PROLOGUE | Intellect and Emotion

Several years ago, while I was on a sabbatical leave, a colleague stopped by my office and asked whether I would like to see an interesting patient. The patient, a 72-year-old man, had suffered a massive stroke in his right hemisphere that had paralyzed the left side of his body.

Mr. V. was seated in a wheelchair equipped with a large tray on which his right arm was resting; his left arm was immobilized in a sling, to keep it out of the way. He greeted us politely, almost formally, articulating his words carefully with a slight Central European accent.

Mr. V. seemed intelligent, and this impression was confirmed when we gave him some of the subtests of the Wechsler Adult Intelligence Test. His verbal intelligence appeared to be in the upper 5 percent of the population. The fact that English was not his native language made his performance even more remarkable.

The most interesting aspect of Mr. V.'s behavior after his stroke was his lack of reaction to his symptoms. After we had finished with the testing, we asked him to tell us a little about himself and his lifestyle. What, for example, was his favorite pastime?

"I like to walk," he said. "I walk at least two hours each day around the city, but mostly I like to walk in the woods. I have maps of most of the national forests in the state on the walls of my study, and I mark all of the trails I've taken. I figure that in about six months I will have walked all of the trails that are short enough to do in a day."

"You're going to finish up those trails in the next six months?" asked Dr. W.

"Yes, and then I'll start over again!" he replied.

"Mr. V., are you having any trouble?" asked Dr. W.

"Trouble? What do you mean?"

"I mean physical difficulty."

"No." Mr. V. gave him a slightly puzzled look.

"Well, what are you sitting in?"

Mr. V. gave him a look that indicated he thought that the question was rather stupid–or perhaps insulting. "A wheelchair, of course," he answered.

"Why are you in a wheelchair?"

Now Mr. V. looked frankly exasperated; he obviously did not like to answer foolish questions. "Because my left leg is paralyzed!" he snapped.

Mr. V. clearly knew what his problem was, but he failed to understand its implications. He could verbally describe his disability, but he was unable to grasp its significance. Thus, he blandly accepted the fact that he was confined to a wheelchair. The implications of his disability did not affect him emotionally or figure into his plans.

The word *emotion* refers to positive or negative reactions to particular situations. There is no such thing as a neutral emotion. For example, being treated unfairly makes us angry, seeing someone suffer makes us sad, and being close to a loved one makes us happy. Emotions consist of patterns of physiological changes and accompanying behaviors—or at least urges to perform these behaviors. These responses are accompanied by feelings. In fact, most of us use the word *emotion* to refer to the feelings, not to the behaviors. But it is behavior, and not private experience, that has consequences for survival and reproduction. Thus, the useful functions served by emotional behaviors are what guided the evolution of our brain.

This chapter is divided into three major sections. The first considers the patterns of behavioral and physiological responses that constitute the negative emotions of fear and anger. It describes the nature of these response patterns, their neural and hormonal control, and the role of emotions in moral judgments and social behavior. The second section describes the communication of emotions—their expression and recognition. The third section examines the nature of the feelings that accompany emotions.

Emotions as Response Patterns

An emotional response consists of three types of components: behavioral, autonomic, and hormonal. The *behavioral* component consists of muscular movements that are appropriate to the situation that elicits them. For example, a dog defending its territory against an intruder first threatens the intruder by adopting an aggressive posture, growling, and showing its teeth. If the intruder does not leave, the defender runs toward the intruder and attacks. *Autonomic* responses facilitate the behaviors and provide quick mobilization of energy for vigorous movement. In this example the activity of the sympathetic branch of the autonomic nervous system increases while that of the parasympathetic branch decreases. As a consequence the dog's heart rate increases, and changes in the size of blood vessels shunt the circulation of blood away from the digestive organs toward the

muscles. *Hormonal* responses reinforce the autonomic responses. The hormones secreted by the adrenal medulla—epinephrine and norepinephrine—further increase blood flow to the muscles and cause nutrients stored in the muscles to be converted into glucose. In addition, the adrenal cortex secretes steroid hormones, which also help to make glucose available to the muscles.

This section discusses research on the control of overt emotional behaviors and the autonomic and hormonal responses that accompany them. Special behaviors that serve to communicate emotional states to other animals, such as the threat gestures that precede an actual attack and the smiles and frowns used by humans, are discussed in the second section of the chapter. As you will see, negative emotions receive much more attention than positive ones. Most of the research on the physiology of emotions has been confined to fear and anger—emotions associated with situations in which we must defend ourselves or our loved ones. The physiology of behaviors associated with positive emotions—such as those associated with lovemaking, caring for one's offspring, or enjoying a good meal or a cool drink of water (or some other beverage)—is described in other chapters but not in the specific context of emotions. Chapter 16 discusses the consequences of situations that evoke negative emotions: stress.

Fear

As we just saw, emotional responses involve behavioral, autonomic, and hormonal components. These components are controlled by separate neural systems. The *integration* of the components of fear appears to be controlled by the amygdala.

RESEARCH WITH LABORATORY ANIMALS

The amygdala plays a special role in physiological and behavioral reactions to objects and situations that have biological significance, including those that warn of pain or other unpleasant consequences. Researchers in several different laboratories have shown that single neurons in various nuclei of the amygdala become active when emotionally relevant stimuli are presented. For example, these neurons are excited by such stimuli as the sight of a device that has been used to squirt either a bad-tasting solution or a sweet solution into the animal's mouth, the sound of another animal's vocalization, the sound of the opening of the laboratory door, the smell of smoke, or the sight of another animal's face (O'Keefe and Bouma, 1969; Jacobs and McGinty, 1972; Rolls, 1982; Leonard et al., 1985). And as we saw in Chapter 9, the amygdala is involved in mediating the effects of olfactory stimulation on reproductive physiology and behavior. This section describes research on the amygdala's role in organizing emotional responses produced by aversive stimuli.

lateral nucleus (LA) A nucleus of the amygdala that receives sensory information from the neocortex, thalamus, and hippocampus and sends projections to the basal, accessory basal, and central nucleus of the amygdala.

central nucleus (CE) The region of the amygdala that receives information from the basal, lateral, and accessory basal nuclei and sends projections to a wide variety of regions in the brain; involved in emotional responses.

The amygdala (or more precisely, the *amygdaloid complex*) is located within the temporal lobes. It consists of several groups of nuclei, each with different inputs and outputs—and with different functions (Amaral et al., 1992; Pitkänen et al., 1997; Stefanacci and Amaral, 2000). The amygdala has been subdivided into approximately twelve regions, each containing several subregions. However, we only need to concern ourselves with three major regions: the *lateral nucleus*, the *basal nucleus*, and the *central nucleus*.

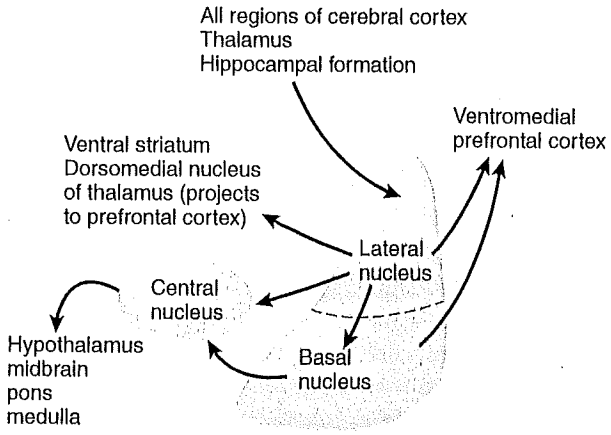

FIGURE 10.1 **The Amygdala.** This much-simplified diagram shows the major divisions and connections of the amygdala that play a role in emotions.

The **lateral nucleus (LA)** receives information from all regions of the neocortex, including the ventromedial prefrontal cortex, the thalamus, and the hippocampal formation. The lateral nucleus sends information to the basal nucleus (B) and to other parts of the brain, including the ventral striatum (a brain region involved in the effects of reinforcing stimuli on learning) and the dorsomedial nucleus of the thalamus, whose projection region is the prefrontal cortex. The LA and B nuclei send information to the ventromedial prefrontal cortex and the **central nucleus (CE)**, which projects to regions of the hypothalamus, midbrain, pons, and medulla that are responsible for the expression of the various components of emotional responses. As we will see, activation of the central nucleus elicits a variety of emotional responses: behavioral, autonomic, and hormonal. (See ***Figure 10.1.***)

The central nucleus of the amygdala is the single most important part of the brain for the expression of emotional responses provoked

by aversive stimuli. When threatening stimuli are perceived, neurons in the central nucleus become activated (Pascoe and Kapp, 1985; Campeau et al., 1991). Damage to the central nucleus (or to the nuclei that provide it with sensory information) reduces or abolishes a wide range of emotional behaviors and physiological responses. After the central nucleus has been destroyed, animals no longer show signs of fear when confronted with stimuli that have been paired with aversive events. They also act more tamely when handled by humans, their blood levels of stress hormones are lower, and they are less likely to develop ulcers or other forms of stress-induced illnesses (Coover, Murison, and Jellestad, 1992; Davis, 1992; LeDoux, 1992). Normal monkeys show signs of fear when they see a snake, but those with amygdala lesions do not (Amaral, 2003). In contrast, when the central amygdala is stimulated by means of electricity or by an injection of an excitatory amino acid, the animal shows physiological and behavioral signs of fear and agitation (Davis, 1992), and long-term stimulation of the central nucleus produces stress-induced illnesses such as gastric ulcers (Henke, 1982). These observations suggest that the autonomic and endocrine responses controlled by the central nucleus are among those responsible for the harmful effects of long-term stress, which are discussed in Chapter 16. Rather than describing the regions to which the amygdala projects and the responses these regions control, I will refer you to Figure 10.2, which summarizes them. (See ***Figure 10.2.***)

A few stimuli automatically activate the central nucleus of the amygdala and produce fear reactions—for example, loud unexpected noises, the approach of large animals, heights, or (for some species) specific sounds or odors. Even more important, however, is the ability to *learn* that a particular stimulus or situation is dangerous or threatening. Once the learning has taken place, that stimulus or situation will evoke fear; heart rate and blood pressure will increase, the muscles will become more tense, the adrenal glands will secrete epinephrine, and the animal will proceed cautiously, alert and ready to respond.

The most basic form of emotional learning is a **conditioned emotional response**, which is produced by a neutral stimulus that has been paired with an emotion-producing stimulus. The word *conditioned* refers to the process of *classical conditioning*, which is described in more detail in Chapter 12. Briefly, classical conditioning occurs when a neutral stimulus is regularly followed by a stimulus that automatically evokes a response. For example, if a dog regularly hears a bell ring just before it receives some food that makes it salivate, the dog will begin salivating as soon as it hears the sound of the bell. (You probably already know that this phenomenon was discovered by Ivan Pavlov.)

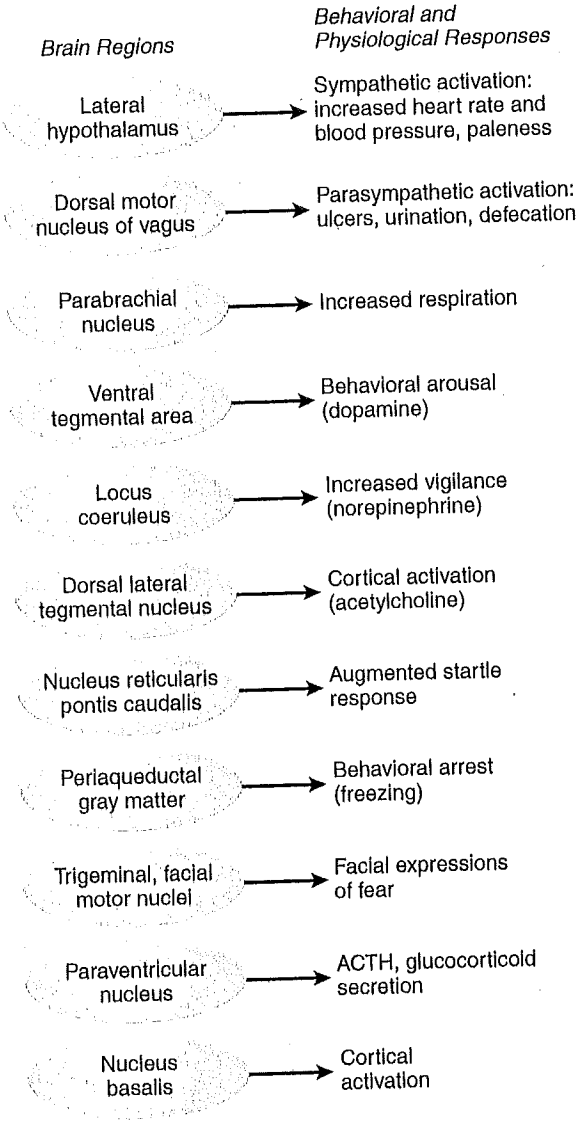

FIGURE 10.2 **Amygdala Connections.** This schematic diagram shows some important brain regions that receive input from the central nucleus of the amygdala and the emotional responses controlled by these regions.

