SWEET

Dreams Are Made of THIS

What are dreams? Why do we have them? The answers are as intriguing as dreams themselves

By Gerhard Klösch and Ulrich Kraft

In his time, Artemidorus Daldianus was a highly regarded man. He was a dream doctor, and in the second century A.D. his fellow Greeks considered dreams to be encoded messages from the gods. Deciphering them required an expert, with Artemidorus chief among them.

Artemidorus declared that all dreams were not created equal, however. If the nocturnal visions could be explained from past events in the sleeper's life, the good doctor wrote them off as meaningless constructions of the individual's experiences and mental orientation; these dreams were not secrets of the gods. Artemidorus himself would never have imagined that, with this idea, he had anticipated a core debate that would arise some 1,700 years later.

The physician who sparked that debate was none other than Sigmund Freud. According to his monumental 1899 work, *The Interpretation of Dreams,* our nighttime hallucinations are activated by subconscious wishes that can burst forth from behind the protective veil of sleep. Freud's contention was just that, however—a hypothesis, one that neurologists of the day could never prove despite a flurry of scientific investigation. Freud lacked the answer to the ancient question, "What does the brain do when we enter the dreamworld?" And it frustrated him. He openly wished for neurological evidence, worked at it himself and even said that such information would likely supersede his psychological theories about dreams. But he lacked the science and tools needed to find it.

(Emotionally loaded dreams fill REM sleep. Subdued visions arise during non-REM sleep.)

Today we have better tools, and modern explanations of dreaming are being turned on their heads, in some cases leading back to age-old theories. But as scientists try to pin down what causes dreams and what they mean, if anything, one lesson has clearly emerged: dreams play a vital role in memory and learning, and it is too early to give up on the proposition that they provide a window into our true emotions as well.

The REM Revolution

As Freud's stature grew in the early 1900s, psychologists the world over strongly embraced his theory of dreams. It was not until the 1950s that we reached the next turning point in our understanding. Nathaniel Kleitman of the University of Chicago and a student assistant in his sleep laboratory, Eugene Aserinsky, began to record the eye movements of sleeping children. Kleitman hoped to find an indicator for when the wee ones would awaken. In 1953 the duo found that during overnight sleep, test subjects went through four to six periods of eye twitching, each lasting from 10 to 50 minutes. The pattern held in adults, too. The scientists named this phase rapid eye movement (REM) sleep.

Kleitman was even more amazed when he looked at the sleepers' brain waves, recorded by electroencephalograms (EEGs). The brain was extremely active during the REM phase; neurons fired about as much as they did when the subjects were awake. Yet their muscles were practically flaccid during REM sleep. Kleitman and Aserinsky wondered what all the activity was about. So they began waking their subjects during the high point of REM sleep and asking them if they had been dreaming. From 80 to 95 percent said yes. If the same people were woken during other sleep phases, however, only 5 to 10 percent reported dreams. Neurologists celebrated the discovery: REM sleep, the high-frequency pattern of brain waves and the reduced muscle tone were objective manifestations of the subjective experience of dreams. The excitement was so great that dream researchers dismissed the rest of the sleep cycle as meaningless "non-REM," an assumption that would later prove premature.

A plethora of experiments about the biochemical mechanisms of REM sleep boosted scientific euphoria for two decades. Proof that REM sleep occurred in almost all mammals—mammals that in labs could be much more comprehensively investigated than humans—added fuel to the fire. In 1962 neurophysiologist Michel Jouvet of the University of Lyon in France discovered that in cats, a relatively small bundle of nerve cells in the brain stem known as the pons was always active when muscles were relaxed during sleep. If he disturbed the pons, muscles stiffened and quick eye movements did not occur.

Jouvet later implanted electrodes into cats' brains and managed to trigger REM phases by electrically stimulating the pons. He also found, to his surprise, that higher-order brain regions had no function in REM whatsoever. Even animals in which all nerve connections from the pons to the cerebral cortex had been severed fell into REM sleep. The REM center appeared to reside in the pons, which lies in the brain stem, an old, primitive brain region that bears responsibility for basic functions such as breathing and heartbeat.

