3

Experiments of Nature

Perhaps it is simply the ghost of Freud that is getting in the way.
—Allen Braun

GROWING UP IN A TINY VILLAGE in a remote corner of Africa that is now part of Namibia, Mark Solms and his brother, Lee, were inseparable, in part because they were among the only English-speaking children in town. They had settled in this former German colony in southwest Africa because it was home to a rich diamond deposit and their father was an executive with the diamond giant DeBeers. But when Lee slipped off a rooftop he'd scrambled onto while playing and suffered a brain injury at the age of six, not only was he himself forever changed, but he indirectly sent his younger brother Mark on a quest that would shape his life.

"In retrospect, I believe my brother's accident led me to study neuroscience in the particular way I did, because I wanted to understand how people are organized by their brain function. Not just the brain's cognitive aspects, such as how we learn to speak or read, but rather how the brain gives us our personality and sense of self," says Solms. "How can it be that a person comes from this lump of tissue, and how

could my brother as a person be changed entirely because of damage to portions of that lump of tissue?" Studying exactly how damage to specific areas of the brain affects behavior was in fact what led Mark Solms to discoveries that uncovered surprising and vital new pieces of the dreaming puzzle.

Though he's now based in South Africa, Solms travels regularly to New York in connection with his work at the New York Psychoanalytic Center, which encourages the exchange of information and research among neuroscientists and psychoanalysts. Over breakfast in an Upper East Side hotel dining room with a decidedly British ambience, Solms's earnest passion for his work is apparent as he explains how he came to focus on dreaming as a window into the workings of the mind.

As a student of neuroscience in Johannesburg in the early 1980s, Solms found that he was being taught much about superficial brain mechanisms but next to nothing about the larger questions that interested him. Nothing, that is, until he was persuaded by friends to take a seminar on Freudian dream theory taught, not by a scientist, but by a professor of comparative literature. The focus of the seminar was a manuscript Freud had written in the late 1800s speculating about what brain mechanisms might underlie the dreaming process. "Most of my friends were in the arts, history, and philosophy and I was the odd man out, studying the brain, so they were excited to find a seminar for me in their world," he recalls. "I was fascinated listening to these speculations about the brain functions underlying dreaming—wishful thoughts, coping with strong emotions—all the meaty stuff about lived lives that I wasn't hearing about in neuroscience."

As a result, for his doctoral dissertation, Solms decided to update Freud, whose speculations about the neurological basis of dreaming relied on a body of knowledge about how the brain works that had changed significantly since his theories on the subject were published in 1895 as "Project for a Scientific Psychology." For starters, Solms closely read Hobson's studies showing that signals from the brainstem during REM sleep triggered dreaming and that the more highly evolved forebrain simply did its best to make sense of the randomly generated chaotic signals it was receiving by producing hallucinatory dream imagery. Though Solms says he thought Hobson's 1977 paper

was "unnecessarily destructive, stacking the cards in ways that made the Freudian effort look more ridiculous than it really was," he maintains that he never set out to disprove Hobson's model. "Hobson's view was *the* view of how the brain generated dreams. It dominated the field and I was just a student, so I had no reason to doubt its fundamentals. All I intended to do was elucidate in greater detail what the forebrain actually does with those dream-activating impulses coming from the brainstem," Solms says.

Solms—who already was on his way to becoming a Freudian scholar and leader in the psychoanalytic field—suspected that dreams were created in a more complex way than Hobson's theory indicated. If Solms could pinpoint exactly how the more highly evolved parts of the brain participated in creating dreams, he might be able to prove that Freud was correct in proposing that dreams draw on our memories from early childhood to the present and symbolically express the strong emotions that drive our inner life.

The young researcher began his quest by doing classic neuroanatomical detective work. Working in the neurosurgery department, first at a hospital in Johannesburg and then in London, Solms had an opportunity to study every patient who had any type of brain damage (known as lesions), whether due to stroke, tumors, or traumatic injuries like his brother's. He questioned each patient about what effect, if any, the illness or injury had on his or her dreaming. His method of inquiry paid off almost immediately: one of the first patients he examined claimed to have ceased dreaming entirely. That patient had a lesion in the parietal lobe, which is the portion of the brain that combines various forms of sensory information to create our sense of spatial orientation and mental imagery. It's what allows us to conjure up a scene of relaxing on a South Pacific beach when we're daydreaming, to envision the route we'll take to stop by the bank, or to imagine how we'll reconfigure the counter space to remodel a kitchen.

