Romanovsky AA. 2014. Skin temperature: its role in thermoregulation. Acta Physiol (Oxf) 210:498–507.

Rossi J, Balthasar N, Olson D, et al. 2011. Melanocortin-4 receptors expressed by cholinergic neurons regulate energy balance and glucose homeostasis. Cell Metab 13:195–204.

Saper CB. 2002. The central autonomic nervous system: conscious visceral perception and autonomic pattern generation. Annu Rev Neurosci 25:433–469.

Saper CB, Romanovsky AA, Scammell TE. 2012. Neural circuitry engaged by prostaglandins during the sickness syndrome. Nat Neurosci 15:1088–1095.

Shah BP, Vong L, Olson DP, et al. 2014. MC4R-expressing glutamatergic neurons in the paraventricular hypothalamus regulate feeding and are synaptically connected to the parabrachial nucleus. Proc Natl Acad Sci U S A 111:13193–13198.

Song K, Wang H, Kamm GB, et al. 2016. The TRPM2 channel is a hypothalamic heat sensor that limits fever and can drive hypothermia. Science 353:1393–1398.

Speakman JR, Levitsky DA, Allison DB, et al. 2011. Set points, settling points and some alternative models: theoretical options to understand how genes and environments combine to regulate body adiposity. Dis Model Mech 4:733–745.

Stricker EM, Hoffmann ML. 2007. Presystemic signals in the control of thirst, salt appetite, and vasopressin secretion. Physiol Behav 91:404–412.

Swanson LW. 2000. Cerebral hemisphere regulation of motivated behavior. Brain Res 886:113–164.

Tan CH, McNaughton PA. 2016. The TRPM2 ion channel is required for sensitivity to warmth. Nature 536:460–463.

Tan CL, Cooke EK, Leib DE, et al. 2016. Warm-sensitive neurons that control body temperature. Cell 167:47–59 e15.

Tanaka M, Owens NC, Nagashima K, Kanosue K, McAllen RM. 2006. Reflex activation of rat fusimotor neurons by body surface cooling, and its dependence on the medullary raphe. J Physiol 572:569–583.

Toates F 1986. *Motivational Systems*. New York: Cambridge University Press.

Williams EK, Chang RB, Strochlic DE, Umans BD, Lowell BB, Liberles SD. 2016. Sensory neurons that detect stretch and nutrients in the digestive system. Cell 166:209–221.

Wong LC, Wang L, D'Amour JA, et al. 2016. Effective modulation of male aggression through lateral septum to medial hypothalamus projection. Curr Biol 26:593–604.

Wu Z, Autry AE, Bergan JF, Watabe-Uchida M, Dulac CG. 2014. Galanin neurons in the medial preoptic area govern parental behaviour. Nature 509:325–330.

Yang CF, Chiang MC, Gray DC, et al. 2013. Sexually dimorphic neurons in the ventromedial hypothalamus govern mating in both sexes and aggression in males. Cell 153:896–909.

Zhang Y, Proenca R, Maffei M, Barone M, Leopold L, Friedman JM. 1994. Positional cloning of the mouse obese gene and its human homologue. Nature 372:425–432.

Zimmerman CA, Lin YC, Leib DE, et al. 2016. Thirst neurons anticipate the homeostatic consequences of eating and drinking. Nature 537:680–684.

42

Emotion

Elation, compassion, sadness, fear, and anger are commonly considered examples of emotions. These states have an enormous impact on our behavior and well-being. But what exactly is an emotion? Distinguishing different emotion states is difficult and requires an account of the environmentally or internally generated challenge an organism faces as well as its physiological responses. For example, before we can conclude that a rat is in a state of fear, we need to know that the rat is evaluating a specific threatening stimulus (a predator in its environment) and is mounting an adaptive response, such as high arousal and freezing.

Emotions are often represented along two dimensions: valence (ie, pleasantness to unpleasantness) and intensity (ie, low to high arousal), called "core affect" in many psychological theories. However, emotions can also be grouped into categories, such as categories of basic emotions (happiness, fear, anger, disgust, sadness) and categories of more complex emotions that help regulate social or moral behaviors (eg, shame, guilt, embarrassment, pride, jealousy). There is considerable debate about whether all the categories that are in common usage (like the ones just mentioned) will correspond to scientifically useful categories in a future neuroscience of emotion.

