

Emotional States and Feelings

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PLEASURE, ELATION, EUPHORIA, ecstasy, sadness, despondency, depression, fear, anxiety, anger, hostility, and calm—these and other emotions color our lives. They contribute to the richness of our experiences and imbue our actions with passion and character. Moreover, as we shall learn in [Chapter 61](#), disorders of emotion contribute importantly to several major psychiatric illnesses. An emotional state has two components, one evident in a characteristic physical sensation and the other as a conscious feeling—we sense our heart pounding *and* we consciously feel afraid. To maintain the distinction between these two components, the term *emotion* sometimes is used to refer only to the bodily state (ie, the emotional state) and the term *feeling* is used to refer to conscious sensation.

Like perception and action, emotional states and feelings are mediated by distinct neuronal circuits within the brain. In fact, many drugs that affect the mind—ranging from addictive street drugs to therapeutic agents—do so by acting on specific neural circuits concerned with emotional states and feelings.

Conscious feeling is mediated by the cerebral cortex, in part by the cingulate cortex and by the frontal lobes. Emotional states are mediated by a family of peripheral, autonomic, endocrine, and skeletomotor responses. These responses involve subcortical structures: the amygdala, the hypothalamus, and the brain stem. When frightened we not only feel afraid but also experience increased heart rate and respiration, dryness of the mouth, tense muscles, and sweaty palms, all of which are regulated by subcortical structures. To understand an emotion such as fear we therefore need to understand the relationship between cognitive feeling represented in the cortex and the

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associated physiological signs orchestrated in subcortical structures.

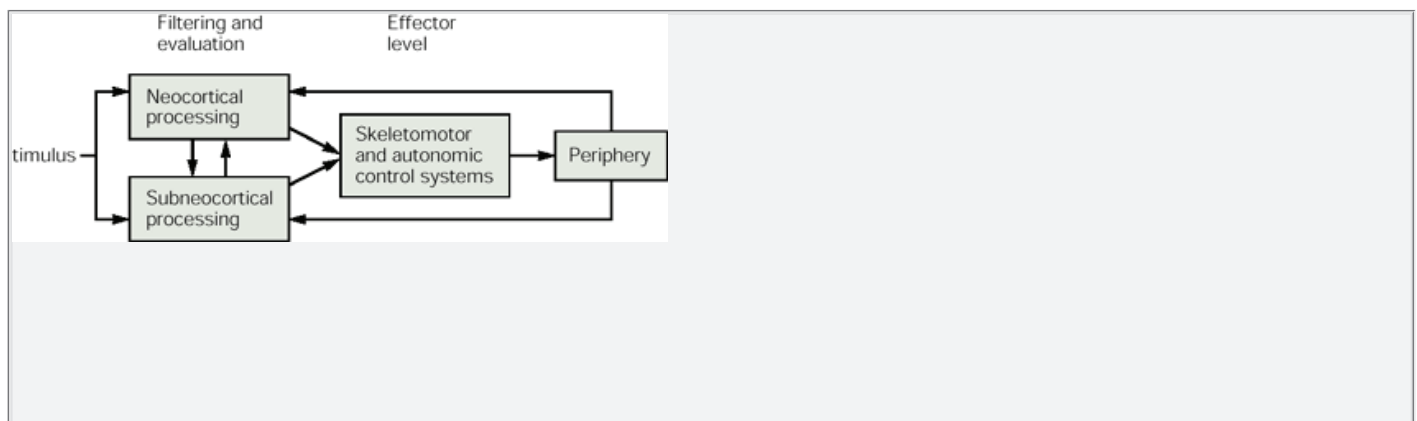


Figure 50-1 Model of the basic neural systems that control emotions. Emotions are typically elicited by a specific stimulus. The stimulus affects both neocortical and subcortical structures, such the amygdala. In turn, cortical structures and the amygdala and other subcortical structures regulate the systems that mediate the peripheral manifestations of emotional behaviors. The particular emotion experienced is a function of cross-talk between neocortical and subcortical structures, as well as feedback from peripheral receptors.

In this chapter we examine how emotion is represented in the brain. A neural analysis of emotion must take into account at least four issues. First, we must understand how stimuli acquire emotional significance and what roles conscious cognitive processes and automatic unconscious processes have in determining whether a particular stimulus at a particular moment will have emotional significance ([Figure 50-1](#)). Second, we must understand how certain autonomic and skeletomotor responses are triggered once a stimulus acquires emotional significance. Third, we must identify the circuits in the cerebral cortex responsible for feelings. Finally, we need to understand how somatic emotional states and conscious feeling states interact—how feedback from peripheral, autonomic, and skeletomotor systems to the cerebral cortex shapes emotional experience. As we will see, various theories of emotion largely differ in their emphasis on the importance of this feedback.

The Peripheral Components of Emotion Prepare the Body for Action and Communicate Our Emotional States to Other People

The peripheral, skeletomotor, and autonomic aspects of emotion have preparatory and communicative functions. The preparatory function involves both *general arousal*, which prepares the organism as a whole for action, and *specific arousal*, which prepares the organism for a particular behavior. For example, sexual arousal involves an increase of heart rate, a change that prepares the organism generally for physical exertion. In addition, it involves more localized changes, such as tumescence, that are specific to sexual behavior. The mechanisms of generalized and specific arousal act synergistically to prepare the periphery (muscles, glands, blood vessels) and the cerebral cortex for ongoing or upcoming events. Unless it is extreme, arousal enhances intellectual and physical performance ([Figure 50-2](#)).

The peripheral component of emotion also communicates emotion to others. In humans social communication of emotion is mediated primarily by the skeletomotor system, in particular by the muscles that control facial and postural expressions.

A Theory of Emotion Must Explain the Relationship of Cognitive and Physiological States

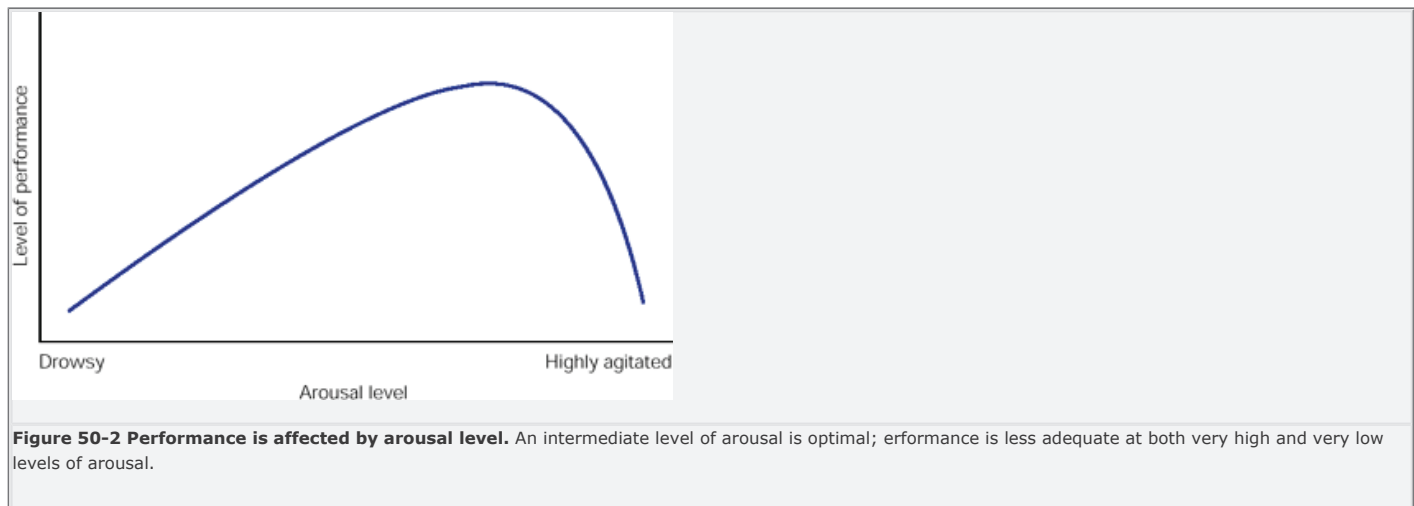
Until the late nineteenth century the traditional view of the evocation and expression of emotion consisted of the following sequence. First, an important event is recognized—for example, you see your house on fire. This recognition in turn produces in the cerebral cortex a conscious emotional experience—fear—that triggers signals to peripheral structures including the heart, blood vessels, adrenal glands, and sweat glands. According to this traditional view, a conscious, emotional event initiates reflexive autonomic responses in the body.