Based on Davis, M., *Trends in Pharmacological Sciences*, 1992, *13*, 35–41.

Several laboratories have investigated the role of the amygdala in the development of classically conditioned emotional responses. For example, these responses can be produced in rats by presenting an auditory stimulus such as a tone, followed by a brief electrical shock delivered to the feet through the floor on which the animals are standing. (See ***Figure 10.3.***) By itself the shock produces an *unconditional* emotional response: The animal jumps into the air, its heart rate and blood pressure increase, its breathing becomes more rapid, and its adrenal glands secrete catecholamines and steroid stress hormones. After several pairings of the tone and the shock, classical conditioning is normally established.

The next day, if the tone is presented alone—not followed by a shock—physiological monitoring will show the same physiological responses they produced when they were shocked during training. In addition, they will show behavioral arrest—a species-typical defensive response called *freezing*. In other words, the animals act as if they were expecting to receive a shock. The tone becomes a *conditional stimulus* (*CS*) that elicits freezing: a *conditional response* (*CR*).

Research indicates that the physical changes responsible for the establishment of a conditioned emotional response take place in the lateral nucleus of the amygdala (Paré, Quirk, and LeDoux, 2004). Neurons in the lateral nucleus communicate with neurons in the central nucleus,

conditioned emotional response A classically conditioned response that occurs when a neutral stimulus is followed by an aversive stimulus; usually includes autonomic, behavioral, and endocrine components such as changes in heart rate, freezing, and secretion of stress-related hormones.

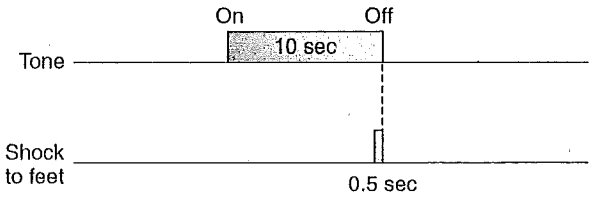

FIGURE 10.3 **Conditioned Emotional Responses.** The diagram shows the procedure used to produce conditioned emotional responses.

which in turn communicate with regions in the hypothalamus, midbrain, pons, and medulla that are responsible for the behavioral, autonomic, and hormonal components of a conditioned emotional response. More recent studies indicate that the neural circuitry responsible for the process of classical conditioning is actually more complex than that (Ciocchi et al., 2010; Haubensak et al., 2010; Duvarci, Popa, and Paré, 2011), but the details of this process will be postponed until Chapter 12, which discusses the physiology of learning and memory.

The neural mechanisms responsible for classically conditioned emotional responses evolved because they play a role in an animal's survival by increasing the likelihood that they can avoid dangerous situations, such as places where they encountered aversive events. In the laboratory, if the CS (tone) is presented repeatedly by itself, the previously established CR (emotional response) eventually disappears—it becomes *extinguished.* After all, the value of a conditioned emotional response is that it prepares an animal to confront (or better yet, avoid) an aversive stimulus. If the CS occurs repeatedly but the aversive stimulus does not follow, then it is better for the emotional response—which itself is disruptive and unpleasant—to disappear. And that is exactly what happens.

Behavioral studies have shown that extinction is not the same as forgetting. Instead, the animal learns that the CS is no longer followed by an aversive stimulus, and as a result of this learning, the expression of the CR is inhibited; the memory for the association between the CS and the aversive stimulus is not erased. This inhibition is supplied by the **ventromedial prefrontal cortex (vmPFC).** Evidence for this conclusion comes from several studies (Amano, Unal, and Paré, 2010; Sotres-Bayon and Quirk, 2010). For example, lesions of the vmPFC impair extinction, stimulation of this region inhibits conditioned emotional responses, and extinction training activates neurons there. Besides playing an essential role in extinction of conditioned emotional responses, the vmPFC can modulate the expression of fear in different circumstances. Depending on the situation, one subregion of the prefrontal cortex can become active and suppress a conditioned fear response, and another subregion can become active and enhance the response.

ventromedial prefrontal cortex The region of the prefrontal cortex at the base of the anterior frontal lobes, adjacent to the midline; plays an inhibitory role in the expression of emotions.

RESEARCH WITH HUMANS

We humans also acquire conditioned emotional responses. Let's examine a specific (if somewhat contrived) example. Suppose you are helping a friend prepare a meal. You pick up an electric mixer to mix some batter for a cake. Before you can turn the mixer on, the device makes a sputtering noise and then gives you a painful electrical shock. Your first response would be a defensive reflex: You would let go of the mixer, which would end the shock. This response is *specific;* it is aimed at terminating the painful stimulus. In addition, the painful stimulus would elicit *nonspecific* responses controlled by your autonomic nervous system: Your eyes would dilate, your heart rate and blood pressure would increase, you would breathe faster, and so on. The painful stimulus would also trigger the secretion of some stress-related hormones, another nonspecific response.

Suppose that a while later you visit your friend again and once more agree to make a cake. Your friend tells you that the electric mixer is perfectly safe. It has been fixed. Just seeing the mixer and thinking of holding it again makes you a little nervous, but you accept your friend's assurance and pick it up. Just then, it makes the same sputtering noise that it did when it shocked you. What would your response be? Almost certainly, you would drop the mixer again, even if it did not give you a shock. And your pupils would dilate, your heart rate and blood pressure would increase, and your endocrine glands would secrete some stress-related hormones. In other words, the sputtering sound would trigger a conditioned emotional response.

Evidence indicates that the amygdala is involved in emotional responses in humans. One of the earliest studies observed the reactions of people who were being evaluated for surgical removal of parts of the brain to treat severe seizure disorders. These studies found that stimulation of parts of the brain (for example, the hypothalamus) produced autonomic responses that are often associated with fear and anxiety but that only when the amygdala was stimulated did people also report that they actually *felt* afraid (White, 1940; Halgren et al., 1978; Gloor et al., 1982).

Many studies have shown that lesions of the amygdala decrease people's emotional responses. For example, Bechara et al. (1995) and LaBar et al. (1995) found that people with lesions of the amygdala showed impaired acquisition of a conditioned emotional response, just as rats do.

Most human fears are probably acquired socially, not through firsthand experience with painful stimuli (Olsson, Nearing, and Phelps, 2007). For example, a child does not have to be attacked by a dog to develop a fear of dogs: He or she can develop this fear by watching another person being attacked or (more often) by seeing another person display signs of fear when encountering a dog. People can also acquire a conditioned fear response through instruction. For example, suppose that someone is told (and believes) that if a warning light in a control panel goes on, he or she should leave the room immediately, because the light is connected to a sensor that detects toxic fumes. If the light does go on, the person will leave the room, and is also likely to experience a fear response while doing so.

We saw that studies with laboratory animals indicate that the vmPFC plays a critical role in extinction of a conditioned emotional response. The same is true for humans. Phelps et al. (2004) directly established a conditioned emotional response in human subjects by pairing the appearance of a visual stimulus with electric shocks to the wrist and then extinguished the response by presenting the squares alone, without any shocks. As Figure 11.4 shows, increased activity of the medial prefrontal cortex correlated with extinction of the conditioned response. (See ***Figure 10.4.***)

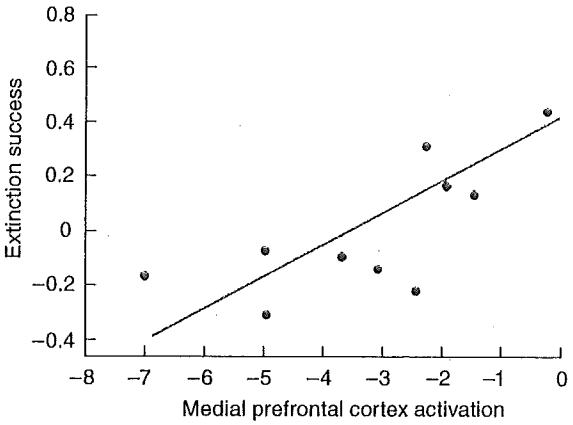

FIGURE 10.4 **Control of Extinction.** This graph shows that activation of the medial prefrontal cortex is related to the extinction of a conditioned emotional response.

Based on data from Phelps et al., 2004.

As we saw in Chapter 7, Patient I. R., a woman who had sustained damage to the auditory association cortex, was unable to perceive or produce melodic or rhythmic aspects of music (Peretz et al., 2001). She could not even tell the difference between consonant (pleasant) and dissonant (unpleasant) music. However, she was still able to recognize the mood conveyed by music. Gosselin et al. (2005) found that patients with damage to the amygdala showed the opposite symptoms: They had no trouble with musical perception but were unable to recognize scary music. They could still recognize happy and sad music. Thus, amygdala lesions impair recognition of a musical style that is normally associated with fear.

Anger, Aggression, and Impulse Control

Almost all species of animals engage in aggressive behaviors, which involve threatening gestures or actual attack directed toward another animal. Aggressive behaviors are species-typical; that is, the patterns of movements (for example, posturing, biting, striking, and hissing) are organized by neural circuits whose development is largely programmed by an animal's genes. Many aggressive behaviors are related to reproduction. For example, aggressive behaviors that gain access to mates, defend territory needed to attract mates or to provide a site for building a nest, or defend offspring against intruders can all be regarded as reproductive behaviors. Other aggressive behaviors are related to self-defense, such as that of an animal threatened by a predator or an intruder of the same species.

RESEARCH WITH LABORATORY ANIMALS

Neural Control of Aggressive Behavior The neural control of aggressive behavior is hierarchical. That is, the particular muscular movements an animal makes in attacking or defending itself are programmed by neural circuits in the brain stem. Whether an animal attacks depends on many factors, including the nature of the eliciting stimuli in the environment and the animal's previous experience. The activity of the brain stem circuits appears to be controlled by the hypothalamus and the amygdala, which also influence many other species-typical behaviors. And, of course, the activity of the hypothalamus and amygdala is controlled by perceptual systems that detect the status of the environment, including the presence of other animals.

Role of Serotonin An overwhelming amount of evidence suggests that the activity of serotonergic synapses inhibits aggression. In contrast, destruction of serotonergic axons in the forebrain facilitates aggressive attack, presumably by removing an inhibitory effect (Vergnes et al., 1988).

A group of researchers has studied the relationship between serotonergic activity and aggressiveness in a free-ranging colony of rhesus monkeys (reviewed by Howell et al., 2007). The researchers assessed serotonergic activity by capturing the monkeys, removing a sample of

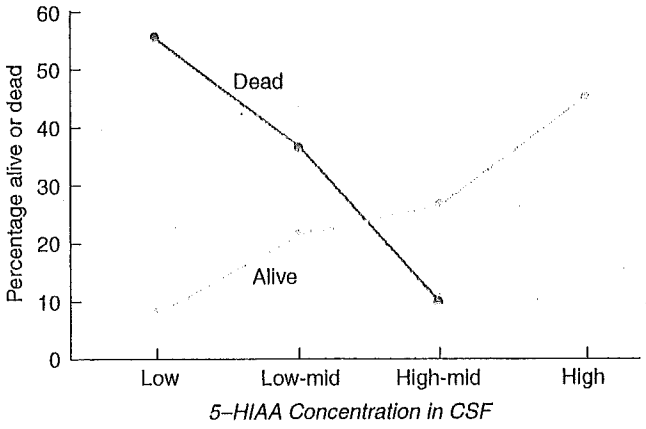

FIGURE 10.5 **Serotonin and Risk-Taking Behavior.** The graph shows the percentage of young male monkeys alive or dead as a function of 5-HIAA level in the CSF, measured four years previously.