Within experimental contexts, the term *emotion* is used in several different ways, often related to the ways in which emotion is measured (Box 42–1). In everyday conversation, most people use the term "emotion" synonymously with "conscious experience of emotion" or "feeling," and most psychological studies in humans have focused on this sense of "emotion" as well. Most research in animals has focused instead on specific behavioral or physiological responses, in good part because it is impossible to obtain verbal reports in animal studies. Yet emotions have been conserved throughout the evolution of species, as Charles Darwin first observed in his seminal book, *The Expression of the Emotions in Man and Animals.* The empirical approach we describe in this chapter thus considers emotions as central brain states that can be studied in humans as well as many other animals, provided that we distinguish between emotions and feelings.

Emotion states typically cause a broad range of physiological responses that occur when the brain

Box 42–1 Ways of Measuring Emotion

Measures Commonly Used in Humans

Psychophysiology. Psychophysiology uses several measures to assay the physiological parameters associated with emotional states. These measures include autonomic responses (Chapter 41) as well as some somatic responses. The most commonly used measure is the galvanic skin response (also known as the skin conductance response), a measure of sympathetic autonomic arousal derived from the sweatiness of the palms of the hands. Other measures include heart rate, heart rate variability, blood pressure, respiration, pupil dilation, facial electromyography (EMG), and the startle response (see below). Some of these measures mostly correlate with basic dimensions of emotion, such as valence (eg, the

Table 42–1 Common Questionnaires Used to Assess Fear in Human Emotion Studies

Questionnaire	Type of fear questions
Fear Survey Schedule II	Probes an individual's level of fear across a range of different objects and situations that commonly evoke fear
Fear of Negative Evaluation Scale	Measures fear of being evaluated negatively by others
Social Avoidance and Distress Scale	Measures fear of social situations
Anxiety Sensitivity Index	Measures fear of experiencing different bodily sensations and feelings
Beck Anxiety Inventory	Measures fear and panic-related symptoms experienced over the prior week
Albany Panic and Phobia Questionnaire	Has the subject estimate the amount of fear they would experience in different situations
Fear Questionnaire	Measures the degree of avoidance due to fear
PANAS-X Fear (general)	Measures how much, in general, a person feels fear-related affective states
PANAS-X Fear (moment)	Measures how much, during the present moment, a person feels fear-related affective states

PANAS, Positive and Negative Affect Schedule.

detects certain environmental situations. These physiological responses are relatively automatic, yet depend on context, and occur within the brain as well as throughout the body. In the brain, they involve changes in arousal levels and in cognitive functions such as attention, memory processing, and decision making. Somatic responses involve endocrine, autonomic, and musculoskeletal systems (Chapter 41). In sum, emotions are neurobiological states that cause coordinated behavioral and cognitive responses triggered by the brain. This can occur when an individual detects a significant stimulus (positively or negatively charged) or has a specific thought or memory that leads to an endogenously generated emotion state.

Some stimuli—objects, animals, or situations—trigger emotions without the organism having to learn anything about those stimuli. Such stimuli have innately reinforcing qualities and are called unconditioned stimuli; examples are a painful shock or a disgusting taste. However, the vast majority of stimuli acquire their emotional significance through associative learning.

When an individual detects an emotionally significant stimulus, three physiological systems are engaged: the endocrine glands, the autonomic motor system, and the musculoskeletal system (Figure 42–1). The endocrine system is responsible for the secretion and regulation of hormones into the bloodstream that affect bodily tissues and the brain. The autonomic system mediates changes in the various physiological control systems of the body: the cardiovascular system, the visceral organs, and the tissues in the body cavity (Chapter 41). The skeletal motor system mediates

magnitude of the startle response) or arousal (eg, the galvanic skin response), whereas others (eg, facial EMG) can provide more fine-grained information about emotions. Facial expression has been used extensively but has no simple relationship to specific emotions.

Subjective ratings. Subjective ratings are often used in human studies and include categorical and continuous ratings (Table 42–1). These ratings can range along emotion dimensions, such as valence (pleasantness/unpleasantness), or the intensity of specific emotions. Subjective ratings necessarily depend on culture-specific words and concepts for emotions.

Experience sampling. Psychologists use experience sampling to quantify the emotions that people actually experience in everyday life. Participants might have their cell phone sound an alarm every few hours, and they then have to stop whatever activity they are doing and fill out a brief questionnaire about what they are feeling at the moment. In this way, a plot of the data can characterize how people's emotions change throughout the day or over longer periods. It turns out that we are actually fairly good at predicting what emotion people will feel next, from knowing how they currently feel.

Hormonal measures. Hormonal responses to emotional states are typically slower than psychophysiological measures. Emotion researchers measure a variety of hormones to assay emotional states over these lengthy periods. Relatively undifferentiated arousal responses are used to evaluate stress. The stress hormone cortisol (Chapter 61) is easily measured from people's saliva.