In the James-Lange Theory Emotions Are Cognitive Responses to Information From the Periphery

In 1884 the American psychologist William James rejected the traditional view that emotions are initiated by

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cognitive activity. In an article entitled *What Is Emotion?* James proposed that the cognitive experience of emotion is secondary to the physiological expression of emotion. He suggested that when we encounter a potentially dangerous situation—for example, a bear sitting in the middle of our path—the evaluation of the bear's ferocity does not itself generate a consciously experienced emotional state. We do not experience fear until after we have run away from the bear. That is, we act instinctively by running away and then consciously explain our action and the changes in our body (the increase in heart rate and respiration) as "driven by fear."



Based on this idea, James and the Danish psychologist Carl Lange proposed an alternative hypothesis: The feeling state, the conscious experience of emotion, occurs *after* the cortex receives signals about changes in our physiological state. Feelings are preceded by certain physiological changes—an increase or decrease in blood pressure, heart rate, and muscular tension. Thus, when you see a fire you feel afraid because your cortex has received signals about your racing heart, knocking knees, and sweaty palms. James wrote: "We feel sorry because we cry, angry because we strike, afraid because we tremble and not that we cry, strike or tremble because we are sorry, angry or fearful as the case may be." According to this view, emotions are cognitive responses to information from the periphery

There is now experimental support for aspects of the James-Lange theory. For example, objectively distinguishable emotions can be correlated with specific patterns of autonomic, endocrine, and voluntary responses. Furthermore, patients in whom the spinal cord has been accidentally severed so that they lack feedback from the autonomic nervous system appear to experience a reduction in the intensity of their emotions.

However, the James-Lange theory fails to explain certain aspects of emotional behavior. For example, one often continues to be emotionally aroused even after the physiological changes have subsided. Were physiological feedback the only controlling factor, the emotions should not outlast the physiological change. Yet a person can sustain a feeling of fear long after a threat has abated. Conversely, some feelings arise faster than the changes in bodily state normally associated with those feelings. Thus there may be more to emotions than just cortical interpretation of feedback information from the periphery.

Perhaps the most serious challenge to the James-Lange theory came in the 1920s from Walter B. Cannon's study of peripheral responses to intense emotion. Cannon's work indicated that intense emotion triggered an emergency reaction—a *fight-or-flight response*—in anticipation of additional behavioral responses and expenditure of energy. Cannon suggested that this flight-or-fight response was mediated by the sympathetic component of the autonomic nervous system and that it acted as a whole, almost in an all-or-none way, independent of the specific emotionally significant stimuli that elicited it. He therefore proposed that the physiological responses to emotionally significant stimuli are too undifferentiated to convey to the cortex specific, detailed information about the nature of an emotional event.

The Cannon-Bard Theory Emphasizes the Role of the Hypothalamus and Other Subcortical Structures in Mediating Both the Cognitive and Peripheral Aspects of Emotion

To deal with the shortcomings of the James-Lange theory, Cannon and Philip Bard suggested that two subcortical structures, the hypothalamus and the thalamus, have a key role in mediating emotions, including regulating the peripheral signs of emotion and providing the cortex with the information required for the cognitive processing of emotion. This idea was based on studies by Cannon and Bard using cats in which the whole cerebral cortex had been removed. Such animals retain fully integrated emotional responses, termed *sham rage* because the responses appear to lack elements of conscious experience that are characteristic of genuine, naturally occurring rage.

Sham rage also differs from genuine rage because responses can be triggered by very mild stimuli, such as a weak touch, or can even occur spontaneously, without provocation. No matter how it is elicited, sham rage subsides very quickly once the stimulus is removed. In addition, sham rage is undirected, and the animals sometimes even bite themselves. When Bard analyzed sham rage by progressive transections he found that the coordinated response disappeared, leaving only isolated elements of the response, when the hypothalamus was included in the ablation ([Figure 50-3](#)).

The question whether conscious feeling follows bodily changes (James-Lange) or bodily changes follow feeling continued to dominate modern discussions of emotional states for many years. Emotions are increasingly viewed as the outcome of a dynamic, ongoing interaction, perhaps at the level of the amygdala, of peripheral factors mediated by the hypothalamus and central factors mediated by the cerebral cortex. This synthesis of two theories, which now seems obvious, has emerged only slowly over the past three decades.

According to the Schachter Theory Feelings Are Cognitive Translations of Ambiguous Peripheral Signals

The James-Lange view of emotion has been refined in important ways, first by Stanley Schachter and more recently

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by Antonio Damasio. In the 1960s Schachter began to emphasize that the cortex actually constructs emotion—much like it does vision—out of often ambiguous signals it receives from the periphery. According to the James-Lange theory emotional experience is the direct consequence of information arriving in the cerebral cortex from the periphery. Instead of this simple relation, Schachter proposed that the cortex actively translates peripheral signals, even nonspecific ones, into specific feelings. He suggested that the cortex creates a cognitive response to peripheral information consistent with the individual's expectations and social context.

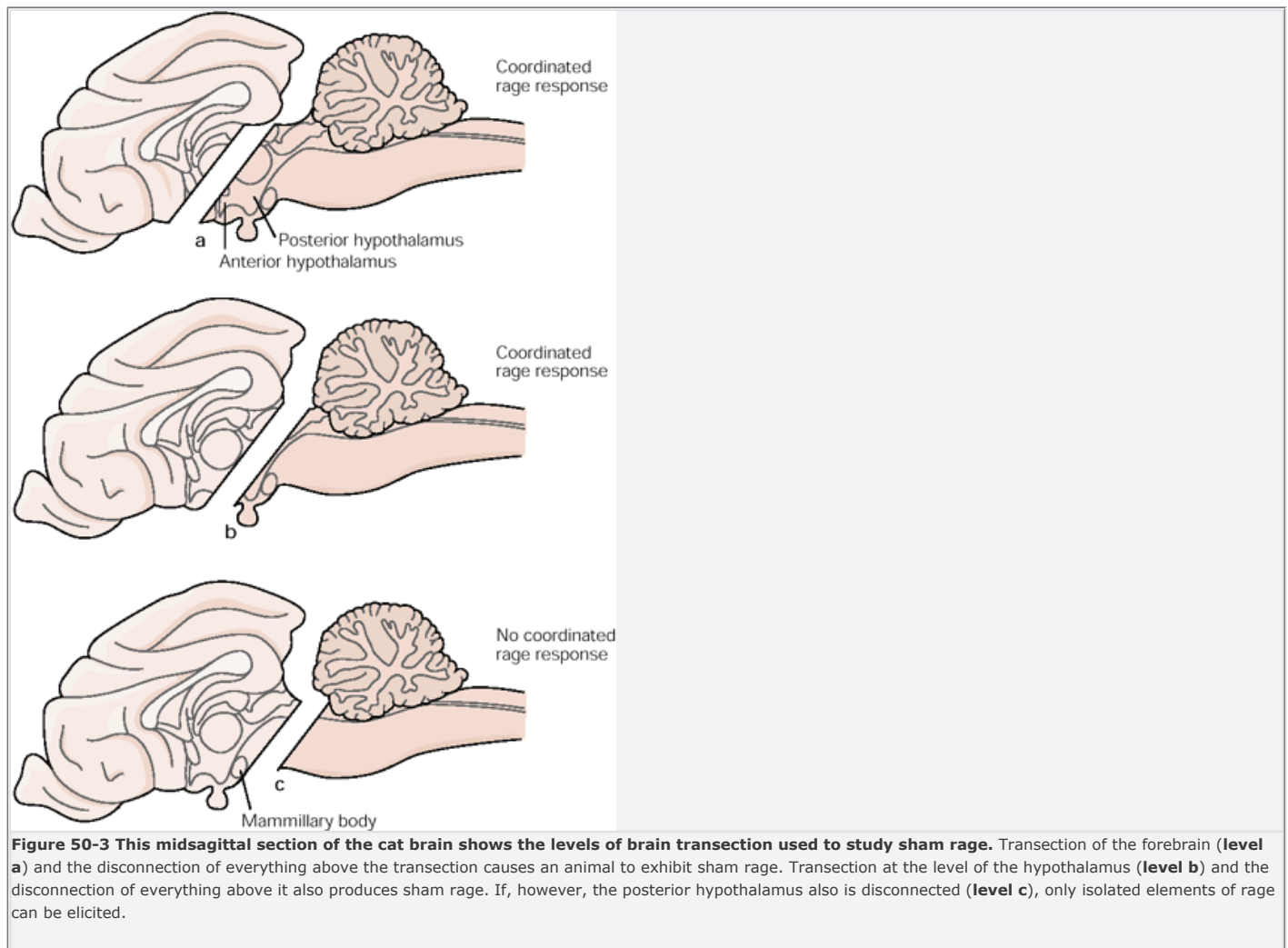
In one study Schachter injected volunteers with epinephrine; some subjects were informed of the side effects (eg, pounding heart), others were not. All of the subjects were then exposed either to annoying or amusing conditions. The subjects who had been warned about the side effects of epinephrine exhibited less anger or less pleasurable feelings. Schachter interpreted this finding as indicating that the informed subjects attributed their arousal to the drug, whereas the other group perceived their arousal as an emotional response—as strong anger or pleasant feelings depending on the conditions. More recent experiments have shown that the general arousal produced by exercise can lead to specific arousal, such as sexual arousal.

