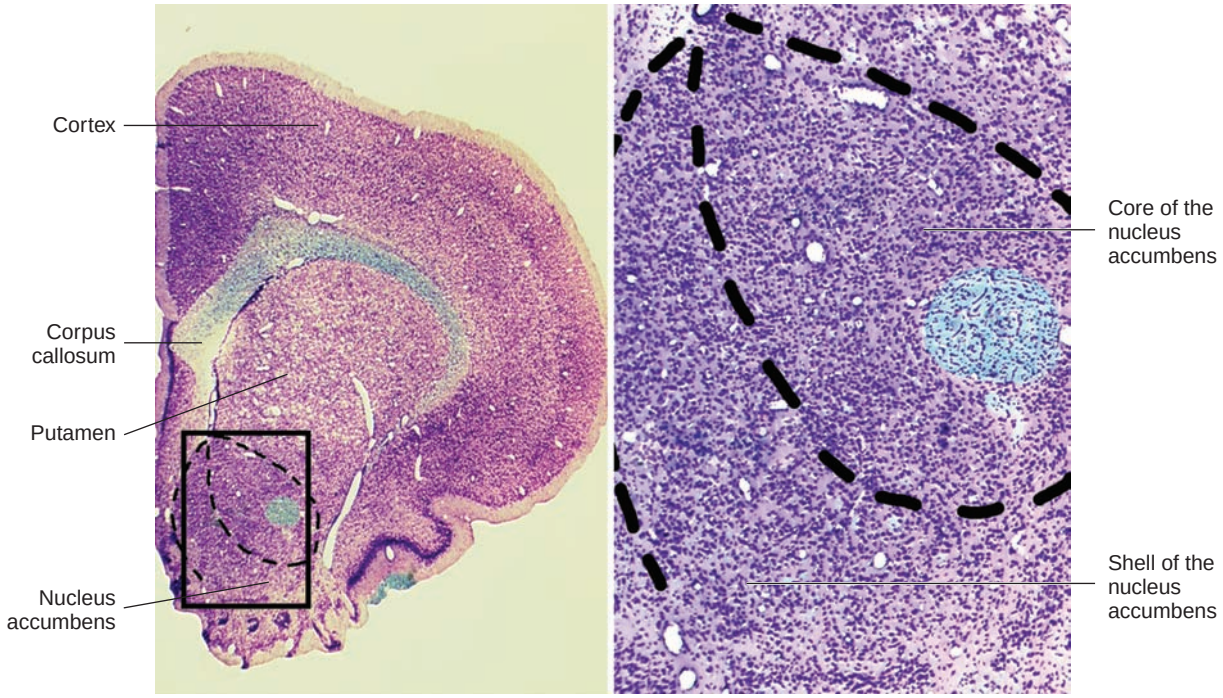


FIGURE 13.8 The nucleus accumbens core and shell regions in the rat brain. The core of the nucleus accumbens is thought to play a role in wanting or desire incentive, whereas the shell is thought to play a role in consummatory pleasure. Courtesy of Harold Prüss

of limitations in the spatial resolution of imaging techniques, most studies of humans are not able to distinguish between the core and the shell of the accumbens.) Interestingly, the accumbens becomes activated in people when they receive a reward, especially when the rewards are unexpected (Berns, McClure, Pagnoni, & Montague, 2001). As you may remember from Chapter 2, dopaminergic responses in the accumbens are largest under such conditions. In contrast, in situations with predictable rewards, the accumbens is activated when the person anticipates the reward before actually receiving it (Knutson, Adams, Fong, & Hommer, 2001). These results suggest that the accumbens is initially sensitive to unexpected rewards; however, as a pattern of rewards emerges, the accumbens begins to anticipate the reward (for similar results in single-cell studies with monkeys, see Fiorillo, Tobler, & Schultz, 2003; Hollerman & Schultz, 1998).

The nucleus accumbens is activated by many stimuli that could be considered rewarding, such as sweet juice (Berns et al., 2001), money (Knutson, Westdorp, Kaiser, & Hommer, 2000), and attractive faces (e.g., Aharon, Etcoff, Ariely, Chabris, O'Connor, & Breiter, 2001; Kampe, Frith, Dolan, & Frith, 2001). This region is also activated by rewarding items that are addictive. For example, smokers show greater accumbens responses to smoking-related imagery than do nonsmokers (David et al., 2005). For obvious reasons, clinically oriented research on the ventral striatum has centered on its role in addiction, to which we will return in Chapter 14.

Cortical Contributions to Emotion

Having just examined the roles that subcortical regions play in emotion, we now turn our attention to the functions of cortical regions. The cerebral cortex is crucial for emotional functions such as deciding whether a particular behavior is likely to lead to a positive outcome, inferring the feelings of others based on facial expression, and using the correct tone of voice to convey to others how we are feeling. Cortical regions are also important in representing bodily signals of emotion, as we will soon learn.

Representing Bodily Cues of Emotion

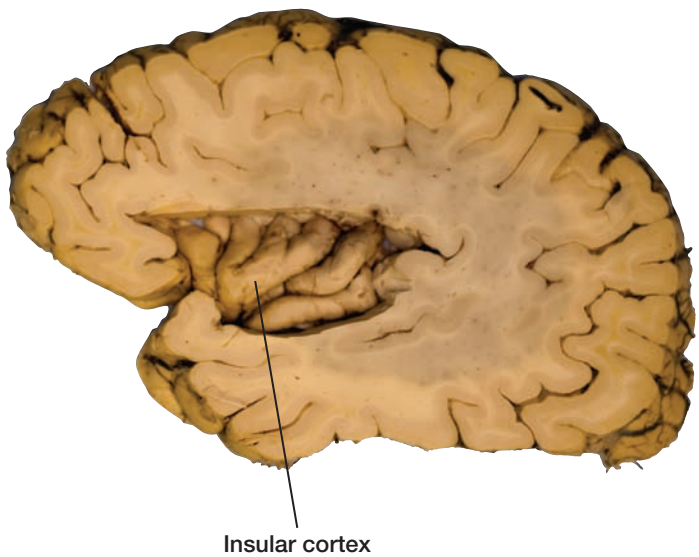
More than 100 years ago, the pioneering psychologist William James argued that conscious experience of an emotion depends upon the ability to mentally represent

the state of the body. According to James, bodily signals provide the brain with information about the emotion that the body is experiencing, and therefore give rise to conscious emotional feelings. The finer points of James's theory have long been debated (see Ellsworth, 1994). Although researchers disagree about whether it is absolutely necessary for the brain to receive information from the rest of the body before a person can feel an emotion, it is true that our minds are able to represent our bodily states in some way. For example, you might be able to tell that you are anxious because you can perceive that your heart is beating very fast.

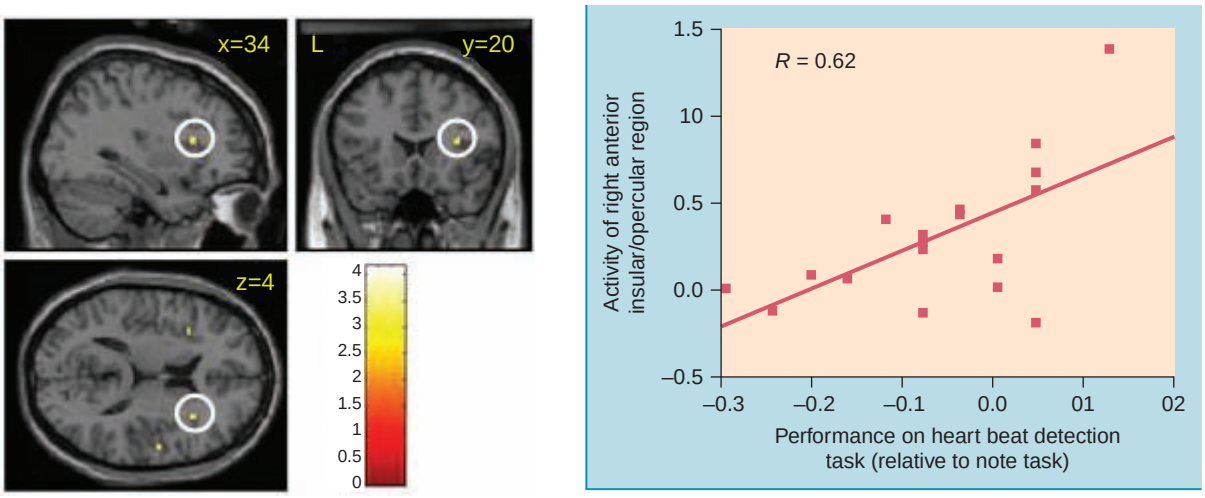
It is important to distinguish between the *control* of bodily states of emotion and the ability to *represent* those states mentally. As already discussed in an earlier section, the hypothalamus is involved in regulating autonomic functions (for example, controlling whether heart rate is high or low). However, the ability to perceive the internal state of the body, a function known as **interoception**, appears to depend upon another region, the *insular cortex* (or *insula*) (Craig, 2002, 2009; Verhagen, 2007). The insula is tucked deep inside the Sylvian fissure (● Figure 13.9), and its anterior region has extensive connections to other structures involved in emotion, including the amygdala and orbitofrontal cortex.

One study of the insula's role in interoception examined participants' ability to detect their own heartbeats (Critchley, Rotshtein, Öhman, & Dolan, 2004). The researchers found that activation was enhanced in the insula during this task, compared to a control condition that involved detecting external stimuli (● Figure 13.10). In addition, people who were more accurate at detecting their own heartbeats had a right insula that was both bigger and more active compared to people with poor accuracy at the task. These data imply that the insula plays an important role in encoding interoceptive cues.

Whereas some research indicates that the insula is important in representing a variety of internal bodily cues of emotion, other research emphasizes its special role in the emotion of disgust. Interestingly, research with nonhuman primates indicates that part of the insula serves as the primary gustatory (taste) area. What does taste have to do with emotion? One clue is provided by the term *disgust,* which literally means "bad taste." Though disgust is a sensation that we associate with rotten food or foul odors, the term has broader significance as well. As Charles Darwin noted, the facial expressions we make in situations of moral repulsion are the same as those we make when recoiling from disgusting food (Darwin, 1873). Researchers have confirmed Darwin's observation by demonstrating that the same facial expression muscles (levator labii muscles) were activated when people tasted unpleasant liquids, viewed photographs of contaminants such as feces or insects, or experienced unfair treatment in a social game (Chapman, Kim, Susskind, & Anderson, 2009).



● **FIGURE 13.9 Location of the insula.** The insula is a cortical region tucked between the frontal and temporal lobes. Here, a lateral section of the brain has been dissected away to reveal the insula. *Source:* Adapted by permission from Macmillan Publishers, LTD. Fig 1 from Craig, A. D. 2009). How do you feel now? The anterior insula and human awareness. Nature Reviews Neuroscience, 10, 59–70.



(A) (B)

● **FIGURE 13.10 Activation of the insula during an interoceptive judgment task.** Participants had to determine whether a series of tones matched their own heartbeat. Those who performed better at the task (compared to performance on a control task involving detection of notes) had more activity in the right insula. Panel A shows the right insula region (in white circle) and panel B shows the correlation between activity in that region and performance. Adapted by permission from Macmillan Publishers, LTD: Fig 3 (a and b) from Critchley, H. D., Rotshtein, P., Ohman, A., & Dolan, R. J. (2004). Neural systems supporting interoceptive awareness. Nature Neuroscience, 7, 189–195. Figure 13.10a & b.