As he came across increasing numbers of patients with damage to this region—all of whom claimed to have stopped dreaming as a result—Solms searched the medical literature and found that there had been previous scattered case reports all pointing to that same conclusion. This all made sense to Solms, and there was nothing in it to

contradict Hobson's theory: EEG recordings showed that the patients still experienced REM sleep, so Solms assumed that the signals from the brainstem that switched on REM were being transmitted as usual but dreaming didn't occur, simply because the picture-making machinery that received them in the forebrain was broken.

But Solms was truly startled by two other patterns that emerged among the patients he encountered. While most people with injuries to the brainstem do not retain sufficient consciousness to be able to report whether or not they dream, he had a few such patients who said they still continued to dream—something that should have been impossible under Hobson's theory about how dreams were triggered. "I started to worry about why I was seeing all of these patients with parietal lesions who had stopped dreaming but none who'd stopped as a result of damage to the brainstem," he says. So Solms again searched the medical literature, assuming that even if he hadn't found such patients himself, surely he'd find other published reports of them. He couldn't find a single one. "I was absolutely dumbfounded," Solms recalls. "If you're going to claim a certain brain structure performs a certain function, then you have to be able to show that damage to that structure leads to loss of that function."

At that point, everything about the prevailing theory on dreaming became open to question. As Solms reviewed earlier work, he read the studies by David Foulkes and Gerald Vogel showing that subjects reported dreams in the majority of awakenings at sleep onset before the first REM period of the night, as well as John Antrobus's research showing vivid dream reports during non-REM at the other end of the cycle, in the morning before awakening. "When I saw that the connection between REM sleep and dreaming wasn't watertight after all, I started to think that maybe the whole process was driven by the forebrain rather than the brainstem." He speculated that REM sleep indeed was switched on when the brainstem was flooded with the neurotransmitter acetylcholine, as Hobson had shown, but that dreaming itself was an entirely separate process that could occur only when specific mechanisms in the higher portions of the brain were also activated.

If this turnabout in thinking proved true, it would establish a physiological basis for viewing dreaming as a mental process driven by

more complex brain structures, rather than simply a chaotic response to the brainstem's random electrical signals. Not only could the age-old fascination with dream content be justified scientifically, but in Solms's view, this new direction for research would prove that the obituaries that had been written for Freudian dream theory were premature. If dreaming and REM sleep were two distinct processes, each with its own on/off switches, then it was possible that dreams could be driven by the motivational regions of the brain, in keeping with Freud's notion that dreams expressed our deepest wishes and fears. And if regions of the forebrain that are strongly linked to memory formation also proved to be involved in dream creation, that would support Freud's suggestion that the characters, settings, and actions in dreams were drawn from the vast stores of the dreamer's personal experiences, including early childhood ones that were outside the realm of conscious recall.

Solms's speculation that dreaming and REM sleep were separate processes controlled by different brain mechanisms soon waxed to conviction when he saw a second unexpected pattern. He discovered reports of dreaming ceasing altogether in yet another group of patients—those with damage to both the right and left sides of a section deep in the middle of the brain's frontal lobe. The tissue in this area is called white matter because it is rich in neural pathways that are coated in a glistening white fatty sheath that enables neuronal signals to travel quickly over long distances. One patient suffered damage in this area from a knife wound in the eye that penetrated both sides of the brain's midsection, while others had lesions in this area due to a specific type of tumor called a butterfly glioma that projects down into the white matter like a pair of wings. All told, Solms had only nine of these patients, which he feared wasn't enough to support any valid scientific conclusions. Damage to this well-protected area due to injury or natural causes was uncommon, but when Solms decided to go back through decades of medical reports to see if other such cases had been described, he struck scientific gold.