Schachter's refinement of the James-Lange theory was elaborated even further by Damasio, who argues that the feeling state, the experience of emotion, is essentially a story that the brain constructs to explain bodily reactions. Indeed, recent studies indicate that autonomic responses are not as uniform and stereotyped as Cannon had originally believed. Different emotional states are typically accompanied by different patterns of autonomic responses, such as changes in blood flow or heart rate.

In the Arnold Theory Autonomic Responses Are Not an Essential Component of Emotion

Magda Arnold has advanced this line of thinking further. She argues that emotion is the product of unconscious evaluation of a situation as potentially harmful or beneficial, while feeling is the conscious reflection of the unconscious appraisal. Feeling is therefore a tendency to respond in a particular way, not the response itself. Emotions differ from one another because they elicit different action tendencies. Thus, unlike the James-Lange theory, Arnold's view does not require that we have an autonomic response to experience emotion.

A consensus is emerging that Arnold's "appraisal" theory provides a good overall description of how emotions are generated: unconscious, implicit evaluation of a stimulus is followed by action tendencies, then peripheral responses, and finally conscious experience. A key finding supporting this idea is that we can have emotional reactions to subliminal stimuli. An important implication of Arnold's view is that emotions may have their own logic, one that is not derived from either conscious cognitive processes or somatic events associated with emotional states.



To what degree do emotions require conscious and unconscious processes or feedback from peripheral organs? To answer these questions we must ground the study of emotion in neural science. During the past decade the neural pathways for the peripheral (autonomic)

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and central (evaluative components of emotion) have been identified with some precision. It is now clear from Cannon's work that the peripheral component involves the hypothalamus, while the central, evaluative component, both unconscious and conscious, involves the cerebral cortex, especially the cingulate and the prefrontal cortex. Central to both of these systems is the amygdala, a subcortical nuclear complex thought to coordinate the conscious experience of emotion and the peripheral expressions of emotions, in particular fear.

Neural studies by Joseph LeDoux, by Michael Davis, and by Michael Fanslow indicate that the unconscious evaluation of the emotional significance of a stimulus begins before the conscious processing of the stimulus. Moreover, the neural systems for storing unconscious memories about emotional states (somatic response) are different from those responsible for the memory of conscious feeling. Damage to the amygdala, a system concerned with the experience and memory of fear, disrupts the ability of an emotionally charged stimulus to elicit an unconscious emotional response. In contrast, damage to the hippocampus, the core of the medial temporal lobe system concerned with conscious memory ([Chapter 62](#)), interferes with remembering the cognitive features of fear—where the fear-provoking stimulus was and in what context it occurred. Whereas cognitive systems present us with a choice of action, unconscious appraisal systems limit the options to a few adaptively important choices.

An attractive feature of this view is that it brings the study of emotion in line with studies of memory storage, which indicate that memory has two major forms: a conscious (*explicit*) memory for facts and personal events and an unconscious (*implicit*) memory for motor and sensory experience ([Chapter 62](#)). Memory of emotional states (autonomic and somatic responses) involves implicit memory storage, whereas memory of feelings involves explicit memory storage.

The Hypothalamus Coordinates the Peripheral Expression of Emotional States

How does the hypothalamus regulate the physiologic expression of emotion? We now appreciate that the hypothalamus acts on the autonomic nervous system by modulating visceral reflex circuitry that is basically organized at the level of the brain stem. This was first shown in 1932 by Stephen Ranson in anesthetized animals, using stereotaxic methods that permit precise and reproducible placement of electrodes in the different regions of the hypothalamus. By stimulating these different hypothalamic regions Ranson evoked almost every conceivable autonomic reaction, including alterations in heart rate, blood pressure, and gastrointestinal motility, as well as erection of hairs and bladder contraction.

In the 1940s Walter Hess extended Ranson's approach to awake, unanesthetized cats and found that different parts of the hypothalamus produce characteristic constellations of reactions that appear to be parts of organized responses characteristic of specific emotional states. For example, electrical stimulation in cats of the lateral hypothalamus and the fibers of passage in this area (see [Chapter 51](#)) elicits autonomic and somatic responses characteristic of anger: increased blood

pressure, raising of the body hair, pupillary constriction, arching of the back, and raising of the tail.

These observations provided the basis for the important conclusion that the hypothalamus is not only a motor nucleus for the autonomic nervous system. Rather, it is a coordinating center that integrates various inputs to ensure a well-organized, coherent, and appropriate set of autonomic and somatic responses. Since many of these responses resemble those seen during various types of emotional states, Hess suggested that the hypothalamus coordinates the peripheral expression of emotional states. This idea is supported by lesion studies that associate different hypothalamic structures with a wide range of emotional states. For example, animals with lesions in the lateral hypothalamus become placid, whereas animals with lesions of the medial hypothalamus are highly excitable and easily become aggressive.

The Search for Cortical Representation of Feeling Has Led to the Limbic System

Emotionally significant stimuli activate sensory pathways that trigger the hypothalamus to modulate heart rate, blood pressure, and respiration. (These observations are consistent with the James-Lange and Schachter-Damasio theories.) In turn, information about emotionally significant stimuli also is conveyed to the cerebral cortex both directly from the peripheral organs whose homeostatic state has been disturbed and indirectly from the hypothalamus, the amygdala, and related structures.

How are feeling and emotion represented in the cortex? In 1937 James Papez proposed that the cortical machinery for feeling involves the *limbic lobe*, a region identified by Paul Broca. The limbic lobe comprises a ring of phylogenetically primitive cortex around the brain stem and includes the cingulate gyrus, the parahippocampal gyrus (which is the anterior and inferior continuation of the cingulate gyrus), and the hippocampal formation, which lies deep in the parahippocampal

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gyrus and is morphologically simpler than the overlying cortex (Figure 50-4). The hippocampal formation includes the hippocampus proper, the dentate gyrus, and the subiculum.

