

THE BRAIN AND THE INNER WORLD
AN INTRODUCTION TO THE NEUROSCIENCE
OF SUBJECTIVE EXPERIENCE

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FOREWORD
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to the man in the bed next to hers as being her husband, and behaved accordingly toward him. Again, the wish-fulfilling properties of such conflation are clear. She wanted her husband to be there. When he was, that was fine; but when he wasn't, it was not at all difficult to ignore or modify her conception of reality to fit with her requirements.

CONCLUDING REMARKS

These clinical phenomena reveal a number of important things about the mind and how it works. But, most important of all, they demonstrate the principle that it *is possible* to find the neurological correlates of some traditional psychoanalytic concepts and thereby to set them on a firm, organic foundation. The cases above show that the capacity of the "ego" to inhibit instinctual drives—the very foundation of rational, reality-constrained behavior—is somehow intimately bound up with the functions of the ventromesial frontal lobes. In subsequent chapters, we repeatedly pick up the strands of the issues raised by these cases and clarify further what they reveal about the functional organization of the mind. For now, having sketched a first, rough map of the neurological correlates of *consciousness* and *the unconscious*, we are in a position to consider in more detail, in the next chapter, what modern neuroscience reveals about "drives."

CHAPTER 4

EMOTION AND MOTIVATION

Our goal-directed actions are ultimately motivated by the biological task of meeting our needs in the outside world. The function of consciousness, described in the previous chapter, contributes a great deal to the successful performance of this task. "Core consciousness" relates information about the current state of the self to the prevailing circumstances in the outside world—the source of all the objects that the self requires to meet its inner needs. This information is *conscious* because it is *intrinsically evaluative*; it tells us how we *feel* about things. This applies especially to the inwardly derived aspect of consciousness—the conscious "state"—which provides our background sense of awareness. This background sense of awareness is not merely quantitative; it always has a particular qualitative "feel" to it. Conscious awareness is therefore grounded in *emotional* awareness.

WHAT IS EMOTION?

Emotion is akin to a sensory modality—an internally directed sensory modality that provides information about the current state of the bodily self, as opposed to the state of the object

world. It adds a sixth sense (a sixth modality of "qualia") to our conscious existence. Emotion is the aspect of consciousness that is left if you remove all externally derived contents. If you were deprived of all sensory images (drawn from present and past perception), you would still be conscious. You would still be aware of your inner state—of your *core self*. Aristotle suggested that there are only five ways of knowing the world, corresponding to the five classical senses, but there is more to the world than the *outer* world.

EMOTION AS AN INTERNALLY DIRECTED PERCEPTUAL MODALITY

The "sense" of emotion is organized in a very different way from the externally directed sensory modalities. This is partly because it is a state-dependent function rather than a channel-dependent one. It reflects changes in your body that are communicated to the somatic monitoring structures of your brain, not only via discrete information-processing channels, but also via the gross chemical-transport mechanisms of the bloodstream and cerebrospinal-fluid circulation. These somatic monitoring structures, in turn, broadcast their outputs widely throughout the forebrain, thereby exerting a global "mass-action" effect on the information-processing channels of consciousness. (We have pointed out already, in chapter 3, that these outputs are not determined only by *actual* bodily events; body-mapping structures generate a *virtual* body, which is subject to all manner of chemical and other influences.)

Emotion is also different from the other sense modalities simply because it is *internally* directed. Only *you* can feel your emotions. This also applies to consciousness in general (cf. "the problem of other minds," chapter 2), but it applies to emotion in a special way. It is not only the *perception* of emotion that is

subjective. *What* emotion perceives is subjective too. What you perceive when you feel an emotion is your *own subjective response* to an event—not the event itself. Emotion is a perception of the *state of the subject*, not of the object world. If a flash of lightning and clap of thunder cause you to feel a fright, it is not the lightning and thunder that you perceive emotionally (you see and hear them visually and aurally); it is your visceral response to those events that you feel emotionally. The same event can therefore make one person feel frightened and another not.

The fact that certain events make just about *everybody* feel roughly the same way is pregnant with significance for our understanding of the neurobiological mechanisms of emotion. We shall return to this fact in a moment.

MAPS OF THE BODY

The structures that form the *core* of the emotion-generating systems of the brain are identical to those that generate the background state of consciousness (see chapter 3). These phylogenetically ancient structures lie in deep regions of the brain, in the middle and upper zones of the brainstem (see Figure 4.1). The brain structures in question include the hypothalamus, ventral tegmental area, parabrachial nuclei, periaqueductal

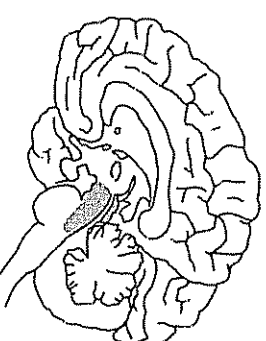


FIGURE 4.1

Location of core emotion-generating structures

gray, raphe nuclei, nucleus locus coeruleus complex, and classical reticular formation. As we discussed in chapter 3, all of these structures are implicated in the monitoring and regulation of visceral states.

Perhaps the most important of all these structures, as far as emotion is concerned, is the **periaqueductal gray** (PAG). This area of gray matter situated deep within the brainstem, surrounding the cerebral aqueduct (hence its name), has a vertical columnar organization (see Figure 3.2). The columns are divided into two broad types: some generate *pleasurable* sensations (in ventral [i.e. lower] PAG) and others generate *unpleasant* (dorsal [i.e. upper] PAG). Degrees of pleasure and unpleasantness calibrate the basic qualitative range within which the "sense" of emotion is experienced. Pleasure and unpleasantness might, therefore, be considered broadly equivalent to light and dark with respect to visual sensation, or high and low tones with respect to auditory sensation. It is important to note that *pain* (as we use the term) is not synonymous with *unpleasantness*. "Unpleasantness" denotes an *emotional* feeling (ultimately derived from the state of the internal milieu), whereas "pain" is a submodality of *somatic sensation*—one of the *externally* directed sensory modalities (see chapter 1).¹ Nevertheless, it is interesting to note that the PAG plays an important part in the generation of both unpleasantness and somatosensory pain. This suggests that our exteroceptive consciousness of pain was, in an evolutionary sense, built upon the existing mechanism for generating unpleasantness (or vice versa).