Specific experimental probes. Several specific behavioral and physiological assays are used to probe emotions with specific stimuli. These assays generally fall within the field of psychophysiology. A common measure is the amplitude of a subject's eyeblink (or other startle reflexes) when a loud sound is presented. This is potentiated when the subject is in a negatively valenced emotional state. Potentiation of the startle reflex is often used to assay the level of anxiety in people, and the same measure has also been validated in animals.

Measures Commonly Used in Nonhuman Animals

Innate behavioral responses. Animals often exhibit stereotyped behaviors as a consequence of certain emotional states. Observing and scoring the behavior is one method of measuring emotional behaviors. Such behaviors can include approaching a stimulus that is rewarding or that promises reward in the future (a positively valenced emotional state), as well as avoiding or defending against threatening stimuli (a negatively valenced emotional state). In addition, analysis of facial expressions can be utilized in many animal model systems, and has even been used for mice.

Psychophysiology and specific experimental probes. As in the case of humans, animal studies can use several psychophysiological measures (eg, heart rate, respiratory rate, galvanic skin response, pupil diameter, startle). In addition, specific behavioral assays have been developed in animals, often derived from initial observations of their innate behavioral responses. Behaviors such as freezing, attacking, exploring, approaching, and hiding can be measured in response to well-controlled experimental stimuli that are designed to induce certain emotional states. The correspondence between human and animal behaviors, which Charles Darwin originally noted in his 1872 book *The Expression of the Emotions in Man and Animals*, provides powerful animal models for investigating human emotions and their pathology.

overt behaviors such as freezing, fight-or-flight, and particular facial expressions. Together, these three systems control the physiological expression of emotion states in the body.

We begin this chapter with a discussion of the historical antecedents of modern research on the neuroscience of emotion. We then describe the neural circuits and cellular mechanisms that underlie the most thoroughly studied emotion, fear, and in so doing, we will focus on the amygdala. However, it is important to note that there does not appear to be any single brain structure that participates in only one emotion. For instance, the amygdala, which has been known to participate in negatively valenced emotions, also plays a central role in positively valenced emotions: Distinct populations of neurons within the amygdala process positively valenced versus negatively valenced stimuli. We briefly review how emotion states can be changed through extinction and regulation and how emotion interacts with other cognitive processes. We conclude with a survey on the relevance of emotion research to understanding psychiatric disorders.

The Modern Search for the Neural Circuitry of Emotion Began in the Late 19th Century

The modern attempt to understand emotions began in 1890 when William James, the founder of American psychology, asked: What is the nature of fear? Do we run from the bear because we are afraid, or are we afraid because we run? James proposed that the conscious