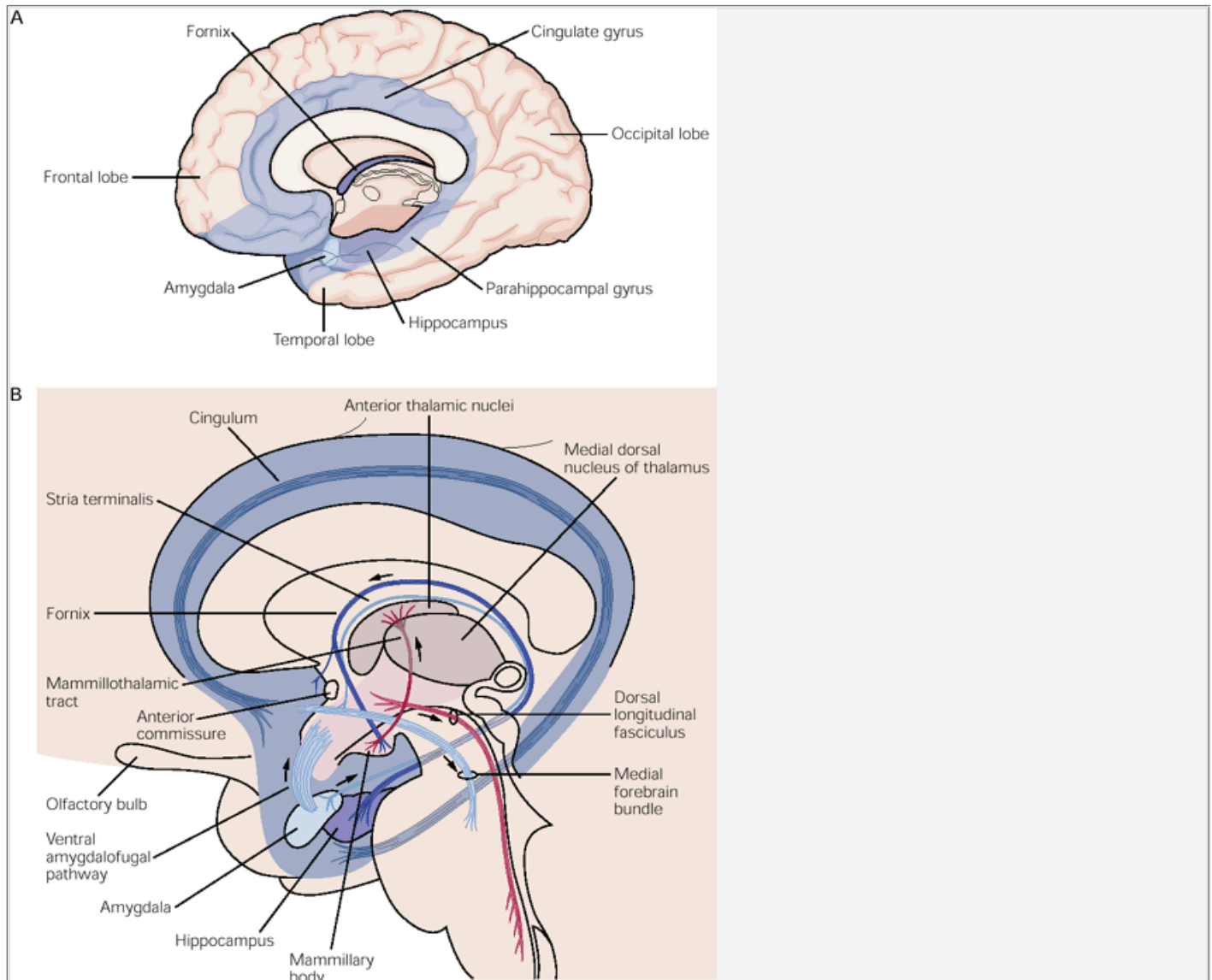


Figure 50-4 The limbic system consists of the limbic lobe and deep-lying structures. (Adapted from [Nieuwenhuys et al. 1988.](#))

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).

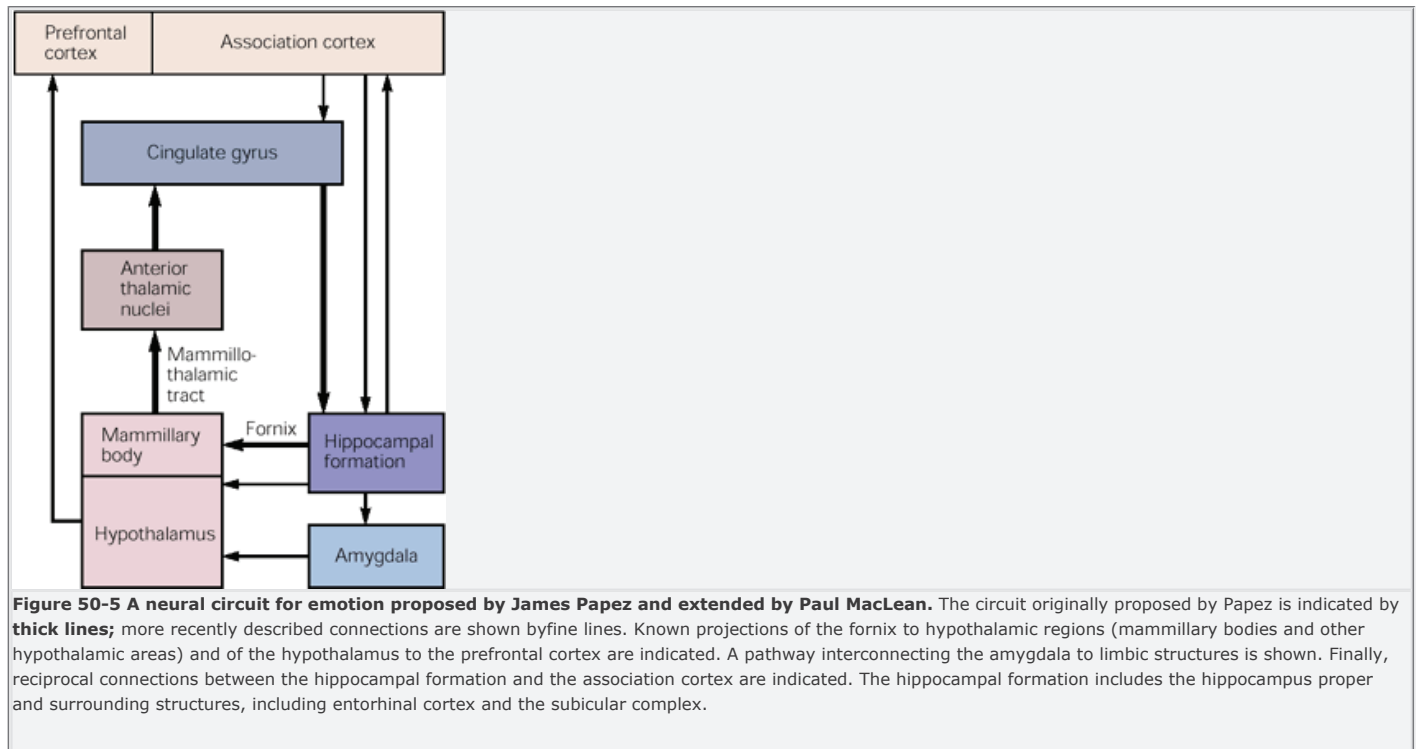
B. Interconnections of the deep-lying structures included in the limbic system. The predominant direction of neural activity in each tract is indicated by an **arrow**, although these tracts are typically bidirectional.

Papez argued that, since the hypothalamus communicates reciprocally with areas of the cerebral cortex, information about the conscious and peripheral aspects of emotion affect each other. Papez proposed that the neocortex influences the hypothalamus by means of connections to the cingulate gyrus and from the cingulate gyrus to the hippocampal formation. According to this idea, the hippocampal formation processes information from the cingulate gyrus and conveys it to the mammillary bodies of the hypothalamus by way of the fornix (a fiber bundle that carries part of the outflow of the hippocampus; see [Figure 50-4](#)). In turn, the hypothalamus provides information to the cingulate gyrus by a pathway from the mammillary bodies to the anterior thalamic nuclei (the mammillothalamic tract) and from there to the cingulate gyrus ([Figure 50-5](#)). Consistent with this idea is the clinical observation that patients who have been infected with the rabies virus—which characteristically attacks the hippocampus—show profound changes in emotional state, including bouts of terror and rage.

The concept of the limbic system was later expanded by Paul MacLean to include parts of the hypothalamus, the septal area, the nucleus accumbens (a part of the striatum), neocortical areas such as the orbitofrontal cortex, and most important, the amygdala. Modern anatomical studies have also shown that there are extensive direct connections between neocortical

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areas, the hippocampal formation, and the amygdala (Figure 50-5).



As we will see below, Papez was correct in attributing an important role to the cingulate cortex and the parahippocampal gyrus in the perception of feeling and emotion. He was incorrect, however, in thinking that the hippocampus coordinates the activity of the hypothalamus with these cortical areas: that coordinating role is carried out by the amygdala.

The first clue to the representation of emotion in the limbic system was found in 1939, when Heinrich Klüver and Paul Bucy showed that bilateral removal of the temporal lobes in monkeys—including the amygdala and the hippocampal formation, as well as the nonlimbic temporal cortex—produced a dramatic behavioral syndrome that included a major change in emotional behavior. After the operation, the monkeys, which had been quite wild before the procedure, became tame and fearless and their emotions flattened. They also exhibited a variety of other behavioral changes not directly related to emotions. They put inedible objects into their mouths and exhibited an enormous increase in sexual behavior, including mounting inappropriate objects and species. Finally, the animals showed a compulsive tendency to observe and react to every visual stimulus but failed to recognize familiar objects.

The Amygdala Is the Part of the Limbic System Most Specifically Involved With Emotional Experience

Because Papez's ideas were so influential, the whole Klüver-Bucy syndrome was for some years ascribed largely to the limbic system. It is now clear that the syndrome can be fractionated and that only some components involve the limbic system. For example, the visual deficits in Klüver-Bucy syndrome are mostly due to damage to the visual association areas of the inferior temporal cortex, the area concerned with, among other things, the recognition of faces and other complex visual forms (Chapter 28). Most important, the hippocampus, the mammillary bodies, and anterior thalamic nuclei, which were central to Papez's thinking about emotion, were found not to be involved in emotion but are critical for cognitive forms of memory storage (Chapter 62). Thus, with the exception of the cingulate and parahippocampal gyri, most parts of the limbic system as originally defined by Papez appear not to play a major role in the emotional components of the Klüver-Bucy syndrome or in emotion in general.