The distinction between unpleasantness and pain reminds us of the fact that there are two sources of knowledge about the body,

¹The fact that pain and unpleasantness are not synonymous is perhaps best illustrated by pointing out that some people (sexual masochists) experience pain as *pleasurable*. Further evidence for this distinction is the fact that pain and unpleasantness can be selectively targeted pharmacologically. Interactions between the somatosensory and emotional aspects of pain underpin the everyday travails of most pain clinics.

derived from its internal and external anatomy, respectively (see chapter 1). The first represents the "visceral" body—that is, the internal milieu. The internal milieu is regulated by various homeostatic mechanisms, which ensure that blood sugar, temperature, oxygen levels, and the like are adequately maintained. The state of these systems is what is monitored by the deep-brain structures listed above. These structures therefore generate a map of the *functions* of the body. The second source of bodily awareness is linked to the musculoskeletal system. This is the sensorimotor apparatus that moves the body around in the outside world. It is projected onto the cortical surface of the forebrain in much the same way as other objects in the external world are projected onto little maps of visual, auditory, etc., space. This generates a map of the *movements* (or potential movements) of the body.

These two sets of representations are not "maps" in a strict topographical sense—that is, they are not scale models of bodily anatomy.² The map of the internal milieu, in particular, does not represent the body topographically at all; it gathers together and represents pertinent information about the body's homeostatic physiology, not its musculoskeletal anatomy.

²On a map of Britain, the ratio of distances from London to Cambridge to Edinburgh is the same as occurs in reality—that is, the map retains an accurate record of the topographical relationship between these spatial elements. However, not all maps work like this. The map of the London Underground system (perhaps the most famous nontopographical map in the world) does not provide spatially accurate topographical information. The ratio of distances between Paddington, Baker Street, and King's Cross stations on the map is not the same as that found in reality—though obviously some spatial properties, such as the sequence of these stations on a line, is retained. Although the metric properties of the map have been distorted (to ensure ease of use), no one would claim that the map of the London Underground is not a "map." It contains relevant information about objects of importance and organizes the information in a way that allows it to be usefully employed.

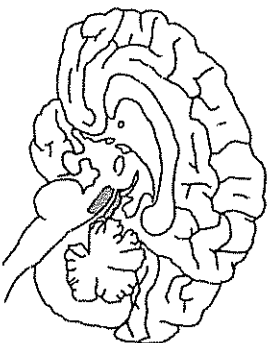


FIGURE 4.2

Tectum and dorsal tegmentum

We have already listed the structures that map the body's physiological functions. Maps of the body in the second (topographic) sense are found in several locations in the brain. One of them is of special interest to us, for reasons that will soon become apparent. This map is located in the **tectum** and **dorsal tegmentum** of the upper brainstem (Figure 4.2)—that is, close behind the *ventral* tegmental area depicted in Figure 4.1. This region of the brain receives input from *all* the sensorimotor modalities, and it is therefore one of the "zones of convergence" mentioned in chapter 2 in connection with the "binding problem." The proximity of this map of the musculoskeletal body to the adjacent projections of its visceral states is important, for two reasons.

First, these two maps together generate a rudimentary representation of the *whole* person, the inner and outer "virtual bodies" combined. Jaak Panksepp—a leading neurobiologist whose conceptualization of the functional anatomy of emotion is very similar to that of Antonio Damasio—therefore (rather boldly) calls this brain region the **SELF** ("Simple Ego-like Life Form"; Panksepp, 1998). (He capitalizes the term to distinguish the technical from the colloquial meaning of the word.) This primal SELF forms the foundational "ego" upon which all our more complex representations of our selves are built (cf. the "autobiographical self" described in chapter 3).

The second reason why the proximity of the two body maps is important is that it gives the emotion-generating part of the brain direct access to one of its *action*-generating mechanisms. The *homunculus* in the dorsal tegmentum provides a combined *sensorimotor* map of the body, which generates primitive action tendencies (e.g., approach and avoidance behaviors, which are closely linked to pleasure and displeasure, respectively). This reminds us of the very important fact that we do not only *experience* our emotions, we also *express* them. Emotion is not only an internally directed perceptual modality, it is also a form of motor discharge.

EXPRESSION OF THE EMOTIONS

The perceptual aspect of emotion has a compulsive effect on us. We cannot simply lie back and feel our emotions. They make us want to *do* something. This "doing something" once again implicates the inner/outer distinction to which we have referred so often. The motor aspect of emotion involves both inwardly and outwardly directed discharge processes. Inwardly, the experience of emotion is accompanied by the release of hormones, changes in breathing and heart rate, vasodilation and vasoconstriction, changes in regional blood supply, and so forth. Outwardly, emotion manifests itself in various ways: through changes in facial expression, baring of teeth, crying, blushing, and the like, but also in complex behaviors like shouting, running away, and lashing out. Some of the inwardly and outwardly directed discharges of emotion are difficult to distinguish from each other (e.g., laughing, crying, blushing). It is also difficult to draw a sharp distinction between the perceptual and motor aspects of emotion; for example, the feeling of a racing pulse is an important part of the *perceptual* complex of fear, as is the urge to run or hide.

We mentioned before that the fact that certain events make just about *everybody* feel roughly the same way is pregnant with significance. This applies equally to the perceptual *and* motor aspects of emotion. Certain situations are apt to evoke certain feelings in all of us, and they are equally apt to make us want to act in certain relatively stereotyped ways. For example, the sight of a snake slithering rapidly toward you is likely to evoke a feeling of fear, no matter who you are, and it also makes you freeze all bodily movement. Such situations appear to have universal significance. Our ability to recognize them, and our reactions to them, appear to be largely innate.³

Neurobiologists call these universal affective reactions **basic emotions**. The "basic emotions" appear to consist in "hard-wired" connections between certain external situations of biological significance and the subjective responses they evoke. This implies that certain patterns of external perceptual stimuli are innately linked to specific internal perceptual stimuli, and that these perceptual links automatically trigger innate (both internal and external) motor responses. The orchestration of these different elements of the "basic emotions" unfolds over concrete anatomical pathways and involves specific physiological mechanisms. Bearing in mind what we have said already about the anatomy and physiology of emotion, the reader can perhaps predict which structures are likely to be involved. For example, we can safely assume that the orchestration of outer and inner perceptions will involve links between exteroceptive forebrain and interoceptive brainstem structures, and that the PAG will be centrally involved in the generation of most (if not all) the basic emotions.