Throughout the 1950s and into the 1960s, one of the treatments for schizophrenics and other delusional patients was a modified version of prefrontal lobotomy, the surgical procedure used to tame McMurphy, the hero in Ken Kesey's *One Flew over the Cuckoo's Nest.*

In the gentler version, known as a prefrontal leukotomy, surgeons found that they could cure their patients' hallucinations by cutting into exactly that area of the brain that had been injured in the nine patients who reported to Solms that they no longer dreamed. So although nature itself doesn't often create damage to that crucial section of white matter, surgeons had done so in vast numbers of people for years, and Solms found that an overwhelming majority of them said they had stopped dreaming. "It was there in black and white to see for decades, and no one noticed," he says.

Prefrontal leukotomies also provided information about the function of the area of white matter, known as the ventromesial forebrain, that lies in the lower and middle portions of the brain's frontal lobes. Both the people who'd had the procedure and the group of nine patients with injuries whom Solms had found experienced similar behavior changes afterward. They became completely apathetic, displaying little spontaneity or curiosity about the world. This wasn't surprising, because the same brain area—sometimes called the seeking system—had been well studied in animals and was known to shift into high gear when an animal was motivated to meet its basic needs or seek pleasure, from foraging for food to pursuing a mate. In humans it was also the area that lights up in brain-imaging studies when an addict is merely shown slides of drug paraphernalia or when a smoker is desperately scavenging for a cigarette. In short, the ventromesial forebrain is the brain's "I want it" system, and Solms was convinced that it—not the primitive brainstem—was the crucial structure needed to generate dreams.

But what was it about this particular brain region that could make it so crucial to dreaming? Solms agreed with Hobson that acetylcholine was the key to turning on REM sleep, but he hypothesized that it was a different brain chemical—dopamine—that switched on dreaming itself. Dopamine levels surge when the brain's reward system is activated in waking by activities an individual finds exciting or pleasurable—from taking drugs such as cocaine or alcohol, to sex, to gambling or thrill-inducing activities like bungee jumping. A plunge in dopamine transmission is associated with a feeling of complete boredom. Though the white matter deep in this midsection of the brain is rich with fibers that transmit both of those neuromodulators,

Solms suspected that dopamine was the one that really triggered dreaming, because medications used to treat schizophrenia worked to eliminate hallucinations in a simple way: they blocked dopamine transmission in that portion of the brain.

If his suspicion were correct, an increase in dopamine transmission should somehow intensify dreaming. In fact, dopamine's ability to do just that had been demonstrated in a 1980 experiment by Tufts University psychiatrist and dream researcher Ernest Hartmann. Hartmann found that giving test subjects drugs that increased dopamine transmission in the brain greatly jazzed up their dreams. Though subjects who had been given the drug had the same number and duration of REM cycles as subjects who were given placebo, the people who'd had the drug reported dreams that were substantially longer, more bizarre, vivid, and emotionally intense than those of the control subjects.

Solms became more convinced that the brainstem alone did not trigger dreaming when he encountered another fascinating group of brain lesion patients: those who couldn't stop dreaming, even when they were awake. These patients suffered damage to a specific group of cells in the base of the forebrain that played a crucial role in Hobson's view of how dreams are created. Hobson contended that the brainstem's dream-generating signals projected onto these cells (called basal forebrain nuclei) and that they in turn activated the forebrain structures needed to create visual images and the other stuff of which dreams are made. If Hobson's theory were correct, then damaging those cells should result in a loss of dreaming, but Solms found just the opposite was true. Damage to those cells and closely related brain structures instead created patients whose nighttime dreams were unusually vivid and frequent and who had difficulty distinguishing between dreams and waking experience during the day. The reality-testing system that goes off-line when we dream—allowing us to fully believe that we're back at the high school prom wearing nothing but our underwear—normally comes back online when we awaken. Not so for patients with damage to these clusters of cells.

For instance, one of Solms' patients was a thirty-two-year-old man who'd suffered a head injury during an auto accident that damaged his basal forebrain nuclei. Not only did his dreams become more

vivid, but frequently he would wake up from a frightening dream only to find that the dream seemed to continue while he was awake. He said the experience was "terrifyingly real," ending only when his wife would awaken and convince him that his visions of ghosts or small animals crawling around the room were merely hallucinations.