Considerable evidence from both humans and experimental animals now indicates that the amygdala rather than the hippocampus intervenes between the regions concerned with the somatic expression of emotion (the hypothalamus and the brain stem nuclei) and the neocortical areas concerned with conscious feeling, especially fear (the cingulate, parahippocampal, and prefrontal cortices).

For example, electrical stimulation of the amygdala in humans produces feelings of fear and apprehension. Conversely, damage to the amygdala in experimental animals produces tameness. Isolated lesions of the amygdala rarely occur in humans, but lesions of the amygdala occur as part of the widespread Urbach-Wiethe disease, a degenerative condition associated with calcium deposition in the amygdala. If the lesion occurs early in life, patients with bilateral amygdala damage fail to learn the cues that normal subjects use to discern fear in facial expressions and to discriminate fine differences in other facial expressions. Thus Urbach-Wiethe disease disrupts the unconscious processing

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of cues to fear in both real faces and imagined faces drawn from memory.

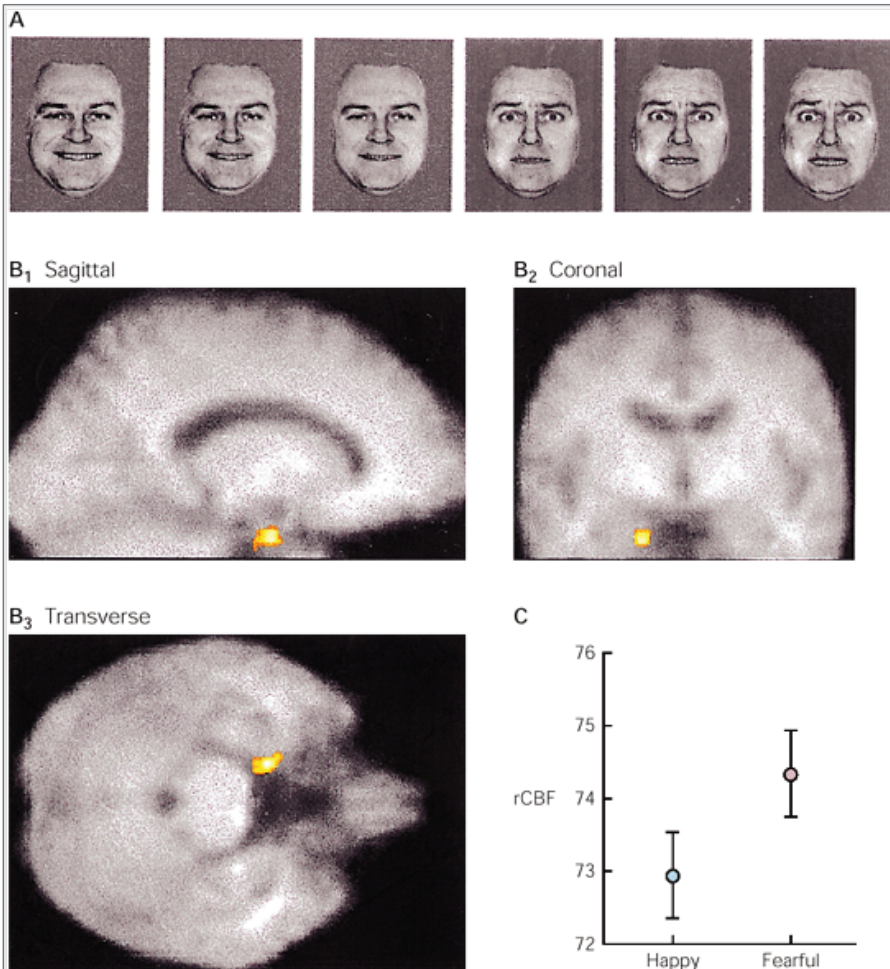


Figure 50-6 Brain imaging studies demonstrate the role of the amygdala in emotional responses. (From [Morris et al. 1996.](#))

A. A series of faces shows a continuum of expression between happiness and fear. Activity in the brain of normal subjects was recorded as they viewed these faces.

B. With the presentation of each of the faces only the left amygdala was found to vary in a systematic fashion. The region with activity that was correlated with the type of face that was shown is indicated in **yellow** and **red**.

C. The mean regional cerebral blood flow (**rCBF**) for the predominantly happy and predominantly fearful expressions. These results are consistent with recording and ablation experiments on animals that suggest the amygdala has a critical role in emotions, particularly in fear.

The disease does not impair the conscious ability to discriminate complex visual stimuli such as faces. Indeed, patients can accurately identify familiar people from photographs. For example, one patient with degeneration of the amygdala was tested for her ability to rate the intensity of human facial expressions of happiness, surprise, fear, anger, disgust, and sadness. She rated fear, anger, and surprise as less intense than did any of the controls, although she was able to recognize the identity of familiar faces, some of which she had not seen for many years

These results suggest that there are two anatomically separate neural systems. One, located in the inferotemporal cortex, is involved in the explicit memory of facial identity. The other, located in the amygdala, is concerned with the implicit memory of the appropriate cues that signal emotions expressed by faces. Consistent with this idea, studies using positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) clearly show that recognition of emotional expression in faces involves the amygdala. When subjects were asked to view photographs of fearful or happy faces, the responses in the amygdala, especially in the amygdala of the left hemisphere, were significantly greater to fearful expressions than to happy expressions. Moreover, the response in the left amygdala increases with increasing fearfulness and decreases with increasing happiness ([Figure 50-6](#)).

How does the amygdala participate in forming an emotional response to visual stimuli? Appropriate responses to the sight of emotionally charged signals are coded by the inferior temporal cortex. Neurons in the inferior temporal cortex respond to facial features, including the direction of gaze. Lesions in this area impair the ability to discriminate the direction of gaze in other faces. Since the amygdala receives input from the inferior temporal cortex and has strong connections to the autonomic nervous system, it can mediate emotional responses to complex visual stimuli.

As Charles Darwin first pointed out in 1872, fearful, angry, and happy facial expressions are virtually universal

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and have not only personal but social significance. Indeed, the recognition of facial expressions is essential for successful social behavior in a complex social environment. Thus, the behavioral impairments resulting from damage to the amygdala suggest that the amygdala may be important for social cognition.