By virtue of these fixed anatomical arrangements, the basic emotions have provided a powerful research paradigm for understanding the brain mechanisms of our inner mental life.

³This does not mean that these inborn emotional action tendencies are not modifiable (see below).

EMOTION IN MAN AND IN OTHER ANIMALS

Needless to say, the "basic-emotion command systems" (as they are called) evolved over eons of time. The basic emotions exist because they have established survival value. In situations of biological significance (e.g., mortal danger, proximity of a fertile mate), these emotions provide ways of reacting that increase the likelihood that the organism will survive and reproduce, and thereby propagate its genes. For this reason, Panksepp (1998) suggests that the basic emotions should be thought of as "e-motions"—"evolutionary motions." Precisely because these mechanisms take so long to develop, and also because they have such potent survival value, they are deeply conserved in mammalian genotypes. They surely evolved long before *Homo sapiens* even appeared on the evolutionary tree, and they will long continue to be preserved.

Accordingly, we share with all other mammals the basic-emotion command systems (and the feelings that correspond to them) described in the following sections of this chapter. Dogs, cats, dolphins, whales, rats, mice—all possess the anatomical and physiological arrangements we describe in the following sections. This shared evolutionary heritage literally embodies the primal experiences of our ancestors, which, even if we cannot reexperience them, have left traces in our "procedural-memory" systems (see chapter 5).⁴ The basic emotions therefore define a

⁴Freud's belief in phylogenetic memories (the "inheritance of acquired characteristics") has often been derided. He seems to have believed (incorrectly) that it was the frequency of occurrence over generations of these primeval events, rather than the survival value of certain ways of responding to them, that led to them being preserved. The figurative language that Freud (1912-13) used to describe this aspect of mental life also created the (false) impression that episodic memories of the events themselves were literally "passed down." The influence of such primeval events on our procedural or instinctual memory systems is certainly a reality, but the indirect mode of transmission and influence does not permit literal "remembering" (see chapter 5).

set of common biological "values" that unite us all in our struggle with the tasks of life. (Cf. the question of consciousness in other animals, in chapter 3.)

THE BASIC EMOTIONS

The scientific data upon which this knowledge is based comes from observations of anatomical structures that produce reliable emotional effects when *modified* in some way. The data come from both animal and human neuroscience, as well as from biological psychiatry.

In animal research, the "modification" might consist of increasing the activity of a structure by stimulating it electrically, or administering quantities of the chemical messenger that is typically employed to excite a system. Neurological results are recorded and linked with observations of the animal's behavior. (It is impossible to monitor an animal's subjective state.) The activation of a structure can also be *reduced* by administering a chemical that blocks its normal activity, or a structure can be eliminated by removing it surgically or ablating it chemically.

When the same brain regions are investigated in humans, the results are typically highly consistent with the animal work. Research with humans lacks the precision of animal research because human researchers do not selectively ablate circumscribed areas of brain tissue. We must investigate people in whom natural events (like strokes or tumors) have produced similar effects. These lesions are usually not circumscribed to a single structure. Likewise, chemical manipulation of the human brain, such as can be observed in drug users and psychiatric patients, usually lacks the specificity and selectivity that is achieved when animal researchers deal with brain tissue more directly. In the case of humans, though, we have access to verbal reports of the *subjective states* that occur when the emotional

parts of the brain are modified. There are also a good few studies of the subjective effects of local brain stimulation during surgical operations and in cases of focal epilepsy.

Readers who are skeptical of the idea that one can simply "turn on" or "turn off" an emotion are encouraged to take a closer look at this literature.⁵ This field of research has produced a far better understanding of the neurobiology of emotion than we could ever have hoped for. Although there are still controversies in the field, especially on the borderlands, the findings reported below are not really controversial; they represent the conservative "basics" that most neuroscientists working in this area would now agree upon.

There appear to be four "basic-emotion command systems" in the brain. In the following sections, we use the nomenclature of Panksepp (1998) to describe these systems: SEEKING, RAGE, FEAR, and PANIC. Some of the terms that other authorities use for these emotion systems are also mentioned. By deriving a common denominator from these alternative terms, the reader should get a good idea of each of the emotions we are talking about.

The SEEKING system

Long known as a "reward" system, the SEEKING system is also associated with the terms "curiosity," "interest," and "expectancy." This system provides the arousal and energy that activates our interest in the world around us. On the perceptual side, it generates the feeling that something "good" will happen if we explore the environment or interact with objects. On the motor side, it promotes exploratory behaviors, like foraging.

⁵Panksepp's (1998) masterful survey of the field is perhaps the best place to start. Joseph LeDoux's highly readable 1996 book *The Emotional Brain* is perhaps more accessible, but it focuses more narrowly on a single emotion: fear.

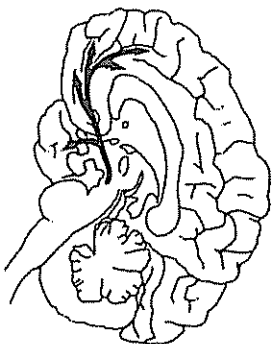


FIGURE 4.3
The SEEKING system

Exploratory behavior varies somewhat from species to species, and it also depends on the particular need that is activating the system at a given moment, but it typically involves sniffing, touching, and oral exploration. This system is heavily activated during sexual arousal and other **appetitive** states (e.g., hunger, thirst, or even craving a cigarette).⁶ It is also associated with play, especially of the rough-and-tumble variety, and some forms of aggression (especially the predatory variety, known as "cold" aggression).

The source cells of the SEEKING system are located in the ventral tegmental area (Figure 4.3). The axons of these cells pass through (i.e., synapse on) the dorsolateral hypothalamus, en route to the nucleus accumbens, which is where most of these axons terminate. They also project further upward to the anterior cingulate gyrus and other cortical areas in the frontal lobes, and downward to the amygdala (in the temporal lobe).

The command neurotransmitter of this system is *dopamine*. (The SEEKING system forms part of the *mesocortical-mesolimbic DA system* mentioned in chapter 1.)

⁶Where Freud used the sexual term "libido" to denote the mental function activated by our bodily needs of all kinds, modern neurobiologists speak of "appetites."