Several lines of research link the insula to disgust. Early studies performed during brain surgery found that stimulation of the insula in humans elicited sensations of unpleasant taste and nausea (Penfield & Faulk, 1955). Neuroimaging studies show that this area is sensitive to processes related to feeding, such as odor, taste, tongue stimulation, swallowing, thirst, and hunger (Small, Zatorre, Dagher, Evans, & Jones-Gotman, 2001). Damage to the insula interferes with both the experience of disgust and the ability to recognize facial expressions of disgust in others (Calder, Keane, Manes, Antoun, & Young, 2000). Likewise, neuroimaging studies have demonstrated activity in the anterior insula when the participant tastes bitter liquids, imagines disgusting scenarios, or sees another person expressing disgust (Jabbi, Bastiaansen, & Keysers, 2008). Additional neuroimaging studies have shown that activity in the insula is correlated with subjective ratings of disgust (e.g., Schienle, Schafer, & Vaitl, 2008; Stark et al., 2007).

How should we integrate these different findings about the insula? The human insula is anatomically complex, and researchers have proposed that posterior regions represent primary sensory representations (such as taste) and more anterior insular regions integrate these sensations with awareness (Craig, 2009; see also Taylor, Seminowicz, & Davis, 2009). A related possibility is that the insula originated as an area that represented taste, but then expanded to represent other bodily signals of emotion, such as heart rate, temperature changes, pain, and visceral sensations. In humans, this region may play a role in even more complex and abstract emotions. For example, one study found that the insula was active when participants imagined a personal event involving the most guilt they had ever experienced (Shin et al., 2000). Although there is clearly not a "guilt center" in the brain, feelings of guilt may involve some of the same interoceptive cues as sensations of disgust, nausea, or other bodily displeasure.

Monitoring for Emotionally Salient Events

From an evolutionary standpoint, it is crucial to be on the lookout for events in the world that could be either advantageous or disadvantageous—the appearance of a dangerous predator or the sight of a juicy fruit. As we have already learned, certain subcortical regions play a role in these basic motivations. That is, the amygdala is especially involved in responding to emotionally arousing stimuli, and the reward pathways are crucial in responding to positive incentives. In addition, another brain structure, the cingulate cortex, appears to be crucial in monitoring for events that have emotional significance and in integrating motivational aspects of behavior.

The cingulate cortex has been viewed as a component of the limbic system since Broca first described *le grande lobe limbique* in 1878 (for a review, see Allman, Hakeen, Erwin, Nimchinsky, & Hof, 2001). The cingulate wraps around the corpus callosum like a collar, or *cingulum* (● Figure 13.11). Traditionally, it is divided into two regions: the anterior cingulate cortex, forward of the central gyrus, and the posterior cingulate cortex, behind the central gyrus.

Because the cingulate is located on the medial surface of the brain, it is rarely damaged in isolation. As a result,

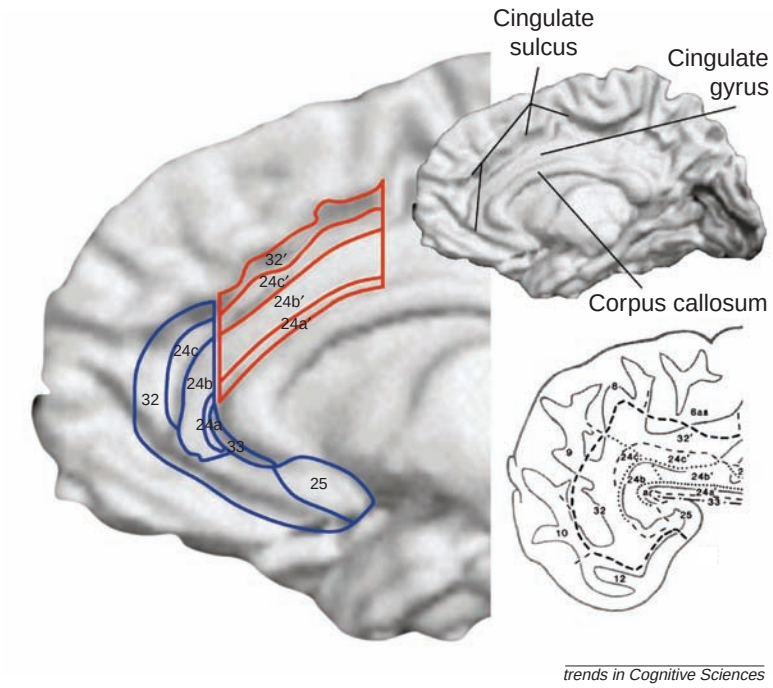


FIGURE 13.11 Cingulate cortex is involved in cognition and emotion. The cingulate lies directly above the corpus callosum. The anterior cingulate cortex has two main parts, a dorsal part (shown in red) and a rostral/ventral part (shown in blue). The rostral portion is more consistently implicated in emotional functions. *Source:* Fig 1 from Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. Trends in cognitive sciences, 4, 215–222. Reprinted by permission of Elsevier.

for many years its organization was a relative mystery. More current research suggests that it has an intricate organization with as many as nine or more distinct subregions (Beckmann, Johansen-Berg, & Rushworth, 2009), the functions of which are still being hotly debated. Despite these debates, a good generalization is that the cingulate is a region where emotion, cognition, and motor control interface (see Paus, 2001, for a review). For example, one theoretical view is that the anterior cingulate cortex is involved in selecting motor actions, considering both the cost and effort entailed in those actions, and weighing how much reward has been gained by taking those actions previously (Rushworth, Buckley, Behrens, Walton, & Bannerman, 2007).

What is clear, however, is that lesions of the anterior cingulate cortex can result in a variety of emotional sequelae, including apathy, inattention, emotional lability, and changes in personality and social interaction (Bush, Luu, & Posner, 2000; Hadland, Rushworth, Gaffan, & Passingham, 2003). In addition, the cingulate is also involved in pain (Tracey, 2005), receiving input from subcortical structures that have neurons specialized to respond to noxious stimuli. Patients who received small cingulate lesions as a treatment for pain reported that the pain still existed but no longer bothered them as much (Cohen et al., 1999). Some portions of the anterior cingulate appear to discriminate between the presence or absence of a painful stimulus but are not sensitive to pain intensity, whereas other portions appear to code the intensity of a painful stimulus (Büchel, Bornhovd, Qunate, Glauche, Bromm, & Weiler, 2002). Fascinating biofeedback research has shown that, in at least some situations, people can use information regarding the activity of their own anterior cingulate to control the intensity of the pain they experience. Whether such a biofeedback technique can be used more commonly in clinical practice remains an open question, but this research provides a potential new means of helping people who experience chronic pain (deCharms et al., 2005).

Certain portions of the cingulate appear to have distinct roles. In particular, researchers have made a distinction between the dorsal and rostral portions of the anterior cingulate cortex (see Figure 13.11) (Bush, Luu, & Posner, 2000). The rostral portion is also sometimes called the *subgenual portion,* because it sits underneath the genu or knee of the callosum. The rostral portion of the anterior cingulate appears to be more intimately involved in emotional functions than the dorsal portion. Anatomically, the rostral cingulate is connected to many other emotion-related areas, including the amygdala, the hypothalamus, the insula, and

the orbitofrontal cortex. Imaging studies suggest that the rostral region is especially activated by tasks that have an emotional component (Bush, Luu, & Posner, 2000; Mohanty et al., 2007). Activity in this region is also correlated with changes in the autonomic nervous system (e.g., Critchley, Tang, Glaser, Butterworth, & Dolan, 2005; Matthews, Paulus, Simmons, Nelesen, & Dimsdale, 2004), and has been linked to depression, as we will learn in Chapter 14. In contrast, the dorsal portion of the cingulate has connections with lateral prefrontal cortex, parietal cortex, and motor areas. As we learned in Chapter 12, this region is more involved in cognitive function, especially executive function.

The dorsal and rostral subdivisions of the anterior cingulate may relate to one another in a reciprocal fashion at times. During cognitive task performance, activity often decreases in the rostral division while increasing in the dorsal division; during emotional conditions, activity often increases in the rostral division while decreasing in the dorsal division (Drevets & Raichle, 1998). These results suggest that there may be a reciprocal dynamic between emotion and cognition, with strong emotion functioning to shut down certain cognitive systems and vice versa. This notion seems intuitively appealing, as many of us have experienced for ourselves how an emotional state can interfere with paying attention to a nonemotional task. Conversely, many of us have also had occasion to "lose ourselves in our work" for the purpose of coping with an emotional stress or trauma.

These findings should not be taken, however, to suggest a strict and rigid dichotomy between the functions of the two cingulate subregions, as other evidence suggests that cognition and emotion may not be so easily separable (Compton et al., 2003; Davis et al., 2005; Phan, Wager, Taylor, & Liberzon, 2002). Indeed, it is probably no coincidence that the rostral and dorsal regions are highly interconnected with one another. That is, it may be adaptive for the rostral region, which responds to the emotional salience of events, to influence the dorsal region, which is involved in governing executive attention. After all, it could make sense to allocate attention depending on the emotional significance of the information at hand.

Studies of the error-related negativity (ERN), which is generated by the cingulate cortex, illustrate the complexity of teasing apart cognitive and emotional functions in the cingulate cortex. As discussed in Chapter 12, the ERN is an electrical response that occurs when a person detects that he or she has made an error, or when a person receives negative feedback about performance. Influential theories describe the ERN as part of a system of cognitive control, a signal that indicates when outcomes are worse than expected (Holroyd & Coles, 2002). Because an error is usually an unpleasant outcome, we could think of the error signal as an emotional signal. In this sense, the fact that the ERN is generated by the cingulate cortex fits with the idea that the cingulate is involved in monitoring for emotionally salient events. At the same time, an error signal also indicates the need for a change in attention or behavior, so as to avoid repeated mistakes. Some source-localization studies suggest that the ERN is generated by the dorsal or "cognitive" subdivision of the cingulate, while others point toward localization in the rostral or "emotional" subdivision (e.g., Herrmann, Römmler, Ehlis, Heidrich, & Fallgatter, 2004; Mathalon, Whitfield, & Ford, 2003; Taylor et al., 2006; van Veen & Carter, 2002). In the end, it may be useless to try to pigeonhole phenomena such as the ERN as either strictly cognitive or strictly emotional, both because the boundaries between cognition and emotion are somewhat artificial and because evaluation of one's behavior involves both cognitive and emotional components.

The cingulate cortex clearly acts as a central hub for both emotional and cognitive processing, but other cortical brain regions are important in specific functions that involve both cognition and emotion. These functions include the influence of emotion on decision making, the top-down regulation of emotion, and the communication of emotion through facial and vocal cues. We discuss each of these functions in turn in the next few sections.

Incorporating Emotion into Decision Making

Common sense tells us that emotions affect decision making. When deciding how to spend your Saturday evening, your choices will be affected by memories of activities that you found to be either pleasant or unpleasant in the past. When choosing to vote for a political candidate, your decision may be influenced in part by the candidate's emotional appeals. Although the influence of emotion on decision making is sometimes considered troublesome because it is "irrational," some researchers argue that emotional signals are actually important cues that effectively guide us toward outcomes that benefit us and away from outcomes that harm us (e.g., Damasio, 1994).

The brain region most implicated in integrating emotion and decision making is the **orbitofrontal cortex (OFC)**. A recent review described the OFC as "among the least understood regions of the human brain" (Kringelbach, 2005, p. 691), partly because of differences in this region across species and notable variation in its anatomical structure from one person to the next. In addition, the OFC includes several different subareas whose functional distinctions are not yet clear. Here we use the term *OFC* to include both regions that directly overlie the eye orbits and areas that extend into the medial wall of the frontal lobes, an area that is sometimes referred to as the *ventromedial prefrontal cortex* (● Figure 13.12). Anatomical connections imply that the OFC is important in emotion, as this region is reciprocally interconnected with many other emotion-related structures such as the hypothalamus, amygdala,

insula, and cingulate cortex. Although the overarching function of the OFC is still in dispute, current research implies that this region plays a role in understanding rewards and punishments and using that understanding to guide adaptive behavior.