Based on data from Higley, J. D., Mehlman, P. T., Higley, S. B., et al., *Archives of General Psychiatry*, 1996, *53*, 537–543.

cerebrospinal fluid, and analyzing it for 5-HIAA, a metabolite of serotonin (5-HT). When 5-HT is released, most of the neurotransmitter is taken back into the terminal buttons by means of reuptake, but some escapes and is broken down to 5-HIAA, which finds its way into the cerebrospinal fluid. Thus, high levels of 5-HIAA in the CSF indicate an elevated level of serotonergic activity. The investigators found that young male monkeys with the lowest levels of 5-HIAA showed a pattern of risk-taking behavior, including high levels of aggression directed toward animals that were older and much larger than themselves. They were much more likely to take dangerous unprovoked long leaps from tree to tree at a height of more than 7 m (27.6 ft). They were also more likely to pick fights that they could not possibly win. Of the preadolescent male monkeys that the investigators followed for four years, a large percentage of those with the lowest 5-HIAA levels died, while all of the monkeys with the highest levels survived. (See ***Figure 10.5.***) Most of the monkeys that died were killed by other monkeys. In fact, the first monkey to be killed had the lowest level of 5-HIAA and was seen attacking two mature males the night before his death. Clearly, serotonin does not simply inhibit aggression; rather, it exerts a controlling influence on risky behavior, which includes aggression.

Study of free-ranging colonies of rhesus monkeys has provided important information about the role of serotonin in risk-taking behavior and aggression.

Genetic studies with other species confirm the conclusion that serotonin has an inhibitory role in aggression. For example, selective breeding of rats and silver foxes has yielded animals that display tameness and friendly responses to human contact. These animals show increased brain levels of serotonin and 5-HIAA (Popova, 2006).

RESEARCH WITH HUMANS

Human violence and aggression are serious social problems. Consider the following case histories:

▶▶

Born to an alcoholic teen mother who raised him with an abusive alcoholic stepfather, Steve was hyperactive, irritable, and disobedient as a toddler .... After dropping out of school at age 14, Steve spent his teen years fighting, stealing, taking drugs, and beating up girlfriends .... School counseling, a probation officer, and meetings with child protective service failed to forestall disaster: At 19, several weeks after his last interview with researchers, Steve visited a girlfriend who had recently dumped him, found her with another man, and shot him to death. The same day he tried to kill himself. Now he's serving a life sentence without parole (Holden, 2000, p. 580).

By the time Joshua had reached the age of 2, ... he would bolt out of the house and into traffic. He kicked and head-butted relatives and friends. He poked the family hamster with a pencil and tried to strangle it. He threw regular temper tantrums and would stage toy-throwing frenzies. "At one point he was hurting himself—banging his head against a wall, pinching himself, not to mention leaping off the refrigerator .... Showering Joshua with love ... made little difference: By age 3, his behavior got him kicked out of his preschool (Holden, 2000, p. 581). ◀◀

Role of Heredity Early experiences can certainly foster the development of aggressive behavior, but studies have shown that heredity plays a significant role. For example, Viding et al. (2005, 2008) studied a group of same-sex twins at the ages of 7 years and 9 years and found a higher correlation between monozygotic twins than dizygotic twins on measures of antisocial behavior and levels of callous, unemotional behavior, which indicates a genetic component in the development of these traits. No evidence was found that a shared environment played a role.

Role of Serotonin Several studies have found that serotonergic neurons play an inhibitory role in human aggression. For example, a depressed rate of serotonin release (indicated by low levels of 5-HIAA in the CSF) are associated with aggression and other forms of antisocial behavior, including assault, arson, murder, and child beating (Lidberg et al., 1984, 1985; Virkkunen et al., 1989).

If low levels of serotonin release contribute to aggression, perhaps drugs that act as serotonin agonists might help to reduce antisocial behavior. In fact, a study by Coccaro and Kavoussi (1997) found that fluoxetine (Prozac), a serotonin agonist, decreased irritability and aggressiveness, as measured by a psychological test. Joshua, the little boy described in the introduction to this subsection, came under the care of a psychiatrist who prescribed monoaminergic agonists and began a course of behavior therapy that managed to stem Joshua's violent outbursts and risk-taking behaviors.

Role of the Ventromedial Prefrontal Cortex Many investigators believe that impulsive violence is a consequence of faulty emotional regulation. For most of us, frustrations may elicit an urge to respond emotionally, but we usually manage to calm ourselves and suppress these urges. As we shall see, the ventromedial prefrontal cortex plays an important role in regulating our responses to such situations. The analysis of social situations involves much more than sensory analysis; it involves experiences and memories, inferences and judgments. In fact, the skills involved include some of the most complex ones we possess. These skills are not localized in any one part of the cerebral cortex, although research does suggest that the right hemisphere is more important than the left. Nonetheless, the ventromedial prefrontal cortex—which includes the medial *orbitofrontal cortex* and the *subgenual anterior cingulate cortex*—plays a special role.

As we saw earlier in the discussion of the process of extinction, the ventromedial prefrontal cortex (vmPFC) plays a role in inhibiting emotional responses. The vmPFC is located just where its name suggests, at the bottom front of the cerebral hemispheres. (See ***Figure 10.6***.) The vmPFC receives direct inputs from the dorsomedial thalamus, temporal cortex, ventral tegmental area, olfactory system, and amygdala. Its outputs go to several brain regions, including the cingulate cortex, hippocampal formation, temporal cortex, lateral hypothalamus, and amygdala. Finally, it communicates with other regions of the prefrontal cortex. Thus, its inputs provide it with information about what is happening in the environment and what plans are being made by the rest of the frontal lobes, and its outputs permit it to affect a variety of behaviors and physiological responses, including emotional responses organized by the amygdala.

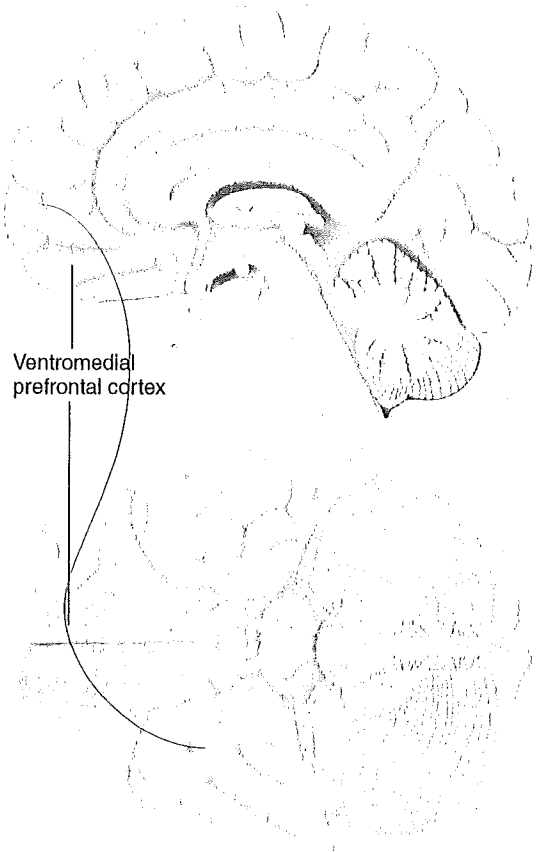

FIGURE 10.6 **The Location of the Ventromedial Prefrontal Cortex.**

The fact that the ventromedial prefrontal cortex plays an important role in control of emotional behavior is shown by the effects of damage to this region.

The first—and most famous—case comes from the mid-1800s. Phineas Gage, the foreman of a railway construction crew, was using a steel rod to ram a charge of blasting powder into a hole drilled in solid rock. Suddenly, the charge exploded and sent the rod into his cheek, through his brain, and out the top of his head. (See ***Figure 10.7***.) He survived, but he was a different man. Before his injury he was serious, industrious, and energetic. Afterward, he became childish, irresponsible, and thoughtless of others. His outbursts of temper led some people to remark that it looked as if Dr. Jekyll had become Mr. Hyde. He was unable to make or carry out plans, and his actions appeared to be capricious and whimsical. His accident had largely destroyed the orbitofrontal cortex (Damasio et al., 1994).

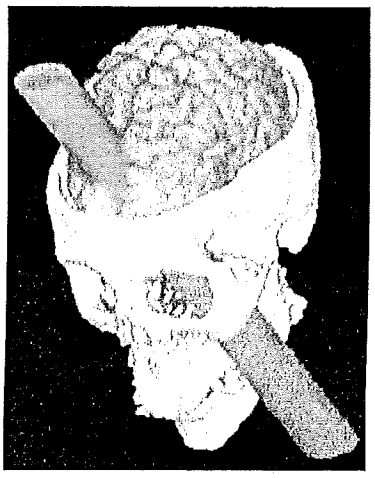

FIGURE 10.7 **Phineas Gage's Accident.** In the accident the steel rod entered his left cheek and exited through the top of his head.

Science Magazine. Damasio, H., Grabowski, T., Frank, R., Galaburda, A. M., and Damasio, A. R. *Science*, 1994, 264, 1102–1105. Copyright © 1994. Reprinted with permission from AAAS.

People whose ventromedial prefrontal cortex has been damaged by disease or accident are still able to accurately assess the significance of particular situations but only in a *theoretical* sense.

Eslinger and Damasio (1985) found that a patient with bilateral damage of the ventromedial prefrontal cortex (produced by a benign tumor, which was successfully removed) displayed excellent social judgment. When he was given hypothetical situations that required him to make decisions about what the

(*continued*)

people involved should do—situations involving moral, ethical, or practical dilemmas—he always gave sensible answers and justified them with carefully reasoned logic. However, his own life was a different matter. He frittered away his life's savings on investments that his family and friends pointed out were bound to fail. He lost one job after another because of his irresponsibility. He became unable to distinguish between trivial decisions and important ones, spending hours trying to decide where to have dinner but failing to use good judgment in situations that concerned his occupation and family life. (His wife finally left him and sued for divorce.) As the authors noted, "He had learned and used normal patterns of social behavior before his brain lesion, and although he could recall such patterns when he was questioned about their applicability, *real-life situations failed to evoke them*" (p. 1737).

Evidence suggests that the vmPFC serves as an interface between brain mechanisms involved in automatic emotional responses (both learned and unlearned) and those involved in the control of complex behaviors. This role includes using our emotional reactions to guide our behavior and in controlling the occurrence of emotional reactions in various social situations. ◀◀

Damage to the vmPFC causes serious and often debilitating impairments of behavioral control and decision-making. These impairments appear to be a consequence of emotional dysregulation. Anderson et al. (2006) obtained ratings of emotional behaviors of patients with damage to the vmPFC—such as frustration tolerance, emotional instability, anxiety, and irritability—from the patients' relatives. They also obtained ratings of the patients' real-world competencies, such as judgment, planning, social inappropriateness, and financial and occupational status, from both relatives and clinicians. They found a significant correlation between emotional dysfunction and impairments in real-world competencies. There was no relation between these impairments and the patients' cognitive abilities, which strongly suggests that emotional problems, and not problems in cognition, lie at the base of the real-world difficulties exhibited by people with vmPFC damage.

Evidence suggests that emotional reactions guide moral judgments as well as decisions involving personal risks and rewards and that the prefrontal cortex plays a role in these judgments as well. Consider the following moral dilemma (Thomson, 1986): You see a runaway trolley with five people aboard hurtling down a track leading to a cliff. Without your intervention these people will soon die. However, you are standing near a switch that will shunt the trolley off to another track, where the vehicle will stop safely. But a worker is standing on that track, and he will be killed if you throw the switch to save the five helpless passengers. Should you stand by and watch the trolley go off the cliff, or should you save them—and kill the man on the track?

Most people conclude that the better choice is to throw the switch; saving five people justifies the sacrifice of one man. This decision is based on conscious, logical application of a rule that it is better to kill one person than five people. But consider a variation of this dilemma. As before, the trolley is hurtling toward doom, but there is no switch at hand to shunt it to another track. Instead, you are standing on a bridge over the track. An obese man is standing there too, and if you give him a push, his body will fall on the track and stop the trolley. (You are too small to stop the trolley, so you cannot save the five people by sacrificing yourself.) What should you do?

Most people feel repugnant at the thought of pushing the man off the bridge and balk at the idea of doing so, even though the result would be the same as the first dilemma: one person lost, five people saved. Whether we kill someone by sending a trolley his way or by pushing him off a bridge into the path of an oncoming trolley, he dies when the trolley strikes him. But somehow, imagining yourself pushing a person's body and causing his death seems more emotionally wrenching than throwing a switch that changes the course of a runaway trolley. Thus, moral judgments appear to be guided by emotional reactions and are not simply the products of rational, logical decision-making processes.

In a functional imaging study, Greene et al. (2001) presented people with the moral dilemmas such as the one I just described and found that thinking about them activated several brain regions involved in emotional reactions, including the vmPFC. Making innocuous decisions, such as whether to take a bus or train to some destination, did not activate these regions. Perhaps, then, our reluctance to push someone to his death is guided by the unpleasant emotional reaction we feel when we contemplate this action.

Considering whether to throw a switch to cause one death but save five other lives evokes a much smaller emotional reaction than considering whether to shove someone on the tracks to

Phineas Gage had a personality change as a result of a rod that traveled through his brain.