The neurobiology of "libidinal drive" The concept of **drive** seems to be unfashionable in psychoanalysis nowadays. It is unclear why this happened, but it has had the unfortunate result of divorcing psychoanalytic understanding of the human mind from knowledge derived from all other animals. We humans are not exempt from the evolutionary biological forces that shaped other creatures. It is therefore difficult to form an accurate picture of how the human mental apparatus really works without using a concept at least something like Freud's definition of "drive".⁷

The psychical representative of the stimuli originating from within the organism and reaching the mind, as a measure of the demand made upon the mind for work in consequence of its connection with the body. [Freud, 1915c, p. 122]

This definition conveys very well the place that the SEEKING system occupies in our mental economy as a whole.

How is the SEEKING system normally activated? There is a range of **need-detector** mechanisms in the hypothalamus (particularly the lateral and ventromedial nuclei, with extensive connections to other brain regions). These detectors constantly "sample" the internal milieu to maintain its delicate economy. Different hypothalamic regions switch these detector systems on (they act like "accelerators") and off (acting like "brakes"). One of these systems, for example, regulates your core body temperature. It ensures that your temperature stays in the (very narrow) correct range. There is also a thirst detector, a hunger detector, even a "sexual-need" detector. To illustrate how these systems work: Lesions of the "brake" hunger system produce a dramatic

⁷Freud used the German term *Trieb* where we say "drive," but his English translators preferred to use "instinct." Where Freud spoke of "libidinal drives," we nowadays use the term "appetitive drives." Appetitive drives activate the SEEKING system, discussed here.

increase in an animal's interest in food. Given unlimited access to supplies, it will eat to the near total exclusion of interest in all other events in the world. Gross obesity follows very rapidly, at which time food consumption slows. Conversely, lesions to the "accelerator" system create an almost total loss of interest in all food. Anorexia follows rapidly, although the animal will occasionally nibble—just enough to remain alive.

It is uncertain how specific each detector system is to its target need—for example, whether the "thirst detector" is concerned only with thirst. They are probably not entirely specific, but the important issue is that these hypothalamic systems generate "needs," and these "needs" activate the SEEKING system. In other words, when the need-detector systems register that one of the homeostatic mechanisms they monitor has moved out of its acceptable range, they activate seeking—"appetitive"—behavior to correct it. This activation can be maintained for long periods of time. The SEEKING system can also be activated by a range of other inputs, both perceptual and cognitive, but the simple "detector" mechanisms provide the best illustration of how this system works.

What does the SEEKING system do? As the name suggests, it seeks. The more difficult question is: *What* does it seek? One might think that it seeks the specific object of a current need, as determined by the need detectors. The reality is slightly more complex. The SEEKING system itself does not appear to know what it is seeking. (In psychoanalytic parlance, one might say that it is "objectless.") The SEEKING system appears to be switched on in the same way by all triggers, and, when activated, it merely looks for *something* in a nonspecific way. All that it seems to know is that the "something" it wants is "out there." A nonspecific system like this cannot by itself meet the needs of an animal. It has to interact with other systems. The mode of operation of the SEEKING system is therefore incomprehensible without reference to the *memory* systems with which it is inti-

imately connected. These systems provide the *representations* of objects (and past interactions between the self and those objects) that enable the organism to *learn* from experience. One of the most basic tasks that these combined systems have to perform is to distinguish which objects in the outside world possess the specific properties that the internal milieu lacks when a particular need detector switches "on." Like any system of learning, this requires a "reward" mechanism. Panksepp labels this extension of the SEEKING system the LUST system.

The LUST subsystem The LUST subsystem has a longer history of being called a "pleasure," "reward," or "reinforcement" system. These terms reveal that the function of the system is associated with *gratification*—that is, with *consummation* of the appetites that activate the SEEKING system. On the perceptual side, this system generates feelings of pleasurable delight: "That feels *good*!" On the motor side, this system switches appetitive behaviors off and replaces them with **consummatory** behaviors. (There is a reciprocal relationship between SEEKING and LUST activation.) Like exploratory behaviors, consummatory behaviors are complex reflexive-action programs that vary somewhat across species (and the sexes) and across needs. These instinctual behavior patterns are automatically released when the object of a biological need is attained. The thirsty cat laps up milk; the sexually aroused male dog rhythmically thrusts its penis.

The LUST system consists of a complex group of structures arising from the hypothalamus and lying mostly in the basal forebrain, close to the main termination of the SEEKING system's ascending projections (see Figure 4.4). The most critical of these structures appear to be parts of the septal region and hypothalamic nuclei (mainly the preoptic area). Stimulation of these structures (in humans) produces orgasmic feelings. The system terminates in the PAG, which is presumably where the

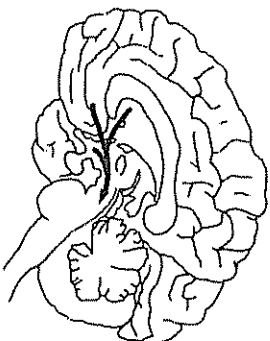


FIGURE 4.4
The LUST system

pleasurable sensations are in fact generated or "perceived" (i.e., where the pleasure centers exert their influence on the virtual body of the primal SELF). The command neuromodulator (actually, neuropeptide) of this system is **endorphin**.

As with the whole concept of "basic-emotion command systems," the idea of "pleasure centers" in the brain might seem overly simplistic—even unlikely—to psychoanalytically minded readers who are familiar with the complexities of human desire. It is therefore important to point out that these crude mechanisms (the existence of which is beyond doubt, and which we share with all other mammals) are subject to a wide range of higher cognitive influences that can modulate, modify, and inhibit them and their associated behaviors in multifarious ways (see below).

The key finding that revealed the existence of these pleasure centers dates back to the 1950s and the work of James Olds (Olds & Milner, 1954). In a series of (initially serendipitous) experiments, Olds demonstrated that animals are prepared to work extremely hard in order to experience electrical stimulation of these brain regions, especially if the electrodes are placed in the septal nuclei. This experimental behavior is called **self-stimulation**. Animals rapidly learn the skilled movements neces-

sary to switch on a neurostimulator when the electrodes are placed in these regions, and they continue to stimulate themselves to exhaustion, and to the near total exclusion of any other behavior. Even when offered a choice between behavior (usually bar-pressing) that produces either food, water, sex, or self-stimulation, they overwhelmingly choose self-stimulation. Typically, such animals self-stimulate for long periods, then occasionally shift to the bar that offers food for a brief snack before quickly returning to self-stimulation. Needless to say, these animals rapidly lose a great deal of weight. The parallel with *addictive* behavior in humans is obvious.