Case studies have shown that people with damage to the OFC exhibit disinhibited behaviors (e.g., grabbing things they want from others), socially inappropriate behaviors (e.g., blurting out tasteless remarks), and irresponsibility. They seem to have difficulty anticipating the consequences of their actions, they make poor decisions that result in negative outcomes, and they do not seem to learn from their mistakes (Bechara, Damasio, Damasio, & Anderson, 1994; Rolls, Hornak, Wade, & McGrath, 1994). Bechara and colleagues (1994) have termed this behavior "myopia for the future." These behaviors are especially remarkable because the patients show no deficits in intellectual ability as measured with standard IQ tests. Some researchers have even suggested that the OFC may provide the substrate for the development of moral behavior, comparing people with OFC damage to those with the psychiatric disorder of psychopathy, a failure of empathy often seen in violent criminals (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999).

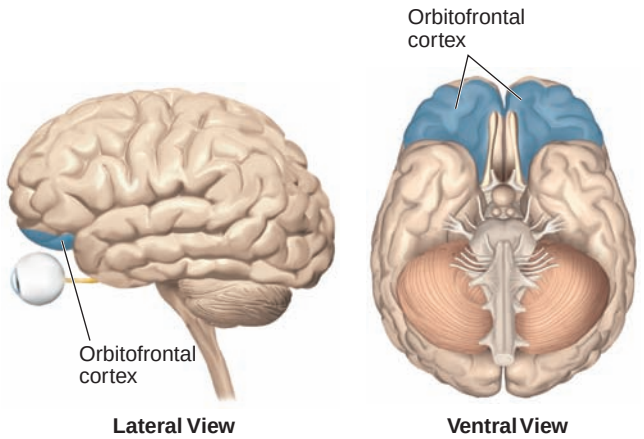


FIGURE 13.12 Location of orbitofrontal cortex. The orbitofrontal cortex is so named because it lies directly above the eye sockets, or orbits. Sometimes the medial portion of the orbitofrontal cortex is referred to as *ventromedial prefrontal cortex*.

People with damage to the OFC perform especially poorly on tasks in which past losses and gains must be considered in order to make appropriate choices in the present. Such deficits have been empirically demonstrated using gambling tasks, in which the participant must choose a particular stimulus that results in either winning or losing money (Bechara, Damasio, & Damasio, 2000; O'Doherty, Kringelbach, Rolls, Hornak, & Andrews, 2001). These tasks are designed so that people cannot simply associate one stimulus with one outcome; rather, the tasks are designed to work probabilistically, so that over time some choices tend to be better than others. In a gambling task, a person might win big by choosing a particular item, but continuing to choose that item over time results in a series of small losses, thus making this choice less profitable than an alternative choice. People with damage to the OFC tend stick with the "big win" stimulus even though it leads to greater losses over time. This behavior resembles that of a child who cannot resist the impulse to eat a huge piece of cake despite knowing that later on it will lead to an upset stomach. As we discuss in more detail in Chapter 14, similar impairments in decision making are also evident in substance abuse, in which people often make decisions on the basis of immediate gratification while ignoring the long-term consequences.

The OFC is especially important for learning in situations that require the individual to respond to changing patterns of reward and punishment. Researchers often study this phenomenon by varying what is called the **reinforcement contingency**, which simply refers to the degree to which a reward or punishment is associated with a particular stimulus or action. Single-cell recording studies in nonhuman primates show that neurons in the OFC respond to the rewarding value of taste, smell, and visual stimuli, and that some neurons respond only when the reinforcement contingencies change (Rolls, 1999). People with OFC damage are impaired in the ability to change their behavior when the contingencies change. One example of such contingency change is referred to as **reversal learning**. For example, let's say you were first rewarded for pressing the left button in response to a red light and the right button in response to a green light. In reversal learning, you are now rewarded for pressing the left button for the green light and the right button for the red light. Reversal learning is deficient following OFC damage in humans and other primates (Roberts, 2006). Neuroimaging studies also support the idea that the OFC tracks the changing reward value of a particular stimulus. For example, food becomes less rewarding as a person becomes satiated (full). Correspondingly, OFC activity decreases as the food becomes less desirable with satiation (Kringelbach, O'Doherty, Rolls, & Andrews, 2003).

Recent studies suggest that different subregions of the OFC respond to rewards versus punishments, an organization that may help the OFC to keep track of changing contingencies (Kringelbach, 2005). The lateral area of the OFC is activated following a punishing outcome in a gambling task, whereas the medial area is activated following a rewarding outcome (O'Doherty et al., 2001). These two regions appear to act in a reciprocal manner: the medial region increases activation to reward and decreases activation to punishment, whereas the lateral orbitofrontal region exhibits the opposite pattern. Furthermore, the larger the reward or punishment delivered, the greater the brain activation. An intact OFC therefore allows us to represent the costs and benefits

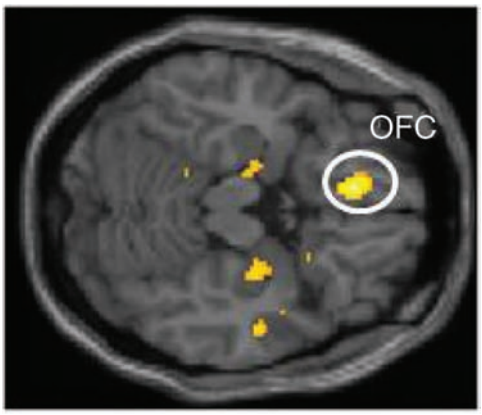
associated with any choice, leading to more informed and effective decision making.

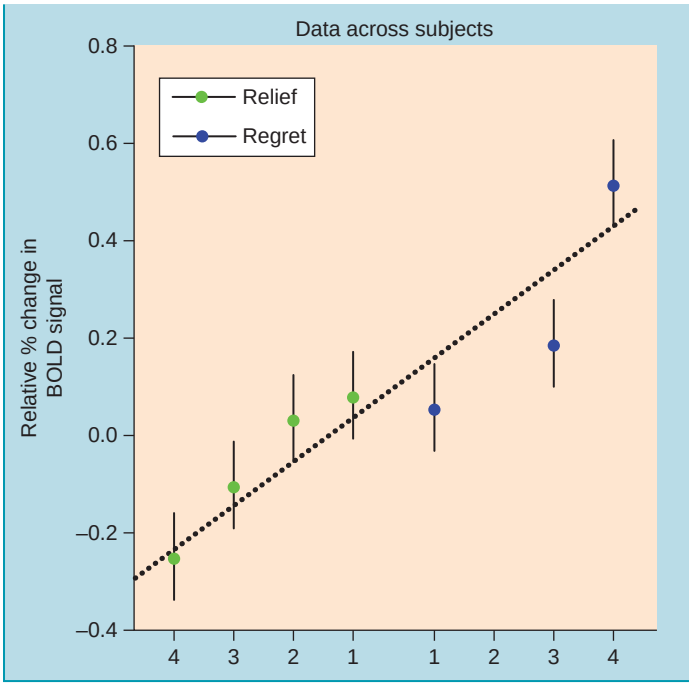
The OFC is also crucial for evaluating the consequences of our choices. One of the ways that we think about the consequences of our own decision making is to consider what might have happened if we had made a different choice. Would I have been happier if I had bought the Honda rather than the Chevy? When we discover that we made the "wrong" choice, we often feel regret. Interestingly, patients with OFC damage do not appear to feel regret (Camille, Coricelli, Sallet, Pradat-Diehl, Duhamel, & Sirigu, 2004). Neuroimaging research with neurologically intact people has also found that the OFC is active in situations of regret. For example, the OFC becomes activated when participants learn that a choice they rejected would have led to a greater benefit (● Figure 13.13) (Coricelli, Critchley, Joffily, O'Doherty, Sirigu, & Dolan, 2005). OFC activity is especially tied to situations in which participants felt agency (responsibility) for the choice, rather than instances in which the undesired outcome was simply a matter of chance. When participants in this study were faced with similar choices again, the OFC became reactivated in anticipation of the choice, presumably as participants reconsidered the regrettable consequences of their previous actions.

Regulating Emotion

An important aspect of emotion is being able to control it. If you've ever cheered yourself up after a bad day, suppressed your anger after a friend made an unfair or callous remark, or practiced meditation to help relieve stress, you've engaged in some form of emotion regulation. Although the term is conceived rather broadly, **emotion regulation** generally refers to attempts to manage the emotions that one experiences, so that they are socially appropriate and do not spiral out of control. Emotion regulation may be disrupted in certain clinical conditions, such as mood disorders. Although many strategies for emotion regulation are conscious, voluntary efforts, emotion regulation may take place at an unconscious level as well.

Studies using ERP methods have shown that emotion regulation strategies can influence how the brain responds to emotional information. Several of these studies have investigated the effect of reappraising





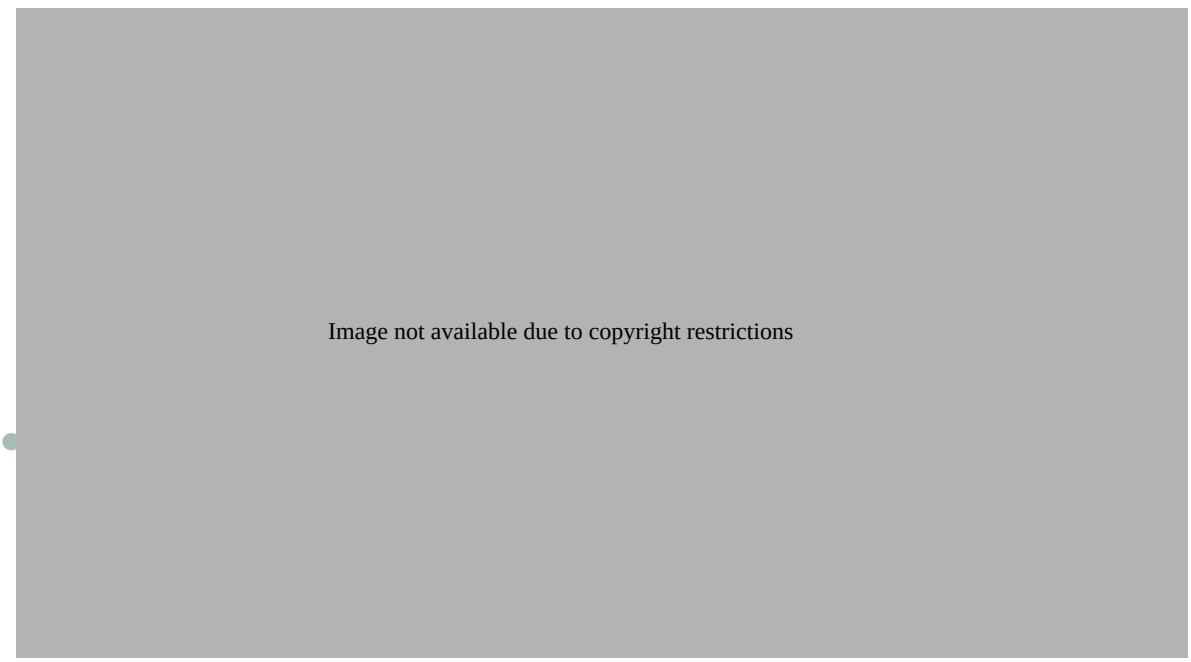
● **FIGURE 13.13 Orbitofrontal cortex responds to degrees of regret in a decision-making task.** Levels of regret (1–4) are defined by the discrepancy between what a participant earned from a particular choice and what she might have earned had she made a different choice. For example, level 4 of regret represents a condition in which the participant discovered that she lost 200 points when she could have gained 200 points if she had picked the other option. Level 1 of regret represents a condition in which the participant lost 50 points but could have earned 50 points. Levels of relief represent conditions in which the participant learned that she made the right choice, such as by earning 200 points when she could have lost 200 points (level 4 of relief) or by earning 50 points when she could have lost 50 points (level 1 of relief). Activity in the orbitofrontal cortex tracks the value of the actual choice relative to the value of the path not taken. Adapted by permission from Macmillan Publishers, LTD: Coricelli, G., Critchley, H. D., Joffily, M., O'Doherty, J. P., Sirigu, A., & Dolan, R. J., (2005). Regret and its avoidance: A neuroimaging study of choice behavior. Nature Neuroscience, 8, 1255–1262. Figure 13.13.

emotional pictures in less emotional ways. For example, participants might see a picture of a snarling dog with teeth bared; however, instead of thinking about how frightening it would be to run into such an animal, they would be instructed to try to view the picture in a more positive way, such as by imagining that the dog was protecting them from an intruder. Negative pictures usually produce a significantly larger P_{300} response in the ERP waveform, compared to neutral pictures, but engaging in reappraisal lessened this effect (Hajcak & Nieuwenhuis, 2006; see also Foti & Hajcak, 2008). Follow-up research demonstrated that the P_{300} response to positive pictures could also be lessened by reappraisal strategies, paralleling the effects shown with negative pictures (Krompinger, Moser, & Simons, 2008). Because the P_{300} is thought to reflect allocation of attention, these studies imply that less attention was allocated to the pictures when participants attempted to view them in a less emotional manner.