Collection of Jack and Beverly Wilgus.

accomplish the same goal. Indeed, considering
activates the vmPFC. Therefore, we might expect
to push the man onto the track in the second dil
demonstrate *utilitarian* moral judgment: Killing
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narios to patients with vmPFC lesions, patients w
normal controls. For example, the switch-thrown
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(*See Table 10.1.*)

Figure 10.8 shows the proportion of the sub
groups who endorsed a decision to act in high-c
mas, such as the lifeboat scenario. As you can
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It might seem that I have been getting awa
anger and aggression. However, recall that mar
pulsive violence is a consequence of faulty emo
plays an important role in provoking anger a
and the prefrontal cortex plays an important r
lor by making us see its negative consequence

TABLE 10.1 **Examples of Scenarios Inv**
Personal Moral Judgment

Brownies (Nonmoral Scenario)
You have decided to make a batch of brownies for
for brownies. The recipe calls for a cup of chopped
damia nuts. As it happens, you have both kinds of

Would you substitute macadamia nuts for walnuts

Speedboat (Impersonal Moral Scenario)
While on vacation on a remote island, you are fishi
board a small boat and set sail for a nearby island.
there is a violent storm brewing, a storm that is su
their safety is to warn them by borrowing a nearby
who would not take kindly to your borrowing his p

Would you borrow the speedboat in order to warn

Lifeboat (Personal Moral Scenario)
You are on a cruise ship when there is a fire on boa
carrying many more people than they were design
low in the water—a few inches lower and it will sir
fill with water. If nothing is done it will sink before t
However, there is an injured person who will not s
the boat will stay afloat and the remaining passeng

Would you throw this person overboard in order to

ng moral, ethical, or practical dilemmas—he always carefully reasoned logic. However, his own life was a gs on investments that his family and friends pointed ther because of his irresponsibility. He became unable ortant ones, spending hours trying to decide where to situations that concerned his occupation and family ce.) As the authors noted, "He had learned and used ain lesion, and although he could recall such patterns ty, *real-life situations failed to evoke them*" (p. 1737). as an interface between brain mechanisms involved d and unlearned) and those involved in the control ur emotional reactions to guide our behavior and in ns in various social situations. ◀◀

l often debilitating impairments of behavioral con- ppear to be a consequence of emotional dysregula- of emotional behaviors of patients with damage to tional instability, anxiety, and irritability—from the the patients' real-world competencies, such as judg- nancial and occupational status, from both relatives ation between emotional dysfunction and impair- o relation between these impairments and the pa- sts that emotional problems, and not problems in ulties exhibited by people with vmPFC damage.

guide moral judgments as well as decisions involv- refrontal cortex plays a role in these judgments as homson, 1986): You see a runaway trolley with five a cliff. Without your intervention these people will itch that will shunt the trolley off to another track, s standing on that track, and he will be killed if you gers. Should you stand by and watch the trolley go the man on the track?

e is to throw the switch; saving five people justifies on conscious, logical application of a rule that it is onsider a variation of this dilemma. As before, the switch at hand to shunt it to another track. Instead, n obese man is standing there too, and if you give op the trolley. (You are too small to stop the trolley, g yourself.) What should you do?

of pushing the man off the bridge and balk at the be the same as the first dilemma: one person lost, y sending a trolley his way or by pushing him off ; he dies when the trolley strikes him. But some- dy and causing his death seems more emotionally the course of a runaway trolley. Thus, moral judg- tions and are not simply the products of rational,

l. (2001) presented people with the moral dilem- l that thinking about them activated several brain ing the vmPFC. Making innocuous decisions, such ation, did not activate these regions. Perhaps, then, guided by the unpleasant emotional reaction we

cause one death but save five other lives evokes a ering whether to shove someone on the tracks to accomplish the same goal. Indeed, considering the second dilemma, and not the first, strongly activates the vmPFC. Therefore, we might expect people with vmPFC damage to be more willing to push the man onto the track in the second dilemma. In fact, that is exactly what they do: They demonstrate *utilitarian* moral judgment: Killing one person is better than letting five people get killed. Koenigs et al. (2007) presented nonmoral, impersonal moral, and personal moral scenarios to patients with vmPFC lesions, patients with brain damage not including this region, and normal controls. For example, the switch-throwing scenario we just considered is an impersonal moral dilemma, and the person-pushing dilemma is a personal moral dilemma. Table 10.1 lists examples of the scenarios that the investigators of the present study presented to their subjects. (See ***Table 10.1.***)

Figure 10.8 shows the proportion of the subjects from each of the three groups who endorsed a decision to act in high-conflict personal moral dilemmas, such as the lifeboat scenario. As you can see, the patients with vmPFC lesions were much more likely to say "yes" to the question posed at the end of the scenarios. (See ***Figure 10.8.***)

I suggested a few paragraphs ago that our reluctance to push someone to his death even though that action would save the lives of others might be caused by the unpleasant thought of what it would feel like to commit that action and see the man fall to his death. If this is true, then perhaps people with vmPFC lesions say that they are willing to push the man off the bridge because thinking about doing so does not evoke an unpleasant emotional reaction. In fact, Moretto et al. (2009) found that people without brain damage showed physiological signs of an unpleasant emotional reaction when they contemplated pushing the man off the bridge—and said that they would not push him. People with vmPFC lesions did not show signs of this emotional reaction—and said that they *would* push him.

It might seem that I have been getting away from the topic of this section: anger and aggression. However, recall that many investigators believe that impulsive violence is a consequence of faulty emotional regulation. The amygdala plays an important role in provoking anger and violent emotional reactions, and the prefrontal cortex plays an important role in suppressing such behavior by making us see its negative consequences. As we saw earlier, antisocial

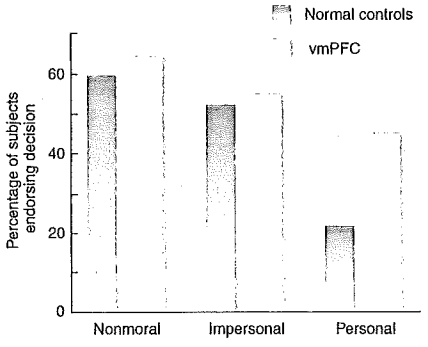

FIGURE 10.8 **Moral Decisions and the vmPFC.** The graph shows the percentage of people with lesions of the ventromedial prefrontal cortex and normal controls who endorse decisions of nonmoral, impersonal moral, and personal moral scenarios like the ones listed in Table 10.1.

Based on data from Koenigs et al., 2007.

TABLE 10.1 **Examples of Scenarios Involving Nonmoral, Impersonal Moral, and Personal Moral Judgments from the Study by Koenigs et al. (2007)**

Brownies (Nonmoral Scenario)
You have decided to make a batch of brownies for yourself. You open your recipe book and find a recipe for brownies. The recipe calls for a cup of chopped walnuts. You don't like walnuts, but you do like macadamia nuts. As it happens, you have both kinds of nuts available to you.
Would you substitute macadamia nuts for walnuts in order to avoid eating walnuts?

Speedboat (Impersonal Moral Scenario)
While on vacation on a remote island, you are fishing from a seaside dock. You observe a group of tourists board a small boat and set sail for a nearby island. Soon after their departure you hear over the radio that there is a violent storm brewing, a storm that is sure to intercept them. The only way that you can ensure their safety is to warn them by borrowing a nearby speedboat. The speedboat belongs to a miserly tycoon who would not take kindly to your borrowing his property.
Would you borrow the speedboat in order to warn the tourists about the storm?

Lifeboat (Personal Moral Scenario)
You are on a cruise ship when there is a fire on board, and the ship has to be abandoned. The lifeboats are carrying many more people than they were designed to carry. The lifeboat you're in is sitting dangerously low in the water—a few inches lower and it will sink. The seas start to get rough, and the boat begins to fill with water. If nothing is done it will sink before the rescue boats arrive and everyone on board will die. However, there is an injured person who will not survive in any case. If you throw that person overboard the boat will stay afloat and the remaining passengers will be saved.
Would you throw this person overboard in order to save the lives of the remaining passengers?

behavior may be associated with decreased volume of the prefrontal cortex; thus, activation of the prefrontal cortex may reflect its role in inhibiting aggressive behavior. Raine et al. (1998) found evidence of decreased prefrontal activity and increased subcortical activity (including the amygdala) in the brains of convicted murderers. These changes were primarily seen in impulsive, emotional murderers. The prefrontal activity of cold-blooded, calculating, predatory murderers—whose crimes were not accompanied by anger and rage—was closer to normal. Presumably, increased activation of the amygdala reflected an increased tendency for display of negative emotions, and the decreased activation of the prefrontal cortex reflected a decreased ability to inhibit the activity of the amygdala and thus control the people's emotions. Raine et al. (2002) found that people with antisocial personality disorder showed an 11 percent reduction in volume of the gray matter of the prefrontal cortex.

Earlier in this chapter we saw that decreased activity of serotonergic neurons is associated with aggression, violence, and risk taking. As we saw in this subsection, decreased activity of the prefrontal cortex is also associated with antisocial behavior. These two facts appear to be linked. The prefrontal cortex receives a major projection of serotonergic axons. Research indicates that serotonergic input to the prefrontal cortex activates this region. A functional imaging study by New et al. (2004) measured regional brain activity of people with histories of impulsive aggression before and after twelve weeks of treatment with a specific serotonin reuptake inhibitor. They found that the drug increased the activity of the prefrontal cortex and reduced aggressiveness. Crockett et al. (2010) found that a single high dose of a 5-HT agonist decreased the likelihood of subjects making a decision to cause harm in scenarios that presented moral dilemmas. In other words, the increased serotonergic activity made them less likely to make utilitarian decisions. It seems likely, therefore, that an abnormally *low* level of serotonin release can result in decreased activity of the prefrontal cortex and increased likelihood of utilitarian judgments or, in the extreme, antisocial behavior.

SECTION SUMMARY

Emotions as Response Patterns

The word *emotion* refers to behaviors, physiological responses, and feelings. This section has discussed emotional response patterns, which consist of behaviors that deal with particular situations and physiological responses (both autonomic and hormonal) that support the behaviors. The amygdala organizes behavioral, autonomic, and hormonal responses to a variety of situations, including those that produce fear, anger, or disgust. It receives inputs from the olfactory system, the association cortex of the temporal lobe, the frontal cortex, and the rest of the limbic system. Its outputs go to the frontal cortex, hypothalamus, hippocampal formation, and brain stem nuclei that control autonomic functions and some species-typical behaviors. Electrical recordings of single neurons in the amygdala indicate that some of them respond when the animal perceives particular stimuli with emotional significance. Stimulation of the amygdala leads to emotional responses, and its destruction disrupts them. Pairing of neutral stimuli with those that elicit emotional responses results in classically conditioned emotional responses. Learning of these responses takes place primarily in the amygdala. Extinction of conditioned emotional responses involves inhibitory control of amygdala activity by the ventromedial prefrontal cortex.

Studies of people with amygdala lesions and functional imaging studies with humans indicate that the amygdala is involved in emotional reactions in our species, too. However, many of our conditioned emotional responses are acquired by observing the responses of other people or even through verbal instruction.

Aggressive behaviors are species-typical and serve useful functions most of the time. These behaviors are organized by circuits in the brain stem, which are modulated by the amygdala and hypothalamus. The activity of serotonergic neurons appears to inhibit risk-taking behaviors, including aggression. Destruction of serotonergic axons in the forebrain enhances aggression, and administration of drugs that facilitate serotonergic transmission reduces it. Low CSF levels of 5-HIAA (a metabolite of serotonin) are correlated with increased risk-taking and aggressive behavior in monkeys and humans.

The ventromedial prefrontal cortex plays an important role in emotional reactions. This region communicates with the dorsomedial thalamus, temporal cortex, ventral tegmental area, olfactory system, amygdala, cingulate cortex, lateral hypothalamus, and other regions of the frontal cortex. People with lesions of the vmPFC show impulsive behavior and often display outbursts of inappropriate anger. Their lack of an emotional response in a situation that has important consequences for them often leads to poor decision making.