Drug addiction The SEEKING and LUST systems are designed to promote learning, and they motivate us to acquire the skills that are necessary to meet our inner needs in the outside world. The need detectors of the hypothalamus activate the SEEKING system so that this system might generate behaviors that are apt to *meet our actual biological needs* (nutritional needs, the need to reproduce, etc.). Likewise, the pleasure centers of the basal forebrain transmit pleasurable sensations to signal to the brainstem SELF that the object required to meet its actual bodily needs has been attained. The "rewarding" quality of these sensations also *motivates* the animal to perform the *work* necessary to attain these biological objectives. The generation of pleasure for its *own sake* serves no biological purpose. Just like self-stimulation behavior in experimental animals, the use of recreational drugs—such as **cocaine** and **amphetamines** (which stimulate the SEEKING system and thereby artificially generate positive expectancies) and **heroin** and other **opiates** (which stimulate the pleasure centers *directly*)—hijacks, or short-circuits, these adaptive mechanisms. These drugs generate pseudoappetitive behaviors (and associated cravings) and pseudoconsummatory

behaviors (and associated pleasurable sensations) that serve no biologically useful purpose.⁸

Other psychopathologies The SEEKING system of a newborn baby is switched on when activated by a need, without the baby knowing *what* is needed. Left to its own devices, it is so helpless that it will never find the objects required to satisfy its needs and will therefore die. For this reason, it has caregivers that function as "intermediaries" between its needs (communicated by the expression of its emotions) and the objects in the outside world. The actions that these intermediaries perform on the baby's behalf—and their effects—are then gradually learned ("internalized") until the child can take care of itself. This, as we all know, is why parenting is so important. Early experiences of satisfaction form the templates of our understanding of how life works; for a child, learning how to adequately recognize its needs and meet them in the world is utterly bound up with the quality of the parenting it receives. There are all sorts of subtle ways in which this process might be disrupted or distorted (for instance, if a baby's needs are routinely neglected or misunderstood or even met too soon, before they can be felt). The foundations can thereby be laid for later psychopathology—in combination with a

⁸ Some of the dangers of recreational drug use arise from the fact that (as with self-stimulation behavior in animals) preoccupation with the drug can become nearly all-consuming, to the exclusion of all other (biologically useful) behaviors. Also, just as laboratory animals are prepared to work extremely hard to activate the pleasure system, addicts will go to great lengths to obtain another "fix"—as demonstrated by the fact that they resort to burglary and prostitution. Other dangers arise from the fact that the SEEKING and LUST systems can become tolerant of these drugs, with the result that greater use is required to produce the same effects. The drugs in question can also have other, more directly damaging effects on the brain and other bodily tissues (e.g., toxic effects).

set of biological "risk factors," such as variation in the inherent "setting" levels of the basic affective systems.

Even in states of rest, the SEEKING system is tonically active to a certain extent—as long as you are alive, you always need something. Sustained underarousal of the SEEKING system is associated with pathological lack (or loss) of interest in the world. Likewise, unmodulated hyperarousal of this system can produce overexcited states or excessive interest in inappropriate objects and activities. Whatever their cause may be, dysregulations of this type can be managed psychopharmacologically. Drugs that modify mesocortical-mesolimbic dopamine transmission, in various complex ways, therefore form the basis for entire classes of psychiatric medication, used in the treatment of not only the schizophrenias, but also attention deficit and hyperactivity disorders (ADHD), tic disorders, and mood disorders, underlining the vital role that this system plays in human motivation and emotion. (Some of these points will be returned to in our discussion of dreams and hallucinations in chapter 6.)

Activation of the LUST system switches the SEEKING system "off," signaling that an inner need has been met. Activation of the other basic-emotion command systems occurs when our drives are *unmet*, in various ways. Accordingly, the activation of these other systems is associated with varieties of *unpleasure*. The particular variety of unpleasure (and associated instinctual discharge) that is released depends on the nature of the biologically undesirable experience that activated it.

The RAGE system

More than any other system, the RAGE (or "anger-rage") system is activated by states of *frustration*—when goal-directed actions are *thwarted*. The term "anger-rage" is used to denote the *feeling* state associated with the arousal of this system. This term is

necessary because not all aggressive behavior is activated by the RAGE system. Neurobiologists distinguish between two (or three) different types of **aggression**. The RAGE system is associated with only one of them: so-called "hot" aggression. The "cold" type of aggression, associated mainly with predatory behavior, has little to do with feelings of anger or rage; rather, it has to do with *appetitive seeking* and is therefore driven by the dopaminergic system described above. (There is a third variety of aggression, associated with *male dominance behavior*. Neurobiologists classify this type of aggression with the "social emotions," some of which will be discussed later.) The fact that aggression has at least two different neural substrates must have some important implications for psychopathology (for forensic psychology and psychiatry). Here, therefore, is another fruitful area for future collaborative research (see chapter 10).

Feelings of anger-rage (the perceptual aspect of this system) release stereotyped motor programs associated with the well-known "fight" (as opposed to "flight") response. The fighting response is also called "affective attack" behavior. Externally, this involves a facial grimace with baring of teeth, commonly accompanied by an aggressive-sounding noise (e.g., a growl). The body adopts a stable, broad-based posture, and the claws (or fists) are extended. Internally, there is a series of modifications in the autonomic nervous system—such as increased heart rate and redirection of blood supply to the skeletal musculature and redirection of blood supply to the expense of the needed for violent "action" situations (at the expense of the "action-irrelevant" digestive system)—which allow the animal better to engage its enemy.

These changes are orchestrated by amygdalar projections to the PAG. As stated above, the amygdala (in the temporal lobe) is one of the terminal projections of the SEEKING system, and it is made up of a number of different nuclei. The key structure involved in triggering anger-rage is the *medial* nucleus of the amygdaloid complex. This system courses through the bed nu-

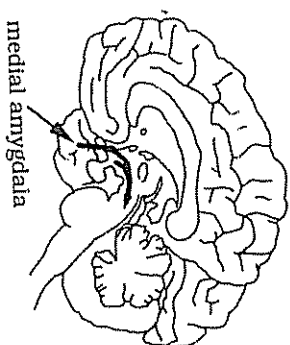


FIGURE 4.5
The RAGE system

cleus of the stria terminalis and (anterior, ventromedial, and perifornical) hypothalamus before it. Like all the other basic-emotion command systems, projects down into the (dorsal) PAG (see Figure 4.5).