According to several studies, when people try to control their emotional responses, activity increases in frontal-lobe regions and decreases in subcortical regions that would normally process that emotion. For example, one study showed sexually provocative pictures to men and instructed them to suppress their sexual arousal responses to the pictures. In this suppression condition, brain activity increased in the right superior frontal gyrus and decreased in the hypothalamus and amygdala, compared to a simple viewing condition (Beauregard, Levesque, & Bourgouin, 2001) (● Figure 13.14). Likewise, when participants were instructed to reevaluate disturbing pictures in a way that would reduce their negative feelings, frontal-lobe activity increased and amygdala activity decreased (Ochsner, Bunge, Gross, & Gabrieli, 2002). Other investigators found that when participants were required to suppress emotional memories, the right inferior and middle frontal gyrus regions became more active, and hippocampal and amygdalar regions less active (Depue, Curran, & Banich, 2007).

Some studies have attempted to distinguish between brain regions that serve as the source of signals to regulate emotional responsiveness and brain regions that serve as targets over which such control is exerted. For example, imagine a situation in which you are about to experience a painful medical procedure. You may try to reduce your anxiety by mentally detaching yourself (for example, by imagining that you are lying on a cozy blanket in a lovely field of flowers with warm sunshine beaming down on you). Such strategies tend to lessen perceived pain. What brain regions are involved in generating the "detachment" experience (the source of emotional control), and how do those brain regions affect the regions that would normally code for pain (the target of control)?

In a study addressing this issue, Kalisch and colleagues (2005) instructed participants to imagine being in a "special place" while knowing that a painful shock might soon be delivered. During the period of anticipation, right lateral prefrontal cortex activity was increased when participants imagined the "special place," compared to control conditions in which no emotion regulation was encouraged; thus, this region was inferred to be the source of emotion regulation. In turn, when pain was actually administered, activity in the anterior cingulate region (which normally responds to pain) was lessened if the participant had used the emotional regulation strategy, indicating that this region was the target of emotion regulation.



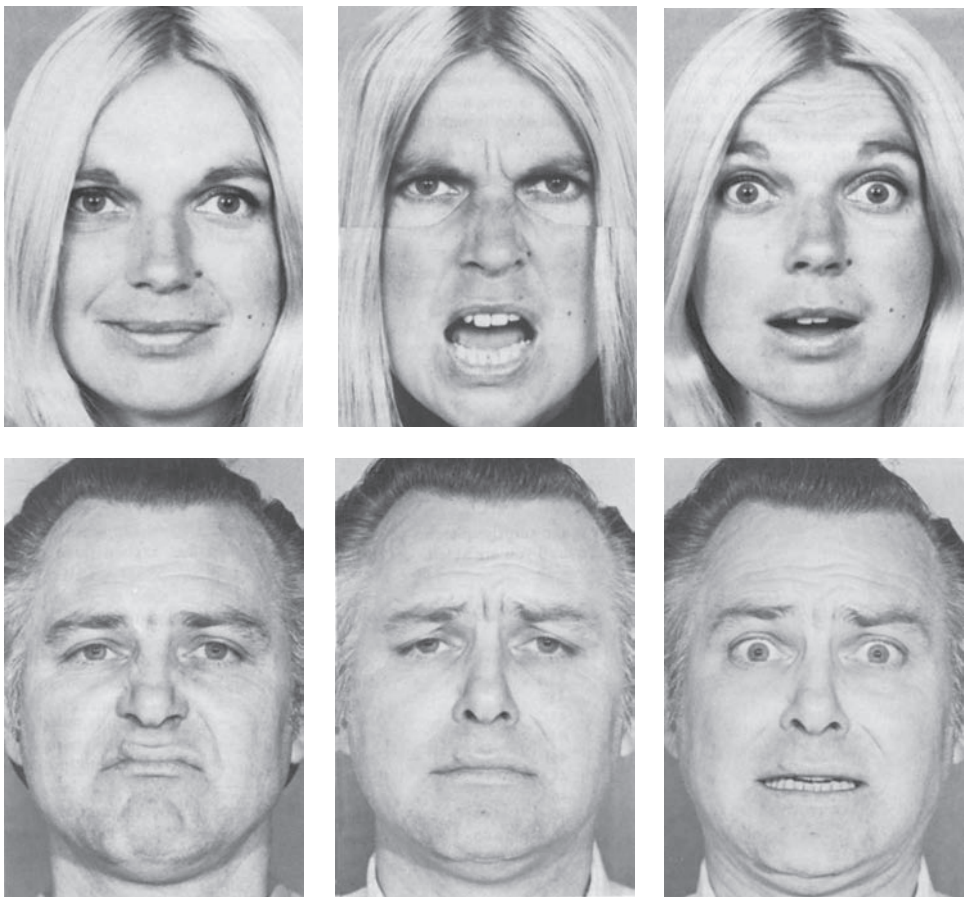
Most research on emotion regulation has focused on suppressing unwanted emotional responses, but future research may also help us to understand how positive and negative responses can be intentionally amplified (see Kim & Hamann, 2007; Ochsner et al., 2004). Though much remains to be learned about emotion regulation, these studies so far tell us that when people adopt voluntary strategies of emotional control, they change how their brains respond to emotional situations.

Communicating Emotion

Our emotions are not just felt internally; they are also conveyed to other people. Although we tend to think of language as the dominant means of communication in our species, nonverbal signals of emotion communicate important information among members of a social group. If you meet a friend and notice that her facial expression is angry, you will interact with her differently than if her face bears a happy expression. Likewise, a phrase such as "Susan and Bill have just eloped" can convey very different sorts of information depending on whether it is spoken in an excited, surprised, sad, or angry tone of voice. In the next two sections, we consider the neural systems involved in both perceiving and producing expressions of emotion.

Facial Expressions

The ability to produce and recognize facial expressions of emotion is nearly universal. Cross-cultural studies have found that similar facial expressions are used across a wide range of cultures to convey basic emotions such as happiness, sadness, anger, fear, surprise, and disgust (● Figure 13.15) (e.g., Ekman, Sorenson, & Friesen, 1969; Elfenbein & Ambady, 2002), although there are some differences across cultures in the exact way that expressions are formed and the social contexts in which they are considered appropriate (e.g., Marsh, Elfenbein, & Ambady, 2003; Matsumoto, Yoo, Hirayama, & Petrova, 2005). Nonetheless, the strong similarity of basic expressions across cultures implies that these expressions are rooted in our species' common biological heritage, as recognized by Charles Darwin more than a century ago (Darwin, 1873). So, what do we know about the neural mechanisms that recognize and produce expressions?



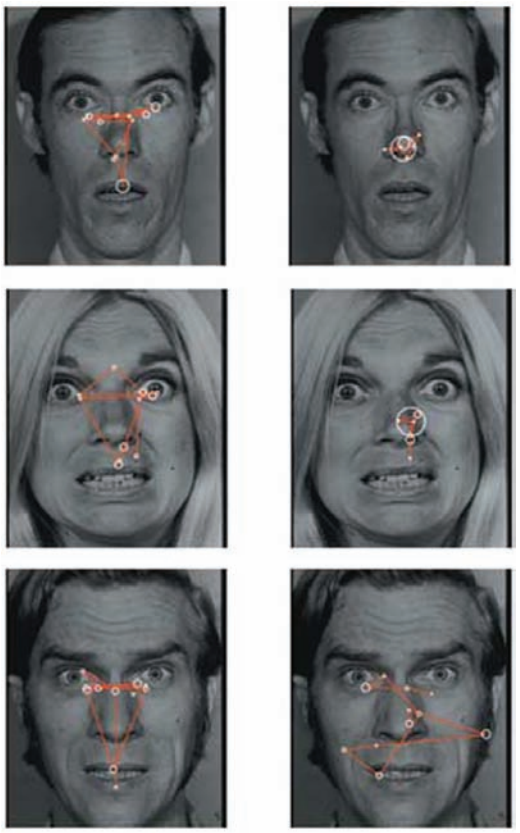
Dr. Paul Ekman

● **FIGURE 13.15 Facial expressions that are universally recognized.** From left to right, the top row shows expressions of happiness, anger, and surprise, and the bottom row shows disgust, sadness, and fear.

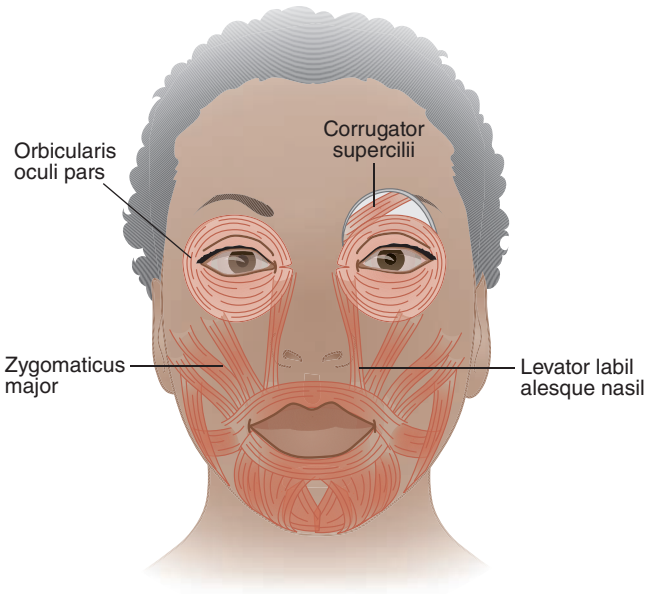
One of the most reliable findings in cognitive neuroscience is the right-hemisphere specialization for both recognizing and producing facial expressions of emotion. Right-hemisphere damage, particularly to temporal and parietal regions of the brain, disrupts the ability to recognize faces much more than does comparable left-hemisphere damage. Borod and coworkers (1998) found that patients with right-hemisphere damage were more impaired than left-hemisphere-damaged patients in tasks that required the patient to name or point to the correct label for an emotional expression depicted on a slide (see also Cicone, Wapner, & Gardner, 1980; DeKosky, Heilman, Bowers, & Valenstein, 1980). The most severe impairments in emotion recognition have been attributed to damage of the right parietal cortex. However, right anterior temporal lobectomy, a treatment for medically intractable epilepsy, has also been shown to cause impairments in processing emotional information, especially negative emotion in faces (see Adolphs, Tranel, & Damasio, 2001). Consistent with these findings from brain-damaged patients, divided visual field studies typically find that people recognize facial expressions of emotion better when the faces are presented to the left visual field (right hemisphere) than to the right visual field (left hemisphere) (e.g., Ladavas, Umilta, & Ricci-Bitti, 1980; Strauss & Moscovitch, 1981).