Evidence suggests that the prefrontal cortex is involved in making moral judgments. When people make judgments that involve conflicts between utilitarian judgments (one person dies but five people live) and personal moral judgments (are you willing to push a man to his death?), the ventromedial prefrontal cortex is activated. People with damage to the vmPFC display utilitarian moral judgments. Evidence suggests that nonpsychopathic people are reluctant to harm others because thinking about doing so produces an unpleasant emotional reaction. The release of serotonin in the prefrontal cortex activates this region, and some investigators believe that the serotonergic input to this region is

Section Summary (continued)

responsible for the ability of serotonin to inhibit a
behavior. A single high dose of a 5-HT agonist dec
that a person will make utilitarian decisions in a mo

Thought Questions

1. Phobias can be seen as dramatic examples o
tional responses. These responses can even be
acquire them without direct experience with a
For example, a child who sees a parent show s
presence of a dog may also develop a fear reac
you think that some prejudices might be learne
2. Suppose a man sustained brain damage that d
prefrontal cortex, and soon afterward he began

Communication of Emotic

The previous section described emotions as organ
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pose a threat to the organism. For our earliest prem
was to emotions. But over time other responses, witl
(including our own) communicate their emotions to
ions, and nonverbal sounds (such as sighs, moans,
functions; they tell other individuals how we feel and
example, they warn a rival that we are angry or tell fri
and reassurance. They can also indicate that a dang
seems to be happening. This section examines such

Facial Expression of Emotions: In

Charles Darwin (1872/1965) suggested that human e
expressions in other animals. He said that emotion
consisting of a complex set of movements, principa
and a wolf's snarl are biologically determined resp
mechanisms, just as coughing and sneezing are. (Of
quite different reasons.) Some of these movements r
evolved from them. For example, a snarl shows one's

Darwin obtained evidence for his conclusion
serving his own children and by corresponding v
around the world. He reasoned that if people all ov
same facial expressions of emotion, then these expre
logical argument goes like this: When groups of pe
different languages. Thus, we can say that the words
basis for using particular words to represent particu
inherited, then they should take approximately the
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Research by Ekman and his colleagues (Ekman
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movements of facial muscles (Darwin, 1872/1965). F
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Westerners readily recognized. Figure 10.9 shows fou
from this tribe reacting to stories designed to evoke fac
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are reluctant to harm others because think-
luces an unpleasant emotional reaction. The
ıe prefrontal cortex activates this region, and
ve that the serotonergic input to this region is

Section Summary *(continued)*

responsible for the ability of serotonin to inhibit aggression and risky behavior. A single high dose of a 5-HT agonist decreases the likelihood that a person will make utilitarian decisions in a moral dilemma task.

Thought Questions

1. Phobias can be seen as dramatic examples of conditioned emotional responses. These responses can even be contagious; we can acquire them without direct experience with an aversive stimulus. For example, a child who sees a parent show signs of fright in the presence of a dog may also develop a fear reaction to the dog. Do you think that some prejudices might be learned in this way, too?
2. Suppose a man sustained brain damage that destroyed his ventral prefrontal cortex, and soon afterward he began exhibiting antisocial behavior. One day, while he was in his car waiting for a red light to change, he saw a man with whom he had previously had a violent argument cross the street in front of him. He suddenly stepped on the accelerator, struck the man, and killed him. Should the fact of his brain damage play any role in his prosecution and judgment in court? Why or why not?
3. From the point of view of evolution, aggressive behavior and a tendency to establish dominance have useful functions. In particular, they increase the likelihood that only the most healthy and vigorous animals will reproduce. Can you think of examples of good and bad effects of these tendencies among members of our own species?

Communication of Emotions

The previous section described emotions as organized responses (behavioral, autonomic, and hormonal) that prepare an animal to deal with existing situations in the environment, such as events that pose a threat to the organism. For our earliest premammalian ancestors, that is undoubtedly all there was to emotions. But over time other responses, with new functions, evolved. Many species of animals (including our own) communicate their emotions to others by means of postural changes, facial expressions, and nonverbal sounds (such as sighs, moans, and growls). These expressions serve useful social functions; they tell other individuals how we feel and—more to the point—what we are likely to do. For example, they warn a rival that we are angry or tell friends that we are sad and would like some comfort and reassurance. They can also indicate that a danger might be present or that something interesting seems to be happening. This section examines such expression and communication of emotions.

Facial Expression of Emotions: Innate Responses

Charles Darwin (1872/1965) suggested that human expressions of emotion have evolved from similar expressions in other animals. He said that emotional expressions are innate, unlearned responses consisting of a complex set of movements, principally of the facial muscles. Thus, a person's sneer and a wolf's snarl are biologically determined response patterns, both controlled by innate brain mechanisms, just as coughing and sneezing are. (Of course, people can sneer and wolves can snarl for quite different reasons.) Some of these movements resemble the behaviors themselves and may have evolved from them. For example, a snarl shows one's teeth and can be seen as an anticipation of biting.

Darwin obtained evidence for his conclusion that emotional expressions were innate by observing his own children and by corresponding with people living in various isolated cultures around the world. He reasoned that if people all over the world, no matter how isolated, show the same facial expressions of emotion, then these expressions must be inherited instead of learned. The logical argument goes like this: When groups of people are isolated for many years, they develop different languages. Thus, we can say that the words people use are arbitrary; there is no biological basis for using particular words to represent particular concepts. However, if facial expressions are inherited, then they should take approximately the same form in people from all cultures, despite their isolation from one another. And Darwin did, indeed, find that people in different cultures used the same patterns of movement of facial muscles to express a particular emotional state.

Research by Ekman and his colleagues (Ekman and Friesen, 1971; Ekman, 1980) tends to confirm Darwin's hypothesis that facial expression of emotion uses an innate, species-typical repertoire of movements of facial muscles (Darwin, 1872/1965). For example, Ekman and Friesen (1971) studied the ability of members of an isolated tribe in New Guinea to recognize facial expressions of emotion produced by Westerners. They had no trouble doing so and themselves produced facial expressions that Westerners readily recognized. Figure 10.9 shows four photographs taken from videotapes of a man from this tribe reacting to stories designed to evoke facial expressions of happiness, sadness, anger, and disgust. I am sure that you will have no trouble recognizing which is which. (See ***Figure 10.9.***)

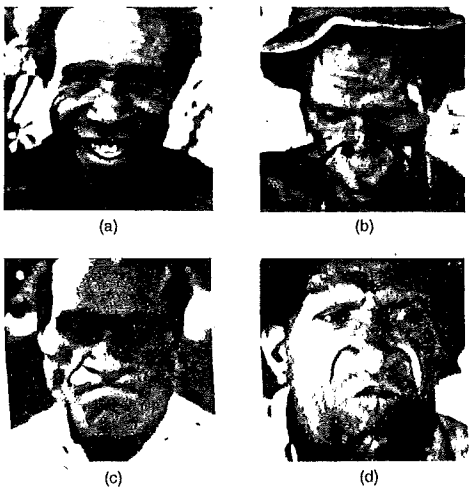

FIGURE 10.9 **Facial Expressions in a New Guinea Tribesman.** The tribesman made faces when told stories: (a) "Your friend has come and you are happy." (b) "Your child had died." (c) "You are angry and about to fight." (d) "You see a dead pig that has been lying there a long time."

From Ekman, P., *The Face of Man: Expressions of Universal Emotions in a New Guinea Village.* New York: Garland STPM Press, 1980. Reprinted with permission.

Because the same facial expressions were used by people who had not previously been exposed to each other, Ekman and Friesen concluded that the expressions were unlearned behavior patterns. In contrast, different cultures use different words to express particular concepts; production of these words does not involve innate responses but must be learned.

Other researchers have compared the facial expressions of blind and normally sighted people. They reasoned that if the facial expressions of the two groups are similar, then the expressions are natural for our species and do not require learning by imitation. In fact, the facial expressions of young blind and sighted children are very similar (Woodworth and Schlosberg, 1954; Izard, 1971). In addition, a study of the emotional expressions of people competing in (and winning or losing) athletic events in the 2004 Paralympic Games found no differences between the expressions of congenitally blind, noncongenitally blind, and sighted athletes (Matsumoto and Willingham, 2009). Thus, both the cross-cultural studies and the investigations of blind people confirm the naturalness of these facial expressions of emotion.

A study by Sauter et al. (2010) reached similar conclusions. The investigators carried out a vocal version of the study by Ekman and Friesen. They presented European English-speakers and natives of isolated northern Namibian villages with recordings of sounds of nonverbal vocalizations to situations that would be expected to produce the emotions of anger, disgust, fear, sadness, surprise, or amusement. The participants were told a story and then heard two different vocalizations (sighs, groans, laughs, etc.), one of which would be appropriate for the emotion produced by the story. Members of both cultures had no difficulty choosing the correct vocalizations of members of their culture and the other culture. (See ***Figure 10.10***.)

Neural Basis of the Communication of Emotions: Recognition

Effective communication is a two-way process. That is, the ability to display one's emotional state by changes in expression is useful only if other people are able to recognize them. In fact, Kraut and Johnston (1979) unobtrusively observed people in circumstances that would be likely to make them happy. They found that happy situations (such as making a strike while bowling, seeing the home team score, or experiencing a beautiful day) produced only small signs of happiness when the people were alone. However, when the people were interacting socially with other people, they were much more likely to smile. For example, bowlers who made a strike usually did not smile when the ball hit the pins, but when they turned around to face their companions, they often smiled. Jones et al. (1991) found that even 10-month-old children showed this tendency. (No, I'm not suggesting that infants have been observed while bowling.)

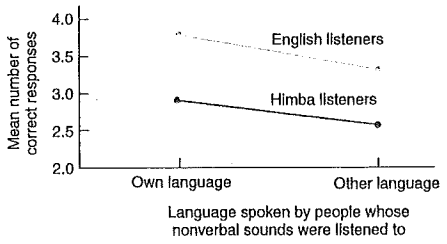

FIGURE 10.10 **Identification of Nonverbal Vocal Expressions of Emotions in a Different Culture.** The graph shows the mean number of correct responses in identifying audio recordings of nonverbal emotional expressions of English speakers and Himba speakers (residents of northern Namibia) by speakers of English and Himba. Accuracy for cross-cultural identification was almost as good as intra-cultural identification.

Based on data from Sauter et al., 2010.

Recognition of another person's facial expression of emotions is generally automatic, rapid, and accurate. Tracy and Robbins (2008) found that observers quickly recognized brief expressions of a variety of emotions. If they were given more time to think about the expression they had seen, they showed very little improvement.

People can express emotions through their body language, as well as through muscular movements of their face (de Gelder, 2006). For example, a clenched fist might accompany an angry facial expression, and a fearful person may run away. The sight of photographs of bodies posed in gestures of fear activates the amygdala, just as the sight of fearful faces does (Hadjikhani and de Gelder, 2003). Meeren, van Heijnsbergen, and de Gelder (2005) prepared computer-modified photographs of people showing facial expressions of emotions that were either congruent with the person's body posture (for example, a facial expression of fear and a body posture of fear) or incongruent (for example, a facial expression of anger and a body posture of fear). The investigators asked people to identify the *facial* expressions shown in the photos and found that the ratings were faster and
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emotion is affected by their body posture as well

LATERALITY OF EMOTIONAL RECOGNITIO

We recognize other people's feelings by means o
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listened to some sentences and identified their
emotional content. In one condition the subjects listened to the meaning of the words and said whether they described a situation in which someone would be happy, sad, angry, or neutral. In another condition they judged the emotional state from the tone of the voice. The investigators found that comprehension of emotion from word meaning increased the activity of the prefrontal cortex bilaterally, the left more than the right. Comprehension of emotion from tone of voice increased the activity of only the right prefrontal cortex. (See ***Figure 10.11***.)

Heilman, Watson, and Bowers (1983) record
disorder called *pure word deafness*, caused by dam
is described in Chapter 13.) The man could not
difficulty identifying the emotion being expressed
imaging study by George et al. (1996), indicates t
tone of voice are independent functions.

