most popular research topics in modern neuroscience. There is, of course, also a lot more that could be said about the implications of these research findings for depth psychology. Nevertheless, we have managed to cover most of what is fundamentally important to our theme. We are now in a good position to tackle our next topic: dreams and hallucinations.



# CHAPTER 6

# DREAMS AND HALLUCINATIONS

Dreams are the primary focus of this chapter. They are hallucinations that we *all* experience—hallucinations that have been regarded by many as a "normal" form of psychosis. Freud was especially interested in dreams because he believed that, if he could understand their mechanism, he would be able to comprehend something fundamental about mental illness.[1] In the later sections of this chapter, other forms of hallucination and delusion, primarily in schizophrenia, are discussed. This chapter focuses on many of the same brain structures that were covered in the previous three chapters on consciousness, emotion, and memory. This is because the brain mechanisms of dreaming (perhaps not surprisingly) overlap a great deal with those of consciousness, emotion, and memory.

## DIFFICULTIES INVESTIGATING DREAMING

Dreams are notoriously difficult to investigate scientifically. This chapter is therefore also concerned with methodological questions regarding *how* brain mechanisms of dreaming have been

[1] Many other physicians and scientists shared this view; see Gottesman (1999, pp. 470, 500) for examples.

investigated. Attention is drawn to the dangers of using inappropriate methods to investigate complex psychological states, and to the advantages of using more than one scientific method to study a difficult and elusive subject. In the past, one of the failings of psychoanalysis was its overreliance, despite the great complexity of its subject matter, on a single method for reaching its conclusions, but this has begun to change somewhat in recent years. Checking the findings of one method against those of another makes it possible to minimize the bias associated with a single method. Our review of the dreaming brain draws on findings from neurophysiological work on animals, sleep studies and functional-imaging studies in neurologically intact humans, and clinical and experimental investigations of patients with focal brain lesions.

## REM SLEEP

Any discussion of the brain mechanisms of dreaming cannot begin before the phenomenon of **rapid-eye-movement** (REM) sleep has been introduced, because REM sleep has become widely known as "dreaming sleep." However, as we shall learn, it is a mistake to equate the two phenomena. Indeed, the conflation of REM sleep with dreaming is one of the most substantial errors that has arisen from methodological impropriety in this field.

When the REM state was discovered in the 1950s, the scientists involved (Aserinsky & Kleitman, 1953; Dement & Kleitman, 1957) immediately suspected that it might be the physiological correlate of dreaming. This was because the REM state involves a period of physiological arousal in the context of otherwise quiescent sleep, just as the dream state involves conscious mental activity in the context of otherwise unconscious sleep. During





REM, it is not only the eyes that are active. An electroencephalogram (EEG)—which provides a measure of the electrical activity in the brain—made during REM would suggest that although you are sleeping, your brain is in a state of heightened activation akin to full wakefulness. There is also activation of other bodily systems. You begin to breathe differently, your heart rate increases, and your genitals (in both males and females) become engorged.[2] One is thus highly excited in several ways during REM sleep. By contrast, however, skeletal muscle tone *drops* dramatically (with the exception of the musculature controlling eye movements). This effectively paralyzes the sleeper, and it apparently prevents him or her from acting out dreams. This cycle appears more or less every 90 minutes in humans, so that we spend some 25% of our sleeping hours in the REM state.

The easiest and most obvious way to test the hypothesis that the REM state is the physiological equivalent of dreaming is to wake people up during both REM and non-REM sleep and compare the frequency of dream reports found in the different awakenings. The first time this hypothesis was tested, it was immediately obvious that many more dream reports are obtained from REM than non-REM (NREM) awakenings. Today, 50-odd years after this issue was originally investigated, there remains some controversy about the exact percentages. The strongest claim is that 90–95% of awakenings from REM sleep produce dream reports, whereas only 5–10% of awakenings from NREM sleep produce equivalent reports. Probably most authorities would agree on a conservative 80:20 (REM:NREM) dream-report ratio.

[2] In fact, penile erection during REM sleep is so reliable that it provides the basis for one of the most widely used investigations of male impotence. If one measures penile tumescence during sleep and the subject has erections during REM, it is likely that his impotence is of psychological origin.

Taking into account the fallibility of human memory in general, let alone memory for *dreams* (which are particularly difficult to recall), it would have been unreasonable for early investigators to expect to obtain a 100% dream recall rate from REM sleep awakenings, or a 0% rate from NREM awakenings. Under the circumstances, the (roughly) 80:20 ratio that *was* observed was therefore interpreted as a near-perfect correlation, and the hypothesis was taken as confirmed: it was concluded that REM sleep and dreaming were literally the *same thing*, considered from two different observational perspectives (see chapter 2). This equation provided an extremely valuable scientific foothold (although it later proved to be a slippery slope): by making the assumption that the REM state is synonymous with the dream state, scientists believed that they had in their grasp an objective measure of the presence or absence of dreaming. As a consequence, they could carry out objective experiments on perhaps the most subjective of all mental functions, the *psychological* study of which had, moreover, provided the theoretical bedrock for the whole discipline of psychoanalysis (which at that time totally dominated American psychiatry). The fact that not only humans but *all mammals* display the REM state made it possible for neuroscientists to go one step further: they could identify the *brain mechanisms* underlying the REM state (read: dream state) by means of animal experiments that were ethically unacceptable in humans. This is where the slippery slope began, for no matter how close the homologue may be between the REM state in humans and other mammals, we have no way of knowing whether the same applies to their *dreams*. The moment investigators switched from studying humans to other animals, the monitoring of their subjects' dreams (as such) was perforce abandoned.





## The biological basis of REM sleep

The main thrust of the ensuing investigations took the form of lesion studies. A French neuroscientist, Michel Jouvet (1967), carried out the first key studies by performing a series of ablation experiments. Although REM sleep occurs in a remarkably wide variety of animals, cats were the main targets of this research—partly because their brains are so similar to ours, but no doubt also because they sleep for so much of the day! Jouvet made a series of slices through the neuraxis of the cat, starting at the highest level of the frontal lobes and moving progressively downward toward the brainstem. He then systematically investigated the effects on the sleep cycle. He wanted to ascertain the key lesion site that would obliterate REM sleep. To his amazement, he found that you could effectively detach the entire forebrain from the brainstem, and the REM state would still remain intact and would punctuate NREM sleep with the same monotonous regularity. The critical incision occurred only in the middle regions of the primitive brainstem, at the level of the *pons* (see chapter 1). Subsequent investigators confirmed that REM sleep can only be obliterated entirely by creating fairly large lesions in the pons (Figure 6.1) (Jones, 1979). In short, these studies demonstrated that, whatever REM sleep was, it was *causally*

FIGURE 6.1
Lesion site to obliterate REM sleep

*generated* by structures in the pontine brainstem. The implications of this finding were enormous. Since the forebrain is the seat of all our higher mental functions (i.e., representational cognition; see chapters 1 and 2), the early investigators concluded that REM sleep (read: dreaming) is an entirely "mindless" activity. This raised serious questions for any psychological theory of the causation of dreams, not least among them being the Freudian theory that dreams are caused by wishful states of mind. The following quotation is from one of the most influential papers in the field:

> If we assume that the physiological substrate of consciousness is the forebrain, these facts completely eliminate any possible contribution of ideas (or their neural substrate) to the primary driving force of the dream process. (Hobson & McCarley, 1977, p. 1338)

### Links between REM sleep, consciousness, and emotion

The role of the pons and other nearby brainstem structures in creating "core" consciousness (discussed in chapter 3) is not incompatible with the notion that dreams are "mindless." Nobody denied that dreaming is a *state of mind*, that you are *conscious* while you are dreaming. The same applies to the fact that many dreams are strongly *emotional* experiences. Although the role of the PAG in generating emotional states had not yet been discovered, the role of the reticular formation in generating consciousness *was* known at that time. But whether these links were understood or not was immaterial: the early investigators did not deny that dreams *took the form* of conscious, emotionally charged experiences; all they asserted was that the mental aspect of dreaming was not *causal* of the dreams. Dreams, they argued, are caused by something happening in the pons that





switches on absolutely *automatically*, every 90 minutes or so, regardless of your state of mind. Since nearby brainstem structures were also known to regulate eye movements, heart rate, and breathing, it seemed perfectly obvious that REM/dreaming was just a basic physiological state. The biological reason for this pontine clockwork was (and remains) unknown, but it was confidently assumed that dreams were merely a byproduct (or *epiphenomenon*) of this causal physiological process.

Philosophically minded readers might have problems with this type of reasoning. You might well ask (as we did in chapter 2) whether it ever makes sense to claim that a physiological process *causes* a mental event, and vice versa, or whether it makes sense to claim that some neurophysiological events are mindless while others are not. From the standpoint of dual-aspect monism (see chapter 2), every neurophysiological event is simultaneously a mental event—albeit, ultimately, an unconscious one. However, although the early neuroscientific investigators of REM dreaming did not address such issues in any depth, they were able to claim that since the generation of REM is an automatic, preprogrammed process, its unconscious mental correlate is as "motivationally neutral" (Hobson & McCarley, 1977, p. 1338) as the brainstem mechanism that generates your heartbeat. This much seemed certain.

### The neurochemistry of REM

By 1975, Hobson and McCarley had narrowed the search for the pontine "dream-state generator" (as they called it) to a set of precisely defined nuclei within the pons. In that year they published a famous paper in which they argued that the REM state is switched on and off by two groups of reciprocally interacting nuclei, one of which excretes a neurotransmitter that switches it on, and the other two neurotransmitters that switch it off

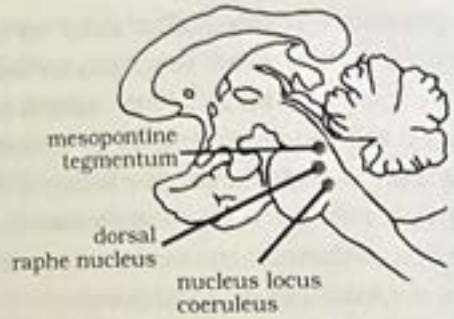



FIGURE 6.2
The "dream-state" generator

(Hobson, McCarley, & Wyzinki, 1975). Although they later changed their minds about some of the specifics of the anatomy, their argument was that the key neurons that switch REM sleep on lie in the **mesopontine tegmentum** (see Figure 6.2). These neurons fire rapidly shortly before the onset of REM, and they stay highly active throughout the REM phase. The neurotransmitter that these neurons produce is **acetylcholine** (see chapter 1). They are therefore described as *cholinergic* cells, and the REM state is considered to be a cholinergically driven phenomenon.

At the transition between REM and NREM sleep, two other sets of neurons, in the **dorsal raphe nucleus** and the **nucleus locus coeruleus**, start firing rapidly (Figure 6.2). The dorsal raphe produces the neurotransmitter **serotonin**; the nucleus locus ceroleus produces *norepinephrine*. When these nuclei become active, the cholinergic system simultaneously (in fact, *consequently*, due to reciprocal inhibition) switches off. This switches off the REM state, and the sleeper falls back into NREM sleep, with a mellowing surplus of serotonin and norepinephrine flowing around the brain. Some 90 minutes later, these two groups of nuclei reciprocally alter their function again—such





that the levels of serotonin and norepinephrine drop, and acetylcholine increases—and the excited REM state reappears.

So, according to this model, acetylcholine switches the REM state on and switches NREM sleep off. Serotonin and norepinephrine switch NREM sleep on and switch the REM state off. For obvious reasons, this model was named *the reciprocal interaction model*. This physiological account of REM sleep is extremely compelling. Some 25 years after it was first proposed, it still completely dominates the field of REM sleep research. By 1975, then, some of the great mysteries surrounding sleep and dreaming appeared to have been resolved.[3]

### Dreams are froth?

Two years after they proposed the reciprocal activation model, Hobson and McCarley published a second paper, which contained a second model—this time not a model of REM sleep but, rather, one of *dreaming* itself (Hobson & McCarley, 1977). This seemed to be a legitimate extension of the first model, because REM sleep and dreaming were thought to be essentially the same thing. They called their second model the *activation synthesis model*. The *activation* aspect of the model argues (no surprise here) that dreaming is activated by cholinergic mechanisms in the pontine brainstem. As we have noted already, this activation—which actually *causes* dreaming—is thought to be "motiva-

[3]It was subsequently discovered that there are interesting periodic changes in our mental state during *waking* life on a 90-minute cycle, which may well relate to the REM/NREM cycle during sleep. However, the effects are far more dramatic during sleep, in part because there is an enormous input of sensory material from external reality during waking life. During sleep (when the other inputs are removed), these intrinsic oscillatory mechanisms appear to dominate.

tionally neutral." The *synthesis* aspect of the model argues that the forebrain, thus activated, lamely attempts to piece together (or synthesize) the meaningless conscious representations (memory images, thoughts, and feelings) that are randomly stimulated from below. Again, note that the forebrain's contribution to the process is secondary to a primary, brainstem-driven events—hence the notion that the dreams themselves are epiphenomenal to the REM state. From the forebrain's point of view, images are being activated during REM as if it were wide awake and experiencing something; it therefore does the only thing it can do, which is to string the images together into a self-object *episode*. In Hobson and McCarley's memorable phrase, the forebrain "does the best of a bad job" by trying to make a sensible experience during REM sleep out of the intrinsically "inchoate" images thrown up by the brainstem (Hobson & McCarley, 1977, p. 1346). Freud had a term for this sort of theory, which actually existed in a speculative form in 1900, prior to the development of modern neuroscience. The expression is "*Träume sind Schäume*," which literally translates as "dreams are froth" (Freud, 1900a, p. 133 [p. 138 in the original German]).

