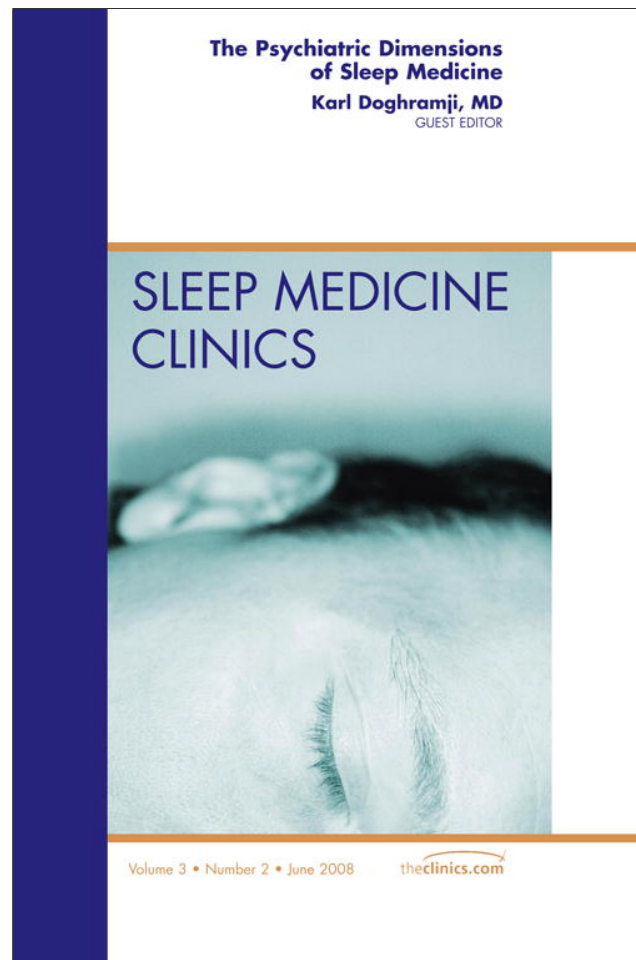


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The Contribution of the Psychology of Sleep and Dreaming to Understanding Sleep-Disordered Patients

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This article reviews the major psychologic functions occurring during sleep and the evidence that patients who present with disorders of sleep have specific psychologic dysfunctions related to the type and degree of their sleep disturbance. Although there are individual differences in the degree to which people are aware of sleep thoughts and dreams, mental activity is actually continuous throughout the waking, sleeping, and dreaming cycles. The neurologic mechanism responsible for this is the cooperation recently identified between the slow wave sleep (SWS) of the first hour and the following rapid eye movement (REM) sleep. As sleep begins, the neural networks that were activated during the waking experience are reactivated and remain active until REM sleep occurs. At that point, the coded representation of that experience is matched to similar older memories, and this combination is displayed as a dream. This is the explanation of how normal sleep is basic to

“learning” the overnight improvement in mood and performance. The organization of new experience, and retention of it in long-term memory, allows waking behavior to be more adaptive. The selection of which experiences are to be saved relates to their emotional relevance to the self-system. The replay of these initiates a hippocampal-neocortical dialog during which negative emotion, generated in response to experience that challenges the sense of self, is downregulated in an across-the-night, sequential processing. If there is a persistent sleep disorder the learning process and mood regulation process is less efficient or stopped altogether. The study of sleep disorders provides insights into this regular nocturnal updating of the self-program, and of the specific psychologic effects of interrupted sleep or abnormalities in the timing of the cycles. The application of these findings to understanding and treating these patients is discussed.

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A brief history

Academic psychology did not recognize dreaming as a legitimate area of study until the early 1950s, when publications from Kleitman's laboratory announced there is a regularly occurring active brain state in sleep, dubbed "rapid eye movement" sleep, which is a reliable indicator of when a distinctive type of mental behavior, dreaming, is ongoing [1,2]. A way was opened to access the psychology of sleep on-line.

At that time psychology was still struggling to be recognized as an independent empiric science devoted to the understanding of human behavior through the methods of hypothesis testing and controls. As a new science, psychology staked out the areas that defined its turf: learning and memory, motivation, and emotion. The idea that these basic aspects of behavior were also operating during sleep, or that they were affected by sleep, was not then considered credible. After all, the sleeper was unconscious and experimental psychologists could neither observe their mental activity directly, nor could they rely on the sleepers' morning memory to be a reliable data source. As a result, the formal study of psychology, with few exceptions, ignored the 8 hours of sleep until the methods of sleep recording made this investigation scientifically feasible.

Freud's *Interpretation of Dreams* [3], originally published in 1900, called attention to the importance of the unconscious as a strong influence on the motives that drive behavior and the accompanying emotions. Freud not only illustrated the many ways the unconscious plays a role in waking behavior, but he pointed to dreaming as the path by which one could tap into this important information [3]. Armed with the guidepost of the strong correlation between REM sleep and dreaming, psychologists set out to test the validity of Freud's premise, that adding information from dreaming enhances one's understanding of human behavior and why it is that even some smart people do not "learn from experience" and continue to make self-destructive behavior choices.

The first twenty years

Despite the drawbacks of the unnatural situation of sleeping in a laboratory, early studies of dreaming managed to address important questions: At what age does the child begin to dream and how does that relate to their waking cognitive development? [4]; Are dreams the normal equivalent of the hallucinations of psychosis? [5]; Do dreams have inherent meaning or is this added to random images as an awakening takes place [6]; Do dreams tell a connected story from REM to REM across the night?