Unlike the SEEKING system, this system is activated only sporadically. However, when it is tonically activated at a low level, we use the word "irritability" to describe its effects. Like anger-rage, irritability is usually caused by the frustration of goal-directed activities. The person experiences background annoyance, and the residue of the motor output of the system manifests in behaviors like scowling and/or tensing of the muscles—especially the hands. This form of chronic low-level activation of the anger-rage system, which **primes** it for full-blown affective attack, seems to be a frequent accompaniment of modern life (perhaps especially in large cities).

It is not difficult to see the evolutionary advantages of having a system like this "hard-wired" into the brain. Instead of each generation having to learn afresh the mechanics of how best to deal with an aggressor, this neurobiological circuit preprograms an entire set of automatic "output" routines. The animal possessing such a system is far more likely to survive its first violent encounter than the one that has to learn all these routines from scratch. The programs that such systems encode were selected

and preserved through evolution because they represent multi-purpose perception and action systems that are applicable to a wide range of typical mammalian life-events—from competing with peers for food and sexual partners to avoiding being eaten by predators.

The FEAR system

The second *negative* emotion command system is probably the most intensively researched system of all (for a review see LeDoux, 1996). It generates (on the perceptual side) feelings of fear-anxiety and (on the motor side) the "flight" response. Just as the different substrates of "hot" and "cold" aggression taught us to distinguish between different forms of violence, neuroscientists have also learned to distinguish between *fear-anxiety* and *panic-anxiety*. (To a certain extent, these two varieties of anxiety correspond to the psychoanalytic distinction between "paranoid" and "depressive" anxiety.) The **benzodiazepines** (minor tranquilizers, like diazepam) are successful in reducing fear-anxiety—by increasing GABA inhibition at certain receptors. Panic-anxiety, by contrast, responds mainly to *antidepressant* medications.

Like the RAGE system, the FEAR system is centered in the amygdala and its connections (see Figure 4.6). The *lateral* and the *central* nuclei of the amygdaloid complex are the hub of this system. (The balance between "flight" versus "fight" responses is apparently determined by interactions between the lateral-central and medial parts of the amygdala.) From there, the circuit projects through the (medial and anterior) hypothalamus before it terminates in the (dorsal) PAG of the brainstem—which is where the feelings in question are actually generated ("perceived" by the SELF) and the motor programs released.

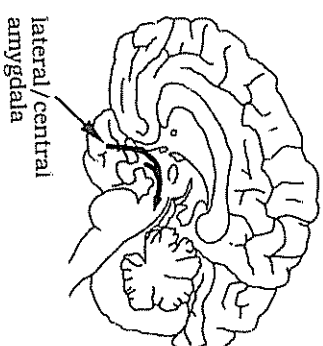


FIGURE 4.6
The FEAR system

The evolutionary advantages of this system are that it allows us to escape rapidly from dangerous situations and to avoid such situations in the future. On the perceptual side, brain stimulation of the sites listed above (in humans) is associated with feelings of extreme anxiety or terror. On the motor side, stimulation of these sites typically makes an animal run away and try to hide. Mild stimulation, on the other hand, leads to a "freeze" response; this is probably because many predators recognize objects through motion, and assuming a motionless posture can therefore help to avoid detection. "Freezing" is, however, only a good strategy under some circumstances. When a predator is quite far away, immobility is generally a useful strategy; however, if an approaching predator is nearby, there is little point in remaining motionless, and flight is the better option. These are the external motor manifestations of fear-anxiety. Internally, there are a range of changes in the viscera, which mirror those seen with activation of the RAGE system: increased heart rate, more shallow and rapid breathing, and redistribution of blood from the gut to the skeletal muscles. The latter change can cause diarrhea in cases of extreme fear, especially when the affect is sustained over a sufficiently long period.

Fearlessness The functional anatomy of the RAGE and FEAR systems allows us to understand some fascinating findings from patients with neurological disorders. A few patients have been described who have selective bilateral lesions of the amygdala, which is usually a result of a rare metabolic condition. These patients are normal individuals in almost all respects. They are, however, distinguished from the norm by the fact that they lack anger-rage and fear-anxiety—the two “negative” emotions mediated by the amygdala. One such case is described by Adolphs, Tranel, and Damasio (1994; see also Damasio, 1999b, pp. 62–67). Their findings—which focus mainly on fear-anxiety—are remarkable. The patient is a bright woman, who understands very well, at a cognitive level, what the concept of “fear” is. At a perceptual-motor level, however, she cannot recognize fear in the faces of others, and she cannot generate a fearful facial expression herself—not even when the examiner models it for her. Her performance in recognizing and producing facial affect is normal for all the other commonly understood emotions, like happiness and sadness. More importantly, her *behavior* is totally devoid of fear. She is exceptionally friendly and is far more prone to touching and hugging than is common among other patients visiting the authors’ laboratory. She is willing to interact with virtually anyone who engages her in conversation and finds it easy to trust people she has only just met. Unfortunately, but not surprisingly, she has frequently had her trust in others abused.

In more severe cases of this condition, which is known as the **Klüver-Bucy syndrome**, in addition to displaying fearless and angerless placidity, such patients (like amygdalectomized laboratory animals) become *hypersexual*. There is a dramatic increase in the *amount* and *variety* of their sexual behavior—so that objects that would not previously have been attractive to them (e.g., members of the same sex, other species, even non-living objects) now are—and there can also be a dramatic in-

crease in masturbation, including in public. These patients (and animals) also become *hyperoral*. They indiscriminately explore objects by mouth and sometimes attempt to eat inedible objects. In addition, they display a symptom called *hypermetamorphosis*—which means that they become hyperdistractable, as everything seems to be of equal interest to them. In animals (but not in humans, due to differences in the anatomy of vision), the symptom of *visual agnosia* (inability to recognize objects visually) is an associated feature of this syndrome.