One important question is whether the perception of emotional expressions relies upon the same neural mechanisms as the perception of facial identity. As you remember from Chapter 7, patients with prosopagnosia (due to occipitotemporal-lobe damage) are unable to recognize the identities of individuals by their faces, but they are sometimes able to recognize emotional expressions (e.g., Tranel, Damasio, & Damasio, 1988). Conversely, some patients have trouble recognizing emotional expressions, but can recognize individuals' identities from their faces (e.g., Young, Newcombe, de Haan, Small, & Hay, 1993). This double dissociation implies that recognition of facial expression and recognition of facial identity rely upon partly separable mechanisms. Of course, both expression and identity recognition are likely to involve some similar steps in visual processing, such as constructing a coherent visual representation of the face structure. For this reason, it is not surprising that viewing emotionally expressive faces leads to activation in the fusiform gyrus of the right hemisphere, the region that is known to be more important for processing faces compared to other visual objects (e.g., Blair, Morris, Frith, Perrett, & Dolan, 1999; Kesler-West et al., 2001). However, the double dissociation tells us that beyond the stage of perceiving the visual image as a face, somewhat different brain regions are implicated in linking that face image with emotional information versus identification information.

Although there is a fair degree of overlap among brain systems that process the six main facial expressions—fear, disgust, anger, surprise, happiness, and sadness—it appears that not all emotional expression are treated equally by the brain (Hennenlotter & Schroeder, 2006). Fear is the expression for which there is the most evidence of a distinct neural substrate. For example, patients with damage to the amygdala are impaired in recognizing facial expressions, but these deficits seem to be most pronounced for fearful faces (Adolphs et al., 1999; Calder, Young, Rowland, Perrett, Hodges, & Etcoff, 1996). Some of the difficulty in recognizing facial expression may arise from the fact that amygdala-damaged patients do not seem to direct their eyes to the most emotionally informative parts of the face, such as the eyes (● Figure 13.16) (Adolphs et al., 2005). In fact, neuroimaging evidence shows that in neurologically intact people, the amygdala is responsive to specific facial features that indicate fear, such as enlarged whites of the eyes (Whalen et al., 2004). Thus, in amygdala-damaged patients, an inability to detect these specific



● **FIGURE 13.16 Patients with amygdala damage look at faces differently than neurologically intact individuals do.** The column on the left shows the eye-movement patterns of a normal participant; the column on the right shows the patterns of an amygdala-damaged patient when viewing fearful faces. Notice that the gaze of the normal participant is centered on examining the eyes and mouth, whereas the individual with amygdala damage tends to focus on the nose. Adapted by permission from Macmillan Publishers, LTD: Fig 2 in Adolphs, R., Gosselin, F., Buchanan, T. W., Tranel, D., Schyns, P., & Damasio, A. R. (2005). A mechanism for impaired fear recognition after amygdala damage. Nature, 433, 68–72.



● **FIGURE 13.17 Muscles of the face that are used to make facial expressions.** The corrugator muscle is used to furrow the brow, as in anger or fear; the orbicularis and zygomaticus muscles are used in smiling, and the levator labii muscles are used to wrinkle the nose in disgust. *Source:* Fig 3 in Niedenthal, P. M. (2007). Embodying emotion. Science, 316, 1002–1005.

cues may lead to the deficit in recognizing fear expressions. While the amygdala clearly contributes to recognition of emotional expressions, the fact that its role is predominantly related to fear expressions indicates that additional brain regions must be important for recognizing other facial emotions.

These studies have focused on perceiving facial expressions in other people; but what about producing emotional expressions in your own face? Several muscles in the face seem to have evolved for the sole purpose of forming emotional expressions (● Figure 13.17). Facial muscles move when they receive input from cranial nerves that are controlled by the brain's various motor systems (see Chapter 5). There are at least two systems for control of facial expressions: a system centered in the basal ganglia that controls spontaneous facial expressions, and a system centered in the motor cortex that controls voluntary facial expressions. Thus, a patient with damage to the basal ganglia (such as a patient with Parkinson's disease) may not make any facial expressions in spontaneous conversation, contributing to a mask-like appearance, but he or she may be able to make posed facial expressions with voluntary effort.

Just like the perception of facial expressions, the production of facial expressions appears to be primarily under the control of the right hemisphere. In one research approach, the facial expressions of patients with left- or right-hemisphere brain damage are photographed or videotaped while the patients are talking, watching emotional films, or doing other tasks. The photographs or videotapes are then rated, either subjectively by judges or by using coding schemes to identify the muscle movements in the face. Typically, patients with right-hemisphere damage are found to be less expressive than those with left-hemisphere damage (e.g., Montreys & Borod, 1998).

In another approach, typically used with neurologically intact individuals, the emotional expression appearing on the left side of the face is compared with that appearing on the right. Often, we can observe facial asymmetries merely by looking at a face, as shown in ● Figure 13.18. However, one way to quantitatively evaluate facial asymmetry is to cut a picture of a person's face in half and to splice each half-face together with its mirror image to create a composite. The result is two chimeras, one consisting of two left half-faces and the other of two right half-faces. When this is done, we can instantly see large differences in the appearance of the two sides of the face (● Figure 13.19). People typically rate left-face composites as more expressive than right-face composites (Sackeim, Gur, & Saucy, 1978). Nonhuman primates, such as macaque monkeys and chimpanzees, also

(A) (B) (C) (D)

● **FIGURE 13.18 Striking asymmetries in facial expression of emotion.** Although we think of people's faces as symmetrical, asymmetries can be seen. Note the asymmetrical expressions on some well-known faces: (A) the Mona Lisa, (B) Marilyn Monroe, (C) Elvis Presley, and (D) John Wayne.

FIGURE 13.19 One method of demonstrating asymmetry of emotional facial expression. An original photograph of the face, shown here in A, is bisected. Then, each half-face is spliced together with its mirror image to create a composite. Note the difference between the two composites depicted in B and C. Which one looks more emotionally intense to you? Usually, individuals choose the composite composed of two left half-faces, depicted in B, as more intense than the composite composed of two right half-faces, depicted in C. This result suggests that the right hemisphere, which controls the lower left half of the face, has a larger role in producing facial emotional expression.

exhibit more dramatic expressions on the left side of the face (Fernandez-Carriba, Loeches, Morcillo, & Hopkins, 2002; Hauser, 1993).

If you've carefully followed our discussion of hemispheric differences in perception and expression of emotion in the face, you may have noticed an odd paradox. Remember that, because of right-hemisphere specialization for emotional expression and perception, emotion is most strongly expressed on the left side of a poser's face (due to right-hemisphere specialization in the poser), and that people are best at understanding emotional expressions seen in the left visual field or left side of space (due to right-hemisphere specialization in the viewer). This means that for two people directly facing each other in a communication context, the most expressive side of the poser's face will fall into the least sensitive half field of the viewer! (Look at Figure 13.20 if you are having some left-right confusion.) This doesn't seem to be optimally adaptive for the purpose of communication. Interestingly, some research has shown that when people wish to communicate emotional information, they turn slightly to show more of the left side of

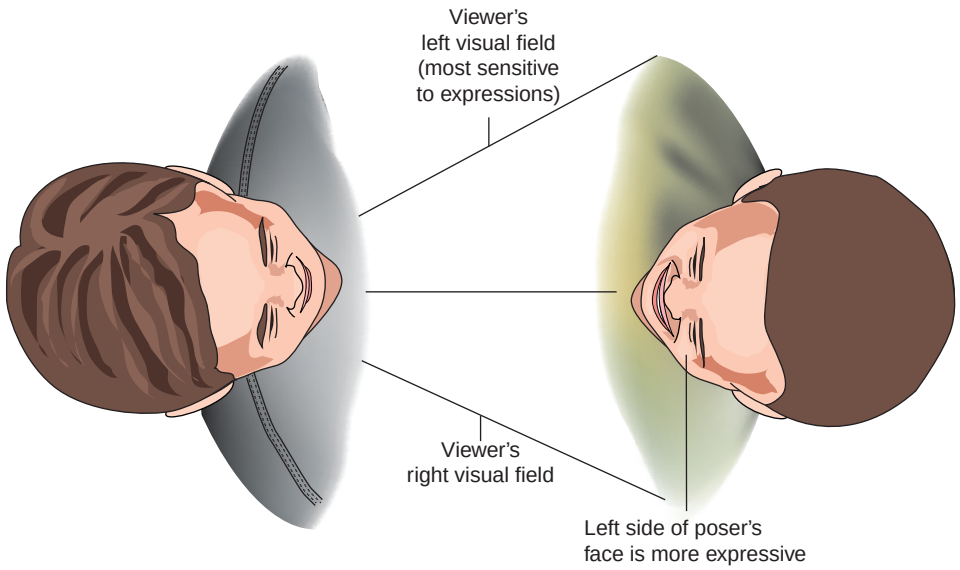


FIGURE 13.20 Expressive asymmetry meets perceptual asymmetry. When two participants interact in face-to-face conversation, the more expressive left side of one person's face is projected onto the less-sensitive right visual field of the other person. This illustrates the paradoxical outcome when both participants have right-hemisphere specialization for emotional expression and perception. However, people tend to adapt to this phenomenon in real life by turning the head to show more of the left side of the face.

the face. Analyses of portraits throughout history reflect a bias toward showing more of the left portion of the face, unless the portraits were made of scientists, who presumably put less emphasis on emotional expressivity (McManus & Humphrey, 1973; Nicholls, Clode, Wood, & Wood, 1999). When participants are asked to pose for a photograph in which they are encouraged to show their emotions, they are more likely to show the left cheek than if asked to pose for an "impassive" photo (Nicholls et al., 1999). This turning bias in portraiture illustrates how cerebral asymmetries for emotion can subtly manifest themselves in everyday life.

Prosody

The tone of voice in which a phrase is uttered is **prosody**. Monrad-Krohn (1947) first coined the term to describe vocal cues such as pitch or frequency, stress, intensity, and timing. Two types of prosody have been described. **Affective prosody** communicates the emotional context or tone of an utterance; for example, "My mother is coming to dinner" could be stated in a way that expresses elation or in a way that expresses dismay. **Propositional prosody** communicates lexical or semantic information—for example, "What's that in the road ahead?" versus "What's that in the road, a head?" Although prosody has been less well studied than facial expression as a means of conveying emotion, there is no question that prosodic cues are important in social interaction. For example, when you talk with a friend on the phone, you have no information about his facial expression, but you can use affective prosodic cues to deduce his emotional state or intent.

Clinical studies have suggested that patients with right-hemisphere lesions are significantly impaired in comprehending prosody, compared to patients with left-hemisphere lesions (e.g., Borod et al., 1998; Ross, 2006). Deficits in comprehension of prosody as a result of brain damage are referred to as **aprosodia**, and tend to be associated with damage to the region around the Sylvian fissure on the right side of the brain (Ross, 2006). This localization makes logical sense, serving as a complement to the role played by left-hemisphere Sylvian regions, which are involved in the auditory processing of language and language comprehension. Right-hemisphere lateralization for prosody is also supported by evidence of a left-ear advantage for comprehending prosody in neurologically intact people (e.g., Grimshaw, Kwasny, Covell, & Johnson, 2003; Ley & Bryden, 1982).

Nonetheless, there is some debate in the literature about how lateralized comprehension of prosody really is, because left-hemisphere damage can also lead to difficulties in interpreting prosody (Pell, 2006; Van Lancker & Sidtis, 1992). Some researchers have suggested that the right hemisphere is important for comprehending affective prosody (e.g., determining the emotional state of a speaker) and the left hemisphere for comprehending propositional prosody (e.g., distinguishing questions from statements based on tone of voice) (Walker, Daigle, & Buzzard, 2002). Other researchers have argued that left-hemisphere contributions to prosody may involve incorporating prosodic cues, which were initially decoded by the right hemisphere, into the overall semantic understanding of language that is dominated by the left hemisphere (Pell, 2006).