This phrase epitomized Hobson and McCarley's conception of dreams. Their work posed a clear threat to Freudian psychoanalysis, and Hobson wasted no time in pointing this out at the 1976 annual meeting of the American Psychiatric Association (APA). After Hobson's presentation, a vote was taken among the membership of the APA as to whether Freud's dream theory was still scientifically tenable, in the light of Hobson's findings. At that time, the APA was still dominated by members sympathetic to psychoanalysis. Nevertheless, the result of the vote went overwhelmingly against Freud—suggesting that this was the end of the road, scientifically speaking, for Freud's (1900a) account of the mechanism of dreams. Given that Freud saw dreams as the "royal road" to an understanding of the unconscious mind, this had serious implications for psychoanalysis in general. It is no





exaggeration to suggest that the tide turned decisively against psychoanalysis in America at that fateful 1976 meeting.

## The dreams of cats

But, as the attentive reader will recall, the activation synthesis theory embodied a critical methodological flaw. Hobson and McCarley's dream theory rested heavily on the *assumption* that dreaming and the REM state are synonymous. The finding that the REM state co-occurs with dreaming in humans, and the fact that the REM state also occurs in cats (and rats), led to a series of experiments on the brains of these lower mammals that sought to identify the brain mechanisms that generate REM sleep (read: dreaming). Having done so, and having demonstrated conclusively that only large pontine-brainstem lesions obliterate REM sleep, the next logical step would be to check whether these lesions also obliterate *dreams*. After all, it was largely the association between REM sleep and dreaming that made it so interesting in the first place. The problem was, of course, that it is impossible to ask a cat (or a rat) whether it is dreaming or not. Some cat lovers might believe that they *do* know when their beloved animals are dreaming, but even behaviorists know that it is dangerous to infer the content of an inner mental state from the form of an external behavior!

The fact is that a reliable method for checking the assumption that the REM state and dreaming are synonymous was available all along, by investigation of dreaming in those who can provide a subjective report. However, once neuroscientists had made the assumption that REM and dreaming were synonymous, it had become such a truism that nobody seemed even to think of checking it, and attention shifted naturally to animal research.

A reliable method for linking a psychological function with a brain structure is the *clinico-anatomical method*, which forms the

basic methodological building-block of human neuropsychology (see chapter 2). This well-established tool was introduced to neuropsychology by Pierre Paul Broca in 1861. The *clinical* side of this method involves making an observation that a mental function is lost following a focal brain lesion. In Broca's famous case, discussed in chapter 2, it was language that was lost. The *anatomical* side of the clinico-anatomical method involves ascertaining the precise extent and location of the brain damage that caused the loss of the mental function in question. In Broca's time, investigators had to "wait" for their patients to die to make this kind of observation. Today, this research can be conducted with living human subjects, using brain-imaging technology.[4] The autopsy of Broca's patient, Leborgne, revealed an area of damage on the lower left-hand side of the frontal lobe. Broca concluded that this is the neurological substrate of the ability to speak—because when it is damaged, speech is lost.

This turned out to be a somewhat oversimplified conclusion. We now know that other parts of the brain participate in a complex functional system subserving speech and language (see chapter 2); however, even these other components of the neural substrate for language were identified using the clinico-anatomical method. From 1861 onwards, therefore, the guiding principle in neuropsychology has remained the same: to demonstrate that activity in a certain part of the brain is the neural correlate of a specific mental function, it is necessary to link damage to that region to a deficit of that function.[5] Jones (1979) demonstrated this, for REM sleep, in cats; subsequent sleep researchers confirmed that this clinico-anatomical correlation held good for humans too (in cases with naturally occurring lesions). The link between the pons and *REM sleep* is therefore clearly established,

[4] Computerized tomography (CT) and magnetic resonance imaging (MRI).

[5] Today it is possible to go further, and check the clinico-anatomical correlation by ensuring that exercising the function in question is linked with increased metabolic activity in that same region (using PET imaging and fMRI).





in humans and other animals. But it is only in humans that the link with *dreaming* could be established—or refuted.

### REM and dreams are not synonymous

Astonishing as it may seem in retrospect, the equation "REM sleep = dreaming" was subjected to systematic clinico-anatomical scrutiny only forty years after the association between REM sleep and dreaming was discovered. And when it was, it was found to be seriously wanting. In a 1997 study, 6 patients who had sustained damage to the REM-generating regions of the pons were asked whether or not they were still dreaming, and their answer was a clear "*yes*." In contrast, more than 40 other patients with damage to specific parts of the *forebrain*, nowhere near the critical REM-generating structures, *did* experience a cessation of dreaming following their brain damage—but in these patients *the REM state was preserved* (Solms, 1997a; see also Solms, 2000a).

## NON-REM DREAMS

The discovery that pontine-brainstem damage does not produce a loss of dreaming in humans led researchers belatedly to take account of previously neglected observations that seemed to point in the same direction (derived from other methods, more suited to human dream research than research on cats and rats). The main victim of this neglect was the work of David Foulkes (a Chicago psychologist) and his collaborators. Foulkes (1962) focused on NREM dreams, which, according to classical teaching, were supposed to be extremely rare. What he found was that by simply rephrasing the question that subjects are asked upon awakening in the sleep laboratory—saying to them

"What was passing through your mind?" rather than "Were you dreaming?"—subjects reported that they were experiencing complex mentation during NREM sleep on 50% of awakenings. However, the REM = dream theorists were quick to point out that dreaming is not the same as thinking.

Our attention therefore shifts to the 5–10% of occasions on which subjects report fully fledged dreams in NREM sleep. These dreams are no different from the dreams of REM sleep. Even Hobson (who has the most to lose from such findings) confirmed that these NREM dreams are "indistinguishable by any criterion" from REM dreams (Hobson, 1988, p. 143). In fact, some REM = dreaming theorists believed that these *were* actually REM dreams that were misattributed to NREM sleep due to the aforementioned vagaries of human memory.[6] Foulkes (1962) showed that this assumption was wrong. He observed that you are most likely to have dreams in NREM sleep *just after you have fallen asleep*, during what is known as the *sleep-onset* phase of NREM sleep (more technically known as Descending Stages 1 & 2). On awakenings from these first few minutes after falling asleep, subjects report dreams some 70% of the time. Most people do not remember these dreams when they wake up in the morning, for obvious reasons, but we have all had the experience of dozing off briefly and then waking up (often with a start) from a dream. These dreams occur before you have entered your first REM period (in fact, roughly 90 minutes before). The 70% of NREM

[6] A similar claim has recently been advanced by Tore Nielsen (2000). Nielsen suggests that although these dreams occur during NREM sleep, as defined by the standard physiological criteria endorsed by the field for over 30 years (Rechtschaffen & Kales, 1968), they are probably generated by intrusions of REM physiology into the NREM state. NREM dreams, according to Nielsen, are therefore actually "covert" REM dreams. Hobson so enthusiastically grasped Nielsen's lifeline to his theory that he actually went so far as to assert (in Hobson, Pace-Schott, & Stickgold, 2000) that "all sleep is REM sleep, more or less"!





dream reports that are obtained from the sleep-onset phase therefore cannot be misremembered REM dreams.