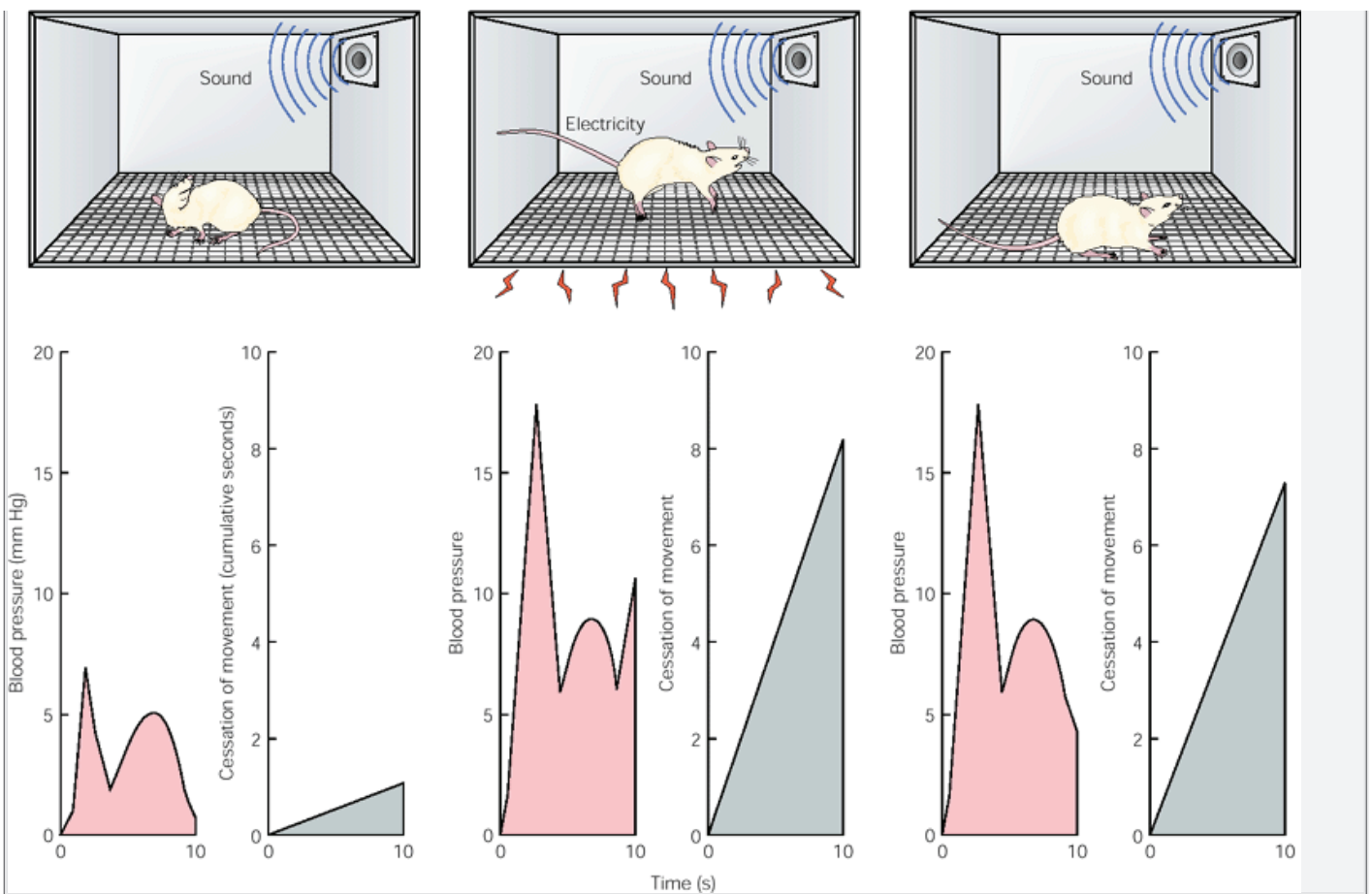


Figure 50-7 Classical fear conditioning can be demonstrated by pairing a sound with a mild electric shock to the foot of a rat. In one set of trials the rat hears a sound (**left panel**), which has relatively little effect on the animal's blood pressure or patterns of movement. Next, the same sound is coupled with a foot shock (**center**). After several pairings the rat's blood pressure rises and the animal freezes; it does not move for an extended period when it hears the sound. The rat has been fear-conditioned. Now, when the sound alone is given, it evokes physiological changes in blood pressure and freezing similar to those evoked by the sound and shock together (**right**). (From [LeDoux 1994](#).)

Learned Emotional Responses Are Processed in the Amygdala

The amygdala is a complex structure, consisting of about 10 distinct nuclei. The sensory inflow for various learned emotional states, particularly fear and anxiety, enters the amygdala by means of a particular set of nuclei: the basolateral complex.

The amygdala mediates both inborn and acquired emotional responses. The best studied example of a learned emotional state is the classical conditioning of fear ([Chapter 62](#)). Bilateral lesions of the basolateral complex of the amygdala in experimental animals abolish this learned response to fear. In this form of learning an initially neutral stimulus, such as a sound that does not evoke autonomic responses, is paired with an electric shock to the feet, which produces pain, fear, and autonomic responses. After several pairings the sound itself elicits a fearful reaction, such as freezing in place or changes in heart rate or blood pressure ([Figure 50-7](#)).

The sensory information about sound is conveyed to the basolateral complex from two sources: directly and rapidly from the auditory sensory nucleus in the

thalamus, and indirectly and more slowly from the primary sensory areas of the cortex. For many types of emotions, particularly fear, information conveyed from the thalamus to the amygdala is especially important because it can initiate short-latency, primitive emotional responses that may be important in situations of immediate danger. This rapidly available information may also prepare the amygdala to receive more highly processed information from cortical centers, which project mainly into the basolateral nuclei but also to the accessory basomedial nuclei ([Figure 50-8](#)). Consistent with their role in memory storage, stimulation of either thalamic or cortical pathways produces long-lasting alterations of synaptic efficacy (long-term potentiation; see [Chapter 63](#)) in the basolateral complex.

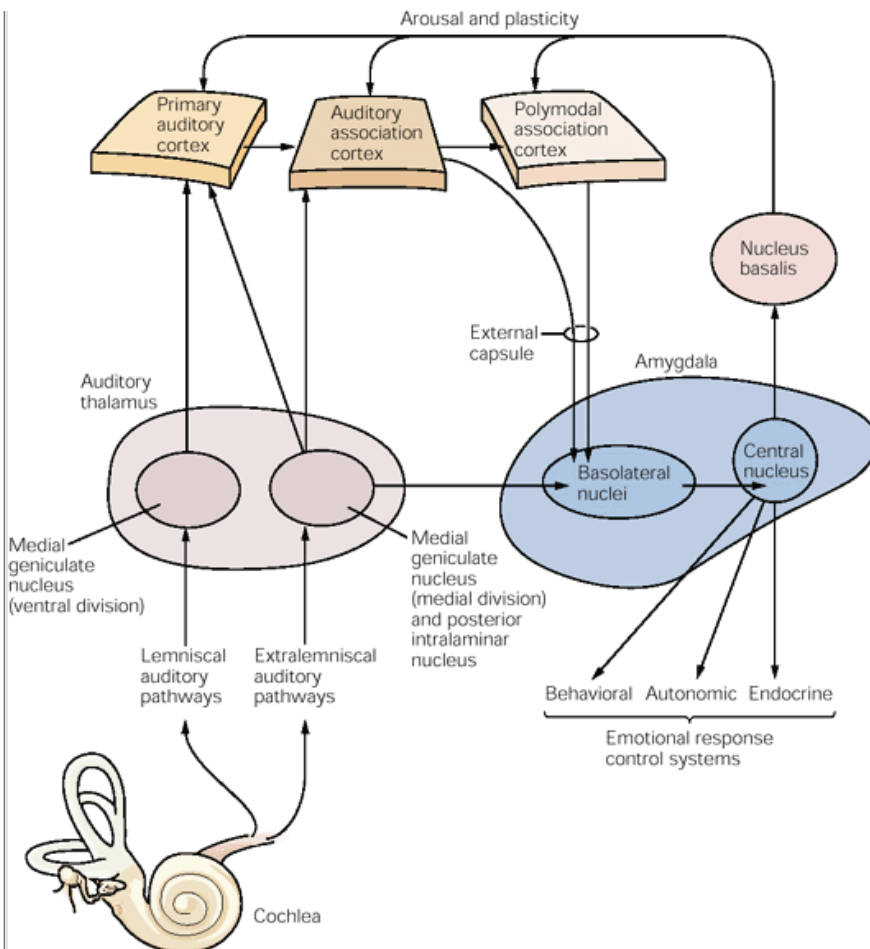


Figure 50-8 Some of the pathways involved in the processing of emotional information. Sensory information is transmitted to the thalamus via lemniscal pathways. The auditory input, for example, arrives in the ventral division of the medial geniculate nucleus. Other extralemniscal pathways deliver auditory information to other parts of the thalamus: the medial division of the medial geniculate nucleus and the posterior intralaminar nucleus. The lemniscal pathway of the ventral division of the medial geniculate nucleus projects only to the primary auditory cortex, but the extralemniscal auditory pathways of the medial geniculate nucleus and posterior intralaminar nucleus project to both the primary auditory cortex and auditory association cortex as well as to the basolateral nuclei of the amygdala. These pathways from the thalamus to the amygdala have been implicated in emotional learning. The anterior nucleus (not shown) projects widely to cortical areas and the central nucleus of the amygdala. The output nucleus of the amygdala, the central nucleus, makes extensive connections with brain stem areas involved in the control of emotional responses. It also projects to the nucleus basalis, which projects widely to cortical areas. The cholinergic projections from the nucleus basalis to the cortex have been implicated in cortical arousal. (Adapted from [LeDoux 1992](#).)

This pattern of responses to a once-neutral sound resembles human anxiety states, as we shall see in [Chapter 61](#). For example, subjects presented repeatedly with a neutral sound together with an offensively loud noise soon show an emotional response—sweating hands, dry mouth, and facial perspiration—to the neutral sound alone. In contrast, patients with damage to the amygdala do not learn to fear the neutral sound even though most were consciously aware that the neutral sound and the offensive noise were paired together.

In addition to simple conditioned fear, both experimental animals and people can also acquire fear-potentiated startle. People and experimental animals will startle more powerfully in response to a loud noise when they are frightened than if they are relaxed. For example, once a rat has learned fear by associating a neutral sound with a foot shock, it will startle much more forcefully to a loud noise heard with the conditioned sound (when the animal is fearful) than it would to the same noise in the absence of the conditioned sound (when the animal is relaxed). Bilateral destruction of the amygdala also eliminates this form of learned fear.