[7]; Why do some dreams repeat over and over? [8]. Some of the more difficult questions addressing the major topics of psychology (what is the role of sleep in learning, memory, and motivation; and is sleep a place where emotion is regulated [9]) remained controversial and only recently have been addressed with more advanced methodology.

The focus on sleep disorders

Something else occurred to divert psychologists from the basic studies. The years 1975 to 2000 saw the emphasis shift from research into the nature and function of normal sleep to that of pathology. That in turn led to the advent of specialized clinical facilities to diagnose and treat those whose sleep was dysfunctional. This development resulted in a tremendous increase in public awareness of the role of sleep in both physical and mental health and an expansion of the disciplines involved in the applied area of clinical sleep medicine [10].

Studies of the mind were left behind; the very existence of mind was denied. It was deemed a fuzzy concept no longer necessary in the age of brain imaging. Some clinical research on the psychologic aspects of sleep and dreaming continued, however, and is now ready to be integrated to better the understanding and treatment of patients who present with problems of sleep.

Current theories of dreaming and the psychology of sleep circa 2007

Although early studies had noted that there was some mental activity that could be retrieved from sleep onset and in sleep stages other than REM [11], most of the research of the first 50 years was focused on REM-related dreaming. This article begins with the legacy from that work, the consensus on one major dream function, before moving to the newer view of the influence of waking states of mind on the mental activity of non-REM (NREM) sleep and on the following dream production in REM and the effect of sleep on tomorrow's behavior, the full 24-hour perspective on continuous mental activity.

Many of the research-based investigators of dreaming [12–17] all place emotion as the key to understanding dreams and their relation to why one generally feels better in the morning. This is referred to as the "mood regulatory function of dreaming." Hartmann [13] states: "overall dreaming makes connections more broadly than waking in nets of the mind, and the connections are not made randomly but guided by the dreamer's emotional concerns." Breger [14] links presleep emotion to postsleep waking adaptation: "(dreaming) serves

a unique function in the assimilation and mastery of aroused material into the solutions embedded in existing memory systems." From my own research [17] I have concluded that "dreaming has an active self-regulatory role in emotional modulation."

How this self-regulating dream function adds to an understanding of four sleep disorders is explored next: sleepwalking (an NREM parasomnia); nightmares and dream enactment (both are REM parasomnias); and major depression (insomnia associated with a mental disorder). These illustrate how specific deficits in the integrity of sleep relate to failures to regulate mood and to change behavior.

Fig. 1 is a model of mental activity throughout night, drawn from reports from normal volunteers awakened in various sleep stages at different times of the night. Freud identified three parallel levels of ongoing mental activity: (1) conscious, (2) preconscious, and (3) unconscious. If one superimposes the results from awakening studies, the model predicts that aspects of the presleep waking thoughts (level 1) are picked up and mingled with the longer-term preconscious emotional concerns of the sleeper (level 2). The mental activity retrieved from the first NREM episode depends on the strength of the emotion evoked in waking and carried into sleep by the reactivation mechanism. As REM sleep is turned on, this activation is "mapped onto previously stored memories" (level 3), matched to a neocortical network with similar feelings. As the first REM period ends and NREM sleep is resumed, some memory bits continue to be carried forward into the next NREM sleep. This process continues throughout the night. The evidence supporting this continuity model has recently been summarized by Ribeiro and Nicolelis [18] and Giuditta and coworkers [19] and is next examined as it applies in several sleep disorders.

Non-rapid eye movement parasomnia: sleepwalking

The continuation of motives and emotions from waking into the first cycle of SWS can be seen

most clearly in the behavior of adult sleepwalkers. This disorder has been difficult to study in the laboratory because of its unpredictability and lack of any distinctive marker of its presence using the standard sleep stage scoring of the polysomnogram (PSG) [20]. What is well established is that the sleepwalker is not acting out a dream. The walk actually begins with an abrupt arousal out of SWS in the first third of the night, not out of REM sleep [21]. Nor can the patient clarify why they were doing what they did, because they have little or no memory of the event. At best they report a sense of urgency. Although this disorder is relatively common in young children with an equal prevalence in both genders, the frequency of these episodes wanes or stops all together during adolescence as SWS is reduced in amount and in the amplitude of the delta waves. There can be a resurgence of this disorder in young adulthood under certain conditions, however, and when it does the prevalence is much higher in males.

What makes a sleepwalker walk?

The strongest predisposing factor is genetics. Those with a family tree loaded with many relatives affected with sleepwalking or sleep terrors are at increased risk for this disorder [22]. Twin studies in Sweden [23] find that monozygotic twins are concordant for sleepwalking at six times the rate of dizygotic twins. Genetic testing by HLA typing shows sleepwalkers more often display a DQB1 marker than do controls [24], although this marker is also found in those affected by narcolepsy and REM behavior disorder. All three of these disorders have in common some motor control dysfunction. More promising is new evidence that scoring the PSG by spectral analysis for the presence of delta frequency activity rather than by the usual sleep stage criteria finds sleepwalkers differ from controls in having less delta activity at the beginning of the night [25–27]. Fig. 2 shows the reduced delta activity in the first sleep cycle of sleepwalkers compared