ROLE OF THE AMYGDALA

As we saw in the previous section, the amygdala pl
a role in emotional recognition as well. For examp
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Krolak-Salmon et al. (2004) recorded electrical
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ROLE OF IMITATION IN RECOGNITION OF E THE MIRROR NEURON SYSTEM

Adolphs et al. (2000) discovered a possible link bet
They compiled computerized information about t
localized brain lesions and correlated this informat
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Adolphs et al. suggest that the somatosensory repre

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ɜed to each other, Ekman and Friesen concluded
unlearned behavior patterns. In contrast, differ-
words to express particular concepts; produc-
ıot involve innate responses but must be learned.
ıave compared the facial expressions of blind
ɔople. They reasoned that if the facial expres-
are similar, then the expressions are natural
ıot require learning by imitation. In fact, the
ng blind and sighted children are very similar
ıberg, 1954; Izard, 1971). In addition, a study
ıions of people competing in (and winning or
ı the 2004 Paralympic Games found no differ-
ɜssions of congenitally blind, noncongenitally
es (Matsumoto and Willingham, 2009). Thus,
studies and the investigations of blind people
of these facial expressions of emotion.
al. (2010) reached similar conclusions. The in-
ʼocal version of the study by Ekman and Friesen.
ı English-speakers and natives of isolated north-
h recordings of sounds of nonverbal vocalizations
e expected to produce the emotions of anger, dis-
ise, or amusement. The participants were told a
different vocalizations (sighs, groans, laughs, etc.),
ɔpropriate for the emotion produced by the story.
s had no difficulty choosing the correct vocaliza-
culture and the other culture. (See ***Figure 10.10.***)

on of Emotions: Recognition

hat is, the ability to display one's emotional state
ɔple are able to recognize them. In fact, Kraut and
circumstances that would be likely to make them
making a strike while bowling, seeing the home
ced only small signs of happiness when the people
acting socially with other people, they were much
made a strike usually did not smile when the ball
e their companions, they often smiled. Jones et al.
ʌen 10-month-old children showed this tendency.
ng that infants have been observed while bowling.)
another person's facial expression of emotions is
, rapid, and accurate. Tracy and Robbins (2008)
s quickly recognized brief expressions of a variety
were given more time to think about the expres-
they showed very little improvement.
ress emotions through their body language, as well
ır movements of their face (de Gelder, 2006). For
ɪ fist might accompany an angry facial expression,
ı may run away. The sight of photographs of bod-
es of fear activates the amygdala, just as the sight
ɜs (Hadjikhani and de Gelder, 2003). Meeren, van
ɪe Gelder (2005) prepared computer-modified pho-
showing facial expressions of emotions that were
ʼith the person's body posture (for example, a facial
ınd a body posture of fear) or incongruent (for ex-
ɔession of anger and a body posture of fear). The in-
eople to identify the *facial* expressions shown in the
photos and found that the ratings were faster and more accurate when the facial and body expressions were congruent. In other words, when we look at other people's faces, our perception of their emotion is affected by their body posture as well as by their facial expression.

LATERALITY OF EMOTIONAL RECOGNITION

We recognize other people's feelings by means of vision and audition—seeing their facial expressions and hearing their tone of voice and choice of words. Many studies have found that the right hemisphere plays a more important role than the left hemisphere in comprehension of emotion. For example, George et al. (1996) measured subjects' regional cerebral blood flow while the subjects listened to some sentences and identified their emotional content. In one condition the subjects listened to the meaning of the words and said whether they described a situation in which someone would be happy, sad, angry, or neutral. In another condition they judged the emotional state from the tone of the voice. The investigators found that comprehension of emotion from word meaning increased the activity of the prefrontal cortex bilaterally, the left more than the right. Comprehension of emotion from tone of voice increased the activity of only the right prefrontal cortex. (See ***Figure 10.11.***)

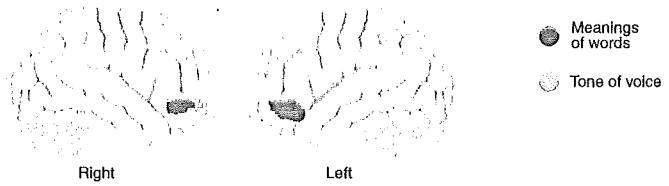


FIGURE 10.11 Perception of Emotions. The PET scans indicate brain regions activated by listening to emotions expressed by tone of voice (green) or by meanings of words (red).

Tracings of brain activity from George et al., 1996.

Heilman, Watson, and Bowers (1983) recorded a particularly interesting case of a man with a disorder called *pure word deafness*, caused by damage to the left temporal cortex. (This syndrome is described in Chapter 13.) The man could not comprehend the meaning of speech but had no difficulty identifying the emotion being expressed by its intonation. This case, like the functional imaging study by George et al. (1996), indicates that comprehension of words and recognition of tone of voice are independent functions.

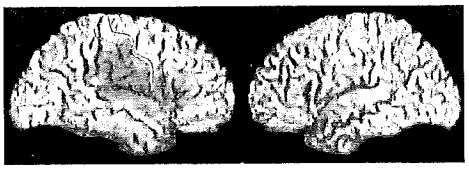
ROLE OF THE AMYGDALA

As we saw in the previous section, the amygdala plays a special role in emotional responses. It plays a role in emotional recognition as well. For example, several studies have found that lesions of the amygdala (the result of degenerative diseases or surgery for severe seizure disorders) impair people's ability to recognize facial expressions of emotion, especially expressions of fear (Adolphs et al., 1994, 1995; Young et al., 1995; Calder et al., 1996). In addition, functional imaging studies (Morris et al., 1996; Whalen et al., 1998) have found large increases in the activity of the amygdala when people view photographs of faces expressing fear but only small increases (or even decreases) when they look at photographs of happy faces. However, amygdala lesions do not appear to affect people's ability to recognize emotions in tone of voice (Anderson and Phelps, 1998; Adolphs and Tranel, 1999).

Krolak-Salmon et al. (2004) recorded electrical potentials from the amygdala and visual association cortex through electrodes that had been implanted in people who were being evaluated for neurosurgery to alleviate a seizure disorder. They presented the people with photographs of faces showing neutral expressions or expressions of fear, happiness, or disgust. The found that fearful faces produced the largest response and that the amygdala showed activity before the visual cortex did. The rapid response suggests that visual information that the amygdala receives directly from the subcortical visual system (which conducts information very rapidly) permits it to recognize facial expressions of fear.

ROLE OF IMITATION IN RECOGNITION OF EMOTIONAL EXPRESSIONS: THE MIRROR NEURON SYSTEM

Adolphs et al. (2000) discovered a possible link between somatosensation and emotional recognition. They compiled computerized information about the locations of brain damage in 108 patients with localized brain lesions and correlated this information with the patients' ability to recognize and identify facial expressions of emotions. They found that the most severe damage to this ability was caused by damage to the somatosensory cortex of the right hemisphere. (See ***Figure 10.12.***) They suggest that when we see a facial expression of an emotion, we unconsciously imagine ourselves making that expression. Often, we do more than imagine making the expressions—we actually imitate what we see. Adolphs et al. suggest that the somatosensory representation of what it feels like to make the perceived



Right hemisphere Left hemisphere

FIGURE 10.12 **Brain Damage and Recognition of Facial Expressions of Emotion.** This computer-generated image shows performance of subjects with localized brain damage on recognition of facial expressions of emotion. The colored areas outline the site of the lesions. Lesions that resulted in good performance are shown in shades of blue; those that resulted in poor performance are shown in red and yellow.

From Adolphs, R., Damasio, H., Tranel, D., Cooper, G., and Damasio, A. R. *The Journal of Neuroscience*, 2000, *20*, 2683–2690. Copyright © 2000 by the Society for Neuroscience. Reprinted with permission.

expression provides cues we use to recognize the emotion being expressed in the face we are viewing. In support of this hypothesis, Adolphs and his colleagues report that the ability of patients with right hemisphere lesions to recognize facial expressions of emotions is correlated with their ability to perceive somatosensory stimuli. That is, patients with somatosensory impairments (caused by right-hemisphere lesions) also had impairments in recognition of emotions.

Hussey and Safford (2009) review a considerable amount of evidence that supports this hypothesis (the so-called *simulationist* hypothesis). For example, neuroimaging studies have shown that brain regions that are activated when particular emotional expressions are observed are also activated when these expressions are imitated. In addition, a study by Pitcher et al. (2008) used transcranial magnetic stimulation to disrupt the normal activity of brain regions involved in visual perception of faces or perception of somatosensory feedback from one's own face. They found that disruption of *either* region impaired people's ability to recognize facial expressions of emotion. Finally, a study by Oberman et al. (2007) had people hold a pen between their teeth, which interfered with smiling. When they did so, they had difficulty recognizing facial expressions of happiness, but not expressions of disgust, fear, and sadness, which involve the upper part of the face more than smiling does.

We are beginning to understand the neural circuit that provides this form of feedback. Research has found that *mirror neurons* play an important role in the control of movement (Gallese et al., 1996; Rizzolatti et al., 2001; Rizzolatti and Sinigaglia, 2010). Mirror neurons are activated when an animal performs a particular behavior or when it sees another animal performing that behavior. Presumably, these neurons are involved in learning to imitate the actions of others. These neurons, which are located in the ventral premotor cortex of the frontal lobe, receive input from the superior temporal sulcus and the posterior parietal cortex. This circuit is activated when we see another person perform a goal-directed action, and feedback from this activity helps us to understand what the person is trying to accomplish. Carr et al. (2003) suggest that the mirror neuron system, which is activated when we observe facial movements of other people, provides feedback that helps us to understand how other people feel. In other words, the mirror neuron system may be involved in our ability to empathize with the emotions of other people.

FIGURE 10.13 **An Artificial Smile.** The photograph shows Dr. Duchenne electrically stimulating muscles in the face of a volunteer, causing contraction of muscles around the mouth that become active during a smile. As Duchenne discovered, however, a true smile also involves muscles around the eyes.

Hulton-Deutsch Collection/CORBIS.

A neurological disorder known as Moebius syndrome provides further support for this hypothesis. Moebius syndrome is caused by defective development of nerves involved in the movement of facial muscles. Because of this paralysis, people affected with this syndrome cannot make facial expressions of emotion. In addition, they have difficulty recognizing the emotional expressions of other people (Cole, 2001). Perhaps their inability to produce facial expressions of emotions makes it impossible for them to imitate the expressions of other people, and the lack of internal feedback from the motor system to the somatosensory cortex may make the task of recognition more difficult.

Neural Basis of the Communication of Emotions: Expression

Facial expressions of emotion are automatic and involuntary (although, as we saw, they can be modified by display rules). It is not easy to produce a realistic facial expression of emotion when we do not really feel that way. In fact, Ekman and Davidson have confirmed an early observation by a nineteenth-century neurologist, Guillaume-Benjamin Duchenne de Boulogne, that genuinely happy smiles, as opposed to false smiles or social smiles people make when they greet someone else, involve contraction of a muscle near the eyes, the lateral part of the orbicularis oculi—now sometimes referred to as Duchenne's muscle (Ekman, 1992; Ekman and Davidson, 1993). As Duchenne put it, "The first [zygomatic major muscle] obeys the will but the second [orbicularis oculi] is only put in play by the sweet emotions of the soul; the . . . fake joy, the deceitful laugh, cannot provoke the contraction of this latter muscle" (Duchenne, 1862/1990, p. 72). (See ***Figure 10.13.***) The difficulty actors have in voluntarily producing a convincing facial expression of

volitional facial paresis Difficulty in moving the facial muscles voluntarily; caused by damage to the face region of the primary motor cortex or its subcortical connections.

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Communication of Emotions:

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emotion is one of the reasons that led Constantin Stanislavsky to develop his system of *method acting*, in which actors attempt to imagine themselves in a situation that would lead to the desired emotion. Once the emotion is evoked, the facial expressions follow naturally (Stanislavsky, 1936).

This observation is confirmed by two neurological disorders with complementary symptoms (Hopf et al., 1992; Topper et al., 1995; Urban et al., 1998; Michel et al., 2008). The first, **volitional facial paresis**, is caused by damage to the face region of the primary motor cortex or to the fibers connecting this region with the motor nucleus of the facial nerve, which controls the muscles responsible for movement of the facial muscles. (*Paresis*, from the Greek "to let go," refers to a partial paralysis.) The interesting thing about volitional facial paresis is that the patient cannot voluntarily move the facial muscles but will express a genuine emotion with those muscles. For example, Figure 10.14a shows a woman trying to pull her lips apart and show her teeth. Because of the lesion in the face region of her right primary motor cortex, she could not move the left side of her face. However, when she laughed (Figure 10.14b), both sides of her face moved normally. (See ***Figure 10.14a*** and ***10.14b***.)

In contrast, **emotional facial paresis** is caused by damage to the insular region of the prefrontal cortex, to the white matter of the frontal lobe, or to parts of the thalamus. This system joins the system responsible for voluntary movements of the facial muscles in the medulla or caudal pons. People with this disorder can move their face muscles voluntarily but do not express emotions on the affected side of the face. Figure 10.14c shows a man pulling his lips apart to show his teeth, which he had no trouble doing. Figure 10.14d shows him smiling; as you can see, only the left side of his mouth is raised. He had a stroke that damaged the white matter of the left frontal lobe. (See ***Figure 10.14c*** and ***10.14d***.) These two syndromes clearly indicate that different brain mechanisms are responsible for voluntary movements of the facial muscles and automatic, involuntary expression of emotions involving the same muscles.