The personalities of these patients are obviously and dramatically skewed by their neurological conditions. They demonstrate how critically important for normal mental life the “negative” functions of the RAGE and FEAR systems are. This sort of evidence helps us to establish the neural correlates of certain aspects of personality (see Kaplan-Solms & Solms, 2000) and may also help us better to understand the ways in which genetic and environmental factors modify the biological systems that control personality.

The PANIC system

The PANIC (or *separation-distress*) system is associated not only with panic-anxiety, but also with feelings of loss and sorrow. This provides neuroscientific evidence for the link that psychoanalysts have long recognized between panic attacks, separation anxiety, and depressive affect. The operation of this system seems to be intimately connected with *social bonding* and with the process of *parenting*—for reasons associated with the neurochemistry of the system and the way it is designed to operate.

The core of the separation-distress system is the *anterior cingulate gyrus*, which has extensive connections with several thalamic, hypothalamic, and other nuclei (see Figure 4.7)—including the bed nucleus of the stria terminalis, preoptic hypo-

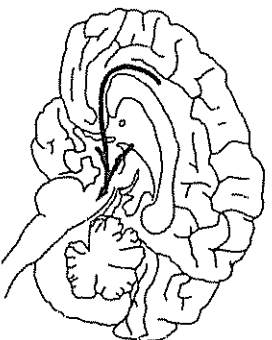


FIGURE 4.7
The PANIC system

thalamus, and ventral tegmental area. These sites are known to be of importance for sexual and maternal behavior in lower mammals. As in all the basic-emotion command systems, there are onward connections from these regions to the (ventral) PAG. The neurochemistry of this system is dominated by the endogenous **opioids**. There is also some evidence that **oxytocin** and **prolactin** are centrally involved in the operation of the system, which, as we shall see in the next section, further underlines the link between the PANIC system and maternal behavior.

Stimulation of some of these structures (in humans) has been associated with sudden onset of panic attacks, and even—in one case—a full-blown clinical depression, which met all the DSM-IV criteria. The depression recovered completely with offset of the stimulation. In animals, stimulation of this system produces “distress vocalizations,” or “separation calls.” These vary from species to species, but they involve actions such as crying, howling, or chirping. Sustained stimulation of this system in freely roaming animals produces an interesting sequence of behaviors. Initially, when the system is first stimulated, it promotes **SEEKING** behaviors, together with distress vocalizations. Presumably this increases the chances of finding the mother, or of being found. After a more or less set period of time, there is a change to *withdrawal* from the environment as the animal retreats into isolation and displays a sort of hibernation behavior

that looks for all the world like depression. This shift in the animal’s behavior, from seeking to withdrawal, is presumably related to the fact that it is not safe to be looking for your mother for too long, due to the risk of attracting the attention of a predator. If she is not around, the best thing to do is to lay low and wait for *her* to find *you*.

The role played by endogenous opioids in this system teaches us an interesting lesson about the nature of attachment. This is best conveyed by describing the various ways in which reinforcement can operate—matters long investigated by animal behaviorists. It is easy to teach animals to do something if they are rewarded when they perform an experimental task. In learning theory parlance, this is called “positive” reinforcement. There is another type of reinforcement that is also a very effective route to learning. This is called “negative” reinforcement. Here, the animal receives *constant* reinforcement, and inappropriate behaviors are associated with the *withdrawal* of this reinforcement.⁹ It is this type of schedule that appears to govern the separation-distress system. Endogenous opioids (like externally administered **morphine**) reduce feelings of pain. Separation from a loved object increases activation of the PANIC system, which reduces the level of opioid activity in this part of the brain. Separation and loss, therefore, are literally “painful.” Young animals rapidly learn to maintain proximity to their caregivers by virtue of this reinforcement strategy.

The CARE subsystem This “social” emotion system influences the *mother’s* behavior no less than the *child’s*. We know that levels of oxytocin and prolactin, two of the key chemicals that

⁹This is not the same as *punishment*—where an action is linked with a bad consequence. As many parents know, punishment is not an effective method of promoting learning.

govern this system, are increased in the period around birth. This increases the extent of mother-infant *bonding* during the immediate postpartum period—again, an aspect of the operation of this system with clear evolutionary advantages. Of particular interest is the fact that these same chemicals are strongly implicated in female sexual behavior. This underscores the sexual underpinnings of mother-infant intimacy, which has long been of interest to psychoanalysis.

There are also interesting clinical implications relating to modifications of this system in some children. This opioid system has been found to be *overactive* in some cases of autism. Consequently, such children experience far less "pain" on separation than their peers, and as a result they bond less well with caregivers and other people. Consistent with this, drugs that block the operation of opiate channels produce more positive social interactions in some cases of autism. But, importantly, the drug only appears to work (to the extent that it can) if it is combined with renewed, facilitating encouragement from the social environment. It is as if the drug opens a window, but by itself it cannot change the nature of the child's object relationships (Panksepp, 1998).

PLAY and other social emotions

As the previous section suggests, neuroscientists are beginning to extend the "basic-emotion" paradigm into other, more complex aspects of human ethology. Perhaps the most interesting avenue of current research in this direction concerns what Panksepp (1998) calls the PLAY system. It is a remarkable fact that all young mammals (including humans) seem to *need* to play, and to need a certain *amount* of play. Whatever its biological purpose may be, play (and especially rough-and-tumble play) seems to function in young children according to homeostatic principles

similar to those that regulate such basic functions as sleep. If a rat pup is deprived of opportunities for rough-and-tumble play, this is followed by a *rebound* effect whereby the pup makes up for the lost time by playing proportionately more whenever it is next given the chance. The fact that this mechanism is so deeply conserved in the mammalian series suggests that play probably performs some critically important developmental functions. Panksepp suggests that the veritable epidemic of ADHD in modern American cities may in part be a consequence of these children being deprived of adequate amounts of rough-and-tumble play.

LEARNING FROM EXPERIENCE

In the foregoing sections, we have repeatedly mentioned the evolutionary advantages of these inherited, emotionally driven behavioral stereotypes. What needs to be emphasized, however, is that it is not enough to have only four emotional responses—SEEKING, RAGE, FEAR, PANIC—coupled with a handful of automatic, stereotyped behaviors to cope with the vast complexities of everyday mammalian life. The world is almost infinitely unpredictable, and we must modulate and regulate ourselves accordingly.