Neuroimaging studies have also implicated the right hemisphere in perceiving affective prosody, although different studies point to different regions within the right hemisphere. Several neuroimaging studies have found that regions in right prefrontal cortex are activated during detection of affective prosody (George et al., 1996; Imaizumi et al., 1997), though other studies have found bilateral activation (Kotz, Meyer, Alter, Besson, von Cramon, & Friederici, 2003). One study compared a condition in which participants had to distinguish the emotional tone of a voice (e.g., angry vs. happy) to another condition in which they had to distinguish different phonemes (e.g., power vs. tower) (Buchanan et al., 2000). Both tasks activated both hemispheres, but the activation was greater in right inferior prefrontal cortex for the emotion task and in the left inferior prefrontal cortex for the phoneme task. This study also found significant activity in the right auditory cortex for the emotional condition. Taken together, the lesion and imaging data indicate that the right hemisphere is more involved in the perception of prosody, but it remains to be seen exactly which regions of that hemisphere are most important.

The production of prosody is also heavily dependent on the right hemisphere (Ross, 2006). For example, some studies have presented brain-damaged patients with neutral sentences and asked them to repeat the sentence in different tones of voice (e.g., happy, sad, angry, or indifferent). Typically, individuals with right-hemisphere damage speak in more of a monotone (e.g., Tucker, Watson, & Heilman, 1977). As you might expect, deficits in producing prosody tend to be associated more with anterior rather than posterior regions within the right hemisphere (● Figure 13.21) (Ross, 2006). Some work with clinical populations has focused on examining whether more specific components of the production of prosody, such as the basic frequency at which an utterance is made (known as the *fundamental frequency*), intensity, and timing parameters, may be differentially lateralized. Evidence suggests that deficits in producing fundamental frequency may be associated with right-hemisphere damage, and deficits in producing timing parameters may be associated with left-hemisphere damage (Pell, 1999). This finding is consistent with some of what we learned in Chapter 4: that the right hemisphere processes global aspects of a stimulus (such as a fundamental frequency that is relatively constant over the entire utterance) and that the left hemisphere processes details such as the changes in timing across an utterance.

In sum, communication of emotion, whether through facial expression or through tone of voice, tends to be dominated by the right hemisphere. This division of

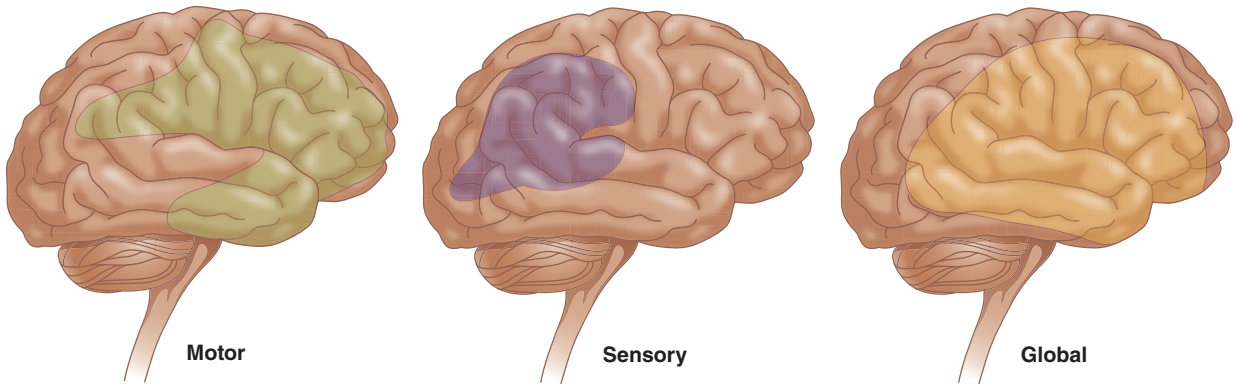


FIGURE 13.21 Areas of the right hemisphere that, when damaged, lead to difficulties in perceiving or producing emotional prosody. The nomenclature used to name these disorders is parallel to that used for aphasia (see Chapter 9). Frontal-lobe damage is associated with poor production but intact comprehension (motor aprosodia), temporoparietal damage is associated with intact production but poor comprehension (sensory aprosodia), and widespread damage to the right hemisphere is associated with deficits in both spontaneous production and comprehension of prosody (global aprosodia).
Source: Fig 21-1 in Ross, E. D. (2006). The aprosodias. In M. J. Farah & T. E. Feinberg (Eds.) Patient-Based Approaches to Cognitive Neuroscience, 2nd ed. MIT Press.

labor between the hemispheres is very efficient, because during a communicative interaction the left hemisphere can take the lead in comprehending and producing appropriate syntax and vocabulary, while the right hemisphere can take the lead in comprehending and producing nonverbal cues. Further, consistent with the basic division of the cortex into anterior motor regions and posterior perceptual regions, production of emotional expressions tends to rely upon frontal regions, while perception of those expressions tends to rely upon posterior regions such as the temporal and parietal cortices.

Emotional Experience

Thus far we have considered the role of various cortical brain regions in representing bodily states of emotion, integrating emotion and cognition, regulating emotion, and communicating emotion through facial and vocal expressions. A final emotional function that we will discuss is the experiential aspect of emotion. When a person experiences a particular emotional state, such as sadness or happiness, what brain regions represent or reflect that experience? This question is difficult to address, because it involves reliably assessing the subjective experience of another person.

Despite the challenges of assessing subjective states of emotion, psychologists have developed models of the basic dimensions of emotional experience. These models attempt to describe emotional experience as existing along several basic dimensions. One type of model assumes that the basic dimensions of emotional experience can be described in terms of approach and withdrawal motivations. For example, happy states involve a tendency to approach and engage with the world, whereas sad states involve a tendency to withdraw from it. Another type of model argues that the basic dimensions of emotion are *valence* (positive versus negative emotions) and *arousal* (low versus high emotional intensity). As we discuss in this section, each of these dimensional models has been related to activity in certain cortical regions.

Approach-Withdrawal Models

The first model that we examine posits that there are distinct brain systems for approach and withdrawal emotions. According to this model, initially proposed by Davidson and colleagues, approach and withdrawal are the most basic and rudimentary actions that organisms take in responding adaptively to the environment (Davidson, 1995; for reviews, see Davidson, 2004; Sutton, 2002). As emotions evolved, they became associated with already established approach or withdrawal action systems. Proponents of this model propose that the left frontal region houses a system involved in approach behaviors. Therefore, increased activity of the left frontal area is associated with emotions that tend to be accompanied by approach behaviors, including most positive emotions. In contrast, the right frontal region is posited to house a system involved in withdrawal behaviors. Increased activity of the right frontal area is associated with emotions, such as fear, disgust, and depression, that are accompanied by withdrawal behaviors.

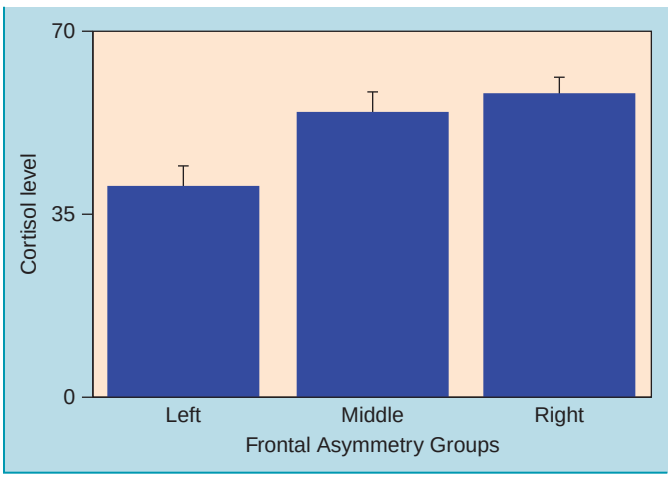
Much of the evidence supporting the **approach-withdrawal model** is based on EEG measures of activity in right or left frontal regions, which vary from person to person depending on the individual's typical outlook or disposition. For example, EEG measures reveal that people differ in the degree to which they show more right versus left prefrontal activity during a resting baseline condition (Coan & Allen, 2004; Davidson, 1995). These

asymmetries predict a person's disposition, with more left frontal activity associated with a more optimistic or positive outlook and more right frontal activity associated with a greater reactivity to negative stimuli. These patterns were replicated in 10-month-old infants, who were more likely to cry when separated from their mothers if they had more right than left prefrontal activation (Davidson & Fox, 1989; see also Buss, Schumacher, Dolski, Kalin, Goldsmith, & Davidson, 2003). Similar patterns of asymmetry exist in rhesus monkeys, who show higher levels of stress hormones if they have more right than left prefrontal activation (● Figure 13.22) (Kalin, Larson, Shelton, & Davidson, 1998; Kalin, Shelton, & Davidson, 2000). Such asymmetries are also associated with transient changes in mood. Increased left frontal activity is observed when people view happy film clips (Davidson, Ekman, Saron, Senulis, & Friesen, 1990) or when infants receive sweet-tasting sugar water (Fox & Davidson, 1986).

A similar relationship between hemisphere of activation and mood state has been observed in clinical populations with affective disorders. For example, during a resting condition, individuals with depression showed more activity in the right prefrontal region than in the left, whereas nondepressed individuals showed the opposite pattern (Schaffer, Davidson, & Saron, 1983; see also Reid, Duke, & Allen, 1998; Shankman, Klein, Tenke, & Bruder, 2007; Thibodeau, Jorgensen, & Kim, 2006). In fact, greater right than left EEG activity in the frontal-lobe regions may reflect a risk factor for depression, as discussed further in Chapter 14.

The approach-withdrawal model is also consistent with studies of the emotional consequences of damage to the left or right hemisphere. Studies of patients with unilateral brain damage found that 60% of the patients with left frontal lobe lesions exhibited symptoms of depression. The more anterior the lesion in the left hemisphere, the more severe the depressive symptoms (e.g., Robinson & Szetela, 1981; Morris, Robinson, Raphael, & Hopwood, 1996). These data fit with the approach-withdrawal model if we assume that damage to the left frontal region impairs the approach motivational system. That is, damage to the approach system (while leaving the withdrawal system intact) may lead to the classic depressive symptoms of apathy, helplessness, and inability to feel pleasure.

Approach and withdrawal might seem synonymous with positive and negative emotions, respectively. However, there is one emotion that does not quite fit this picture: anger. Anger is certainly a negative emotion, but it can be expressed either by "approach" behaviors, such as lashing out, or by withdrawal behaviors, such as giving someone the cold shoulder. For this reason, anger presents a unique test case for the approach-withdrawal model. If the model is correct, then people who tend to act out when angry should exhibit more left frontal activity. Studies have yielded results generally consistent with this prediction, supporting the approach-withdrawal model (Harmon-Jones, 2004, 2007; Harmon-Jones & Sigelman, 2001).



● FIGURE 13.22 Relationship between frontal-lobe activation asymmetry and stress hormones. Rhesus monkeys with greater left than right frontal lobe activity (left bar) have lower levels of the stress hormone cortisol than monkeys with greater right than left frontal activity (right bar) or those with balanced asymmetry (middle bar). *Source:* Fig. 4 in Kalin, N. H., Larson, C., Shelton, S. E., & Davison, R. J. (1998). Asymmetric frontal brain activity, cortisol, and behavior associated with fearful temperament in rhesus monkeys. Behavioral Neuroscience, 112, 286–292.

Valence-Arousal Models

Another cognitive neuroscience model of emotional experience (Heller, 1993; Heller & Nitschke, 1998) is based on psychological models arguing that emotions are best described by two fundamental dimensions: valence (pleasant vs. unpleasant) and arousal (high vs. low intensity) (e.g., Feldman-Barrett & Russell, 1999). According to the valence-arousal model, frontal regions are asymmetrically involved in the valence aspect of emotion, whereas the posterior right hemisphere is involved in the arousal aspect (● Figure 13.23).

How does the valence-arousal model differ from the approach-withdrawal model? The two models are very similar in their predictions for frontal regions. According to the valence-arousal model, the left frontal region is specialized for positive emotions and the right for negative emotions. In the previous section, we reviewed evidence that ties approach (typically positive) emotions to the left frontal region, and withdrawal (typically negative) emotions to the right frontal region. This evidence fits with both models.