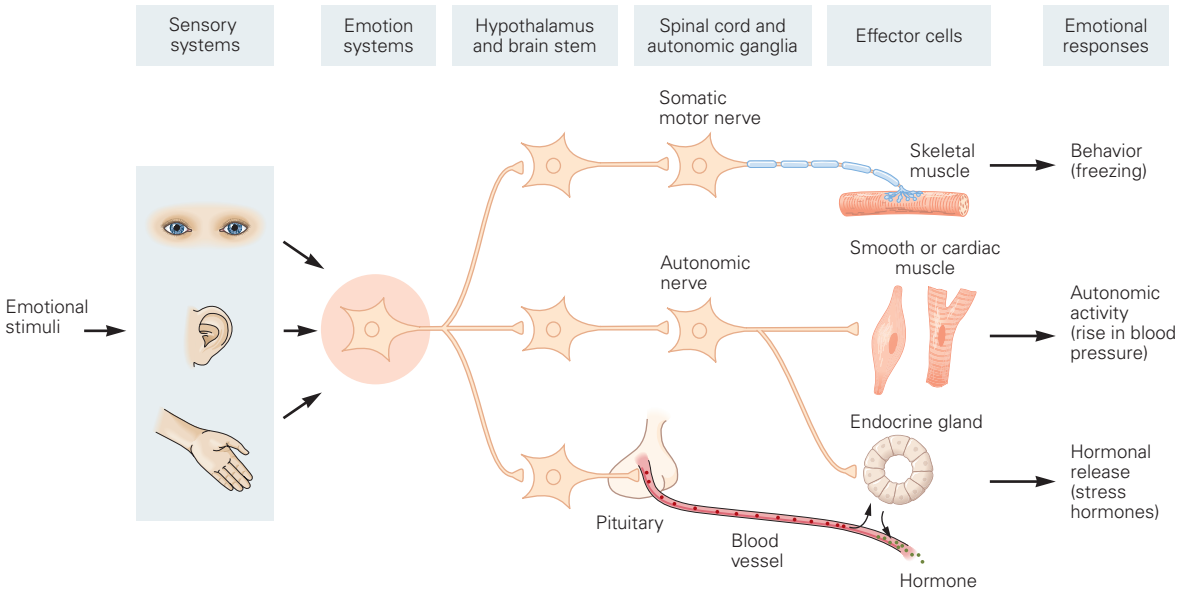

Figure 42–1 Neural control of emotional responses to external stimuli. External stimuli processed by sensory systems converge on "emotion systems" (eg, the amygdala). If the stimuli are emotionally salient, the emotion systems are activated, and their outputs are relayed to hypothalamic and brain stem regions that control physiological responses, including skeletomuscular action, autonomic nervous system activity, and hormonal release. The figure shows some responses associated with fear. It omits many of the complexities of emotion (eg, the effects of emotion states on cognition).

feeling of fear is a consequence of the bodily changes that occur during the act of running away—we feel afraid because we run. James's *peripheral feedback theory* drew on the knowledge of the brain at the time, namely, that the cortex had areas devoted to movement and sensation (Figure 42–2). Little was known at that time about specific areas of the brain responsible for emotion and feeling, but James's view is still debated to this day.

At the turn of the 20th century, researchers found that animals were still capable of emotional responses after the complete removal of the cerebral hemispheres, demonstrating that some aspects of emotion are mediated by subcortical regions. The fact that electrical stimulation of the hypothalamus could elicit autonomic responses similar to those that occur during emotional responses in an intact animal suggested to Walter B. Cannon that the hypothalamus might be a key region in the control of fight-or-flight responses and other emotions.

In the 1920s, Cannon showed that transection of the brain above the level of the hypothalamus (by means of a cut that separates the cortex, thalamus, and anterior hypothalamus from the posterior hypothalamus and lower brain areas) left an animal still capable of showing rage. By contrast, a transection below the hypothalamus, which left only the brain stem and the spinal cord, eliminated the coordinated reactions of natural rage. This clearly implicated the hypothalamus in organizing emotional reactions. Cannon called the hypothalamically mediated reactions "sham rage" because these animals lacked input from cortical areas, which he assumed were critical for the emotional experience of "real" rage (Figure 42–3).

Cannon and his student Phillip Bard proposed an influential theory of emotion centered on the hypothalamus and thalamus. According to their theory, sensory information processed in the thalamus is sent both to the hypothalamus and to the cerebral cortex. The projections to the hypothalamus were thought to produce emotional responses (through connections to the brain stem and spinal cord), while the projections to the cerebral cortex were thought to produce conscious feelings (Figure 42–2). This theory implied that the hypothalamus is responsible for the brain's evaluation of the emotional significance of external stimuli and that emotional reactions depend on this appraisal.

In 1937, James Papez extended the Cannon-Bard theory. Like Cannon and Bard, Papez proposed that sensory information from the thalamus is sent to both the hypothalamus and the cerebral cortex. The descending connections to the brain stem and spinal cord give rise to emotional responses, and the ascending connections to the cerebral cortex give rise to feelings. But Papez

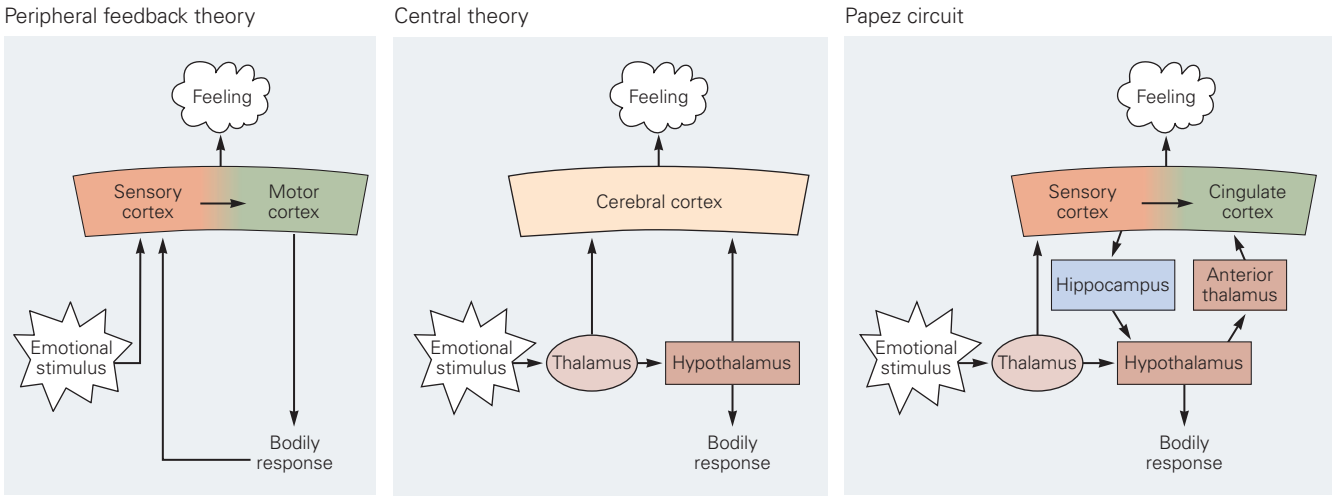

Figure 42–2 Early theories of the emotional brain. (Adapted, with permission, from LeDoux 1996.)