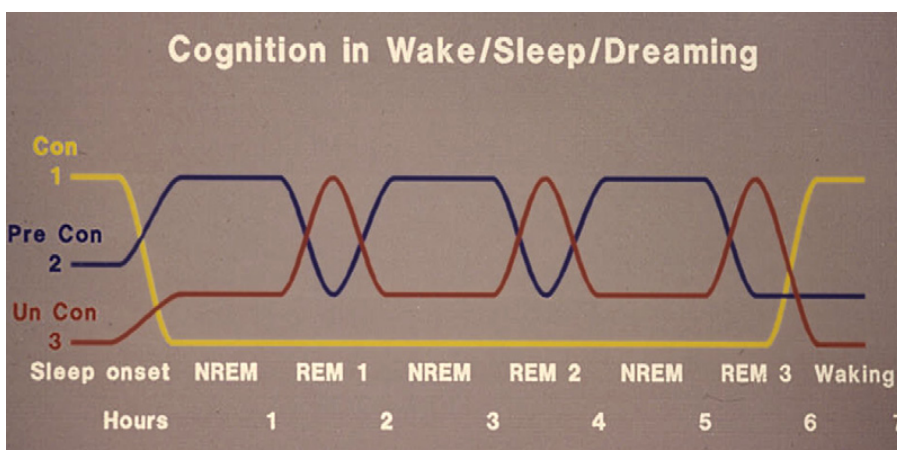


Fig. 1. R. Cartwright graph.

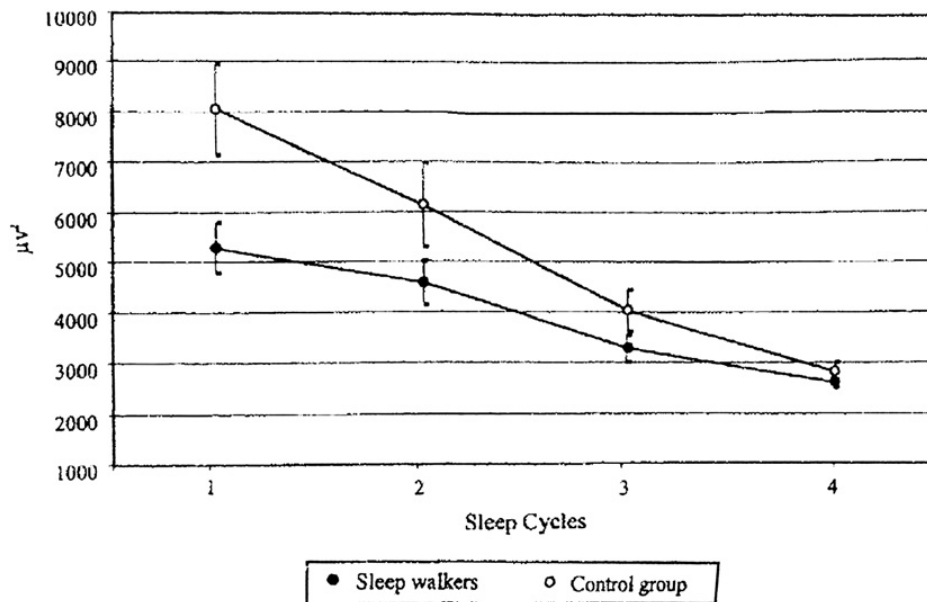


Fig. 2. The reduced delta activity in the first sleep cycle of sleepwalkers compared with controls. (From Guilleminault C, Poyares D, Abat F, et al. Sleep and wakefulness in somnambulism: a spectral analysis study. *J Psychosom Res* 2001;51:411–16; with permission.)

with controls [25]. Another feature of the PSG is that sleepwalkers have multiple arousals from delta sleep in the first two sleep cycles, making this sleep time very unstable.

An episode of adult sleepwalking typically begins with an abrupt arousal during this unstable first hour of sleep, when the brain is simultaneously partially asleep and partially awake. One imaging study caught a 16-year-old habitual sleepwalker shortly after he had a behavioral arousal during a PSG study [28]. The single-photon emission CT showed a 25% increase in blood flow in the posterior cingulate cortex and decreases in the frontoparietal cortices. In this mixed state, a sleepwalker is able to perform complex motor behaviors, including driving a car sometimes for long distances, but is not able to recognize faces, even those of loved ones. This suggests that the area of the brain identified with face recognition, the fusiform gyrus, is not active [29]. Once an episode is over, which may take as long as an hour, the sleepwalker typically returns to sleep spontaneously, although this may be in a strange or even dangerous location.

If startled by a touch or sound while in this state a sleepwalker may become swiftly aggressive, however, behaving as if they are in a fight or flight survival mode. In a large epidemiologic study the rate of those adults reporting that they currently have episodes of aggressive sleepwalking was 2.1%, much higher than expected [30]. These vary in severity from benign to lethal, such as jumping out a window or thrusting an arm through a glass door.

There are many behaviors other than aggression that have been recently reported performed without

conscious awareness, or any later memory, following an arousal from SWS within the first 3 hours of sleep. Some sleepwalkers eat, and even prepare food; often these are strange combinations of foods, such as raw bacon with chocolate bar sandwiches. Others attempt to rescue someone from an imaginary danger, such as pulling their wife out of bed because the mattress is thought to be on fire. Recently, there have been patients who have presented because of inappropriate sexual behavior, such as lying on top of a sleeping child. Exploring new territory is another sleepwalking behavior, as it was in the 15 year old who climbed a 130-foot crane. All of these are instances of basic motives necessary for our survival: eating, procreating, fighting or fleeing in response to danger, protecting the family, and exploring new territory. All show these remain active during SWS, the realm of the unconscious. If not for the arousal that aborts the transmission into REM, the activation of these drives would most likely be expressed in dream imagery and down-regulated by morning.