Antrobus and his colleagues made a related observation, at the opposite end of the sleep cycle (Kondo, Antrobus, & Fein, 1989). They demonstrated that the closer you get toward awakening in the morning—that is, at the end of a night's sleep, after the last REM phase (more technically: in the rising morning phase of the diurnal rhythm)—the more likely you are to obtain a REM-like NREM dream report. This is called the "late-morning effect." The implications of this finding are similar to those for sleep-onset dreaming: the *farther away* from the last REM period you get, the more likely you are to have an NREM dream. In the classical "reciprocal interaction model," these transitional phases between wakefulness and sleep (sleep onset and the late morning) were described—physiologically speaking—as *maximally distinct from the REM state*: they were characterized by very high levels of norepinephrine and serotonin and very low levels of acetylcholine. Clearly, then, dreaming is *not* causally dependent on the unique physiological characteristics of the REM state. However, most NREM dreams do share *another* crucial characteristic with the REM state, which probably casts important light on their true causal physiology. We shall mention this characteristic in a moment.

Before doing so, it is worth asking in passing why the findings contradicting the REM = dreaming doctrine were neglected for so long. The answer may have something to do with the difference between "brain" observations (concerning the state of a piece of physical tissue), and "mind" observations (concerning the contents of subjective reports). The reaction of the scientific community to findings in dream research, and perhaps in other aspects of neuroscience, have often been distorted by the fact that we are more prepared to accept evidence derived from precisely measurable physiological and anatomical variables than from the complicated field of clinical and subjective reports. Understandable

as this bias may be, the example of dream research shows that it is essential for modern neuroscientists to take serious account of the data derived from *both* observational perspectives in the mind–body equation.

## DREAMS AND AROUSAL

The feature that most NREM and REM dreams have in common is *arousal*. This term is not implied in the narrow sexual sense, but with reference to levels of brain activation. Shortly after you fall asleep, your brain is still relatively aroused, as you begin the gradual decline from full wakefulness into sleep.[7] As mentioned earlier, the REM state is characterized, perhaps above all, by sustained periods of (cholinergic) brain activation interrupting an otherwise quiescent sleep state.[8] The rising morning phase, too, is characterized (indeed, defined) by relative arousal—albeit hormonally rather than cholinergically mediated. The three periods of sleep during which you are most likely to experience a dream, therefore, are characterized not by the unique physiology of the REM state (which characterizes only one of the three periods) but by *various types* of arousal. This suggests that a certain *amount* rather than a certain *type* of arousal is a necessary precondition for dreaming.

In the activation synthesis theory, the arousal that accompanies dreams was not only thought always to be of the same type (i.e., *cholinergic* arousal), it was also thought always to arise from the same place (namely, the *brainstem*). If this were true, it might still be possible to claim that dreams are "mindless" and

[7]The contribution that these remnants of wakefulness make to dreaming may be one source of what Freud (1900a) called the "day residues" in dreams.

[8]This striking coexistence of heightened brain activation with ongoing sleep led the early investigators of what later came to be known as "REM sleep" to call it "paradoxical sleep."





"motivationally neutral." But, in fact, good evidence exists that suggests that dreams *can* be causally generated by *forebrain* mechanisms.

## DREAMS AND EPILEPSY

There is a form of epilepsy that involves *partial* seizures that are entirely localized to the limbic regions of the forebrain. Partial seizures occur when the abnormal neuronal activity that causes a seizure does not spread to the rest of the brain (which normally causes seizures to *generalize* into the familiar form known as "tonic clonic convulsion"). Partial seizures reflect their localization: if epileptiform neuronal firing occurs in the visual cortex of the right occipital lobe, the seizure takes the form of flashes of light (or "phosphenes") in the left visual field; if the abnormal activity is in the left motor cortex, the seizure takes the form of twitches in the right arm or leg. Similarly, when epileptiform brain activity is localized to the *limbic* parts of the forebrain, which subserve emotional and memory functions (e.g., the amygdala and hippocampus; see chapters 4 and 5), the resultant seizure takes the form of a *complex mental experience* (e.g., a reminiscence accompanied by a strong feeling of emotion). This limbic form of partial seizure is called *complex*-partial to distinguish it from the elementary sensations and movements that are characteristic of the *simple*-partial seizures just described.

Seizures occur quite frequently during sleep, and typically during the NREM phases—which are characterized by rhythmic, slow waves of electrical activity of a kind that are apt to set off seizures in predisposed brains. These seizures assume various forms (depending on the location and extent of the epileptogenic focus), but not infrequently they take the form of complex-partial seizures. This implies (by definition) that the abnormal brain activity causing the seizure is *wholly confined to the limbic*

*regions of the forebrain.* Specifically, the seizure focus does not spread to the core brainstem structures that regulate the sleep cycle (if it did, the resultant seizure would be neither complex nor partial). It is therefore of considerable interest to observe that these NREM seizures are frequently accompanied by dreams. In fact, they are typically associated with highly distinctive dreams and take the form of recurring nightmares (which reflects the involvement of limbic emotional and memory mechanisms). Given what we know about the underlying physiology of these dreams (which are, in fact, seizures—unequivocally *caused* by focal activation of specific limbic forebrain structures during NREM sleep), one may confidently conclude that the arousal mechanism that triggers dreams is not *necessarily* located in the brainstem at all. Dreaming, it seems, can be triggered by arousal of *any type* arising from *any place*—including the emotion- and memory-generating structures of the limbic forebrain. This casts further serious doubt on the assertions of the old REM/brainstem dream theorists who claimed that the activation of ideas, memories, and emotions cannot, to quote Hobson and McCarley (1977) again, be "the primary driving force of the dream process."

## WHAT IS THE "PRIMARY DRIVING FORCE" OF THE DREAM PROCESS?

If it is no longer tenable to assert that the pontine brainstem contains the primary causal generator of dreaming, then what *is* the primary driving force behind dreams? We said earlier that clinico-anatomical studies revealed that lesions of the pons did *not* cause cessation of dreaming (evidence *against* an exclusive causal role for the pons), but we also said that lesions in two forebrain regions *did* have that effect. Do *these* regions perhaps contain the long-sought "dream-state generator"?





The first of these regions is the transitional zone between the occipital, temporal, and parietal cortex, at the back of the forebrain, in the very hub of the functional unit for receiving, analyzing, and storing information (see chapter 1). Lesions in this area (on *either* side of the brain) produce a total cessation of dreaming (the precise location of these lesions, though, is still uncertain: see Yu, in press).