Where is the memory for learned fear stored? One possibility is that emotional memories such as fear are directly stored in the amygdala itself since lesioning of [P.992](#)

the amygdala abolishes the emotional component of the learned response. Ablation of the amygdala, however, eliminates not only the learned response to fear, but also the innate (unconditioned) response to fear. It eliminates the very ability to express emotion. This leaves open the possibility that emotional memories are not stored in the amygdala directly but are stored in the cingulate and parahippocampal cortices, with which the amygdala is interconnected.

The Amygdala May Be Involved in Both Pleasurable and Fearful Responses to Stimuli

In addition to its role in fear and other negative emotional reactions, the amygdala may also play a role in pleasure or other appetitive, emotional reactions. When a neutral discriminative stimulus such as a tone is paired with a positive reinforcing stimulus such as food, the tone can become associated either with rewarding attributes of the food (its taste) or with nonrewarding attributes (its visual appearance). Lesions of the basolateral nuclei leave intact the learned association between the tone and the nonrewarding aspects of the food, but they disrupt the association of the tone with rewarding attributes of the food. Recall that animals with Klüver-Bucy syndrome frequently take inedible (nonrewarding) objects into their mouths.

Finally, the amygdala is required for a type of learning termed *context conditioning*, (or *place-preference*) by which an animal learns to increase its contact with environments in which it has previously encountered stimuli essential for survival and to minimize contact with environments that are aversive or dangerous. The positive preferences for place can be conditioned not only to food or sexual partners but also to drugs, such as stimulants.

Place preference can be used to measure the rewarding properties of stimuli ranging from simple rewards (sweets) to complex ones (sexual partners). The constellation of stimuli that make up the distinctive environment in which a reward is obtained becomes associated with the reward. As a result, these *place cues* later take on positive values and increase the likelihood that the animal will again seek out this place and maintain contact with it, even in the absence of the primary reward. Presumably, place cues gain positive properties in part by means of classical conditioning ([Chapter 62](#)). There is considerable evidence that the amygdala, particularly the basolateral complex, which integrates incoming sensory input, is involved in associating place cues with reward value. Contextual conditioning also involves acquiring and binding together a variety of sensory information about place, a process that requires the hippocampus ([Chapter 62](#)).

The Amygdala Mediates Both the Autonomic Expression and the Cognitive Experience of Emotion

The amygdala appears to be involved in mediating both the unconscious emotional state and conscious feeling. Consistent with this dual function of emotion, the amygdala has two projections. Many of the autonomic expressions of emotional states are mediated by the amygdala through its connections to the hypothalamus and the autonomic nervous system. The influence of the amygdala on conscious feeling is mediated by its projections to the cingulate gyrus and prefrontal cortex.

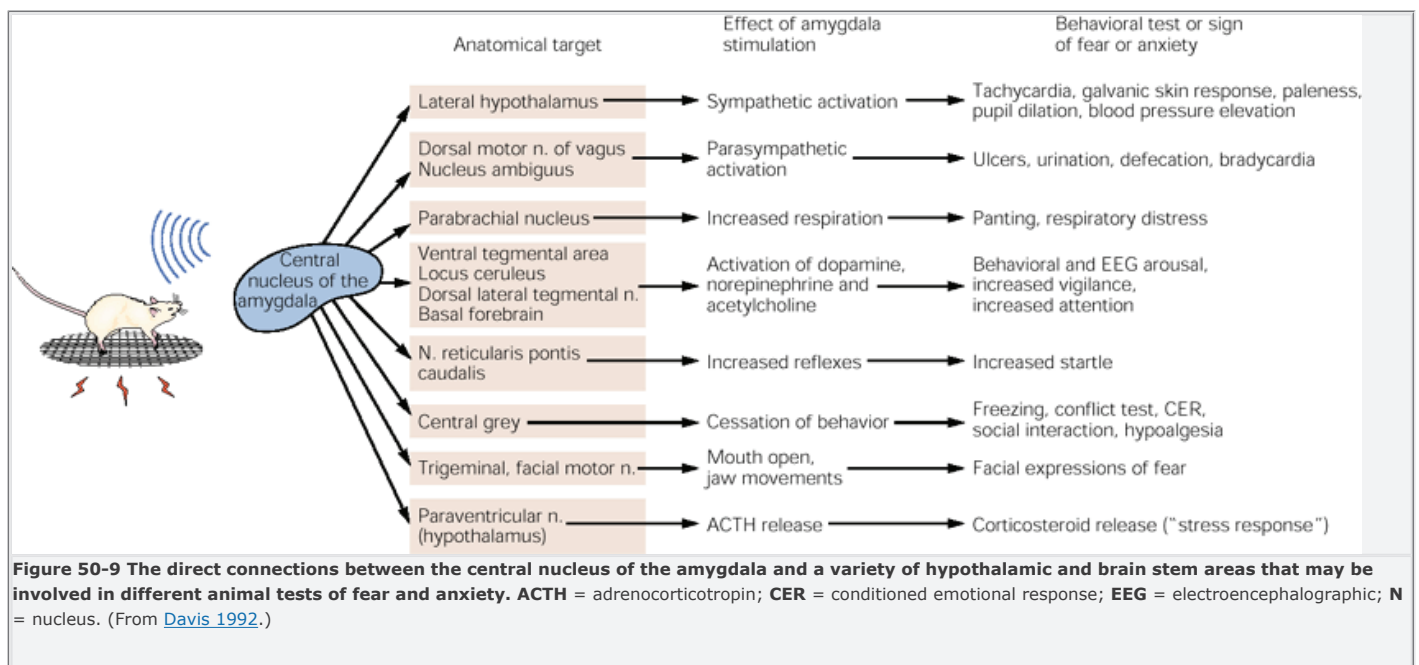
The nuclei of the amygdala are reciprocally connected to the lateral hypothalamus, brain stem, hippocampus, thalamus, and neocortex. The basolateral nucleus of the amygdala receives important afferent information from all sensory modalities and relays this information to the major output region, the central nucleus, both directly and by way of the basolateral and accessory basal nuclei. The central nucleus is reciprocally connected to its target structure by means of two major efferent projections: the *stria terminalis* and the *ventral amygdalofugal pathway* (see [Figure 50-4B](#)). As one might expect from its dual role, the output of the amygdala influences both the autonomic and cognitive components of emotion. The stria terminalis projects to the hypothalamus as well as to the bed nucleus of the stria terminalis and the nucleus accumbens. The ventral amygdalofugal pathway projects to the brain stem, the dorsal medial nucleus of the thalamus, and the rostral cingulate gyrus of the cortex and the orbitofrontal cortex.

Electrical stimulation of the central nucleus produces increases in heart rate, blood pressure, and respiration via its two output pathways to the lateral hypothalamic and brain stem regions ([Figure 50-9](#)). Conversely, lesions of this nucleus block these autonomic changes. The central nucleus also projects directly and indirectly (via the bed nucleus of the stria terminalis) to the paraventricular nucleus of the hypothalamus, which may be important in mediating neuroendocrine responses to fearful and stressful stimuli

The central nucleus also plays an important role in arousal and the conscious perception of emotion. It does this by means of its projections to the association areas of the cortex, especially the rostral cingulate gyrus and the orbitofrontal cortex. Projections from the central nucleus are thought to mediate these aspects of arousal, not only by direct projections to various nuclei ([Figure 50-9](#)) but also through indirect projections to the nucleus basalis.

In mice and other animals β -adrenergic mechanisms in the limbic system are known to be involved in the storage of emotional events. Lawrence Cahill, James McGaugh, and co-workers investigated the effect of propranolol, a β -adrenergic receptor blocker, on the

long-term memory of an emotionally arousing short story or a closely matched, but more emotionally neutral, story. The β -adrenergic blocker selectively impaired the memory for the emotional story, suggesting that nonspecific effects of the drug on arousal or attention could not account for the result. Furthermore, the drug did not block the subjects' initial emotional reaction to the story when it was first presented, but selectively blocked the subjects' memory of it.



The Frontal, Cingulate, and Parahippocampal Cortices Are Involved in Emotion

Electrical stimulation of the orbitofrontal cortex produces many autonomic responses (increases in arterial blood pressure, dilation of the pupils, salivation, and inhibition of gastrointestinal contractions), suggesting that this area is involved in generalized arousal. Lesions of the orbitofrontal cortex reduce the normal aggressiveness and emotional responsiveness of primates, and lesioned animals sometimes fail to show anger when they do not receive expected rewards in a training task. Lesions that include the anterior cingulate cortex also reduce chronic intractable pain, suggesting still another effect of the limbic cortex on emotional behavior.