Looking for Work

But how did the pons control REM and non-REM states? Did dreams have nothing to do with the brain's emotional centers? If not, where did dreams' fantastic visions and delightful story lines, their chase scenes and terrors, their sexual exploits and tensions, come from? In the 1970s, building on Jouvet's results and their own extensive work in sleep labs, J. Allan Hobson and Robert W. McCarley of Harvard Medical School presented two complementary theories: the reciprocal-interaction and the activation-synthesis models. According to the former, REM sleep and

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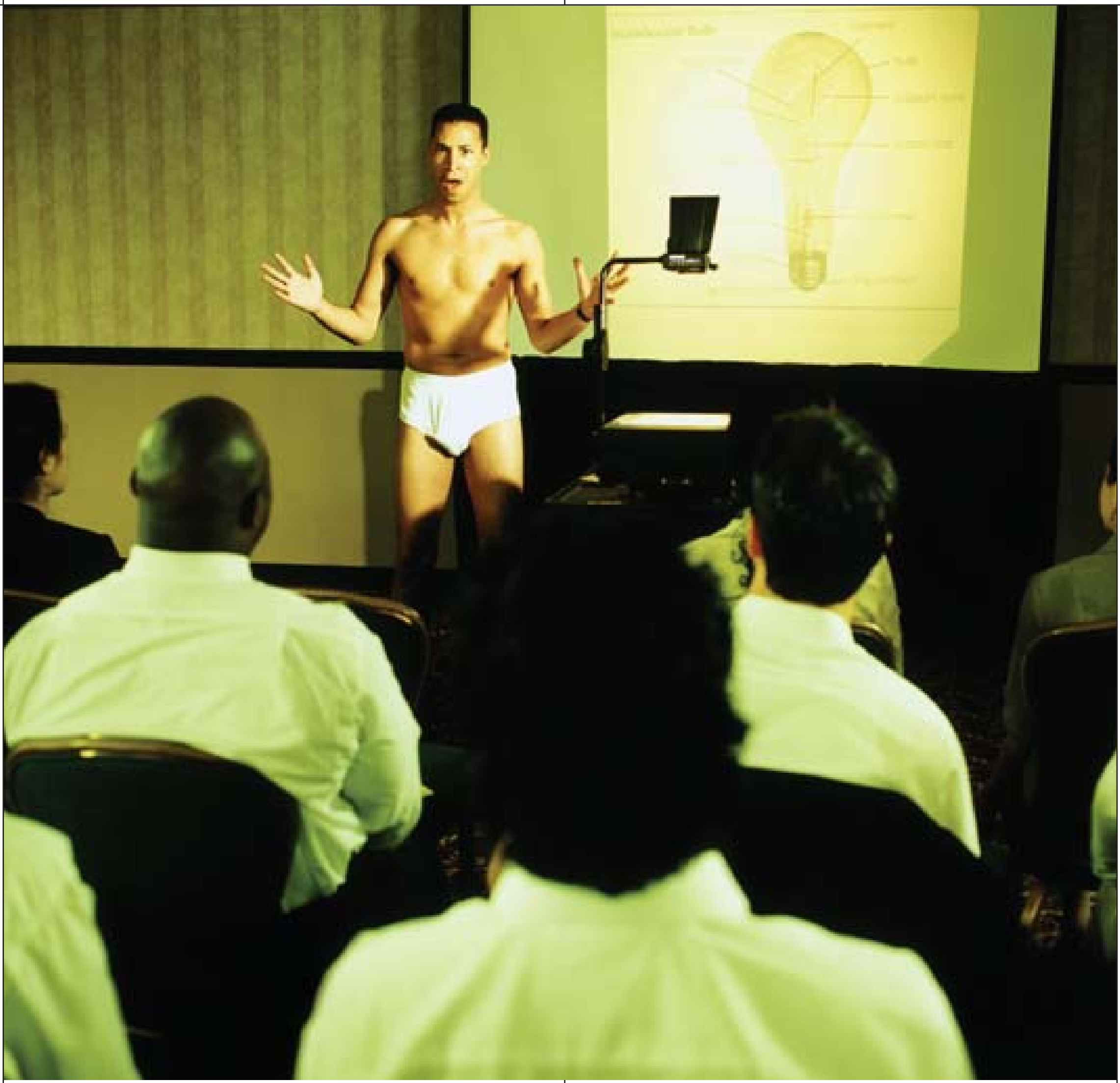

ANXIETY
Do dreams lay bare hidden apprehensions or simply reflect known worries?