This is reflected in the fact that all the basic-emotion command systems discussed above are, to variable degrees in different species, but to a very great degree in humans, open to influence by *learning* mechanisms. In other words, although these systems are innate, they are by no means "hard-wired" in the sense of being *unmodifiable*. On the contrary, they appear to be specifically designed in a way that requires "blanks" to be filled in by life experience (and especially early experience). This general topic is discussed in detail in chapters 5 and 7, so only a few specific points are made here.

We have already illustrated the essential points we need to make when we described the role of learning mechanisms in relation to the "objectless" drives of the SEEKING system. The young animal knows *that* it needs but not *what* it needs—it has to learn from experience which objects in the world satisfy its needs and which do not. The evolutionary advantage of this is that it enables the animal to adapt to whatever environment it is born into, where the types of available satisfying object may vary widely. Youngsters (and especially those with protracted periods of infantile motor helplessness, like humans) are unlikely to survive this early learning process without the mediation of adult caregivers, who actively *teach* the little one what it has to do to meet its inner needs and survive the attendant dangers. We have also mentioned how easily this mediation process can go awry, and how devastating the consequences of this might be for the future mental health of the child.

Similar considerations apply to the other basic-emotion command systems. In the FEAR system, for example, although some dangerous objects and situations *do* appear to be hard-wired into the system (hence the stereotyped nature of most phobias), the representational (or "object") aspect of the system is left largely blank, to be filled in by early experience. LeDoux (1996) describes in detail how this process works. Two points are of special interest.

The first point is that the connections that link the noxious stimulus (the object-to-be-feared) with the fear-anxiety responses are made with *extreme rapidity*, and they are thereafter maintained *outside extended consciousness*. Once a stimulus (thing or place) is associated with a painful experience (sometimes on the basis of just *one* exposure to the noxious stimulus), the FEAR system is immediately and automatically activated whenever that stimulus is encountered again, even before it is consciously recognized as such. We therefore do not have to

think before we *act* in dangerous situations (although we can *subsequently* reflect on what has transpired). On this basis, LeDoux distinguishes two aspects of fear-anxiety. The first is the one that we have just described, which is mediated by a "quick and dirty" (LeDoux, 1996, p. 163) pathway from the amygdala to the PAG and excludes cortical consciousness altogether. The existence of such a pathway has important implications for psychoanalytic clinicians, in that it explains how it can happen that patients feel anxious in certain situations *without knowing why* (i.e., on the basis of "repressed" past experiences or other unconscious associations). The second, slower pathway includes the cortical tissues of the hippocampus—which is of critical importance for episodic memory (see chapter 5). This enables the autobiographical self to consciously *recognize* what has happened and to deliberate reflexively upon it. This "extended consciousness" pathway also links the FEAR system to the executive systems of the brain, which leads us to the second of LeDoux's important points.

THE TAMING OF AFFECT

Once connections of the type just described have been made, they are *indelible*; nothing can extinguish the fact that the noxious object, place, or situation has been incorporated into the FEAR system's lexicon of "dangerous" things (LeDoux, 1996, pp. 250–252). The fact that such things cannot be forgotten has definite evolutionary biological advantages. However, they can also be *maladaptive*. An object, place, or situation that was dangerous in early childhood—when the subject was helpless and vulnerable—might not be equally dangerous or even dangerous at all to the adult. Under these circumstances, it would be highly inexpedient if repeated or renewed exposure to the once-

feared object continued forever to unleash full-blown anxiety attacks (feelings of overwhelming trepidation, freezing, fleeing, hiding, palpitations, rapid breathing, etc.).

For this reason, although the link between such objects and the FEAR system is indelible, the *output* of the system can nevertheless be **inhibited**.¹⁰ In other words, although the association still exists unconsciously, its influence on extended consciousness and voluntary behavior is damped down or even blocked entirely. As mentioned in chapters 1 and 3, the apparatus for such inhibitory control is located in the frontal lobe—in particular, in the ventromesial and orbital frontal areas (Figure 4.8). When the *outward manifestations* of fear-anxiety reactions are extinguished in laboratory animals (through behavior-modification techniques), what functional brain imaging reveals is that the FEAR system *continues* to be highly activated, to almost the same extent as it is in animals displaying full-blown fear-anxiety responses. What differs dramatically between the two groups is that the frontal lobes are *concurrently* highly activated in the fear-inhibited group. As we discussed in chapter 1, the extent of frontal-lobe development is what distinguishes us humans most from other mammals. This is also what most distinguishes the brain of the adult human from that of the child. The frontal lobes develop rapidly during the first few years of life and continue to do so until late adolescence. These neuroanatomical facts explain the enormous differences with respect to flexibility and degree of emotional control that distinguish the human adult from the child and from other mammals. The implications for some forms of psychopathology are again obvious.

Presumably, similar mechanisms exist for all the basic-emotion command systems, although they have been less thoroughly studied. Unbridled affective responses of the types released by

¹⁰For an encyclopedic and multidisciplinary survey of research relevant to this topic, written especially for psychoanalytic readers, see Schore (1994).

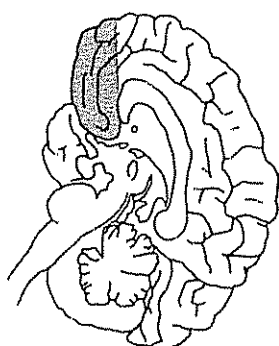


Figure 4.8
Ventromesial and orbital frontal areas

the SEEKING, RAGE, and PANIC systems are no less maladaptive, in principle, than overwhelming FEAR reactions. The balance between these primitive instinctual mechanisms and the voluntary programming, regulation, and verification of action (chapter 1) contributed by frontal-lobe mechanisms, therefore, seems to provide a direct parallel with the balance between what psychoanalysts classically referred to as the "id" and "ego" mechanisms. Damage to these frontal mechanisms is what led Harlow (1868) to observe in the case of Phineas Gage (described in chapter 1) that "the equilibrium or balance, so to speak, between his intellectual faculties and animal propensities, seems to have been destroyed." However, by the same token, the inhibitory brain mechanisms just described can also be *operative*—and this might provide the neural basis of some pathological varieties of repression and other forms of inhibition. Here, once again, there are very fertile fields for future interdisciplinary research.

In discussing such issues, we are leaving the terrain of the basic-emotion command systems themselves and moving deeper into the functional architecture of the *experience-dependent* systems of the brain.