In addition to a genetic predisposition, it takes added factors for an overt episode of sleepwalking to occur. The most powerful of these is sleep deprivation. When a genetically vulnerable person has inadequate amounts of sleep over an extended period of weeks, because of either an internal or external arousing stimulus, sleepwalking is highly likely to occur. Sleep deprivation increases the drive for more SWS. Recently this has been used to stimulate sleepwalking under laboratory conditions. The study required both sleepwalkers and controls to undergo 25 hours of sleep deprivation before

a PSG test. All the sleepwalkers had one or more episodes, whereas none occurred in the controls [31]. Under home sleep conditions, the degree of sleep loss patients report having before a sleepwalking episode occurs is usually more chronic. This protocol, a combination of acute sleep deprivation beforehand, videotaping of any events during the sleep study, and power density scoring of the PSG, can now be used to confirm this diagnosis in the laboratory. This will be important in future forensic cases when sleepwalking is invoked by the defense in the case of a serious assault.

In one such case, which received widespread media attention [32], PSG studies were ordered by the court, but were conducted before the publication of the work showing the usefulness of power density scoring and the triggering effect of prior sleep deprivation. Although the studies demonstrated that K.P., a tall, well built, 23 year old, had very little delta sleep and many abrupt arousals whenever SWS was reached, he made no attempt to sleepwalk in the laboratory and so these tests were inconclusive. There was, however, other evidence that supported the argument that he may have been sleepwalking. K.P. had a strong family history of sleepwalking detailed by a court-appointed expert, and was known to be a sleepwalker as a child. He was also experiencing an ongoing major stress that resulted in extended loss of sleep, and he no memory for the event that brought him to trial.

The back story of this case reveals the psychology of this event. K.P. a high school graduate, married, with a new first baby, had begun to gamble on horse races and quickly found himself in serious debt. Without his wife's knowledge he continued to bet and lose many thousands of dollars. He emptied their joint bank account, took out an additional home loan, and then embezzled from his employer. When this was discovered he was fired. Over the next 4 months he became increasingly immobilized; he stopped seeking work and seeing his friends; and did not visit his in-laws, with whom he had a close relationship. His mother-in-law called him her Gentle Giant. His sleep quality was so poor he often did not go to bed at all but stayed on the couch watching television.

The violent episode started late on a Saturday night. That afternoon he and his wife had quarreled about his behavior. She insisted he seek help and that he accompany her the following day for a Sunday visit to her parents. This would mean revealing his financial crisis to them, which he dreaded and had avoided. His in-laws were modest people, not able to help him financially, and had trusted him to care for their daughter. Now that he was unable to find work and was at risk of losing their home, he feared he would be seriously diminished in their

eyes. He did promise his wife that he would go with her next day. That night he fell asleep on the couch while watching television and, as he reported, "I woke up over the body of a woman." He did not recognize this as his mother-in-law, whom he had stabbed to death, nor did he remember that he had driven 15 km to their house, attacked his sleeping father-in-law, and killed the mother-in-law with a knife from her kitchen.

When I interviewed K.P. while he awaited trial, he asked me in a bewildered voice really wanting an answer: "Why would I do that, when I had everything to lose and nothing to gain?" He was right: this episode made no rational sense. It was an emotionally driven, complex set of responses to an identity crisis. He fell asleep with the visit on his mind. He was determined to go, to carry through on his promise to his wife, but once there was the threat of exposing himself to her parents as a failure. This prospect mobilized his drive to survive by attacking those who would destroy a positive part of his self-system. He would no longer be the beloved Gentle Giant. The jury's task was to decide if the ensuing aggression was committed when he was in a nonconscious state. Their verdict was to acquit on the grounds that he was in a state of "non-insane automatism." Because his arousal from NREM sleep aborted REM, he lost the opportunity for dreaming to perform a dampening of his rage, and no mood regulation took place.

Sleepwalking in obstructive sleep apnea

Cases of NREM parasomnia precipitated by the sleep loss associated with obstructive sleep apnea are not common but three cases have been published; two of these involved serious violence [33–35]. In all three the obstructive sleep apnea was assessed to be severe by laboratory studies and also showed the reduced delta and increased number of arousals typical of obstructive sleep apnea and of patients diagnosed with NREM parasomnia. In the case published by Nofzinger and Wettstein [33] a man shot his wife to death following an arousal from sleep. In this case the sleep expert testified for the prosecution. He argued that sleepwalking was unlikely because obstructive sleep apnea is common but there had been no reports of the disrupted sleep being associated with violence. Also it did not help the defendant's case that he may have had conscious "motivation" for this act. The police found a note written by the wife in her handbag stating that she intended to leave him. The jury found him guilty and he was convicted.