MICHAEL COGLIANTRY *Getty Images*

the dreams related to it are turned on and off by a tug-of-war between special networks of neurons in the pons.

The neurophysiologists determined that so-called REM-on neurons used the neurotransmitter (a messenger chemical) acetylcholine to send impulses to various brain regions, triggering arousal. Acetylcholine caused neurons to fire not only in the pons but also in parts of the cortex and in the limbic system, the emotional center of the brain. According to the researchers' activation-synthesis model, dream images arise randomly from neurons that fire in these various regions. The sleeping brain tries to do with these signals exactly what it does in its waking state with sensory inputs: make sense of them.

Hobson and McCarley said that dreams are the vain attempt of the brain to concoct coherent story lines that link random signals. As part of this effort, the frontal cortex connects the senseless impulses of the pons with feelings, sensory impressions and experiences from memory, composing a narrative that fits the stimuli—a narrative the sleeper experiences as a dream.

After 50 minutes at most, the REM-off nerve cells bring this exercise to an end. They release the neurotransmitters norepinephrine and serotonin, both of which counter the effect of acetylcholine. The sleeper stops dreaming. For the average person the entire cycle repeats every 90 minutes or so throughout the night.

The activation-synthesis model made Freud's basic assumptions untenable. Psychological phenomena such as emotionality, motivation or subconscious desires did not prompt dreams. Self-regulating biochemical feedback loops in the primitive brain did. When Hobson and McCarley introduced their heretical model in the December 1977 issue of the *American Journal of Psychiatry,* they caused an uproar among psychologists. Stating that dreams were nothing more than a by-product of brain chemistry was seen as a vehe-

FREE FALL
Are you plummeting helplessly toward disaster or feeling freed from daily bonds?

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ment attack on Freud and therefore on all of psychoanalysis, widely held as the best way to "cure" people with all degrees of mental illness. The renowned journal received more letters about that article than any that had come before it—most of them expressing outrage. Hobson would later acknowledge that he and McCarley had invented fire when light might have been more useful, but until the fire was lit the scientific community had grossly neglected the brain chemistry that was undeniably fundamental to dreaming.

Indeed, the activation-synthesis model spawned a wide body of research into the neurological stuff that dreams are made of, and the model was confirmed again and again through experimentation. For example, test subjects who were injected with acetylcholine shortly after falling asleep progressed into dream sleep much faster than usual. And administering an acetylcholine inhibitor delayed REM sleep and dreams.

Lobbing a Grenade

But had Hobson and McCarley completely solved the riddle? Dream researcher W. David Foulkes, then at the University of Chicago, decided to find out by systematically waking his subjects during different sleep phases; his results showed that equating REM sleep with dreaming and non-REM sleep with a dreamless state was too simplistic. Although only 5 to 10 percent of sleepers who were woken during a non-REM phase reported dreams, the picture changed drastically when Foulkes reformulated the standard question of sleep research from "Were you dreaming just now?" to "What was going through your head just now?" Suddenly 70 percent described dreamlike impressions during non-REM periods.

Similar experiments into the 1990s showed that REM sleep was not even necessarily the most dream-intensive segment of overnight rest. The phases of falling asleep in late evening and the brief interval shortly before waking in the morning were especially rich in dreams. In addition, it seemed that non-REM dreams were relatively short and rationally constructed in terms of facts and logic, whereas REM dreams were more visual, emotional and detailed.

All these findings made it appear unlikely that REM sleep exclusively drives dreams. Dreaming seemed to be more of a continuous process, not one sequestered within certain sleep phases. This new view raised doubt that the pons in the brain stem was the exclusive source of our dream visions. Scientists who searched back into medical literature found an unusual case that supported their suspicion. In 1982 a man had arrived at the sleep laboratory of Peretz Lavie at the Technion-Israel Institute of Technology. The reason: since he had incurred a head injury during a grenade explosion, he had suffered regularly from terrible nightmares. The sleep doctors wired his brain to an EEG and the next morning were astonished: the man had not gone into a single REM episode the entire night. That omission seemed impossible.

Lavie immediately resorted to computer imaging, which revealed that a small grenade splinter had bored into the man's pons and had destroyed exactly the area that supposedly controlled REM sleep and the dream trigger. So a complete lack of REM sleep made sense. But how, then, could the man be regularly tormented with nightmares? Did dreaming and the control of REM sleep rely on separate mechanisms?