Another of Solms's patients was a forty-four-year-old widow who suffered damage to this area due to an aneurysm, which led her to have much more vivid dreaming at night, as well as daytime experiences in which she says her "thoughts just turned into reality." She was lying in bed one morning thinking of her late husband, when he suddenly appeared in the room with her. After they'd spoken together for a while, he helped her to bathe. Then, in an instant, she realized she was still in bed and totally alone in the room. It was difficult to believe that nothing she'd imagined had actually happened. The woman explained that she wasn't asleep and hadn't been dreaming then or during similar daytime dreamlike experiences she'd had: "It wasn't just seeing things. It was as if it was real—as if it was really happening—and many times I couldn't make out what did happen from what didn't."

Commenting on her case, Solms says, "It's the same feeling we all get occasionally when we awaken from a particularly vivid dream and it takes a while to realize that the events occurred only in the dream. Instead of just thinking wouldn't it be nice if my husband were here, her thoughts turned into what she perceived as a real experience. That is, in essence, what dreaming is."

Looking at the sum of the evidence, Solms now firmly believed he had the makings of a new theory of dream creation. The fact that most dreams occur during REM sleep was actually a misleading coincidence, he argued. REM sleep and dreaming were in fact two separate processes with different off/on switches and probably different biological purposes. As earlier studies by Antrobus, Foulkes, and others had shown, REM was the sleep phase most conducive to dreaming, but dreaming also occurred outside REM, especially when we were first falling asleep and again in the morning as our bodies were preparing to wake. What all three states had in common was an elevated state of brain activation, which was only the first step needed to create dreaming. "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," Solms says.

The high level of activation needed to dream occurred most frequently during REM, as demonstrated in studies by various researchers from the 1960s on in which 80 percent of awakenings during REM produced dream reports. But several studies also showed that dreams were reported in 5 to 20 percent of awakenings from non-REM sleep, and Solms argued that dreaming itself could not occur even during REM unless the elevated level of brain activation in turn switched on the seeking system in the forebrain. That system, driven by dopamine, would then activate the more complex structures needed to construct dream imagery and plot. As a physiological underpinning for Freud's theory that dreams have their roots in subconscious wishes, this dream-generating system was the perfect fit. "It was fascinating to see that the part of the brain that appeared to be crucial for switching dreams on is exactly the part of the brain that you would have predicted on the basis of Freud's most unlikely theory," concluded Solms.

ENTIRELY BY COINCIDENCE, just a few months after Solms's new theory and supporting evidence appeared in print in 1997, two American researchers using sophisticated brain-imaging technology to map the dreaming brain in action published their own groundbreaking findings, which provided the most detailed view yet of what happened in the brain as it moved from waking to dreaming consciousness and back again.

Tom Balkin was conducting sleep deprivation experiments in 1989 when he first met Allen R. Braun, a neurologist at the National Institutes of Health who specialized in Parkinson's disease and other movement disorders. At the time, Balkin was chief of the department of behavioral biology at the Walter Reed Army Institute of Research, and both he and Braun were fascinated by the many unanswered questions about the brain during sleep. When EEG recordings were the only way to view changing brain activity, scientists assumed that

the entire brain became charged up during REM, but Braun suspected that only selected areas were zooming into action and that discovering which areas those were would provide greater understanding of what the brain really was up to. "It seemed to me this was one last great mystery, but to solve it, you'd have to get a total picture of changes occurring simultaneously in all parts of the brain from sleep to waking," says Braun. So in 1991, by which time neuroimaging technology had advanced to provide the level of detail he and Balkin would need to achieve their goal, the two began collaborating on studies that yielded a series of fascinating three-dimensional portraits of the brain at work.

They used a technique called PET (positron-emission tomography) scanning, which measures blood flow to indicate which regions of the brain are most active at a specific point in time. The PET scan produces an image of the brain that can be displayed on a computer screen, with areas of greater and lesser activity coded in different shades of vibrant color. Over a period of two and a half years, Braun and Balkin met regularly in all-night sessions at the NIH lab in Bethesda, Maryland, to scan subjects before they went to sleep, during REM and non-REM sleep stages, and then again after awakening in the morning.

What they saw opened a new window on exactly what happens in specific regions of the brain during our nightly internal odysseys. As we move into the deepest stages of non-REM sleep, activity in nearly all parts of the brain decreases, but the areas that take the first and steepest plunge (activation levels drop by about 25 percent) are the prefrontal cortical regions that we use for the highest order of information processing, such as planning, logical thinking, and problem solving. "These areas are the first to fall asleep and the last to come back online," says Balkin.