William James's peripheral feedback theory. James proposed that information about emotionally competent stimuli is processed in sensory systems and transmitted to the motor cortex to produce responses in the body. Feedback signals to the cortex convey sensory information about the body responses. The cortical processing of this sensory feedback is the "feeling," according to James.

The Cannon-Bard central theory. Walter Cannon and Philip Bard proposed that emotions are explained by processes within the central nervous system. In their model, sensory information is transmitted to the thalamus where it is then relayed to both the hypothalamus and the cerebral cortex. The hypothalamus evaluates the emotional qualities of the stimulus, and its descending connections to the brain stem and spinal cord give rise to somatic responses, while the thalamocortical pathways give rise to conscious feelings.

The Papez circuit. James Papez refined the Cannon-Bard theory by adding additional anatomical specificity. He proposed that the cingulate cortex is the cortical region that receives hypothalamic output in the creation of feelings. The outputs of the hypothalamus reach the cingulate via the anterior thalamus, and the outputs of the cingulate reach the hypothalamus via the hippocampus.

went on to expand the neural circuitry of feelings considerably beyond the Cannon-Bard theory by interposing a new set of structures between the hypothalamus and the cerebral cortex. He argued that signals from the hypothalamus go first to the anterior thalamus and then to the cingulate cortex, where signals from the hypothalamus and sensory cortex converge. This convergence accounts for the conscious experience of feeling in Papez's theory. The sensory cortex then projects to both the cingulate cortex and the hippocampus, which in turn makes connections with the mammillary bodies of the hypothalamus, thus completing the loop (Figure 42–2).

The hypothalamus is currently receiving intense interest in studies of emotion in animals, particularly in experiments using optogenetics to manipulate the activity of precise cell populations. These studies have shown that specific populations in the mouse ventromedial hypothalamus are necessary and sufficient for defensive emotion states. Thus the hypothalamus does not merely orchestrate emotional behaviors, but is part of the neural circuitry that constitutes the emotion state itself. The role of the hypothalamus in emotion is much less studied in humans, in part because functional magnetic resonance imaging (fMRI) does not have the spatial resolution to investigate specific hypothalamic nuclei, let alone neuronal subpopulations within them.

In the late 1930s, Heinrich Klüver and Paul Bucy removed the temporal lobes of monkeys bilaterally, thus lesioning all temporal cortex as well as subcortical structures like the amygdala and hippocampus, and found a variety of psychological disturbances, including alterations in feeding habits (the monkeys put inedible objects in their mouth) and sexual behavior (they attempted to have sex with inappropriate partners, like members of other species). In addition, the monkeys had a striking lack of concern for previously feared objects (eg, humans and snakes). This remarkable set of findings came to be known as the Klüver-Bucy syndrome and already suggested that the amygdala might be important for emotion (although it was not the only structure lesioned in these experiments).

Building on the Cannon-Bard and Papez models and the findings of Klüver and Bucy, Paul MacLean suggested in 1950 that emotion is the product of the "visceral brain." According to MacLean, the visceral brain includes the various cortical areas that had long been referred to as the limbic lobe, so named by Paul