The second case [34] was one of a night terror during a PSG study while continuous positive airway pressure was being titrated. A return of SWS in which

a desaturation occurred prompted an abrupt arousal. The patient jumped up and stood on the bed in a state of fear but no aggression was involved. In the third case [35], a 54-year-old woman with a 5-year history of weight gain and excessive daytime sleepiness awoke one morning to find her hands bloody. She then found more blood on a kitchen cutting board. The remains of her cat were found by the trash can. She was diagnosed with severe obstructive sleep apnea and treated with continuous positive airway pressure and had no further episodes. What her motivation was for dissecting the cat could not be explored because she lived alone and had complete amnesia for this act. A desaturation episode may have triggered an arousal into a sleepwalk to the kitchen where perhaps the cat interrupted her by a touch or sound, but that is a guess.

Two other studies indicate that mild breathing disorders are more common in those who have a history of sleepwalking than they are in matched controls [36,37]. In the Guilleminault study [37], a surgical intervention to improve the respiration, a tonsillectomy, or opening the nasal passages eliminated the sleepwalking in a group of children with a history of sleepwalking.

Rapid eye movement parasomnia: nightmares

Patients suffering from chronic nightmares present another opportunity to examine the impact of a sleep disorder on the psychological function of dreaming. In contrast to the NREM parasomnias, the definition of nightmares includes an abrupt arousal from REM sleep, usually toward the end of the night, into a fully awake state with a clear memory of a highly emotional dream. The dream scenario usually involves a fear-invoking situation in which the dreamer has a strong sense of powerlessness. This may explain why they are more common in young children and become less frequent as youngsters acquire more coping skills. Adult nightmares have been studied extensively where they are found to be associated with psychopathology [38–41]. Nightmare frequency increases suicide potential in the depressed [42], and is a defining symptom of posttraumatic stress disorder following major disasters and personal traumas, such as physical abuse and rape [43–46].

Epidemiologic studies show a high prevalence of nightmares in adults, particularly in inner city women. Between 2% and 6% of the general population report experiencing nightmares at least once per week. Here too, the family and twin studies [47] implicate a genetic basis for this disorder. A variety of explanatory theories link the

neurobiology of fear to a psychological trait of poor capacity to regulate stress [48,49].

In a recent review [48] the authors propose there is a common link between emotion-evoking dream imagery and the profound lack of muscle tone characteristic of REM sleep. Being scared to death, while literally unable to move the major muscles, mimics the situation in the behavioral treatment called “desensitization.” In this program, visually imagining the fear-evoking memory while awake is coupled with training in physical relaxation [50]. The parallel in sleep is that the exposure from REM to REM over 1 or more nights to the feared stimulus while the atonia prevents a motor response “wears out” the arousal response. In chronic nightmares, however, the negative affect exceeds the capacity of REM to sustain sleep. This may be because there is no match in memory to the current stressful event to help disperse the affect load and an arousal occurs instead that prevents completion of the dream. It is the failure to complete the dream that is responsible for the repeated bad dream scenario rather than its extinction. Evidence supporting this model comes from three sources.

1. Nightmares are more common in those identified as having anxiety disorders, neuroticism, schizophrenia-spectrum symptoms, posttraumatic stress disorder, and maladaptive coping.
2. The usual flattening of emotion indicators during fearful dreams (low heart rate, respiratory rate, and low eye movement counts during REM) are even lower in the sleep just before a nightmare arousal [41].
3. The usual reduction in negative mood following the morning awakening fails to occur for the nightmare sufferer [49].

Dream enactment in rapid eye movement behavior disorder

REM behavior disorder is another sleep disorder in which there is a failure to maintain REM sleep. These patients not only have abrupt arousals from REM with vivid recall of a fearful dream, they also act it out [51]. Swinging at imaginary intruders they punch holes in the bedroom walls, upset lamps, hurt themselves, and damage property. Their sleep recordings show bursts of muscle activity in the chin leads during REM sleep and other indicators of a failure of motor control: bruxism (tooth grinding), hypnic jerks, and periodic leg movements are all common throughout the sleep in the PSGs of these patients. When this disorder was originally described it was most often identified in the elderly who also had some neurodegenerative disease, primarily Parkinson’s disease. Then,

younger patients meeting the diagnostic criteria began to be seen in sleep centers, when no clinical Parkinson's disease symptoms were apparent. Longitudinal follow-up of these patients showed it might be decades later before the neurologic symptoms were documented in waking [52].

Although rare, a milder form of one REM behavior disorder symptom called "dream enactment" has been documented in healthy young adults. It is possible that these may also be at risk for the later appearance of Parkinson's disease. Again, there is a mixed picture of psychologic precipitating factors on top of a genetic vulnerability caused by a flaw in the REM motor inhibition system. One young patient, a recently married medical student, presented with a self-diagnosis of dream enactment with a 3-month history. He described his sleep episodes as now occurring three to six times per night on 5 or 6 nights a week. A typical recent event began with an abrupt sit up in bed in a fearful state imagining an anaconda in the bed. He described himself as screaming to his wife "you grab the tail, I'll grab the head." His PSG recording showed multiple bursts of activity in the submental muscle and periodic leg movements during REM sleep. Clearly, he needed some insight into the added stress his marriage just 3 months ago imposed while he was preparing for his second-year board examination. He also needed some help with stress management. The breakthrough of muscle activity during REM is usually successfully treated with a small dose of clonazepam at bedtime. In the case of this patient where the psychologic function of sleep was so clearly disturbed the treatment also needed to include the rules of good sleep hygiene, especially the importance of avoiding sleep deprivation and training in relaxation techniques.