Deactivation of these areas is accompanied by sharp drops in levels of serotonin and norepinephrine, which help us focus attention and solve problem when we're awake. Then a surge of the neuromodulator acetylcholine (which fosters free-wheeling associations) turns on REM sleep. As that happens, remarkable changes visible in the PET scan images occur, and Braun believes that they explain much of the phenomenology of dreaming. All of the areas that were geared down

during slow-wave sleep charge back up again except for one: that logical, reasoning portion of the prefrontal cortex that is humankind's latest evolutionary acquisition. Its inactivity would explain why time and space orientation often are jumbled and why we have no reality testing: we don't question the fact that our dead grandfather is dressed in knight's armor and driving a taxicab.

Because the portions of the brain that normally order our thinking are off-line, what we're experiencing as reality in a dream sometimes can be a hallucinatory world, much like what a schizophrenic experiences in waking consciousness. In fact, subsequent brain-imaging studies show that the functional anatomy of dreaming is almost identical to that of schizophrenic psychosis, with the major difference being that for dreamers, the visuospatial system is most highly charged, while for schizophrenics, the audioverbal system is activated. It's no wonder delusional patients often claim to hear voices that dictate their behavior.

Most surprising, though, Braun and Balkin's PET scans revealed that selected portions of the brain are much more active during REM than they are in waking consciousness. The primary visual cortex, which is our portal of visual information from the outside world, is shut down, which is why sleeping subjects whose eyes were taped open in early dream experiments never incorporated into their dreams any images of objects that were flashed before their eyes. But the visual association areas involved in creating mental images and recognizing faces are wildly active, above normal waking levels, making dream imagery richly visual. Braun and Balkin also saw increased activity in an area of the prefrontal cortex that would light up when waking subjects were creating a story based on a series of events from memory. Braun speculates that activation of this area in dreaming reflects the brain's attempt to assemble the visual images into a narrative form.

The area of the brain that allows us to put memories in sequential order and to temporarily store ongoing events in short-term or working memory remains off-line during REM. But the brain structures involved in long-term memory formation and retrieval are actually more active than when we're awake, he says, which creates ideal conditions for REM to play a critical role in long-term memory processing. "REM may provide a situation in which long-term memory traces

can be processed off-line, either consolidated or pruned, while the brain is not actively processing information generated during REM itself," Braun says.

This paradoxical situation in which long-term memory processing centers are firing wildly, while the areas needed for storing current experience (the dream itself) in working memory for later retrieval are not, helps explain why we can easily remember what we had for breakfast at 8 A.M. but not what we were dreaming at 4 A.M. Braun maintains that dream content actually is encoded in the brain, as demonstrated by the fact that we can spontaneously remember fragments of a dream if we see or feel something during the day that is associated with what we dreamed the night before. Our poor dream recall really reflects an impairment in our ability to retrieve that memory.

Perhaps most significant, Braun and Balkin found that the structures in the brain that light up when we are feeling strong emotions or craving the object of our desires also are more supercharged during REM than in waking. Operating full tilt is the limbic system, which is the brain's long-term emotional memory center. When we're dreaming, emotions appear to be behind the steering wheel while the brain's attention-directing, decision-making machinery is snoozing in the passenger seat. Similar results were seen in brain-imaging studies by Pierre Maquet and his research group at the Universite de Liege in Belgium. Maquet concluded that the patterns of activation in the amygdala (which generates the body's fight-or-flight response and other intense emotional reactions) and other cortical areas provided a biological basis for the processing of memory during REM, particularly emotionally influenced memories.

As for the question of what actually triggers the dreaming process, the PET scans could provide no clear answer. The pons area of the brainstem, the key to dream generation in Hobson's model, was significantly activated during REM, but so was the motivational area of the forebrain that played a crucial role in Solms's model.

