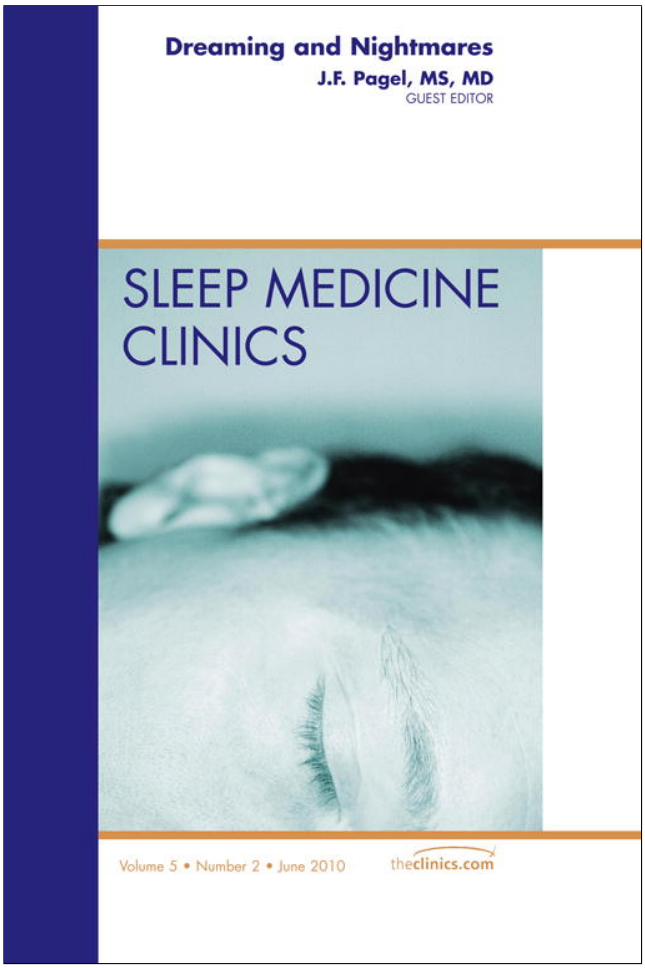

Disturbed Dreaming and Emotion Dysregulation

Ross Levin, PhD[a,*], Gary Fireman, PhD[b], Tore Nielsen, PhD[c,d]

KEYWORDS

• Disturbed dreaming • Emotion dysregulation
• AMPHAC/AND model • REM sleep

Perhaps because of their highly emotive nature and often memorable visual imagery, dreams have long been linked to our innermost emotional functioning, ideas that go back to the earliest use of dreams (as in the Bible) as well as Freud[1,2] and Jung's[3] original psychoanalytic conceptualizations of the human mind. More recently, work by such investigators as Maquet and colleagues[4,5] and Hobson and colleagues[6] have demonstrated an isomorphism between the neurophysiologic components of rapid eye movement (REM) sleep (muscle atonia, heightened limbic activation, particularly in the amygdala, the threat detection control center of the brain, deactivation of the prefrontal dorsolateral cortex, and the reciprocal activation of the amenergic and cholinergic systems) with the phenomenal qualities of dreaming (paralysis, heightened expressed emotion, the absence of metacognition and state-dependent amnesia for the dream on awakening). Thus, the neural qualities of REM sleep seem to be particularly suited for the activation of emotionally charged memorial components that may offer clues into a possible function.

Previous work by Kramer[7] and Cartwright[8,9] has long assigned a mood-regulatory function to dreaming. A review of this work is beyond the scope of the present paper, but Kramer and Cartwright have proposed that dreaming serves a type of emotional thermostat that serves to regulate inner well-being. In a series of studies, Kramer (reviewed in Ref.[7]) demonstrated clear markers of emotion shift from evening to morning that was mediated by dream variables. For example, Kramer demonstrated that the absence of dream characters in the last REM dream of the night was the strongest predictor of a downshift of mood from evening to morning. This finding is consistent with the observation that morning mood is often the worst time for individuals with depression and the findings that depressed individuals have more total REM time and significantly shorter REM latency than individuals without depression or the same individuals after remission of symptoms.[10]

Closely related but independent work by Cartwright and colleagues[8,9,11] with samples of women undergoing midlife divorce and marital separation with and without accompanying depression has shown that the level of intensity of dysphoric mood in their dreams as well as dreaming of the ex-spouse reliably predicted waking depression but that the presence of these variables also predicted enhanced recovery on follow up 1 year after divorce, suggesting that the dreams were integral in processing these negative emotions.

Another area of promising work along these lines pertains to the small literature on recurrent dreaming, loosely defined as dreams with a high degree of replicative content. Some studies in this area[12–14] suggest that the active presence of

The first articulation of the AMPHAC/AND model of disturbed dreaming was presented by Levin & Nielsen (2007) in *Psychological Bulletin* and Nielsen & Levin (2007) in *Sleep Medicine Reviews*.