In 1935 John Fulton and Carlyle Jacobsen first reported that removing the frontal cortex (*lobotomy*) had a calming effect in chimpanzees. Within a few months of Fulton and Jacobsen's report, Egas Moniz, a Portuguese neuropsychiatrist, performed the first prefrontal lobotomy in humans. In an attempt to treat the emotional impairment that often accompanies severe mental illness, Moniz cut the limbic association connections, thereby isolating the orbital frontal cortex.

The early results of frontal lobotomy appeared favorable; many patients seemed less anxious. However, later, more controlled studies led to abandonment of this procedure, in part because lobotomy was associated with a high incidence of complications, including the development of epilepsy and abnormal personality changes, such as a lack of inhibition or a lack of initiative and drive. In addition, the advent of effective psychotherapeutic drugs made radical surgical intervention unnecessary.

The reciprocal connections between the amygdala and the neocortex could permit learning and experience to be incorporated into the cognitive aspects of emotion. Cortical mechanisms provide a means by which memory and imagination, not just external stimuli, can evoke emotional feelings and they enable us to use emotional information generally in cognitive processing. Cortical structures also provide the means by which conscious thought can suppress reflex emotional responses. Once we know that a "bear" is only a shadow that looks like a bear, the fear subsides. The ventromedial frontal cortex is thought to provide one source of cognitive control of

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emotional responses, but we still understand relatively little of the role of the forebrain in complex feeling states.

Lesions to the ventral sector of the frontal lobe result in disinhibition of inappropriate behavior in social situations. This lack of restraint has frequently been noted in patients after psychosurgery to the frontal lobes. It was a prominent behavioral feature in the historical case of Phineas Gage, who survived a traumatic lesion to the anterior part of his brain when a metal bolt was blown through his skull in a mining accident. Gage made a remarkable recovery from this horrendous accident, but he was a changed person. He could no longer plan for the future, conduct himself according to the social rules he had followed previously, or decide on a course of action that would be most advantageous to his survival. At his death more than a decade later no autopsy was performed, but fortunately his skull, with the bolt hole, was kept in a museum. Medical detective work by Hannah Damasio using modern skull measurements led to the conclusion that the bolt almost certainly destroyed

the ventromedial aspect of his frontal lobe (See [Figure 19-2C](#)).

Rigorous neuropsychological tests have been used on patients with ventromedial frontal lobe damage to evaluate the influence of affective information on behavior. One such test is a "gambling experiment" in which a player sits in front of four decks of cards, labeled A, B, C, and D. The player is given a loan of \$2000 (play money looking like the real thing) and is told that the game is about losing as little as possible and trying to make more money. Play consists of turning one card at a time from any of the four decks until the experimenter says "stop." The player is told that turning every card results in earning more money, but occasionally a card will be turned that results in paying back money to the experimenter. No information is given about the size of the gains or losses or about the cards to be found in the different decks. Only when a card is turned does the player learn its value. No tally of gains and losses is available except in the subject's mind. The undisclosed rules are that A and B cards yield \$100 but occasionally require the subject to repay \$1250. Cards C and D yield \$50 but only require repayment of small sums (less than \$100).

Normal people, lured by high rewards, initially play decks A and B, but gradually, usually within 30 of the designated 100 trials of the game, they switch to a preference for decks C and D. Thus normal subjects appear to develop a hunch that decks A and B are more "dangerous" than the others. Patients with frontal lesions behave in quite a different way. After an early general sampling of the card decks they prefer cards from decks A and B and, despite the high penalties and the need to borrow from the bank, they hold to this preference throughout the test. They clearly know which decks are riskier but they continue to behave in this inappropriate way even when retested at a later time.

In patients with either amygdala or frontal lobe damage there is a clear dissociation between autonomic responses to emotive stimuli and cognitive evaluation of those stimuli. Lesions of the amygdala do not impair autonomic responses to aversive stimuli, but they do prevent the subject from learning to associate a particular stimulus with a negative consequence. Patients with frontal lesions have normal galvanic skin responses (sweating measured electrically) to "startle" stimuli, such as unexpected loud noises or bright lights, indicating a normal autonomic response mechanism. However, when patients with frontal lobe lesions were presented with disturbing images interspersed among a series of slides showing bland scenes or abstract patterns, they failed to show the expected autonomic responses to these emotionally charged stimuli. These patients sometimes commented that they knew they should have been disturbed by certain pictures but found themselves unmoved.

Two clinical syndromes dramatically illustrate the dissociation between conscious processing of visual information and unconscious processing of emotional information associated with an image. Patients with prosopagnosia ([Chapter 25](#)) cannot consciously identify faces, even those of familiar associates and relatives. Yet they exhibit autonomic responses (eg, skin conductance change) to familiar faces but not to unfamiliar faces. Conversely, patients who suffer from the rare Capgras syndrome can readily recognize familiar faces but apparently do not have emotional responses to them. Remarkably, these patients report that the face shown to them is that of an imposter who looks identical to the person they know.

The Hippocampus Has Only an Indirect Role in Emotion

Early theories of the neural control of emotional states accorded the hippocampus a major role in coordinating the activity of the hypothalamus and cerebral cortex (see [Figure 50-5](#)). Subsequent experimental studies on both monkeys and humans showed that the coordinating role is carried out by the amygdala rather than the hippocampus. The hippocampal system is involved in explicit (declarative) memory ([Chapter 62](#)).

The distinctive roles of the amygdala and the hippocampus were clearly demonstrated in a study of three patients with selective damage to the amygdala, the hippocampus, or both. These patients were shown monochromatic slides (green, blue, yellow, or red) and

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their autonomic responses were measured. After some of the colored slides a frightening loud horn was sounded. Patients with the amygdala lesion did not become conditioned to the associated color. Yet when asked how many different colors they observed and how many were followed by the loud horn, the patients responded correctly and had clearly acquired explicit knowledge about the testing situation. Patients with hippocampal damage, on the other hand, became conditioned to colors associated with the loud horn but did not learn how many colors were associated with the sound of the loud horn. Patients with lesions in both the amygdala and hippocampus showed neither autonomic conditioning nor knowledge of the testing situation.

An Overall View

The emotional experiences that we perceive as fear, anger, pleasure, and contentment reflect an interplay between higher brain centers and subcortical regions such as the hypothalamus and amygdala. This is illustrated dramatically in patients in whom the prefrontal cortex or the cingulate gyrus has been removed. These patients are no longer bothered by pain. They experience pain as a sensation and exhibit appropriate autonomic reactions, but the sensation is not felt as a powerful unpleasant experience.

Thus, noxious and pleasurable stimuli have dual effects. First, they trigger autonomic and endocrine responses, integrated by subcortical structures, that immediately alter internal states, thereby preparing the organism for attack, flight, sex, or other adaptive behaviors. These behaviors are relatively simple to execute and require no conscious control. Thereafter a second set of mechanisms come into play, involving the cerebral cortex. Cortical processing of emotionally significant stimuli results in a conscious experience of emotion (feeling) as well as in signals to lower centers that can suppress or enhance the somatic manifestations of emotions.

Many aspects of our primary emotional responses are learned, and during this learning visceral feedback probably has an important role. But with experience we depend increasingly on cognition to evaluate the significance of our environment, and visceral sensations probably play a less important role. The anatomical connections of the amygdala with the temporal (cingulate gyrus) and frontal (prefrontal) association cortices provide the means by which visceral sensations trigger a rich assortment of associations and narratives, the cognitive interpretation of emotional states.

Nevertheless, emotional states may contribute to conscious feeling in a less direct way than originally proposed by William James. Antonio Damasio has suggested that when we think about the potential consequences of a behavior, the memory of our emotional state (visceral experiences) in similar circumstances may provide useful information for evaluating the behavior. The memory may activate ascending noradrenergic and cholinergic projections of the brain stem and basal forebrain, thereby activating the cortex and replicating the conscious sensations of the remembered emotional state, bypassing the feedback of the autonomic nervous system. This may be the basis of what we refer to as "gut feelings."

As discussed in the next chapter, emotions and feelings are closely linked to motivated behaviors such as feeding, drinking, and sexual behaviors. Appropriately motivated animals seek particular stimuli in the environment: food, water, warmth, and novelty. These stimuli are related to survival and consequently are particularly meaningful. Almost by definition they evoke pleasure and pain and generate emotional responses.

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