However, the valence-arousal model also posits that arousal, or emotional intensity, is reflected in activity of posterior sections of the right hemisphere. Studies examining perceptual asymmetries have shown that emotional stimuli have a greater influence on heart rate, blood pressure, and the release of stress hormones when they are presented to the right hemisphere rather than the left (Wittling, 1990; Wittling & Pflüger, 1990; Wittling,

Block, Schweiger, & Genzel, 1998). Further, directing participants' attention toward the left visual field, presumably activating the right hemisphere, leads to greater changes in self-reported arousal than directing attention to the right visual field (Compton, 1999). Also, the higher a person's self-reported level of energy or arousal, the larger his or her leftward perceptual bias on a face perception task (Heller, Nitschke, & Lindsay, 1997).

The valence-arousal model of emotion has been particularly helpful in differentiating the patterns of brain activity that characterize depression and anxiety (Heller, Koven, & Miller, 2003). Although both depression and anxiety are certainly unpleasant rather than pleasant mood states, they differ in the arousal dimension. Depression is typically a low-arousal state, whereas anxiety is often a high-arousal state, and therefore they are likely to differentially involve the right hemisphere's posterior regions. We consider the brain regions involved in anxiety and depression in more detail in Chapter 14.

As you may have noticed, neither the approach-withdrawal model nor the valence-arousal model is very specific about exactly which portions of the frontal or parietal lobe are involved in the experience of emotion. Rather, both emphasize a more general pattern of activity that is involved in emotional experience. This likely reflects the limitation of some of the methodologies used to examine the effect of mood. For example, EEG measures are not ideal for determining precisely where in the brain activity is occurring. It also likely reflects an important conceptual point: that there may be no single "mood" center in the brain. Instead, changes in mood are associated with modulation of activity across a wide range of brain areas, which affect not only the subjective feeling that one experiences, but also how one processes perceptual information, pays attention, values reward, and makes decisions.

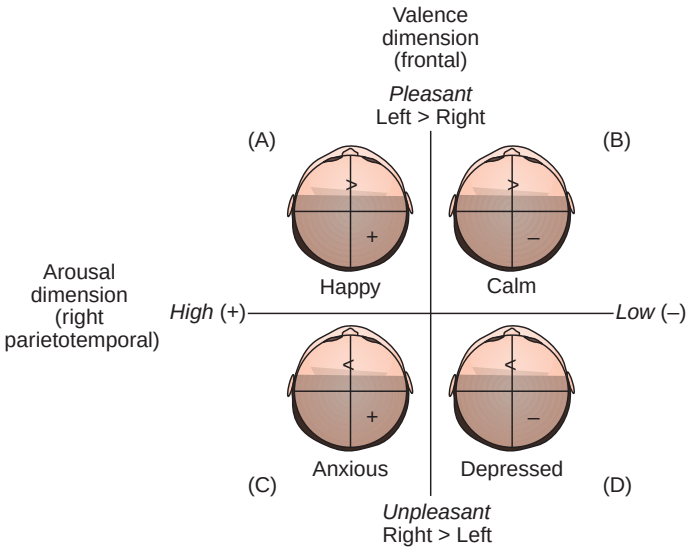


FIGURE 13.23 Model of regional brain activity and mood proposed by Heller (1993). This model posits that the valence dimension of mood (positive or pleasant vs. negative or unpleasant; *y* axis) is mainly affected by activation of frontal regions of the brain, whereas the arousal dimension (high vs. low; *x* axis) is mainly affected by activation of the right posterior region. Depicted here are the patterns of brain activation for four mood states. (A) When activation of left frontal regions is greater than that of right frontal regions (which leads to pleasant emotion) and the right posterior region is highly activated (which leads to arousal), the individual is happy. (B) The pattern of activation is identical over frontal regions for a calm state, but the activity over the right posterior region is reduced, which leads to a lower level of arousal for a calm state than for happiness. (C) In anxiety, higher right than left frontal activation leads to a negative valence, and high activity in the right posterior region leads to increased arousal. (D) Finally, the brain activation in depression is similar to that in anxiety, in that higher right than left frontal activation leads to a negative valence. However, the low activity in the right posterior region causes the decreased arousal that differentiates depression from anxiety. © 2011 Cengage Learning

From Emotion to Social Cognition

The study of emotion is closely tied to the study of social behavior, particularly in social species such as humans and other primates. If you think back over the material covered in this chapter, you can see that many of our examples of emotional processing involved social stimuli or situations. For example, facial expressions and emotional prosody are necessarily social, because they take place in a situation in which two or more people are communicating with one another. We've also considered how patients with orbitofrontal damage are unable to inhibit socially inappropriate behavior, which reminds us that normally people have an internal understanding of how to control their behavior to meet social norms and expectations. As another example, we've noted that social stimuli, such as attractive faces, can engage the subcortical reward systems, much as very tasty food can.

Research on emotion has long involved social aspects, but in recent years there has been growing interest in applying the methods of cognitive neuroscience to understand social phenomena more broadly (Cacioppo, Visser, & Pickett, 2006; Lieberman, 2007). Here, we discuss two lines of research to illustrate the growing subfield of social cognitive neuroscience. First, we consider theories about how our minds are able to represent the mental states of other people. Second, we consider studies that attempt to apply cognitive neuroscience approaches to the topic of prejudice and discrimination.

Understanding the Mental States of Others

People are constantly trying to infer what other people are thinking about. You may wonder whether your roommate likes you, whether your boss intends to fire

IN FOCUS: The Pain of Rejection

There is nothing quite so bad as feeling excluded. Think back to junior high school: remember when the popular kids shunned you? Or think about any time that you tried to join a social group and were rebuffed. It hurt, right? For members of social species like humans, inclusion in a social group is thought to be part of the road to happiness and well-being, while exclusion is associated with a loss of power, esteem, and resources.

Scientists have spent years elucidating the neural underpinnings of physical pain, but recently affective neuroscientists have considered the underpinnings of social pain (see MacDonald & Leary, 2005, for a review). Does the pain of rejection "hurt" in the same places in the brain that physical pain does?

One influential study suggests that it does (Eisenberger, Lieberman, & Williams, 2003). In this study, participants played a virtual ball-tossing game with two other participants, depicted as cartoon characters on a computer screen. (Actually, the other "participants" were just a rigged computer program.) First the participant watched the two others toss the virtual ball back and forth. Then, the participant was drawn into the game, and all three players tossed the ball around. In the third portion of the game, suddenly the other two "players" stopped tossing the ball to the participant. Not surprisingly, participants tended to say that they felt ignored and excluded. At the same time, they showed increased activity in the anterior cingulate cortex and right ventral prefrontal cortex compared to earlier when they had been participating in tossing the ball. Because the anterior cingulate is also activated by physical pain, these results could be interpreted as indicating that social exclusion "hurts" because it activates the same area in the brain as does physical pain.

However, as other researchers pointed out, the exclusion condition probably differed from the inclusion condition in another way: namely, that the exclusion condition violated expectations (Somerville, Heatherton, & Kelley, 2006). That is, the participant was probably surprised when the others stopped throwing the ball to him or her. Was the cingulate cortex activated because of the perceived social pain, or simply because of the violated expectation? Other studies have shown that the cingulate cortex is activated by unexpected outcomes and other cognitive conflicts even in situations that do not involve pain of any sort. To differentiate between these two possibilities, Somerville and colleagues developed an experiment that included two different kinds of conditions: one that involved a violation of expectations and another that involved social feedback (indicating whether another "participant" reported liking the actual participant or not). They found that social rejection activated a different region of the cingulate cortex than did expectancy violation, indicating that these two processes are not one and the same, but rather are separable. Bolstering the idea that physical and social pain are connected, researchers have shown that participants who are more sensitive to social rejection in the ball-tossing game also tend to have lower thresholds for physical pain (Eisenberger, Jarcho, Lieberman, & Naliboff, 2006). This finding implies that physical pain and the pain of social rejection may indeed share a common basis.

you, or whether your study partner really understands the course material he or she is explaining to you. All of these examples involve attempting to understand what is going on in the mind of another person.

There are two main theories of how we understand others' thoughts and feelings. One theory, sometimes called **theory of mind**, assumes that we have a cognitive representation of other people's mental states, including their feelings and their knowledge. Through these cognitive representations, we are able to hold in mind two different sets of beliefs: what we know, believe, or feel, and what we think another person knows, believes, or feels. For example, a teacher might know how action potentials propagate in a neuron, while at the same time knowing that her students do not yet know this on the first day of class. (This can go even one step farther: imagine a student who has already learned about action potentials, thinking "The teacher doesn't know that I know this already!") Because of the high level of cognitive sophistication required for this kind of theorizing about another's knowledge, this ability is not thought to develop until the late preschool years (Flavell, 2004). Some argue that the ability to represent the mental states of others in this way is unique to humans and possibly great apes, though this topic is much disputed (e.g., Povinelli & Vonk, 2003; Tomasello, Call, & Hare, 2003; see also Brüne & Brüne-Cohrs, 2006).

Another theory suggests that we understand the mental states of others through simulation. In the simplest sense, **simulation** just means acting like another person. For example, if you see another person crying, you might understand his mental state by starting to tear up yourself. By mimicking that other person's actions and expressions, you feel as he does, and therefore you comprehend his mental state. This means of understanding another person is closely related to the concept of empathy.

It should be obvious that these two ways of understanding other people—theory of mind and simulation—are not mutually exclusive. For example, the theory-of-mind approach can more easily explain

how we represent mental states that do not have an obvious outward expression, such as beliefs and knowledge. In contrast, simulation can best explain emotional behaviors and motor actions that can be easily mimicked. It can also explain how emotions (and behaviors like laughing) can be "contagious" even among small children and less cognitively sophisticated animals. At the same time, if we only used simulation to understand other people, it could be difficult to separate our own feelings from those of others. Therefore, it is likely that we rely on both means of representing others' mental states, though perhaps in different circumstances.

So, what do we know about the neural processes underlying these skills? As you might expect, it is not easy to localize theorizing about another person's internal knowledge to a particular brain region, because it is so abstract and probably involves several component operations. However, some studies have examined brain activity when people are required to make inferences about the beliefs of other people. Such studies have found activity in a network of areas, including medial prefrontal cortex, temporal poles, superior temporal sulcus, and the temporoparietal junction (Frith & Frith, 2003; Saxe, Carey, & Kanwisher, 2004). However, the precise operation carried out by each of these regions in the network has yet to be fully understood. There are some potential reasons that each of these regions might play a role. For example, the superior temporal sulcus plays a role in coding for the gaze direction of other people (Hoffman & Haxby, 2000; Jellema, Baker, Wicker, & Perrett, 2000; Pelphrey, Singerman, Allison, & McCarthy, 2003), which may be related to inferring their mental states. For example, if someone turns her gaze away from you, you may infer that she is bored or bothered by you.

The simulation notion of understanding others is easier to relate to brain processes. Remember that in Chapter 5, we learned about mirror neurons, which fire when an animal carries out an action or observes another carrying out the same action. Following the initial discovery of mirror neurons in motor regions of the brain, researchers began to consider the concept of *neural mirroring* more broadly. Indeed, in many situations the same brain regions seem to be activated when a person experiences a particular state as when he or she observes another person experiencing that same state. Motor areas are activated when we see another person perform actions (e.g., van Schie, Mars, Coles, & Bekkering, 2004), pain areas of the brain are activated when we see another person in pain (e.g., Jackson, Meltzoff, & Decety, 2005; Singer, Seymour, O'Doherty, Kaube, Dolan, & Frith, 2004), and disgust-related areas of the brain (the insula) are activated both when we smell foul odors and when we see another person smelling them (Wicker, Keysers, Plailly, Royet, Gallese, & Rizzolatti, 2003). In these examples, activation of sensory, motor, and emotional systems by observation of another's experience may help us to simulate, and therefore understand, that experience.