The other region with this property is the limbic white matter of the ventromesial quadrant of the frontal lobes. Damage to this area of the brain (on *both* sides simultaneously) also produces a total cessation of dreaming. Damage to other parts of the brain causes other characteristic *changes* in dreaming (e.g., increased frequency of dreaming, increased nightmares, defective visual dream imagery). This suggests that these regions, too, form part of the complex "functional system" that generates dreams (see chapter 2). The parts of the brain in question include the entire *limbic system* (including all the "limbic" components of the frontal and temporal lobes, but excluding most of their "higher cognitive" components), as well as most of the *visual system* (excluding the visual "projection" cortex). However it seems likely that one of the two structures that are *essential* for the generation of dreams (i.e., either the occipito-temporo-parietal junction or limbic frontal white matter) embody the "primary driving force" behind dreaming.

## FUNCTIONAL-IMAGING FINDINGS

We said earlier that clinico-anatomical findings nowadays are typically checked against functional-imaging findings for accuracy. This is in keeping with the view that scientific conclusions regarding something as complicated and experimentally elusive as human mental life should—wherever possible—be confirmed

by multiple, converging methods of investigation before they can be accepted with confidence.

Through functional brain imaging, it is possible to obtain a graphic representation of the brain of a healthy living subject and to observe where neural activity is greatest during certain mental states. In the last few years, this procedure has been applied to sleep and dreaming by a number of pioneering investigators. The definitive studies in this regard were published by Alan Braun from the National Institutes of Health in Washington, DC, who, together with colleagues (Braun et al., 1997, 1998), used PET to investigate what the brain looks like during REM sleep—the time when one is *most likely* to be dreaming.[9]

During such investigation of the state of the brain during REM sleep, one is probably imaging two different states simultaneously: the REM state and dreaming. There is an 80% chance that dreaming will occur during REM, so the average of the data spread across several REM phases will almost certainly also capture the dream state. (With PET imaging it is always necessary, for technical reasons, to study the *average* picture.) The picture that emerges is therefore a combination of the dreaming and the REMing brain. Not surprisingly, then, Braun found that the pontine brainstem mechanisms that switch on the REM state were highly active during REM sleep. More interesting is what *else* he found.

The activation synthesis theory would have predicted that the brainstem activation of REM should globally activate the entire forebrain—thereby generating the random sensory, motor, emotional, memory, and thought images that comprise the supposed

[9]Other investigators have carried out similar studies, and all have produced findings compatible with Braun's. Due to technological constraints, there have, as yet, been no functional-imaging studies of the brain during sleep onset, or the late morning, when dreaming is dissociated from the REM state. However, these constraints will soon be removed when fMRI technology is applied to dreaming sleep.





"froth" of dreams. This is not what Braun found. Instead, he observed that only highly specific parts of the forebrain were activated during REM dreaming, while other parts were completely inactive. This is evidence of a striking pattern of dissociation between the levels of activation of various parts of the forebrain during sleep, suggesting that dreams are constructed by highly specific forebrain mechanisms. Moreover, the parts of the forebrain that Braun found were most active during dreaming were precisely the parts that obliterated or otherwise altered dreaming when they were damaged by brain lesions—and, conversely, the least active parts were the parts that had no effect on dreaming when damaged (Solms, 1997a). Braun therefore observed the very same pattern of dissociation that the lesion studies had found: the parts of the forebrain involved in the construction of dreams are the entire *limbic system* (including all the "limbic" components of the frontal and temporal lobes but excluding their "higher cognitive" components) as well as most of the *visual system* (excluding visual "projection" cortex). This implies, among other things, that the brain mechanisms of dreams are the same as those for the basic emotions discussed in chapter 4.

## THE DREAMING BRAIN AND THE EMOTIONAL BRAIN

Let us quickly review the basic-emotion command systems. There is the SEEKING system, which runs from the transitional area between brainstem and forebrain to the limbic components of the frontal and temporal lobes (Figure 6.3). It is a nonspecific motivational system engaged in looking for something to satisfy needs. The SEEKING system is linked to the pleasure/lust subsystem, involving nearby basal forebrain nuclei—especially the nucleus accumbens. The RAGE system involves the amygdala (in the limbic temporal lobe) and the hypothalamus and upper

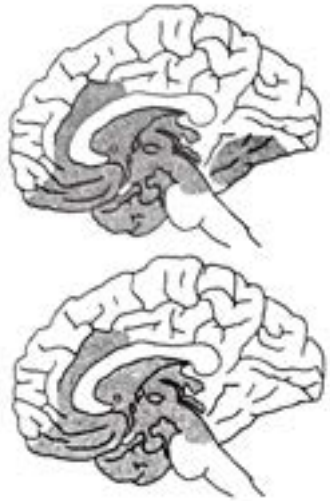


FIGURE 6.3
*Top:* The dreaming brain. *Bottom:* The emotional brain

brainstem structures. The FEAR system follows a very similar course. The PANIC system courses down from the anterior cingulate gyrus (in the limbic frontal lobe) to the same upper-brainstem structures. All of these emotional systems (together with the hippocampus, which subserves episodic memory, and parts of the visual system) are highly active during REM dreaming. But which of them provides the "primary driving force" of dreaming?

## THE PRIMARY DRIVING FORCE BEHIND DREAMING, REVISITED

We said earlier that a certain degree of *arousal* was a necessary precondition for dreaming. We also said that two *forebrain* structures are essential for the generation of dreams (i.e., the





occipito-temporo-parietal junction and the limbic frontal white matter). One of these two regions, we said, therefore probably contains the "primary driving force" behind dreaming.

While arousal is a *necessary* precondition for dreaming, it is not a *sufficient* condition to produce dreaming. We know this to be the case from the observation that patients with damage in the occipito-temporo-parietal junction or the limbic frontal white matter cannot dream, no matter how aroused they may become during sleep (even in the REM state). The necessary and sufficient conditions for dreaming are (1) forebrain arousal and (2) integrity of the occipito-temporo-parietal junction and limbic frontal white matter. So, which of the latter two structures provides the primary driving force? One way of addressing this question is to consider what else these latter two structures are known to do.

The occipito-temporo-parietal junction is heavily implicated in the generation of visuospatial imagery (see Kosslyn, 1994), and it is therefore no surprise that it should be implicated in dreaming—which is, after all, a special type of visuospatial imagery. The limbic frontal white matter, on the other hand, has no known function that is obviously implicated in dreaming, though a link to it may lie in a formerly common surgical procedure.

## FRONTAL LOBOTOMY AND DREAMING

From the 1940s until the 1960s the dramatic procedure of frontal lobotomy (surgical disconnection of the prefrontal lobes) was performed on thousands of patients for the treatment of serious mental illness, especially schizophrenia.[10] In the early

[10]The procedure was developed in 1935 by Egas Moniz and Pedro Almeida Lima but only became widely used in the late 1940s. The term "lobotomy" was later replaced by "leucotomy," when the surgical target was reduced from the whole lobe to only a part of the underlying white matter.

days, this procedure involved a near-total disconnection of the prefrontal lobes from the rest of the brain. This certainly seemed to improve psychotic symptoms—especially the so-called positive symptoms of schizophrenia, such as delusions and hallucinations—but it also produced a range of debilitating side-effects. The most commonly reported adverse effects were inertia and apathy, intellectual decline, personality change, and postoperative epilepsy. The patients who underwent these operations lost not only their psychotic symptoms but also a great deal of what it means to be human.