Allan Hobson responded to the flood of new data from the PET scans by applauding the technological advances in brain imaging that made it possible to describe the activation pattern of the brain in REM. He acknowledged that the consistent results of the various

brain-imaging studies required a rethinking of his model, noting that "dream emotion may be a primary shaper of dream plots rather than playing the secondary role in dream plot instigation," as he had originally proposed. He suggested that although the focus of a dream's plot shifts from the dreamer feeling lost, to missing a train, to not having proper credentials or suitable clothing, they all satisfy the driving emotion—in this case, most likely anxiety. And he also observed that evidence was fast accumulating to indicate that both non-REM and REM sleep benefit learning and memory, a feature that had not been part of his model.

He seized upon the imaging studies' evidence that quite different mechanisms underlie waking and dreaming consciousness to support his quest for the Holy Grail: proof that brain and mind are one, that our state of consciousness is nothing more than a reflection of the particular mix of brain chemicals and neural connections that happen to be active at any given moment. "The initial loss of contact with the outside world at sleep onset, with its flurry of fleeting hypnagogic images, the deeply unconscious oblivion of sleep early in the night, and the gripping hallucinoid scenarios of late-night dreams, all have such strong and meaningful underpinnings in brain physiology as to make all but certain the idea that our conscious experience is the brain-mind's awareness of its own physiological states," he said.

He even came up with a new model explaining our periodically shifting states of consciousness, saying that just as it was no longer acceptable to equate REM sleep with dreaming, it was no longer possible to regard waking as a single state. The narrow categories into which consciousness had been categorized previously—REM sleep, non-REM sleep, and waking—weren't adequate to describe the many variations that humans actually experience, ranging from focused thought needed to perform mathematical calculations to waking hallucinations experienced by schizophrenics or people tripping on LSD. In Hobson's new model, there were three variables that determine our state of mind at any given point in time. The first was the overall activation level of the brain (based on EEG measures of brain waves). The second was the specific mix of neuromodulators that are predominant in any given state. And the third was whether the brain

is processing information generated in the outside world (as it would in alert, waking consciousness) or internally generated information (as it would when dreaming or meditating in silence with closed eyes).

As for Solms's studies, Hobson praised the contribution the neuropsychologist had made using "experiments of nature in the form of strokes" to correlate the locale of brain lesions with changes in the dreaming process and to shed light on how the forebrain participated in constructing dreams. He invited Solms to Harvard to present a paper to his research group. Impressed by Hobson and his colleagues, Solms in turn invited Hobson to appear with him to speak about dreaming at the New York Psychoanalytic Center. Shortly thereafter, Solms received a message from Hobson saying that he was happy to accept Solms's evidence from the brain lesion studies, but if Solms intended to use that research to try to support Freudian dream theories, "that's where we have to part company."

Says Solms, "Prior to that I'd been pleasantly surprised by how scientifically correct in his attitude he was. I don't know why he's got such a bee in his bonnet about psychoanalysis, but it is really like Lucifer to him: if you mention Freud, he gets out his cross. It's unfortunate because it creates a black spot in his vision." Critics contend, though, that Solms has his own pro-Freudian bee-in-the-bonnet, given that he is an active voice in the psychoanalytic community and has served as editor and translator for collections of Freud's complete works.

In any case, Solms was encouraged that Braun's work validated many of his findings. "If you look at the PET images, you see that when we're most likely to be dreaming, the parts of the brain that have to do with memory, visuospatial image generation, motivation, and all of the structures that have everything to do with the emotional life of a mammal are lit up like a Christmas tree. If you put that together with my lesion studies and had to guess what's going on here, you'd say here's a type of cognition that's intensely motivated and emotional. It has to do with memory and is not guided by the self-reflecting structures that normally give our behavior its rational and civilized veneer," he says.

While the new scientific evidence didn't prove Freud right, Solms maintains, it was at least compatible with many of Freud's ideas.

Thus ensued a persistent war of words between Hobson and Solms over Freud and their own conflicting theories of how dreams are triggered. It was in essence the culmination of the battle that had brewed for decades between neurophysiologists on one side of the divide and psychologists and psychiatrists on the other whose interest in dream content and dream analysis as a psychotherapy tool had come to be viewed as misguided by many. For decades, neuroscientists regarded psychotherapy as unscientific, while therapists viewed most neuroscience as simplistic because it excluded the psyche. Neuroscientists were interested in *how* we dream, while psychologists were preoccupied with *why* we dream.