Major depression

In major depression the sleep marker in the PSG is in the timing of the onset of the first REM period of the night. The number of minutes of sleep before the first REM episode, normally about 90 minutes, is most often reduced to less than 65 minutes displacing the SWS [53] or is skipped lengthening the first NREM cycle. This robust finding suggests dreams too may be abnormal. In a series of studies volunteers, all of whom were undergoing the same negative emotional experience (the breakup of a first marriage), were followed longitudinally tracking the early REM marker and their dreams. The aim was to examine whether those who met depression criteria would restore mood regulation over time (wake in a better morning mood and remit from depression) following a change in dream scenarios. The focus was on two dream

variables: the expression of negative emotion and the inclusion in the dream story aspects of both the present life event and older memory images [54]. Twenty men and women who met depression criteria and 10 nondepressed divorcing controls all had their dreams collected from each REM period on three occasions over a 5-month period. At the follow-up visit, 12 of the depressed were in remission without any intervening psychotherapy or pharmacologic treatment and 8 remained depressed. All controls remained free of depression symptoms.

The major difference between the depressed who improved and those who did not was the degree to which the dreams integrated fragments of the recent emotional experience with older memories relating to the same emotion associated with the divorce [54]. One example of this is a dream reported by a depressed woman who when awakened from REM gave this report: "My ex-spouse appeared at my parent's home where I was having my 16th birthday party. He embarrassed me by exposing himself." The dreams of those who were in remission by the end of the study were longer than those who did not change. They were also more complex and included multiple characters and shifts of scene. This heightened dream complexity was characteristic of their REM reports from the start of the study [55]. Their remission could be predicted from the dream-like quality of their reports on the first night of REM awakenings. They seemed to be putting things together in new ways, "changing their minds" during sleep. Those who failed to remit without treatment had short rather stark dreams or no recall at all. These are the depressed that require some treatment intervention, antidepressant medication, psychotherapy, or a therapy directed to dream change [56].

Summary

This article has looked into how one psychologic function of sleep applies to expand the understanding of a number of sleep disorders. Overall, this function integrates new waking experience relevant to the organized self-system, and modulates negative emotion invoked by experience that threatens this program. The reactivation mechanism supports the update of the "underlying strategies that guide behavior" [57] and thereby prepares for more appropriate responses to any challenges the next day. In sleepwalking the arousal out of SWS aborts REM and so stops this process at the beginning of the night's sleep. In nightmare disorder the reactivation matches some current experience to a reminder of an earlier threat to the self-structure. The dream constructed in the late night sleep, when REM is

most intense, has the additive effect of the old and new threats leading to a spontaneous awakening delaying any modification of the underlying self-system. In dream enactment dreams are acted out in response to an overload of new challenges to a system with a genetic deficit in sustaining sleep motor atonia. All these are examples of a failure to sustain sleep because of a high level of some disturbing affect in those with an inherited biologically weakened motor control system either in early NREM sleep, late night REM sleep, or both. In major depression the problem is not primarily one of sustaining sleep but of its timing. The early REM displaces the SWS responsible for the reactivation of relevant waking experience. The REM dream content is either empty or at best simple and bland and is not functional for change in morning mood.

Future research

There is still much work to be done to understand with more precision all of the interactions of brain and behavior in the different organizational states humans cycle through each 24-hour period.

1. The evidence of a role for genetics in several sleep disorders needs further work. As yet there has been no application of the newer SNIPS technique to differentiate between narcolepsy, REM behavior disorder, and sleepwalking, all of which have a common DBQ1 marker using HLA typing.
2. There is likely a genetic differentiation within the group of adult sleepwalkers between those who are and those who are not violent [58]. This should be addressed with better genetic testing.
3. The question of why the male gender is so dominant in adult sleepwalkers also is a topic for further study.
4. Besides the evidence that sleep deprivation and zolpidem are precipitating factors in NREM parasomnias, there is controversy about the role of substances, such as caffeine and alcohol [59–61], which lighten the first NREM sleep and so challenge the old model that it is increased SWS with high thresholds for arousal that are responsible for this disorder rather than low thresholds and the reduced delta activity. Further work exploring the effect of these and other sleep medications is needed to test their potential as triggers for sleepwalking in prone individuals.
5. There is also controversy about the continuity of cognition, motivation, and emotion throughout the sleep-wake cycle based on the data from sleep interruptions across the night. This method may induce continuity because of the memory of what was reported when awakened.

New protocols are needed to control for this experimental artifact.

6. There is evidence that there are individual differences in both the speed and amount of overnight improvement in behavior within normal subjects related to sleep variables other than number of arousals or timing of cycles. The eye movement density is one predictor of learning ability [62]. Further work on the role of more specific sleep variables, such as sleep spindles and K complexes, on learning and memory is indicated [63].

These are a few of the topics for future research to clarify the proposition put forth here: that when sleep is intact, of adequate length, and undisturbed by abnormal arousals, information from waking continues to be actively carried forward through neural circuits allowing it to be sorted, stored, and tempers cooled. Sleep likely performs several psychologic functions. This article focuses on the REM and dream function, for which there is the most data. This explores how sleep processes waking experience that has a negative emotional impact. Recent work studying how more neutral experimental learning tasks effect sleep and subsequent performance is providing information about specific changes in sleep characteristics that take place as one learns. Correcting sleep disorders has the potential of restoring the neuropsychologic system to the fine balance between stability and flexibility of behavior characteristic of humans at their best.