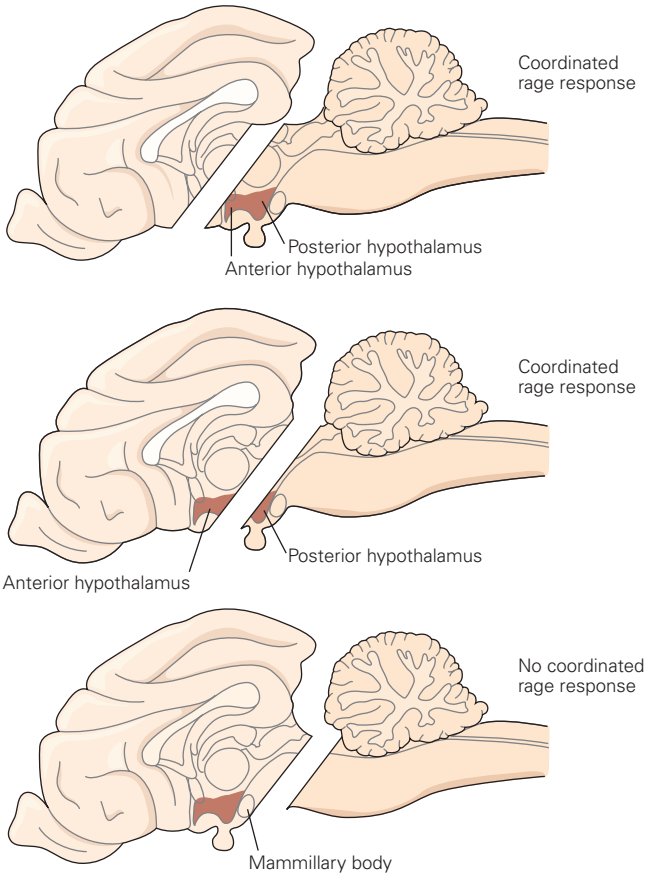

Figure 42–3 Sham rage. An animal exhibits sham rage following transection of the forebrain and the disconnection of everything above the transection (**top**) or transection at the level of the anterior hypothalamus and the disconnection of everything above it (**middle**). Only isolated elements of rage can be elicited if the posterior hypothalamus also is disconnected (**bottom**). This work derives from historical lesion studies in animals. More recent work suggests a more complex picture, in which the hypothalamus is intimately involved in creating the emotion state itself, not merely its behavioral expression.

Broca because these areas form a rim (Latin *limbus*) in the medial wall of the hemispheres. The visceral brain was later renamed the *limbic system*. The limbic system includes the various cortical areas that make up Broca's limbic lobe (especially medial areas of the temporal and frontal lobes) and the subcortical regions connected with these cortical areas, such as the amygdala and hypothalamus (Figure 42–4).

MacLean intended his theory to be an elaboration of Papez's ideas. Indeed, many areas of MacLean's limbic system are parts of the Papez circuit. However, MacLean did not share Papez's view that the cingulate cortex was the seat of feelings. Instead, he thought of the hippocampus as the part of the brain where the external world (represented in sensory regions of the lateral cortex) converged with the internal world (represented in the medial cortex and hypothalamus), allowing internal signals to give emotional weight to external stimuli and thereby to conscious feelings. For MacLean, the hippocampus was involved both in the expression of emotional responses in the body and in the conscious experience of feelings.

Subsequent findings raised problems for MacLean's limbic system theory. In 1957, it was found that damage to the hippocampus, the keystone of the limbic system, produced deficits in converting short- to long-term memory, a function that is distinct from emotions. In addition, animals with damage to the hippocampus are able to express emotions, and humans with hippocampal lesions appear to express and feel emotions normally. In general, damage to areas of the limbic system did not have the expected effects on emotional behavior.

Several of MacLean's other ideas on emotion are nevertheless still relevant. MacLean thought that emotional responses are essential for survival and therefore involve relatively primitive circuits that have been conserved in evolution, an idea already proposed by Charles Darwin almost a century earlier. This notion is key to an evolutionary perspective of emotion. It is now clear that emotions are processed by many subcortical and cortical regions and that the limbic system is by no means the primary system for emotion. Nonetheless, one component of the original limbic system, the amygdala, has received the most attention in studies of both humans and animals. Today, the role of the amygdala in learned fear is probably the best worked-out example of emotion processing in a specific brain structure, and therefore, we consider it next.

The Amygdala Has Been Implicated in Both Learned and Innate Fear

In Pavlovian fear conditioning, an association is learned between an unconditioned stimulus (US) (eg, electric shock) and a conditioned stimulus (CS) (eg, a tone) that predicts the US. For example, if an animal is presented with an emotionally neutral CS (a tone) for several seconds and then shocked during the final second of the CS, especially if this pairing of tone and shock is repeated several times, presentation of the tone alone will elicit defensive freezing and associated changes in autonomic and endocrine activity. In addition, many defensive reflexes, such as eyeblink and startle, will be facilitated by the tone alone.