Interestingly, the degree to which we simulate another's experiences may depend on social factors, such as how much we like that person or whether we see ourselves as similar to that person. One study found that the ventral striatum (nucleus accumbens) was activated when participants viewed someone else receiving a large reward, but the activity was greater when the reward recipient was deemed to be socially desirable, likeable, and similar to the actual participant (Mobbs et al., 2009). Likewise, in a study in which participants watched a confederate perform a simple computer task, the neural response to errors made by the confederate was influenced by the participant's judgment about the similarity between himself and the confederate (Carp, Halenar, Quandt, Sklar, & Compton, 2009). These studies suggest that although we have the capability to mirror other people's states, we may do so preferentially for others whom we see as similar to ourselves.

Cognitive Neuroscience Approaches to Prejudice

Prejudice and discrimination against other people, particularly against minority "out-groups," is a pervasive issue in all societies. The problems of prejudice and discrimination have been at the core of modern social psychology since its founding. Why do people engage in prejudice and discriminatory behavior, and what mechanisms sustain it? Clearly, dealing with the full scope of this problem would involve understanding behavior at the level of the group, inequalities in social structure and power, and how cultural norms and values are taught and learned. It would be naïve to think that we can fully understand prejudice by looking deep within the brain. But can cognitive neuroscience contribute anything to knowledge in this important area?

One thing we have learned from cognitive neuroscience approaches is that the brain distinguishes between in-group and out-group categories fairly rapidly. In one study, researchers measured ERPs in response to faces that belonged to different racial categories (Ito, Thompson, & Cacioppo, 2004). Some early ERP peaks appeared to respond equally to all faces, regardless of their racial category. For example, the face-specific N_{170} peak, which occurs about 170 ms after the presentation of a face, was bigger for faces than for other pictures, but did not differ for faces of different racial categories. However, at around 250 ms, the ERP response was larger in response to faces of in-group members than to faces of out-group members. Thus, within about a quarter of a second, people's brains distinguish between in-group and out-group members. Although early research was limited to white participants, subsequent studies confirmed the same basic pattern—early neural differentiation of

in-group and out-group—in both white and black participants (Dickter & Bartholow, 2007).

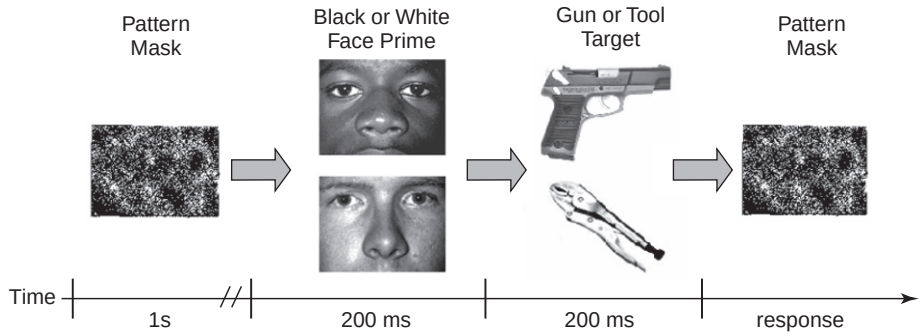
People often seem to feel afraid or uncertain when interacting with those of other races. Could their brains be interpreting out-group members as threats? Some research has found that people acquire a conditioned fear to other-race faces more quickly than to faces of their own race (Olsson, Ebert, Banaji, & Phelps, 2005). In a neuroimaging study, researchers showed that unconscious racial bias was correlated with activity in the amygdala (Phelps et al., 2000). Unconscious bias was measured using a behavioral method that quantifies the speed of association between pictures of other-race faces and negative words (compared to other-race faces and positive words). The researchers found that the higher the measure of unconscious racial bias, the more the amygdala, particularly on the left, became activated. Of course, this does not tell us why some people exhibit stronger unconscious racial biases than others; it just tells us that those who do are activating the amygdalar circuitry of the brain when viewing other-race faces. Interestingly, patients with amygdala damage still show unconscious racial biases (Phelps, Cannistraci, & Cunningham, 2003), indicating that this brain structure is not solely responsible for sustaining racial prejudice.

Although these studies have focused on in-groups and out-groups based on race—a very salient category in our society—social psychologists have long known that in-groups and out-groups can be formed very easily based on virtually any kind of distinction between people. When people are randomly assigned to groups, even through a coin toss, they later show evidence of favoring their own group (Tajfel, 1970). In one neuroimaging study (Van Bavel, Packer, & Cunningham, 2008), participants were randomly assigned to belong to either the "Leopards" or the "Tigers" team, and they were encouraged to learn which other participants belonged to their team versus the other team, supposedly for a later phase of the study. Brain imaging results showed increased activity in a number of regions, including amygdala, orbitofrontal cortex, and fusiform gyrus, when participants viewed their own team members versus members of the other team. In addition, orbitofrontal cortex activity predicted how much the participants favored their in-group when asked to rate how much they liked each face. The difference between the response to in-groups versus out-groups in this study could not be accounted for by prior experience with the group, because the participants did not know each other before the study and the researchers controlled how long participants viewed each face. These results show that even the simplest kinds of social categorization, established within a single experimental session, can affect how the brain responds to other people.

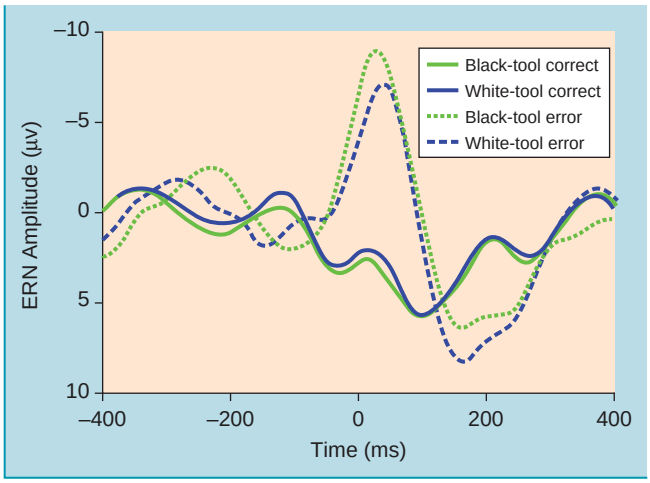
In this day and age, most people know that overt racial bias is socially unacceptable, and are uncomfortable with the thought that they might act or think in a racist way. One study focused on this phenomenon by studying people's responses to their own errors that might imply they harbored a racial bias (Amodio, Harmon-Jones, Devine, Curtin, Hartley, & Covert, 2004). In this study, people had to quickly press a button to indicate whether a picture was a gun or a tool. Just before the picture, participants were primed with a picture of an African-American or Caucasian person. Overall, participants tend to be more likely to mistakenly press "gun" when primed with an African-American face than when primed with a Caucasian face, indicating an implicit biased association between African-Americans and guns. Researchers examined the error-related negativity (ERN) evoked when participants made errors in this task, and found that the ERN was significantly higher when participants made racially charged errors (e.g., mistakenly pressing the button for "gun" rather than "tool" when primed by an African-American face), compared to errors that would not imply racial bias (● Figure 13.24) (Amodio et al., 2004; Amodio, Kubota, Harmon-Jones, & Devine, 2006). Participants with a larger ERN to racially charged errors were more likely to slow down and become more accurate on the next trial, suggesting that they were trying to compensate to avoid such errors in the future. Based on what we know about the source of the ERN, we can assume that this cognitive control process involves the anterior cingulate cortex and related frontal-lobe regions.

Other findings by this same research group have tied feelings of guilt about racial prejudice to EEG asymmetries in the frontal lobe (Amodio, Devine, & Harmon-Jones, 2007). In this study, participants were given false feedback indicating that they had responded in a racially prejudiced way. The feedback altered patterns of frontal-lobe EEG asymmetry, toward less left-sided activity. According to the approach-withdrawal model, this is consistent with reduced approach motivation; perhaps the sense of guilt at being prejudiced made people want to pull back. However, participants who reported more guilt were also subsequently more interested in reading articles about prejudice reduction, which in turn was associated with an increase in approach-related left-frontal activation. In other words, the feeling of guilt was first associated with withdrawal-related EEG asymmetries, but taking advantage of the opportunity to make amends was associated with approach-related EEG asymmetries.

Once again, these results do not really tell us why some people are more or less prone to racial prejudice. However, they do give clues about what is going on in the brain when people perceive racial cues and try to regulate their own responses to those cues. Clearly, brain structures involved in emotion and emotion regulation play an important role in these social processes.



(A)



(B)

● **FIGURE 13.24 Error-related brain processes are influenced by racial meaning.** Panel A illustrates a task in which participants had to decide if a visual image was a gun or a tool. The image was preceded by the quick presentation of an African-American or Caucasian face. In Panel B, the error-related negativity was greater when participants made black-tool errors (that is, mistakenly calling a tool a gun after being primed with an African-American face), compared to white-tool errors or correct responses. These data indicate that errors reflecting racial bias elicit a stronger neural response than other errors. *Source:* Fig 1 from Amodio, D. M., Harmon-Jones, E., Devine, P. G., Curtin, J. J., Hartley, S. L., & Covert, A. E. (2004). Neural signals for the detection of unintentional race bias. Psychological Science, 15, 88–93 and Fig. 3a from Amodio, D. M., Harmon-Jones, E., Devine, P. G., Curtin, J. J., Hartley, S. L., & Covert, A. E. (2004). Neural signals for the detection of unintentional race bias. Psychological Science, 15, 88–93. Reprinted by permission of John Wiley.

Summary

Subcortical Contributions to Emotion

- The hypothalamus mediates some of the physiological phenomena associated with emotional states, such as changes in autonomic nervous system and endocrine function that are associated with fleeing or fighting.
- The amygdala is involved in learning the emotional significance of information and in producing a quick, instinctive, emotional response. The amygdala can also influence how attention is directed to emotionally significant events.
- The ventral striatum, or nucleus accumbens, is important in reward-seeking behavior. It is especially responsive to unpredicted rewards and becomes active when a person is anticipating a reward.

Cortical Contributions to Emotion

- The insula is involved in coding for unpleasant tastes, and also plays a role in the experience and perception of disgust. It is important in representing internal body states that are relevant to emotion.
- The rostral region of the anterior cingulate is involved in monitoring for emotionally salient events.
- Orbitofrontal cortex is involved in evaluating reward and punishment contingencies and in responding adaptively to changes in these relationships. Damage to the orbitofrontal cortex can lead to deficits in controlling behavior and emotion in a socially appropriate manner.
- Control of emotions depends upon interactions among cortical and subcortical brain regions.

Suppressing an emotion appears to involve top-down control over subcortical systems such as the amygdala and hypothalamus.

- Temporoparietal regions of the right hemisphere are important for comprehending emotional information expressed in tone of voice or facial expression.
- The right hemisphere plays a predominant role in producing prosody that is related to emotional affect and in governing the expression of emotion on the face.
- Positive affect is associated with more activity over the left than the right prefrontal cortex, whereas negative affect is associated with the reverse pattern (greater right prefrontal than left prefrontal activity).
- States of high arousal appear to differentially involve the right hemisphere, particularly in posterior regions.

From Emotion to Social Cognition

- Many aspects of social behavior are closely related to emotional processes.
- Two main theories address how we understand the mental states of others. "Theory of mind" is a cognitive representation of others' knowledge or beliefs, and involves a network of regions including medial prefrontal cortex, superior temporal sulcus, and the temporoparietal junction. "Simulation" refers to representing the mental states of others by activating the same brain regions as the other person is likely to be activating.
- Researchers have examined neural correlates of prejudice. The amygdala is activated by faces of other races, suggesting that those faces may be coded as threatening. People are especially sensitive to mistakes that might reflect racial bias, and try to correct themselves. Guilt associated with racial prejudice is associated with changes in approach-withdrawal systems in the two hemispheres.

Key Terms

affective prosody 386
approach-withdrawal model 387
aprosodia 386
emotion regulation 380
interoception 375
limbic system 367
nucleus accumbens 373
orbitofrontal cortex (OFC) 378
propositional prosody 386
prosody 386
psychic blindness 368
reinforcement contingency 379
reversal learning 379
simulation 390
theory of mind 390

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