For these reasons, some of the surgeons involved modified the procedure. They developed a more limited operation that damaged a far smaller region of the brain, with the intention of achieving the same therapeutic benefit but without the side-effects. Several different approaches involving different parts of the frontal lobe were attempted. They finally settled on the white matter underlying the ventromesial quadrant of the frontal lobes (for a review see Walsh, 1985, pp. 158–168). This modified procedure was called *ventromesial leucotomy* and involved using a custom-designed surgical "leucotome" to create bilateral lesions beneath the ventromesial surfaces of the frontal lobes (Figure 6.4).

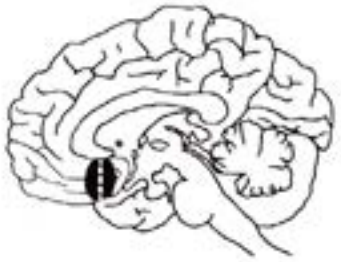


FIGURE 6.4
Modified frontal leucotomy





The area targeted by this modified procedure is exactly the one that the lesion studies mentioned above discovered was essential for the preservation of dreaming. In fact, we should say *re*discovered, for, incredible as it may seem, the practitioners of frontal leucotomy had already observed in the 1950s that the operation resulted in a cessation of dreaming in the vast majority of patients (for a review see Solms, 1997a, pp. 45–53). Psychiatrists knew this long ago, but, after the operation fell out of use, the knowledge was never transferred to the neuroscience literature. One psychiatrist even went so far as to observe that preservation of dreaming after the operation was a poor prognostic sign—continued dreaming seemed to indicate that the psychosis had not been successfully treated (Schindler, 1953). So, we may conclude that whatever it is that generates positive psychotic symptoms might well be the "primary driving force" behind dreams. As already noted, many psychiatrists have observed that dreams and psychotic illness must somewhere share a common mechanism.

## DRUGS AND DREAMING

There are a number of reasons why the psychosurgical treatment of schizophrenia fell out of favor, including ethical considerations. However, it is widely accepted that the most important reason for the shift was the development of *pharmacological agents* that were just as successful at controlling positive psychotic symptoms (if not more so), but with fewer side-effects. These drugs are the "major tranquilizers," also referred to as "neuroleptics" or "antipsychotics." Psychiatrists still employ versions of these drugs today for the treatment of schizophrenia. All of these agents have one core feature in common—they block *dopamine* transmission, and mesocortical–mesolimbic dopamine

transmission in particular (see chapter 1; for reviews see Lickey & Gordon, 1997, or Snyder, 1999). The mesocortical–mesolimbic pathways course through exactly the area of white matter that was targeted in ventromesial leucotomy (see Figure 4.3). For this reason, some neurobiologists have irreverently remarked that antipsychotic drugs function as "chemical leucotomies" (Panksepp, 1985, p. 273).

In chapter 4 we pointed out that one of the basic-emotion command systems is embedded in precisely these same pathways. This is the dopaminergic SEEKING system. Antipsychotic medications therefore block activity in this system, just as the old ventromesial leucotomy procedure did.[11] This blocking treats the positive symptoms of schizophrenia because, for reasons not well understood, overactivation of the SEEKING system seems to generate the positive symptoms of schizophrenia. This association is demonstrated, among other things, by the fact that pharmacological *stimulation* of this system can artificially produce psychotic symptoms in psychiatrically normal subjects. *Cocaine* and the *amphetamines* are two other classes of pharmacological agent that act on this dopamine system. Lower doses of both of these drugs produce a great boost of energy and increased interest in objects in the world. This is consistent with increased activation of the SEEKING system. In higher doses, a "stimulant psychosis" (Snyder, 1999, pp. 138–140) is initiated. Long before dosage levels produce psychosis, however, users develop the feeling that some events in the world have a "special significance" for them, and they exhibit a degree of suspiciousness about the behavior of others. In the more extreme state, patients almost invariably become paranoid, and they sometimes suffer auditory hallucinations. Such stimulant psychoses can be rapidly and effectively treated by the administration of the antipsychotic medications usually given to schizophrenics.

[11] The system is not typically described by psychiatrists as the SEEKING system—this term is employed only by those working on the neurobiology of emotion. However, both literatures know this set of pathways as the mesocortical–mesolimbic ascending dopamine system. Psychiatrists often describe this set of pathways as the D2 (or 2nd dopamine) system. Thus, different disciplines have developed separate terminologies to describe the same neuroanatomical and neurochemical systems.





The same thing can happen with the administration of dopamine agonists (stimulants) in the treatment of Parkinson's disease (the drug *L-dopa*, for example, is notorious for inducing psychotic symptoms). On this basis, Ernest Hartmann conducted a study that might be considered a direct test of the hypothesis that the mesocortical–mesolimbic dopamine (SEEKING) system is the "primary driving force" behind dreams (Hartmann et al., 1980). He administered either L-dopa or a placebo to neurologically and psychiatrically normal subjects, shortly after the first REM period. The effect was immediate and dramatic. The subjects who received the L-dopa experienced a massive increase in the frequency, vivacity, emotional intensity, and bizarreness of dreaming. The frequency, density, and length of their REM periods was, by contrast, completely unchanged. This provides further evidence for the dissociation between dreaming and REM sleep discussed above and suggests that the dopaminergic SEEKING system might well be the "primary driving force" we have been looking for.[12]

In summary, when the SEEKING system is damaged, patients lose interest in objects in the world, dreaming ceases, and positive psychotic symptoms (hallucinations and delusions) decrease. Conversely, when the system is stimulated, energy levels increase, dreaming increases, and psychosis may ensue. There

[12] This conclusion is controversial and is still hotly contested by Hobson and his school. See Pace-Schott et al. (in press) for an overview of all the arguments for and against this view.

is therefore a clear series of links between dreaming, psychosis, and the operation of the SEEKING system.[13] In Hobson's original argument against the Freudian dream theory, he said: "these facts completely eliminate any possible contribution of ideas (or their neural substrate) to the primary driving force of the dream process," and he argued that the real driving force behind dreams was "motivationally neutral" (Hobson & McCarley, 1977, p. 1338). In the light of the present-day neuroscientific evidence, it seems quite inappropriate to claim that dreams are not caused by "ideas" and that they are instigated by a "motivationally neutral" process. On the contrary, dreaming and motivated ideas (akin, perhaps, to Freudian "wishes") appear to be inextricably interlinked.