Solms argued that since the new evidence was forcing Hobson to revise the dreaming model that he'd used for years to publicly demolish Freudian theory, he now should be willing to admit that Freud may have been at least partly right. Hobson had made adjustments to his theory to account for the more active role of complex brain structures in dreaming, but he still contended that neither Solms's data nor the brain-imaging studies could provide "the faintest modicum of support" for Freud's ideas that the meaning of dreams was disguised or censored or that dreaming provided some special access to unconscious motives via the technique of free association to bizarre dream material. Nor could he tolerate Solms's linking the role of the brain's seeking system in dream creation to Freud's idea that dreams equaled wish fulfillment. "In dreams I'm running away from things half the time. Is that wish fulfillment? Freud is very hard to kill. It is part of our culture now to think this way," Hobson says.

He insisted that the pons area of the brainstem was the generator of both REM sleep and dreaming but that dreaming in fact might be merely an accidental by-product of REM sleep, which he suggested had its own functions, such as regulating body temperature, maintaining the immune system, and performing a crucial balancing act with serotonin and other crucial neuromodulators. If dreaming is indeed simply a by-product of the brain's need to enter a state intended to serve these other purely physiological functions, "dream content might be quite irrelevant, telling us only what a subject's mental state might be like if he or she were to become delirious," said Hobson in an article he wrote in 1999 for a journal edited by Solms that was read

primarily by psychoanalysts. In a statement that couldn't help but push every button imaginable for a Freudian, he concluded: "In this sense, the interpretation of dreams in terms of unconscious motives would make about as much sense as interpreting the ravings of an alcoholic in the throes of delirium tremens."

Perhaps the most objective insight on the matter came in a commentary by Allen Braun published in the same journal. Braun agreed with Solms that the imaging map of the dreaming brain was compatible with psychoanalytic theory in some important ways. The fact that the emotional and long-term memory systems were supercharged while the centers for rational thought were dozing could be viewed in Freudian terms as the "ego" relinquishing its command and giving the unconscious freedom to romp freely. And the activity in the motivational region could support Freud's idea that dreams were powered by our most basic drives and wishes. But Braun sided with Hobson when he argued that since the symbol-creating portion of the brain (the prefrontal cortex) was off-line, dream content could not possibly reflect unconscious desires that were censored and disguised via symbols requiring elaborate decoding and interpretation. "I think you can use dream content on the face of it, what Freud called the manifest content—for self-therapy or psychotherapy," he says, "but there's no interpretation needed, because nothing is disguised."

Neatly summing up his view of the Hobson-Solms debate, Braun wrote in his commentary: "Stepping back a short distance, this is what I see: Hobson, a consummate biological psychiatrist, now argues against reductionism and passionately advocates the study of subjective conscious experience. Solms, a psychoanalyst, is attempting to recast dynamic psychology in neurochemical terms. It sounds to me like these gentlemen are approaching common ground. Perhaps it is simply the ghost of Freud that is getting in the way."

JUST TWO YEARS LATER, in one of those truth-is-stranger-than-fiction twists of fate, Hobson himself became one of the "experiments of nature" upon which Solms's neuroanatomical dreaming study was based. While traveling with his wife in the south of France in February 2001, Hobson suffered a stroke. Ironically, it affected only the brainstem—the portion of the brain that had been the focus of much

of his life's work. His wife, Lia, a neurologist, recognized his sudden difficulty in swallowing and other initial symptoms as signs of a stroke and quickly got him to the emergency room of the Princess Grace Hospital in Monaco, where he remained for ten days before being transferred by air ambulance to Brigham & Women's Hospital, in Boston, just a block away from his neurophysiology lab.

Ever the curious scientist, Hobson became an observer of his own condition while hospitalized, dictating his perceptions for journal entries that chronicled what often became a waking nightmare. Because the stroke was confined to the brainstem, he suffered no lasting cognitive impairments. But among the most striking immediate results of his brainstem lesion was that for the entire ten days he was hospitalized in Monaco, he was unable to sleep at all.