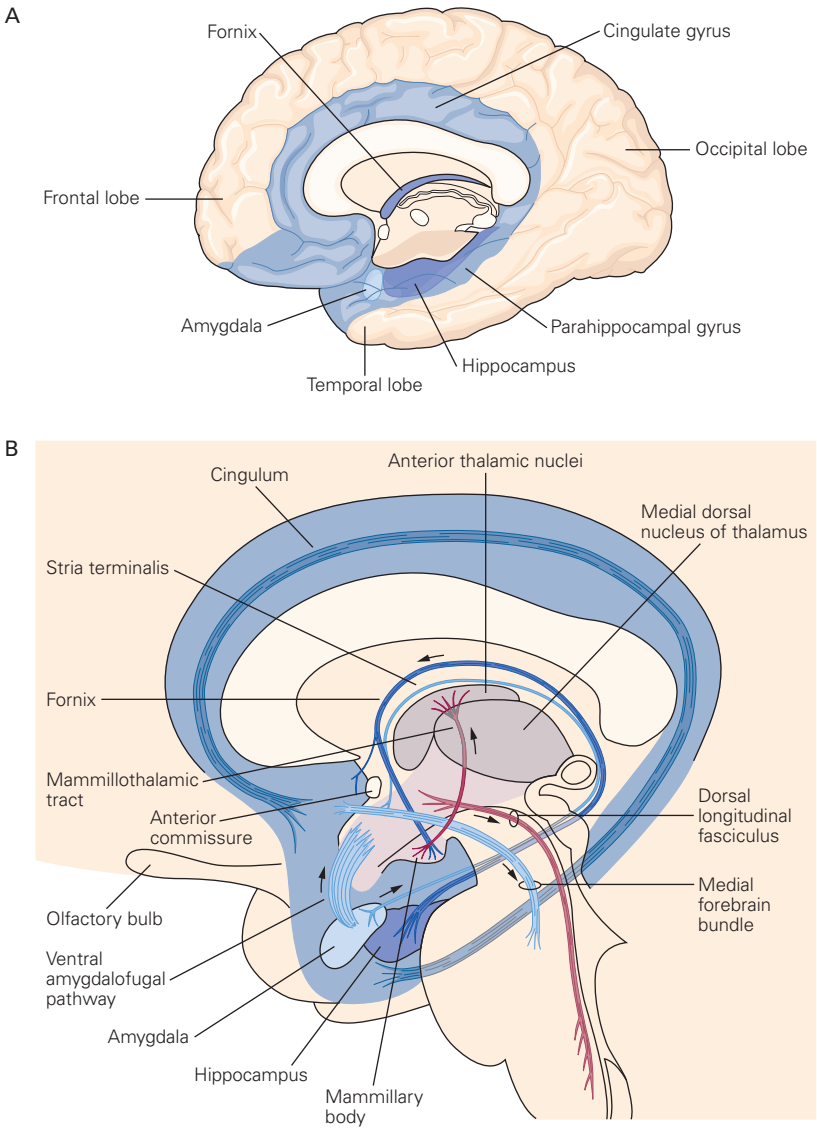

Figure 42–4 The limbic system consists of the limbic lobe and deep-lying structures. (Adapted, with permission, from Nieuwenhuys et al. 1988.)

A. This medial view of the brain shows the prefrontal limbic cortex and the limbic lobe. The limbic lobe consists of primitive cortical tissue (**blue**) that encircles the upper brain stem as well as underlying cortical structures (hippocampus and amygdala).

B. Interconnections of the deep-lying structures included in the limbic system. The **arrows** indicate the predominant direction of neural activity in each tract, although these tracts are typically bidirectional.

Research in many laboratories has established that the amygdala is necessary for Pavlovian fear conditioning: Animals with amygdala damage fail to learn the association between the CS and the US and thus do not express fear when the CS is later presented alone.

The amygdala consists of approximately 12 nuclei, but the lateral and central nuclei are especially important in fear conditioning (Figure 42–5). Damage to either nucleus, but not to other regions, prevents fear conditioning. The lateral nucleus of the amygdala receives most sensory input (but the medial nucleus receives olfactory input), including sensory information about the CS (eg, a tone) from both the thalamus and the cortex. The cellular and molecular mechanisms within the amygdala that underlie learned fear, especially in the lateral nucleus, have been elucidated in great detail. The findings support the view that the lateral nucleus is a site of memory storage in fear conditioning. Neurons in the central nucleus, by contrast, mediate outputs to brain stem areas involved in the control of defensive behaviors and associated autonomic and humoral responses (Chapter 41). The lateral and central nuclei are connected by way of several local circuits within the amygdala, including connections with the basal and intercalated masses. The actual circuitry for Pavlovian learning is thus considerably more complex than what is indicated by Figure 42–5, involving multiple relays among amygdala regions.