## VISUAL AREAS INVOLVED IN DREAMING

We have said that there is a second forebrain area that appears to be critical for dreaming, but it seems less likely that this area is the primary *driving force* behind dreams. The precise role that the occipito-temporo-parietal junction plays in the dream process is not entirely clear. It may well be that lesions to these sites produce loss of dreaming because of the role of these sites in mental imagery. If the patient loses the ability to generate a mental image, then inability to dream seems a logical consequence. If this argument is valid, then the effects of this second

[13] In this context, it is of some interest that Freud (1924b [1923], 1940a [1938]) believed that psychotic states resulted from an overwhelming of the ego by the libidinal (appetitive) drives (i.e., by the system that motivates our interest in objects in the world). Freud's position is therefore quite consistent with the fact that some aspects of psychosis (no less than dreams) appear to flow from an overactivation of the SEEKING system. A full discussion of this interesting possibility is beyond the scope of this book.





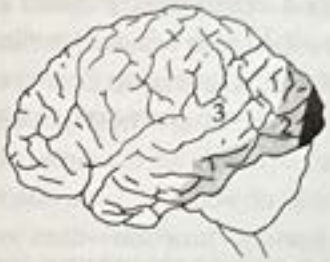



FIGURE 6.5
The three visual zones

lesion site are of less theoretical interest than is the important issue of the role of motivation in dreams.

A more significant finding is the *isolated loss* of visual dream imagery (or *aspects* of visual dream imagery, such as color or movement) after damage to the brain's visual areas. This suggests something about the "direction of flow" of information in dreams. The visual regions of the brain can be regarded as involving three hierarchically organized zones (see Figure 6.5):

1. The first is an area more or less directly connected to the retina, known as the primary visual cortex, lying at the back of the occipital lobes. This region is the "input" end of the system.
2. Next to this zone lies the "middle" part of the system, which is dedicated to a range of specialized visual-processing tasks. Color and motion processing, object recognition, and so on appear to take place here.
3. Finally, the zone in front of this is the highest level of the visual system, running the most abstract aspects of visual processing, which are also dependent on several other sensory modalities. This zone is involved in arithmetic, writing, constructional operations, and spatial attention. It represents the "output" end of the normal perceptual system.

Damage to Zone 1—the primary visual area—causes cortical blindness. Visual experience ceases, because the "input" end of the system is disrupted. Damage to Zone 2 causes more complex disorders of visual processing. These patients lose the ability to perceive color or movement, for example, or they lose the ability to recognize specific objects or faces. Damage to Zone 3—the occipito-temporo-parietal junction—does not affect visual perception *per se* but, rather, causes more abstract disorders that transcend concrete perception: acalculia (inability to calculate), agraphia (inability to write), constructional apraxia (inability to construct), and hemispatial neglect (inability to attend to one side of space).

In *dreaming*, however, this hierarchy seems to be reversed. Damage to the primary visual cortex, Zone 1, has (perhaps surprisingly) no effect on dreaming at all. Although these patients cannot see in waking life, they see perfectly well in their dreams. It seems that this aspect of the system is no longer the "input" end. Damage to the middle zone of the system, Zone 2, causes exactly the same deficits in dreams as it does in waking perception: these patients continue to dream in various sense modalities, especially somatosensory and auditory, but their visual dream imagery is deficient in specific respects. For example, they no longer dream in color, or they dream in static images (loss of visual movement), or they cannot recognize any of the faces in their dreams. Damage to the higher zone in the occipito-temporo-parietal junction, Zone 3, on the other hand, produces complete loss of dreaming. Recall that, in normal waking life, damage here does not affect perception *per se*, only higher visual cognition. That is, damage to the part of the system that is the "output" end in normal waking life seems to function as the "input" end during dreaming.

This "inverted" relationship has been proposed in the cognitive neuropsychological literature as a suitable model for the





organization of the imagery system in waking cognition.[14] It seems to apply equally well to the case of dreaming. Freud called this mode of organization "regression" and wrote that "in dreams the fabric of thought is resolved into its raw material" (Freud, 1900a, p. 543).

## A SUMMARY OF THE NEUROBIOLOGY OF DREAMS

The following description of the mechanism of dreaming integrates the evidence reviewed above. Some speculation is required, however, to fill in the gaps, although this will no doubt be less of a problem in the near future, as research in this exciting area is proceeding apace.

First and foremost, dreaming depends on a critical degree of activation of the basic mechanisms of core consciousness. If this inner source of consciousness is not aroused, then you cannot have a dream. It does not seem to matter what the trigger of the arousal is. Frequently, it is simply the residue of waking thoughts, as you drift off to sleep. The most reliable trigger is the REM state, which provides a sustained source of activation at regular intervals throughout sleep. As you begin to wake up, hormonal mechanisms gradually activate the forebrain. All these triggers activate (or "prime") consciousness, which is a necessary precondition for dreaming, but is not dreaming itself.

The activation of the motivationally charged SEEKING system, which drives our appetitive interest in the object world, appears to begin the dream process proper. It is probably accurate to say that *an arousal stimulus only triggers dreaming proper if it attracts appetitive interest*. When this happens, the

[14] Kosslyn (1994) calls it "backward projection." See also Zeki (1993).

subjective feeling is something along the lines of: "What could this be? I want to know more about this."

Activation of the SEEKING system during sleep is commonly, but not exclusively, triggered by the REM state. A thought process occurring during any stage of sleep can presumably also activate the SEEKING system. This thought process could be linked to an episodic memory from the previous day, or even just to a feeling. If the memory or feeling activates the interest of the SEEKING system, this would be enough to begin the dream process. This explains the observation that although *most* dreams occur at sleep onset, or during REM sleep, or just before waking, it *is possible* to have a dream at almost any time during the night—even during deep ("Stage 4") sleep. Recall, in this context, that these NREM dreams are indistinguishable from REM dreams.

When you sleep, you cannot go about exploring or seeking what you are motivationally interested in. This sort of behavior is not compatible with sleep, and it is probably for this reason that we dream. It seems a reasonable hypothesis that *the dream occurs instead of a motivated action*. That is, instead of doing something in the real world, you have a dream. The frontal lobes (the "action" end of the brain; see chapter 1) is normally a central "scene of action" in waking cognitive activity. However, this system is dormant (i.e., inhibited or underactivated) during dreaming sleep. The "scene of action" of cognitive activity therefore shifts to the posterior forebrain, with activation of the parietal, temporal, and occipital lobes. This is experienced as imaginative perception and cognition—which, however, differs from waking thought in that it is unconstrained by frontal inhibition. In the absence of the ability of the frontal lobes to program, regulate, and verify our cognition, affect, and perception, subjective experience becomes bizarre, delusional, and hallucinated.





In our dreams, the focus of motivated cognition is therefore removed from our goal-directed action systems and shifts toward the perceptual systems—especially the visuospatial component. The functional anatomy of dreaming is therefore almost identical to that of schizophrenic psychosis, as revealed by functional-imaging studies. One substantial difference is that in schizophrenia it is primarily the *audioverbal* component of the perceptual systems that is activated, rather than the visuospatial. The basis of this difference is unknown.