"The worst time is the night because I am alone, usually from 7 P.M. to 7 A.M., and because I cannot sleep a wink. I'm just awake, my mind working actively in the dark all night long," he dictated in notes on day six of his insomnia. Of course, dreaming also ceased. Instead, when he closed his eyes even momentarily he would see a vault over his body upon which he would view projected images of hallucinations of geologic forms, inanimate sculptures, and disconnected human body parts. He also had horrific hallucinatory experiences in which he felt that he was being catapulted through space, as he described in his journal: "The illusion that I was actually moving at very high speed for at least 100 meters through space was so completely convincing and so terrifying that I said to myself, 'So this is what death is like.'"

Hobson did not have what he terms a vivid, sustained dream until thirty-eight days following the stroke. In that dream, he was traveling with his wife abroad and discovered she had given another man a drill bit from a treasured tool Hobson kept at their weekend home, a farm in Vermont. Describing the dream in his journal, Hobson said: "It seemed to me odd that she would give a stranger one of my most precious tools without asking me. I was feeling very vexed and apprehensive." In the dream, his wife confides to him that she needs to have a secret life, and for much of the rest of the dream, he is wandering alone, unable to find her.

Though the dream contains elements that obviously are tailor-made for Freudian analysis, Hobson observes: "I didn't need the

dream to tell me that I was worried about my ability to maintain my relationship with my wife as an impaired person. It is a strong emotion and it creates the whole scenario. Emotion is running the dream show." He argues that those who say he proclaimed that dreams are meaningless misunderstood him. "Dreams are dripping with meaning, but they don't have to be interpreted. Dreaming is the way it is, probably for some reasons that are more like Freudian ideas than it might first appear. There must be some kind of reworking of memory going on, but it isn't buried stuff you can't manage. It's probably just the opposite: in dreaming we're trying to integrate and come to terms with the difficulties of having emotions."

Drawing conclusions from his stroke experience, Hobson says Solms's "findings have reopened debate about the relative importance of brainstem and forebrain structures in the neurogenesis of dreaming." As for the correlation between REM sleep and dreaming, Hobson already had acknowledged in 1988 that at least 5 percent of non-REM dreams were indistinguishable from dreams in REM, and today he summarizes his stance by saying, "Dreamlike mentation occurs in all states, but REM is the best state to study dreaming."

In the end, however, Hobson comes back to his own bedrock position on how dreams are generated: "Once recovery began and my brain stem had recovered its ability to support sensorimotor functions, my dreaming recovered, too. I have no doubt that normal dreaming requires a normal forebrain and that severe damage to the forebrain leads to a loss of dreaming, which may even be permanent. But based on my experience, I also have no doubt that a normal forebrain cannot sustain normal dreaming as long as the brainstem is dysfunctional."

For his part, Solms concedes that Freud may have been wrong about the meaning of dreams being disguised and censored, noting that the bizarreness of dreams may simply be due to the fact that the frontal lobes of the brain aren't performing their usual executive decision-making functions. But he is convinced that it's the motivational circuits of the forebrain—not the brainstem—that trigger the dreaming process. Certainly it's clear from study after study conducted by researchers around the world that the overwhelming majority of dreams—particularly the vivid memorable ones we recall and recount

to others—occur during REM sleep, probably because it provides the high level of overall brain activation that is a prerequisite for dream creation. But it's also indisputable that such dreams also can occur during other sleep phases, albeit less frequently. Solms has begun brain-imaging studies aimed at identifying what brain regions are activated during these non-REM dreams, and that kind of evidence may eventually answer the question of what really triggers dreaming.

While the Freudian Wars may be drawing to a close, another long-standing unresolved question about dreaming continues to be a subject of hot debate among researchers. Does dreaming serve any biological purpose at all? Many scientists who wouldn't agree on much else about dreaming—such as Hobson and David Foulkes—maintain that dreams are merely an accidental by-product of other evolutionary developments, without any specific function of their own. We happen to periodically experience high levels of brain activation, and when that happens, our neuronal networks can't help but process information and spin narratives, because that's what they're designed to do. Researchers such as Tom Balkin argue further that these periods of activation during which dreams occur actually serve only one simple biological purpose: to keep the neural networks tuned so that the brain is prepared to return to fully alert, waking mode when the need arises.

But there's another scientific camp contending that REM sleep evolved for reasons that were crucial to mammalian survival and that, as a result, dreaming itself has come to serve a variety of important biological functions. Mounting evidence from recent studies indicates that clues to support this viewpoint actually are right before our eyes—in the content of our dreams.