References

- [1] Aserinsky E, Kleitman K. Regularly occurring periods of eye motility and concomitant phenomena during sleep. *Sciences* 1953;118:273–4.
- [2] Dement W, Kleitman N. Cyclic variations in EEG during sleep and their relation to eye movements, body motility and dreaming. *Electroencephalogr Clin Neurophysiol* 1957;9:673–90.
- [3] Freud S. *The interpretation of dreams*. (English translation). New York: Basic Books; 1954. p. 608.
- [4] Foulkes D. *Children's dreams: longitudinal studies*. New York: John Wiley and Sons; 1982.
- [5] West LJ. A general theory of hallucinations and dreams. In: West LJ, editor. *Hallucinations*. New York: Grune and Stratton; 1962. p. 275–91.
- [6] Hobson JA, McCarley R. The brain as a dream state generator: an activation-synthesis hypothesis of the dream process. *Am J Psychiatry* 1977; 134:1335–48.
- [7] Rechtschaffen A, Vogel G, Shaikun G. Interrelatedness of mental activity during sleep. *Arch Gen Psychiatry* 1963;9:536–47.
- [8] Cartwright R. The nature and function of repetitive dreams: a survey and speculation. *Psychiatry* 1979;42:131–7.

- [9] Kramer M. The selective mood regulatory function of dreaming: an update and revision. In: Moffitt A, Kramer M, Hoffman R, editors. *The functions of dreaming*. Albany (NY): State University of New York Press; 1993. p. 139–95.
- [10] Cohen H, Altevogt B, editors. *Sleep disorders and sleep deprivation: an unmet public health problem*. Washington, DC: National Academies Press; 2006.
- [11] Foulkes D. Dream reports from different stages of sleep. *J Abnorm Soc Psychol* 1962;65:14–25.
- [12] Foulkes D. *Dreaming: a cognitive-psychological analysis*. Hillsdale (NJ): Erlbaum; 1985.
- [13] Hartmann E. Dreaming. In: Lee-Chiong T, Sateia M, Carskadon M, editors. *Sleep medicine*. Philadelphia: Hanley and Belfus; 2002. p. 223.
- [14] Breger L. Function of dreams. *J Abnorm Psychol* 1967;72:1–28.
- [15] Reiser M. The dream in contemporary psychiatry. *Am J Psychiatry* 2001;158:351–9.
- [16] Kramer M. *The dream experience: a systematic exploration*. New York: Routledge; 2007. p. 167–87.
- [17] Cartwright R, Young M, Mercer P. The role of REM sleep and dream variables in the prediction of remission from depression. *Psychiatry Res* 1998;80:249–55.
- [18] Ribeiro S, Nicolelis M. Reverberation, storage, and post-synaptic propagation of memories during sleep. *Learn Mem* 2004;11:686–96.
- [19] Giuditta A, Mandile P, Montagnese P, et al. The role of sleep in memory processing: the sequential hypothesis. In: Maquet P, Smith C, Stickgold R, editors. *Sleep and brain plasticity*. New York: Oxford Press; 2003. p. 157–78.
- [20] Cartwright R. Sleep related violence: does the polysomnogram help establish the diagnosis? *Sleep Med* 2000;5:25–32.
- [21] Broughton R. Sleep disorders: disorders of arousal? *Science* 1968;159:1070–8.
- [22] Kales A, Soldates C, Bixler E. Hereditary factors in sleepwalking and night terrors. *Br J Psychiatry* 1980;137:111–8.
- [23] Hublin C, Kaprio J, Partinen M. Prevalence and genetics of sleepwalking: a population based sleep study. *Neurology* 1997;48:177–81.
- [24] Lecendreux M, Bassetti C, Dauvilliers Y. HLA and genetic susceptibility to sleepwalking. *Mol Psychiatry* 2003;8:114–7.
- [25] Guilleminault C, Poyares D, Abat F, et al. Sleep and wakefulness in somnambulism: a spectral analysis study. *J Psychosom Res* 2001;51:411–6.
- [26] Espa F, Ondze B, Deglise P, et al. Sleep architecture, slow wave activity and sleep spindles in adult patients with sleepwalking and sleep terrors. *Clin Neurophysiol* 2000;11:929–39.
- [27] Gaudreau H, Joncas S, Zadra A, et al. Dynamics of slow wave activity during the NREM sleep of sleep walkers and control subjects. *Sleep* 2000; 23:755–60.
- [28] Bassetti C, Vella S, Donati S, et al. SPECT during sleepwalking. *Lancet* 2000;356:484–5.
- [29] Cartwright R. Sleepwalking violence: a sleep disorder, a legal dilemma and a psychological challenge. *Am J Psychiatry* 2004;161:1149–58.
- [30] Ohayon M, Guilleminault C, Priest R. Night terrors, sleep walking and confusional arousals in the general population: their frequency and relationship to other sleep and mental disorders. *J Clin Psychiatry* 1999;60:268–76.
- [31] Pilon M, Zadra A, Adam B, et al. 25 hours of sleep deprivation increases the frequency and complexity of somnambulistic episodes in adult sleepwalkers. *Sleep* 2005;28:A257.
- [32] Broughton R, Billings R, Cartwright R, et al. Homicidal somnambulism: a case report. *Sleep* 1994;17:915–25.
- [33] Nofzinger E, Wettstein R. Homicidal behavior and sleep apnea: a case report and a medicolegal discussion. *Sleep* 1995;18:776–82.
- [34] Pressman M, Meyer T, Kendrick-Mohamed J. Night terrors in an adult precipitated by sleep apnea. *Sleep* 1995;8:773–5.
- [35] Lateef O, Wyatt J, Cartwright R. A case of non-REM parasomnia that resolved with treatment of obstructive sleep apnea. *Chest* 2005; 28:461S.
- [36] Espa F, Dauvilliers Y, Ondze B. Arousal reactions in sleep-walking in adults: the role of respiratory events. *Sleep* 2002;25:871–5.
- [37] Guilleminault C, Palombini L, Pelayo R, et al. Sleepwalking and sleep terrors in prepubertal children: what triggers them? *Pediatrics* 2003; 111:17–25.
- [38] Spoomaker VI, Schredl M, Bout JV. Nightmares: from anxiety symptom to sleep disorder. *Sleep Med Rev* 2005;10:19–31.
- [39] Levin R. Nightmares and schizotypy. *Psychiatry* 1998;61:206–16.
- [40] Zadra A, Donderi DC. Nightmares and bad dreams: their prevalence and relationship to well-being. *J Abnorm Psychol* 2000;109:273–81.
- [41] Nielsen T, Zadra A. Nightmares and other common dream disturbances. In: Kryger M, Roth T, Dement W, editors. *Principles and practice of sleep medicine*. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 926–35.
- [42] Agargun M, Cartwright R. REM sleep, dream variables and suicidality in depressed patients. *Psychiatry Res* 2003;119:33–9.
- [43] Ohayon M, Morselli P, Guilleminault C. Prevalence of nightmares and their relationship to psychopathology and daytime functioning in insomnia subjects. *Sleep* 1997;20:340–8.
- [44] Mellman T, Pigeon W. Dreams and nightmares in posttraumatic stress disorder. In: Kryger M, Roth T, Dement W, editors. *Principles and practice of sleep medicine*. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 573–8.
- [45] Mellman T, David D, Bustemente V. Dreams in the acute aftermath of trauma and their relationship to PTSD. *J Trauma Stress* 2001;14: 241–7.