## DREAMS AS THE GUARDIANS OF SLEEP

In addition to claiming that dreams were motivated by wishes, Freud famously argued that they "serve the purpose of prolonging sleep instead of waking up. Dreams are the guardians of sleep, and not its disturbers" (Freud, 1900a, p. 223). This means that the sleeper is "protected" from the disturbing influence of motivational urges that emerge during sleep. This hypothesis seems reasonable in the light of all we have learned above. But reasonable hypotheses are frequently proved wrong, and therefore untested hypotheses have limited value in science. One of the criticisms most frequently thrown at psychoanalysis is that its core hypotheses are untestable. It is here that the advantages of interdisciplinary collaboration become apparent: now that we know that certain unfortunate individuals with damage to specific parts of their brains are unable to dream, the sleep-protection hypothesis can easily be tested. Nondreaming neurological patients should experience sleep that is more disturbed than (say) that of neurological patients with equivalent degrees of brain damage in whom dreaming persists.

This critical test will soon be performed. It has only been possible so far to gather some preliminary data on the question,

by simply asking patients who do not dream whether the quality of their sleep is unchanged, is better, or is worse than before their neurological problems began. The data collected so far (on a sample of 361 patients) supports Freud's sleep-protection theory at the required levels of statistical significance (Solms, 1995, p. 63). However, a sleep-laboratory study (instead of a bedside assessment) is required before this issue can be addressed with confidence.

## DREAM CENSORSHIP

People who misunderstand Freud's theory of dream censorship (Braun, 1999; Hobson, 1999) mistakenly believe that the theory predicts that the (inhibitory) frontal lobes should be more rather than less active during dreams than in waking life (which is what they are). But Freud's dream theory states only that the "censorship" function of the executive ego is *not completely inactive* during sleep, not that it is *more active* during sleep than in waking life. In fact, according to Freud's theory, it is the *weakened* state of the inhibitory systems of the mind that makes our instinctual drives so unruly during sleep, and it is this that causes us to think and do in our dreams things that would be inconceivable in our waking lives. The theory therefore predicts exactly what functional brain imaging reveals—namely, that the inhibitory systems of the brain are *relatively* but not *completely* inactive during dreaming sleep (see Yu, 2000). However, this is far from proving Freud's censorship theory correct.

Freud's theory was designed to explain the differences between two components of the dream process. On the one hand, the manifest (or "explicit") content of the dream is often illogical and bizarre. On the other, the patient's associations to the individual elements of the dream suggest that the underlying, latent (or "implicit") content of the dream involved a motivational





impulse that was not at all illogical or bizarre. In this regard, the neuroscientific evidence is compatible with Freud's model. Freud went on to question why the two levels of dream content differed so dramatically. His answer, of course, was the censorship. Here he may have been wrong. The apparent illogicality and bizarreness of dreams may be due to the inherently "regressive" nature of the dream process. The mere fact that the system is forced to function in the way that it does, where the executive systems of the frontal lobes cannot program, regulate, and verify the output of the posterior forebrain, may well produce the difference between the latent and manifest contents—with no need to introduce the additional function of censorship. The symbolic transformations to which Freud drew attention might, therefore, simply be the product of unconstrained parietal-lobe mechanisms operating in reverse, "resolving the fabric of thought into its raw material," as it were (Freud, 1900a, p. 543).

However, most observers would agree that the neuroscientific evidence does not yet have a decisive bearing on these important questions. The available evidence cannot tell us whether the distortions that appear to be introduced between latent and manifest dream thoughts are tendentiously motivated or not. For now, we shall have to rely on purely psychological techniques to assess the validity of this aspect of Freud's dream theory. Although multiple, converging lines of evidence are desirable in science, certain types of psychological question cannot be pertinently answered by neuroscientific methods.

In conclusion, modern neuroscience has come to understand a great deal about the biological basis of dreams, particularly the brain regions and attendant psychological processes that seem to be most central to the dreaming state. This knowledge is broadly consistent with Freud's psychoanalytic theory of dreams—although it would be inappropriate to say that his theory has been directly *proven*. The neural mechanisms of dreaming appear to overlap in several important respects with

the neural mechanisms of certain core features of psychosis, especially the positive symptoms such as hallucinations. This confirms a long-standing hunch harbored by Freud (and many others) to the effect that understanding dreams might provide us with a key to understanding mental illness in general. Dreams truly do appear to be "the insanity of the normal man."



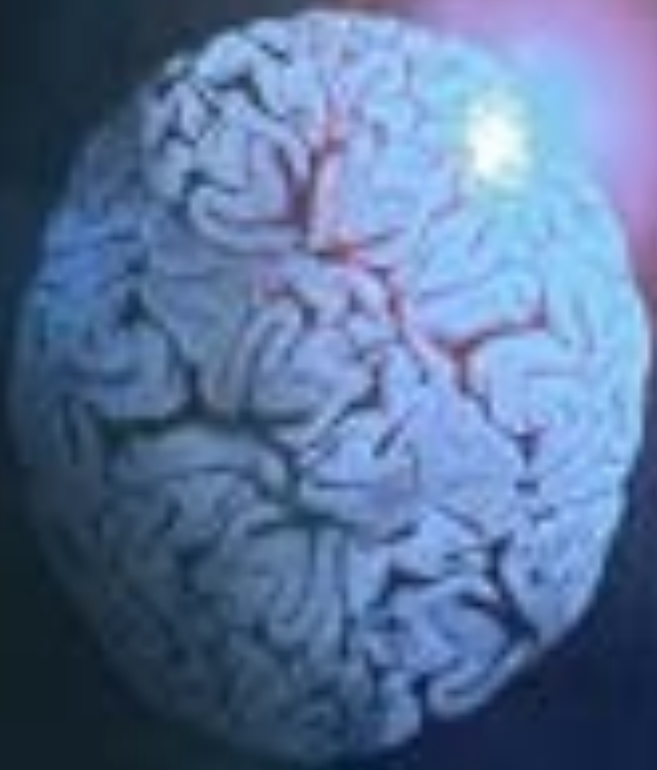
CHAPTER 7

# GENETIC AND ENVIRONMENTAL INFLUENCES ON MENTAL DEVELOPMENT

The overwhelmingly vast topic of nature–nurture influences on the brain has the potential to include everything that neuroscience knows about the developmental sequence, in every psychological domain. The previous few chapters covered only *individual* mental functions, and they focused primarily on their organization in the mature, adult brain. This chapter broadens our focus considerably. We therefore want to emphasize at the outset that our goals in this chapter are very limited: to introduce some of the basic principles about genes and their workings and to discuss their implications for the broader theme of this book. The best way we could think of doing this was to begin by summarizing the main principles and then illustrating these with reference to *a single aspect of mental life*—thereby reverting to the structure of the previous four chapters. We decided to use *sexual difference* as our example, thereby enabling us to cover from a neuroscientific point of view (at least in part) another topic that has been a traditional stomping ground of psychoanalysis.

# THE BRAIN AND THE INNER WORLD

An introduction to the neuroscience of subjective experience



MARK SOLMS OLIVER TURNBULL

FOREWORD BY OLIVER SACKS