As we saw in the previous subsection, the right hemisphere plays a more significant role in recognizing emotions in the voice or facial expressions of other people—especially negative emotions. The same hemispheric specialization appears to be true for expressing emotions. When people show emotions with their facial muscles, the left side of the face usually makes a more intense expression. For example, Sackeim and Gur (1978) cut photographs of people who were expressing emotions into right and left halves, prepared mirror images of each of them, and pasted them together, producing so-called *chimerical faces* (from the mythical Chimera, a fire-breathing monster, part goat, part lion, and part serpent). They found that the left halves were more expressive than the right ones. (See ***Figure 10.15***.) Because motor control is contralateral, the results suggest that the right hemisphere is more expressive than the left.

Moscovitch and Olds (1982) made more natural observations of people in restaurants and parks and found that the left side of their faces appeared to make stronger expressions of emotions. They confirmed these results in the laboratory by analyzing videotapes of people telling sad or humorous stories. A review of the literature by Borod et al. (1998) found forty-eight other studies that obtained similar results.

Left hemisphere lesions do not usually impair vocal expressions of emotion. For example, people with Wernicke's aphasia (described in Chapter 13) usually modulate their voice according to mood, even though the words they say make no sense. In contrast, right-hemisphere lesions do impair expression of emotion, both facially and by tone of voice.

We saw in the previous subsection that the amygdala is involved in the recognition of other people's facial expression of emotions. Research indicates that it is *not* involved in the *expression* of facial emotions.

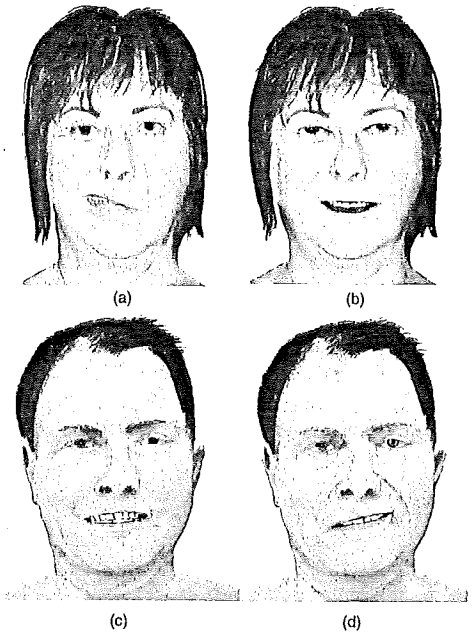

FIGURE 10.14 **Emotional and Volitional Paresis.** (a) A woman with volitional facial paresis caused by a right hemisphere lesion tries to pull her lips apart and show her teeth. Only the right side of her face responds. (b) The same woman shows a genuine smile. (c) A man with emotional facial paresis caused by a left-hemisphere lesion shows his teeth. (d) The same man is smiling. Only the left side of his face responds.

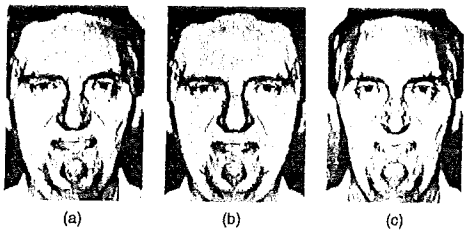

FIGURE 10.15 **Chimerical Faces.** (a) Original photo. (b) Composite of the right side of the man's face. (c) Composite of the left side of the man's face.

Reprinted with permission from *Neuropsychologia, 16*, H. A. Sackeim and R. C. Gur, Lateral asymmetry in intensity of emotional expression. Copyright 1978, Pergamon Press.

emotional facial paresis Lack of movement of facial muscles in response to emotions in people who have no difficulty moving these muscles voluntarily; caused by damage to the insular prefrontal cortex, subcortical white matter of the frontal lobe, or parts of the thalamus.

Anderson and Phelps (2000) reported the case of S. P., a 54-year-old woman whose right amygdala was removed to treat a serious seizure disorder. Because of a preexisting lesion of the left amygdala, the surgery resulted in a bilateral amygdalectomy. After the surgery, S. P. lost the ability to recognize facial expressions of fear, but she had no difficulty recognizing individual faces, and she could easily identify male and female faces and accurately judge their ages. What is particularly interesting is that the amygdala lesions did not impair S. P.'s ability to produce her own facial expressions of fear. She had no difficulty accurately expressing fear, anger, happiness, sadness, disgust, and surprise. By the way, when she saw a photograph of herself showing fear, she could not tell what emotion her face had been expressing.

SECTION SUMMARY

Communication of Emotions

We (and members of other species) communicate our emotions primarily through facial gestures. Darwin believed that such expressions of emotion were innate—that these muscular movements were inherited behavioral patterns. Ekman and his colleagues performed cross-cultural studies with members of an isolated tribe in New Guinea. Their results supported Darwin's hypothesis. We also receive information about people's emotions from their body posture or movement.

Recognition of other people's emotional expressions involves the right hemisphere more than the left. Functional imaging indicates that when people judge the emotions of voices, the right hemisphere is activated more than the left. In addition, some people with pure word deafness, caused by damage to the left hemisphere, cannot recognize words but can still recognize the emotions portrayed by tone of voice. The amygdala plays a role in recognition of facial expressions of fearfulness; lesions of the amygdala disrupt this ability, and functional imaging studies show increased activity of the amygdala while the subject is engaging in this task. The ability to judge emotions by a person's tone of voice is not affected.

The amygdala receives more primitive visual information from subcortical regions of the brain, and this information is used in making judgments about fearful expressions. There is a natural tendency to imitate the emotional expressions of other people—a response that involves mirror neurons in the ventral premotor cortex. Feedback from this activity, which is transmitted to the somatosensory cortex, appears to help us to comprehend the emotional intentions of other people.

Facial expression of emotions (and other stereotypical behaviors such as laughing and crying) are almost impossible to simulate. For example, only a genuine smile of pleasure causes the contraction of the lateral part of the orbicularis oculi (Duchenne's muscle). Genuine expressions of emotion are controlled by special neural circuits. The best evidence for this assertion comes from the complementary syndromes of emotional and volitional facial paresis. People with emotional facial paresis can move their facial muscles voluntarily but not in response to an emotion, whereas people with volitional facial paresis show the opposite symptoms. In addition, the left halves of people's faces tend to be more expressive than the right halves.

Thought Questions

1. Do you think it is important to be able to recognize other people's emotions? Why? Suppose that you could no longer recognize people's facial expressions of emotions. What consequences would that loss have for you?
2. Novelists will sometimes say that a person's smile did not reach his or her eyes. What do they mean by that?

Feelings of Emotions

So far, we have examined two aspects of emotions: the organization of patterns of responses that deal with the situation that provokes the emotion and the communication of emotional states with other members of the species. The final aspect of emotion to be examined in this chapter is the subjective component: feelings of emotion.

The James-Lange Theory

William James (1842–1910), an American psychologist, and Carl Lange (1834–1900), a Danish physiologist, independently suggested similar explanations for emotion, which most people refer to collectively as the **James-Lange theory** (James, 1884; Lange, 1887). Basically, the theory states that emotion-producing situations elicit an appropriate set of physiological responses, such as trembling, sweating, and increased heart rate. The situations also elicit behaviors, such as clenching of the fists or fighting. The brain receives sensory feedback from the muscles and from the organs that produce these responses, and it is this feedback that constitutes our feeling of emotion.

James-Lange theory A theory of emotion that suggests that behaviors and physiological responses are directly elicited by situations and that feelings of emotions are produced by feedback from these behaviors and responses.

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Or did you ever find tears coming to your eye
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In one of the few tests of James's theory, Hohm
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> scrounge around and put it out. I could have b
> shook up about it. I just didn't feel afraid at all,

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> Now, I don't get a feeling of physical animatio
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uine smile of pleasure causes the contraction of the
orbicularis oculi (Duchenne's muscle). Genuine ex-
ion are controlled by special neural circuits. The best
ssertion comes from the complementary syndromes
volitional facial paresis. People with emotional facial
their facial muscles voluntarily but not in response to
eas people with volitional facial paresis show the op-
In addition, the left halves of people's faces tend to be
an the right halves.

ons
it is important to be able to recognize other people's
y? Suppose that you could no longer recognize peo-
ressions of emotions. What consequences would that
ou?
sometimes say that a person's smile did not reach his
hat do they mean by that?

ons: the organization of patterns of responses that
n and the communication of emotional states with
t of emotion to be examined in this chapter is the

chologist, and Carl Lange (1834–1900), a Danish
explanations for emotion, which most people refer
ies, 1884; Lange, 1887). Basically, the theory states
propriate set of physiological responses, such as
The situations also elicit behaviors, such as clench-
sensory feedback from the muscles and from the
is feedback that constitutes our feeling of emotion.

James says that our own emotional feelings are based on what we find ourselves doing and on the sensory feedback we receive from the activity of our muscles and internal organs. Thus, when we find ourselves trembling and feel queasy, we experience fear. Where feelings of emotions are concerned, we are self-observers. Thus, the two aspects of emotions reported in the first two sections of this chapter (patterns of emotional responses and expressions of emotions) give rise to the third: feelings. (See ***Figure 10.16.***)

James's description of the process of emotion might strike you as being at odds with your own experience. Many people think that they experience emotions directly, internally. They consider the outward manifestations of emotions to be secondary events. But have you ever found yourself in an unpleasant confrontation with someone else and discovered that you were trembling, even though you did not think that you were so bothered by the encounter? Or did you ever find yourself blushing in response to some public remark that was made about you? Or did you ever find tears coming to your eyes while you watched a film that you did not think was affecting you? What would you conclude about your emotional states in situations like these? Would you ignore the evidence from your own physiological reactions?

James's theory is difficult to verify experimentally because it attempts to explain *feelings* of emotion, not the causes of emotional responses, and feelings are private events. Some anecdotal evidence supports the theory. For example, Sweet (1966) reported the case of a man in whom some sympathetic nerves were severed on one side of the body to treat a cardiovascular disorder. The man—a music lover—reported that the shivering sensation he felt while listening to music now occurred only on the unoperated side of his body. He still enjoyed listening to music, but the surgery altered his emotional reaction.

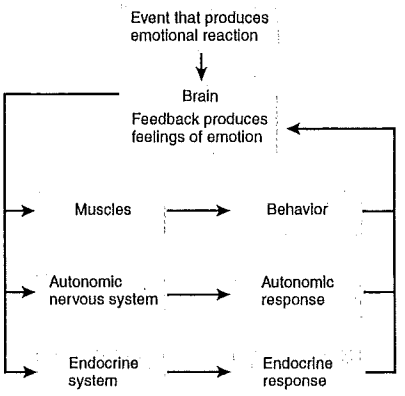

FIGURE 10.16 **The James-Lange Theory of Emotion.** This schematic diagram indicates that an event in the environment triggers behavioral, autonomic, and endocrine responses. Feedback from these responses produces feelings of emotions.

In one of the few tests of James's theory, Hohman (1966) collected data from people with spinal cord damage. He asked these people about the intensity of their emotional feelings. If feedback is important, one would expect that emotional feelings would be less intense if the injury were high (that is, close to the brain) than if it were low, because a high spinal cord injury would make the person become insensitive to a larger part of the body. In fact, this result is precisely what Hohman found: The higher the injury, the less intense the feeling was. As one of Hohman's subjects said:

> I sit around and build things up in my mind, and I worry a lot, but it's not much but the power of thought. I was at home alone in bed one day and dropped a cigarette where I couldn't reach it. I finally managed to scrounge around and put it out. I could have burned up right there, but the funny thing is, I didn't get all shook up about it. I just didn't feel afraid at all, like you would suppose. (Hohman, 1966, p. 150)

Another subject showed that angry behavior (an emotional response) does not appear to depend on *feelings* of emotion. Instead, the behavior is evoked by the situation (and by the person's evaluation of it) even if the spinal cord damage has reduced the intensity of the person's emotional feelings.

> Now, I don't get a feeling of physical animation, it's sort of cold anger. Sometimes I act angry when I see some injustice. I yell and cuss and raise hell, because if you don't do it sometimes, I've learned people will take advantage of you, but it doesn't have the heat to it that it used to. It's a mental kind of anger. (Hohman, 1966, p. 151)

Feedback from Emotional Expressions

James stressed the importance of two aspects of emotional responses: emotional behaviors and autonomic responses. As we saw earlier in this chapter, a particular set of muscles—those of the face—helps us to communicate our emotional state to other people. Several experiments suggest that feedback from the contraction of facial muscles can affect people's moods and even alter the activity of the autonomic nervous system.