Mark Solms of the University of Cape Town in South Africa became one of the first experts to say yes. For years the neuroscientist had sought cases of patients whose brain stems had been damaged by accidents or disease. If dreaming and REM sleep were connected anatomically, a defect in this region would cripple both phenomena. After Lavie's find, Solms and others looked harder and compiled 26 cases of patients who no longer experienced REM sleep because of damage to the pons. Only one patient reported a total loss of dreams, however. All the others experienced nocturnal interludes without REM sleep. At the same time, Solms's group uncovered more than 100 cases of people who said they never dreamed, even though their pons was intact and they slept through completely normal REM phases.

Finally, Independence

Those 100-plus people did, however, have lesions in other brain regions. Solms identified two areas in which damage could cause complete loss of the dream experience, and those areas had no anatomical or functional connection to the pons. The first is the so-called white matter of the frontal lobes, above the eye sockets. Impulses arrive there from various parts of the brain with the aid of the neurotransmitter dopamine, which influences motivation and drive.

Solms noted from clinical drug trial results that medications reducing the brain's dopamine level also decreased dream activity. And dopamine enhancers, such as L-dopa used in treating Parkinson's patients, caused more frequent and intense dreams. But neither regimen affected the frequency or length of REM sleep.

The second area of damage that Solms found could cause a complete loss of dreams was in the occipitotemporoparietal cortex, behind and above the ears. This region is responsible for processing perceptions and abstract thinking. Its role in dreaming remains unclear.

What Solms's research did make clear, though, is that dreaming often takes place independently of REM sleep and of REM's generators in the pons. And it seems that only damage to the frontal lobes of the higher cortex causes dreams to disappear. Damage to lower-level information-processing areas, such as the visual system, may affect only parts of dream images, such as their visual quality. Solms had inverted the modern model of dreams. During sleep, according to Solms, higher-level areas of the cortex generate dream images that then waft through the memory and emotion centers before they are finally perceived by our sleeping senses.

> (Dreams may etch daytime learning into memory and erase informational refuse.)

Was Freud Right?

By 2002 or so it seemed that neuroscientists, psychiatrists and psychologists were falling into one of two camps led by Solms and by Hobson. Public debate became heated, including in the pages of *Scientific American* magazine. Although Solms agreed that the primitive pons stimulated REM sleep, he also believed the origin of dream content lay in the highest-level brain regions, which Hobson characterized as passive recipients of meaningless signals from the brain stem. Solms's view allowed that dream content could be shaped by hidden emotions and motives or forgotten memories, and legions of Freudians—psychoanalysts who based their practices on Freud-like theories—came running into the fold.

This time the critical volleys came from neuroscientists. They claimed that Solms had developed his model from the beginning under the premise of confirming Freud's dream theory and that he was simply looking for the brain regions

(The Authors)

GERHARD KLÖSCH is a psychologist and sleep researcher in the department of clinical neurology at the University of Vienna. ULRICH KRAFT is an editor at *Gehirn & Geist*.

that best fit that preconceived notion. Hobson challenged Solms's theory with several arguments, one of them the plain fact that we almost always forget our dreams by morning. If dreams were really problem-solving or processing functions of the brain, then we should easily remember them when we wake up. Neither researcher, though, could produce unambiguous neurological proof of his claim.

Modern imaging techniques, however, had already begun to influence the stalemate. In 1997 neuroscientist Allen R. Braun of the National Institutes of Health had succeeded in taking positron-emission tomography (PET) pictures of the human brain during REM sleep. Braun's images demonstrated that in REM sleep, the regions that process sensory information are less active than they are in the waking state. This made sense because the slumbering brain is receiving no signals from the senses. But the frontal cortex, responsible for integrating information from other brain areas, also remained relatively calm during the REM phases—a contradiction to Solms's theory that dream content originates there. The limbic system, in particular the amygdala, was very active but only during REM rather than non-REM dreams. That did not directly support Solms or Hobson, but it did explain the differing dream content reported by test subjects in these two dream states: emotionally loaded experiences during REM sleep and emotionally subdued experiences during non-REM sleep.