Sensory inputs reach the lateral nucleus from the thalamus both directly and indirectly. Much as

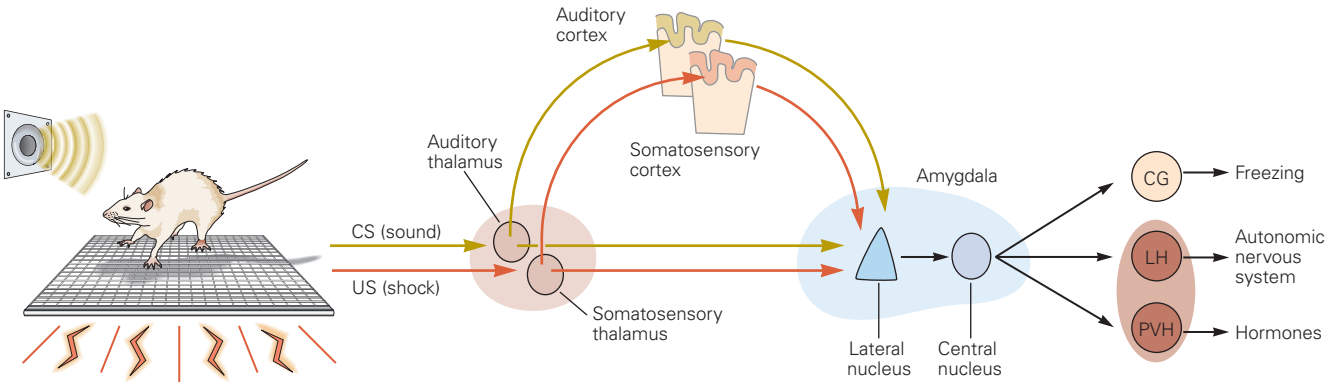

Figure 42–5 Neural circuits engaged during fear conditioning. The conditioned stimulus (**CS**) and unconditioned stimulus (**US**) are relayed to the lateral nucleus of the amygdala from the auditory and somatosensory regions of the thalamus and cerebral cortex. Convergence of the CS and US pathways in the lateral nucleus is believed to underlie the synaptic changes that mediate learning. The lateral nucleus communicates with the central nucleus both directly and through intra-amygdala pathways (not shown) involving the basal and intercalated nuclei. The central nucleus relays these signals to regions that control various motor responses, including the central gray region (**CG**), which controls freezing behavior; the lateral hypothalamus (**LH**), which controls autonomic responses; and the paraventricular hypothalamus (**PVH**), which controls stress hormone secretion by the pituitary–adrenal axis. (Adapted from Medina et al. 2002.)

predicted by the Cannon-Bard hypothesis, sensory signals from thalamic relay nuclei are conveyed to sensory areas of cerebral cortex. As a result, the amygdala and cortex are activated simultaneously. However, the amygdala is able to respond to an auditory danger cue before the cortex can fully process the stimulus information. This scheme is well worked out only for auditory fear conditioning in rodents, and it remains unclear how it applies to other cases, such as visually evoked fear in humans.

The lateral nucleus is thought to be a site of synaptic change during fear conditioning. The CS and US signals converge on neurons in the lateral nucleus; when the CS and US are paired, the effectiveness of the CS in eliciting action potentials is enhanced. This basic mechanism for a form of associative learning is similar to cellular mechanisms that underlie declarative memory in the hippocampus as well (Chapter 54). In particular, the synaptic plasticity found in the hippocampus has also been demonstrated in specific central amygdala circuits. The central amygdala thus does not simply drive motor outputs but is also part of the circuitry through which fear associations are formed and stored, very likely by transmitting information about the CS and US from the lateral nucleus. Neural plasticity likely also occurs in the basal and accessory basal nuclei during fear learning. As with the hypothalamus, recent work in rodents using tools such as optogenetics to manipulate specific subpopulations of amygdala neurons has begun to dissect this circuitry in further detail.

The emotional charge of a stimulus is evaluated by the amygdala together with other brain structures, such as the prefrontal cortex. If this system detects danger, it orchestrates the expression of behavioral and physiological responses by way of connections from the central amygdala and parts of prefrontal cortex to the hypothalamus and brain stem. For example, freezing behavior is mediated by connections from the central nucleus to the ventral periaqueductal gray region. In addition, the basal and accessory basal nuclei of the amygdala send projections to many parts of the cerebral cortex, including the prefrontal, rhinal, and sensory cortices; these pathways provide a means for neural representations in the amygdala to influence cognitive functions. For example, through its widespread projections to cortical areas, the amygdala can modulate attention, perception, memory, and decision making. Its connections with the modulatory dopaminergic, noradrenergic, serotonergic, and cholinergic nuclei that project to cortical areas also influence cognitive processing (Chapter 40). Given these very widespread connections and functional effects, the amygdala is well situated to implement one of the key features of an emotion: its coordinated and multicomponent responses.

The Amygdala Has Been Implicated in Innate Fear in Animals

Although the majority of stimuli acquire their emotional significance through learning, especially in