Ekman and his colleagues (Ekman, Levenson, and Friesen, 1983; Levenson, Ekman, and Friesen, 1990) asked subjects to move particular facial muscles to simulate the emotional expressions of fear, anger, surprise, disgust, sadness, and happiness. They did not tell the subjects what emotion they were trying to make them produce, but only what movements they should make. For example, to simulate fear, they told the subjects, "Raise your brows. While holding them raised, pull your brows together. Now raise your upper eyelids and tighten the lower eyelids. Now stretch your lips horizontally." (These movements produce a facial expression of fear.) While the subjects made the expressions, the investigators monitored several physiological responses controlled by the autonomic nervous system.

The simulated expressions *did* alter the activity of the autonomic nervous system. In fact, different facial expressions produced somewhat different patterns of activity. For example, anger increased the heart rate and skin temperature, fear increased the heart rate but decreased skin temperature, and happiness decreased the heart rate without affecting skin temperature.

Why should a particular pattern of movements of the facial muscles cause changes in mood or in the activity of the autonomic nervous system? Perhaps the connection is a result of experience; in other words, perhaps the occurrence of particular facial movements along with changes in the autonomic nervous system leads to classical conditioning, so that feedback from the facial movements becomes capable of eliciting the autonomic response—and a change in perceived emotion. Or perhaps the connection is innate. As we saw earlier, the adaptive value of emotional expression is that they communicate feelings and intentions to others. The research presented earlier in this chapter on the role of mirror neurons and the somatosensory cortex suggests that one of the ways we recognize the feelings of others is through unconscious imitation.

A study by Lewis and Bowler (2009) found that interfering with muscular movement associated with a particular emotion decreased people's ability to experience that emotion. As you know, injections of a very dilute solution of botulinum toxin (Botox) into facial muscles can reduce wrinkling of the skin caused by chronic contraction of facial muscles. Lewis and Bowler studied people who had been treated with injections of Botox into the corrugator muscle, whose contraction is responsible for a large part of the facial expression of frowning, which is associated with negative emotions. They found that these people showed significantly less negative mood, compared with people who had received other forms of cosmetic treatment. These results, like those described earlier in this subsection, suggest that feedback from a person's facial expressions can affect his or her mood.

A functional imaging study by Damasio et al. (2000) asked people to recall and try to re-experience past episodes from their lives that evoked feelings of sadness, happiness, anger, and fear. The investigators found that recalling these emotions activated the subjects' somatosensory cortex and upper brain stem nuclei involved in control of internal organs and detection of sensations received from them. These responses are certainly compatible with James's theory. As Damasio et al. put it,

> [Emotions are part of a neural mechanism] based on structures that regulate the organism's current state by executing specific actions via the musculoskeletal system, ranging from facial and postural expressions to complex behaviors, and by producing chemical and neural responses aimed at the internal milieu, viscera and telencephalic neural circuits. The consequences of such responses are represented in both subcortical regulatory structures . . . and in cerebral cortex . . . , and those representations constitute a critical aspect of the neural basis of feelings. (p. 1049)

I suspect that if James were still alive, he would approve of these words.

The tendency to imitate the expressions of other people appears to be innate. Field et al. (1982) had adults make facial expressions in front of infants. The infants' own facial expressions were videotaped and were subsequently rated by people who did not know what expressions the adults were displaying. Field and her colleagues found that even newborn babies (with an average age of 36 hours) tended to imitate the expressions they saw. Clearly, the effect occurs too early in life to be a result of learning. Figure 10.17 shows three photographs of the adult expressions and the expressions they elicited in a baby. Can you look at them yourself without changing your own expression, at least a little? (See ***Figure 10.17.***)

Perhaps imitation provides one of the channels by which organisms communicate their emotions—and evoke feelings of empathy. For example, if we see someone looking sad, we tend to assume a sad expression ourselves. The feedback from our own expression helps to put us in the other person's place and makes us more likely to respond with solace or assistance. And perhaps one of the reasons we derive pleasure from making someone else smile is that the other person's smile makes *us* smile and feel happy.

(a)

FIGURE 10.17 Imitation in an Infant. The photographs [illegible] by the infant.

Tiffany Field.

SECTION SUMMARY

Feelings of Emotions

From the earliest times people recognized that emotion[s] panied by feelings that seemed to come from inside t[he] probably provided the impetus for developing physio[logical] of emotion. James and Lange suggested that emotion[s] responses to situations. Feedback from the physiologica[l] reactions to emotion-producing situations gave rise t[o] emotion; thus, feelings are the *results*, not the *causes*, o[f] tions. Hohman's study of people with spinal cord dama[ge] James-Lange theory; people who could no longer feel t[he] most of their body reported that they no longer exp[erienced] emotional states.

Ekman and his colleagues have shown that eve[n] emotional expression causes changes in the activity o[f]

EPILOGUE | Mr. V.

After our visit to Mr. V. (described in the chapter [opening]) were discussing the case. A student asked why Mr. [V.] continuing his walking schedule when he obviously couldn't walk. Did he think that he would recover s[omeday]?

"No, that's not it," said Dr. W. "He *knows* what his [problem] he doesn't really *understand* it. The people at the r[ehab] having trouble with him because he keeps trying t[o take] a walk. The first time, he managed to wheel his ch[air] the stairs, but someone caught him just in time. N[ow] chain across the door frame of his room so that he c[an't] hall without an attendant.

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(a) (b) (c)

FIGURE 10.17 Imitation in an Infant. The photographs show happy, sad, and surprised faces posed by an adult and the responses made by the infant.

Tiffany Field.

SECTION SUMMARY

Feelings of Emotions

From the earliest times people recognized that emotions were accompanied by feelings that seemed to come from inside the body, which probably provided the impetus for developing physiological theories of emotion. James and Lange suggested that emotions were primarily responses to situations. Feedback from the physiological and behavioral reactions to emotion-producing situations gave rise to the feelings of emotion; thus, feelings are the *results*, not the *causes*, of emotional reactions. Hohman's study of people with spinal cord damage supported the James-Lange theory; people who could no longer feel the reactions from most of their body reported that they no longer experienced intense emotional states.

Ekman and his colleagues have shown that even simulating an emotional expression causes changes in the activity of the autonomic nervous system. Perhaps feedback from these changes explains why an emotion can be "contagious": We see someone smile with pleasure, we ourselves imitate the smile, and the internal feedback makes us feel at least somewhat happier. The tendency to mimic the facial expression of others appears to be a consequence of activity in the brain's system of mirror neurons.

Thought Questions

1. Some people are more conscious of their own bodily reactions than most people. What effect would this sensitivity have on their mood?
2. Can you think of any evolutionary advantage of the fact that human infants can mimic the facial emotional expressions of adults?

EPILOGUE | Mr. V. Revisited

After our visit to Mr. V. (described in the chapter prologue), we were discussing the case. A student asked why Mr. V. talked about continuing his walking schedule when he obviously knew that he couldn't walk. Did he think that he would recover soon?

"No, that's not it," said Dr. W. "He *knows* what his problem is, but he doesn't really *understand* it. The people at the rehab center are having trouble with him because he keeps trying to go outside for a walk. The first time, he managed to wheel his chair to the top of the stairs, but someone caught him just in time. Now they have a chain across the door frame of his room so that he can't get into the hall without an attendant.

"Mr. V.'s problem is not that he can't verbally recognize what's going on; it's that he just can't grasp its significance. The right hemisphere is specialized in seeing many things at once: in seeing all the parts of a geometric shape and grasping its form or in seeing all the elements of a situation and understanding what they mean. That's what's wrong. He can tell you about his paralyzed leg, about the fact that he is in a wheelchair, and so on, but he can't put these facts together and realize that his days of walking are over.

"As you could see, Mr. V. can still express emotions." We all smiled at the thought of the contemptuous look on Mr. V.'s face. "But the

right hemisphere is especially important in assessing the significance of a situation and making conclusions that lead to our being happy or sad or whatever. People with certain right-hemisphere lesions are not bothered at all by their conditions. They can *tell* you about their problems, so I guess they verbally understand it, but their problems just don't affect them emotionally."

He turned to me. "Neil, do you remember Mr. P.?" I nodded. "Mr. P. had a left-hemisphere lesion. He had a severe aphasia and could hardly say a word. We showed him a picture of some objects and asked him to try to name them. He looked at them and started crying. Although he couldn't talk, he knew that he had a serious problem and that things would never be the same for him. His right hemisphere was still working. It could assess the situation and give rise to feelings of sadness and despair."

Dr. W. suggested that the right hemisphere's special role in emotional processes is related to its ability to deal with perception and evaluation of patterns of stimuli that occur simultaneously. His suggestion is plausible, but we still do not know enough about hemispheric differences to be sure that it is correct. In any event, many studies have shown that the right hemisphere does play a special role in evaluating the emotional significance of a situation. I described some of these studies in the chapter, but let's look at a few more examples. Bear and Fedio (1977) reported that people with seizures that primarily involve the left hemisphere tend to have thought disorders, whereas those with right-hemisphere seizures tend to have emotional disorders. Mesulam (1985) reported that people with damage to the right temporal lobe (but not the left temporal lobe) are likely to lose their sensitivity to social cues. Obviously, this observation is meaningful only for patients who were sensitive to social cues before the brain damage; if someone is socially insensitive *before* having a stroke, we can hardly blame the behavior on brain damage. In particular, people with right temporal lobe lesions tend to show bad manners. They talk when they feel like it and do not yield the floor to someone else who has something to say; they simply ignore the social cues that polite people observe and follow. They also adopt a familiar conversational style with people to whom they would normally be deferential (for example, the physician who is treating them).

Perhaps my favorite example of possible hemispheric specialization is hypnosis. Sackeim (1982) reported that when people are hypnotized, the left side of their body is more responsive to hypnotic suggestion than the right side is. Because the left side of the body is controlled by the right hemisphere, this observation indicates that the right hemisphere may be more susceptible to hypnotic suggestion. In addition, Sackeim, Paulus, and Weiman (1979) found that students who are easily hypnotized tend to sit on the right side of the classroom. In this position they see most of the front of the room (including the teacher) with their right hemispheres, so perhaps their choice represents a preference for right-hemisphere involvement in watching another person.

One of the reasons I enjoy writing these epilogues is that I can permit myself to be more speculative than I am in the text of the chapter itself. Why might the right hemisphere be more involved in hypnosis? One explanation of hypnosis that I find appealing is that it derives from our ability to get emotionally involved in a story—to get wrapped up in what is happening to the characters in a film or a novel (Barber, 1975). When we become involved in a story, we experience genuine feelings of emotion: happiness, sadness, fear, or anger. We laugh, cry, and show the same sorts of physiological changes that we would if the story were really happening to us. Similarly, according to Barber, we become involved in the "story" that the hypnotist is creating for us, and we suspend our disbelief and act it out. According to this explanation, hypnosis is related to our susceptibility to social situations and to our ability to empathize with others. In fact, people with the ability to produce vivid mental images, a high capacity for becoming involved in imaginative activities, and a rich, vivid imagination are those who are most likely to be susceptible to hypnosis (Kihlstrom, 1985).

As we saw in this chapter, the right hemisphere appears to play a special role in assessing social situations and appreciating their emotional significance. If Barber's explanation of hypnosis is correct, then we can see why the right hemisphere might play a special role in hypnosis, too. Perhaps researchers interested in hypnosis will begin studying patients with right- or left-hemisphere damage, and neuropsychologists already studying these people will start investigating hypnosis and its possible relation to social and emotional variables. Through their research, we can either confirm or disprove these speculations.

KEY CONCEPTS

EMOTIONS AS RESPONSE PATTERNS

1. Emotional responses consist of three components: behavioral, autonomic, and hormonal.
2. The amygdala plays a central role in coordinating all three components in response to threatening or aversive stimuli. In particular, the lateral nucleus is involved in acquisition of conditioned emotional responses.
3. Species-typical aggressive behaviors are controlled by neural circuits in the brain stem, which are modulated by the circuits in the hypothalamus and the amygdala.
4. The ventromedial prefrontal cortex plays a special role in the inhibitory control of emotional responses and in evaluation of situations that involve moral judgments.

COMMUNICATION OF EMOTIONS

5. Facial expressions of emotions appear to be species-typical responses, even in humans.
6. Recognition of facial expressions of emotions may involve imitation, which gives rise to feedback from the somatosensory cortex.
7. The amygdala is involved in visual recogr
pressions of emotion but not in their produ
8. Difficulty in faking true expressions of em
istence of emotional and volitional facial pa
special neural circuits are involved in expre
9. Expression and recognition of emotions
plished by neural mechanisms located in the

EXPLORE the Virtual Brain

STRESS

Brain structures, endocrine structures and neuro
response are explored in this module. Learn abou
HPA axis, as well as long-term effects of stress on h