To Sleep, Perchance to Learn

The debate over exactly how dreams are initiated and sustained still roars today. In the meantime, researchers are trying to answer the related question of why we dream at all. Recent imaging studies show that during REM phases the hippocampus, a brain region key to creating memories, is extremely active. This insight lends strong evidence to a notion Hobson and others had raised—that dreams help the brain lay down memories and hardwire new information. Perhaps, they postulated, dreams were a tool the sleeping brain used to link events from the prior day's milieu to what the brain had already stored and to etch these new wrinkles into long-term memory. Each night, dreaming would help the brain update its lifelong store of memories and learning. Different experiments have demonstrated that animals as well as humans retain new knowledge better after an undisturbed nap or night of sleep. If researchers prevent test subjects from sleeping during REM, they do not retain new information as well as those who are allowed to sleep.

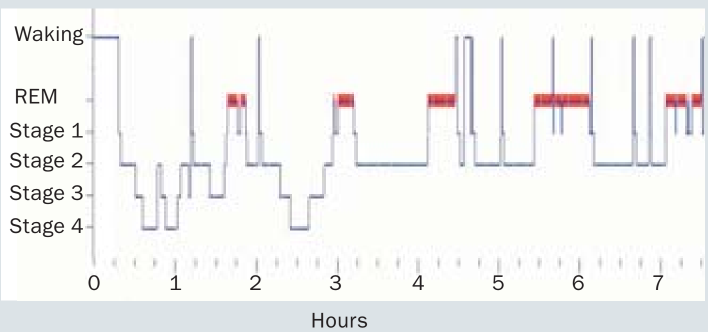
REM sleep appears to be especially important for strengthening visual and motor skills. If someone practices a new set of tennis strokes on a given day, for example, the REM segment of his or her sleep will increase dramatically that night. If one wakes this person repeatedly during REM phases, the retention is hindered—more so than if only non-REM sleep is disturbed.

A mounting number of experiments show that during sleep, the brain makes new connections between neurons, especially in regions that were active in learning during the day. Neurologist Pierre Maquet of the University of Liège in Belgium has demonstrated that this connection occurs most aggressively during REM sleep. And yet other studies indicate that the retentiveness of people who have taken REM-suppressing medications for years is not affected. Patients who do not enter REM phases because of brain damage do not seem to lack in learning ability either.

The famous co-discoverer of DNA who also became renowned for his work in neuroscience, the late Francis Crick of the Salk Institute for Biological Studies in San Diego, and molecular biologist Graeme Mitchison of the University of Cambridge have maintained that we actually dream to forget. According to their theory, dream sleep is a self-cleansing program. Unencumbered

REM Zone

Most adults have a similar sleep cycle that recurs every 90 minutes or so, from four to six times a night. The graph below for one test subject is typical. The individual falls asleep (stage 1) and reaches stage 2 soon after. In another 20 minutes or so deep sleep begins (stages 3 and 4). A REM phase ends the first sleep cycle. In the course of the night the dream-rich REM periods lengthen in duration, while deep-sleep stages shorten. For optimal physical and mental recuperation, it is most important that sleep during the first third of the night is undisturbed. Most people also wake up a number of times without realizing it.



GERHARD KLÖSCH

PANIC
Is someone looking to run you down, or are you just having trouble staying ahead?

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by the constantly flowing signals of the waking state, the brain uses the calm of the night to free the system from informational refuse. Superfluous and disturbing images, memories and associations are brought up in dreams, checked for value, then erased from the cortex.

Crick said that this "reverse learning" prevents the neuronal network from being flooded with data, making it possible for us to once again have an orderly commerce with memories the next morning. Dreaming as unlearning also explains why we are so poor at remembering our nocturnal images. And yet Crick himself admitted that his model, like those of Solms and Hobson, is just a hypothesis. All three theories are only partially supported by experimental results.

Two millennia after Artemidorus Daldianus, there is still plenty to learn. Until conclusive evidence falsifies one of these theories or substantiates a new one, we can simply go along with a paraphrase of French playwright Victor Hugo, which has been neither proved nor contradicted: Thought is the labor of the intellect; dreaming is its pleasure.

(Further Reading)

- **The Neuropsychology of Dreams: A Clinico-Anatomical Study.** Mark Solms. Lawrence Erlbaum Associates, 1997.
- **Dreaming: An Introduction to the Science of Sleep.** J. Allan Hobson. Oxford University Press, 2004.