- [46] Wood J, Bootzin R, Rosenhan D. Effects of the 1989 San Francisco earthquake on frequency and content of nightmares. *J Abnorm Psychol* 1992;101:219–24.
- [47] Hublin C, Kaprio J, Partinen M. Nightmares, familial aggregation and association with psychiatric disorders in a nation wide twin cohort. *Am J Med Genet* 1999;88:329–36.
- [48] Nielson T, Levin R. Nightmares: a new neurocognitive model. *Sleep Med Rev* 2007;11:295–310.
- [49] Kramer M. The nightmare: a failure of dream function. *Dreaming* 1991;1:277–85.
- [50] Perlis M, Nielsen T. Mood regulation, dreaming and nightmares: evaluation of a desensitization function for REM sleep. *Sleep* 1993;3:243–57.
- [51] Schenck C, Bundlie S, Ettinger M, et al. Chronic behavioral disorders of REM sleep: a new category of parasomnia. *Sleep* 1986;9:293–308.
- [52] Schenck C, Bundlie S, Mahowald M. Delayed emergence of a parkinsonian disorder in 38% of 29 older men initially diagnosed with idiopathic rapid eye movement sleep disorder. *Neurology* 1996;46:388–93.
- [53] Kupfer D, Foster F. Interval between the onset of sleep and rapid eye movements as an indicator of depression. *Lancet* 1972;11:684–6.
- [54] Cartwright R, Agargun M, Kirkby J, et al. Relation of dreams to waking concerns. *Psychiatry Res* 2006;141:261–70.
- [55] Cartwright R, Baehr E, Kirkby J, et al. REM sleep reduction, mood regulation and remission in untreated depression. *Psychiatry Res* 2003;12:159–67.
- [56] Cartwright R, Lamberg L. *Crisis dreaming*. New York: Harper Collins; 1992. [reprinted by iUniverse.com Inc. 2000, Lincoln, NB].
- [57] Winson J. *Brain and psyche: the biology of the unconscious*. Garden City (NJ): Anchor Press/Doubleday; 1985.
- [58] Moldofsky H, Gilbert R, Lue F, et al. Sleep-related violence. *Sleep* 1995;18:731–9.
- [59] Pressman M. Factors that pre-dispose, prime and precipitate NREM parasomnias in adults: clinical and forensic implications. *Sleep Med Rev* 2007;11:5–30.
- [60] Cartwright R, Pressman M. Factors that pre-dispose, prime and precipitate NREM parasomnias in adults: clinical and forensic implications [letter]. *Sleep Med Rev* 2007;11:327–9.
- [61] Pressman M. Response to Rosalind Cartwright's letter to the editor. *Sleep Med Rev* 2007;11:329–33.
- [62] Smith C, Nixon M, Nader R. Posttraining increases in REM sleep intensity implicate REM sleep in memory processing and provide a biological marker of learning potential. *Learn Mem* 2004;11:714–9.
- [63] Fogel S, Nader R, Cote K, et al. Sleep spindles and learning potential. *Behav Neurosci* 2007;121:1–10.