[a] 25 West 86th Street, Suite #3, New York, NY 10024, USA
[b] Department of Psychology, Suffolk University, Boston, MA, USA
[c] Department of Psychiatry, University of Montreal, Montreal, Canada
[d] Dream and Nightmare Lab, Sacre-Coeur Hospital, Montreal, Canada
* Corresponding author.
E-mail address: rosslevin@drrosslevin.com

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recurrent dreams (roughly defined as dreaming the same dream over and over again) connotes a psychological/emotional obstacle that is associated concomitantly with poorer daytime functioning. Once recurrent dreams end, improved waking psychological functioning is often noted.[12]

One obvious limitation to this work is the inability to disentangle the effects of dreaming from their neurophysiologic background, namely REM sleep, the sleep stage most highly associated with dreaming. There is mounting evidence that healthy sleep is integral to healthy emotional functioning and that waking states marked by mood disturbances of emotional dysregulation are often accompanied by and causally preceded by poor sleep.[15–18] In addition, it seems that intact sleep is crucial to the encoding and consolidation of intact memories (eg, Ref.[19]). This is important given the intimate connection between memory and affect regulation, particularly for such clinical disorders as posttraumatic stress disorder (PTSD) and depression.

The present paper focuses on another type of dreaming often implicated in emotion functioning, namely disturbed dreaming (DD), and a recently formulated model of the pathogenesis of these dreams specifically and all dreaming more generally is reviewed (see Refs.[20–23] for more comprehensive discussion of the model). A primary assumption of this model is that dreams have an adaptive function independent from REM sleep (although closely entrained to REM): the reduction or even extinction of fear memories. DDs, vivid dreams often marked by intense dysphoric emotion (predominantly fear but also including rage, guilt, and grief), represent a dysfunction of this regulatory process and are often engendered by high levels of waking stress. For our purposes, the domain of DD includes the broad spectrum of dysphoric dreaming ranging from dreams that are remembered only on awakening (bad dreams) to dreams that result in a nocturnal awakening (nightmares). Although occasional DDs are nearly ubiquitous in the general population,[24] high DD incidence rates (usually defined as weekly episodes) are strongly associated with poorer waking psychological well-being. In addition, DDs (recurrent nightmares in particular) are a defining symptom of PTSD.[20] The authors thus consider all forms of disturbed dreaming to be observable variants of a common underlying process, namely dysphoric imagery produced during sleep, and that the consequences of these variants are largely dictated by waking responses to the imagery (eg, distress).

Perhaps the most robust finding in the DD literature is the strong association between DD frequency and waking psychopathology.[20,21,23,25–31] Because most of these clinical disorders are marked by considerable waking emotional distress, their association with DD suggests that these dreams are related to a personality style characterized by intense reactive emotional distress.[20,23,26,28,32,33] Furthermore, DDs are often precipitated by stressful life events[25,27,34] and there is a strong link between trauma exposure and subsequent DD.[35,36]

A naturalistic study by Wood and colleagues,[37] clearly showed the relation between heightened life stress and increased DD production. They found the incidence of DD to be twice as high immediately after the 1989 San Francisco earthquake in 2 groups in the San Francisco Bay area than in a sample from Arizona, despite equal baseline frequencies. These differences were dose-response specific to proximity to the earthquake epicenter; those who lived closer to the epicenter reported more nightmares.

THE AMPHAC/AND MODEL OF DD GENERATION

Despite a recent proliferation of experimental work on DDs, their pathogenesis has remained largely unexplained. A recently proposed model incorporating advances in cognitive neuroscience, sleep neurophysiology, and fear conditioning, particularly in relation to PTSD and sociocognitive-based diathesis (ie, vulnerability) stress models of psychopathology, supports a multilevel model of dream function and DD production that unites neural and cognitive processes in waking and sleeping.[20,21,23,29,38] The neurophysiologic branch of this model is termed the AMPHAC network, after its presumed underlying neurophysiologic centers: the amygdala (A), the medial prefrontal cortex (MP), the hippocampus (H), and the anterior cingulated cortex (AC). The cognitive branch is termed the Affect Network Dysfunction (AND) model. Together, the 2 branches integrate explanatory concepts at a neural level (ie, a cohesive and interconnected network of limbic and forebrain centers underlying emotional expression and representation) and a cognitive level (ie, a dream production system that transforms fear memories into dream and nightmare imagery) to account for a variety of features associated with nightmare imagery (lack of emotional control, bizarre features, or replay of traumatic memories).

The AMPHAC/AND model stipulates that DD results from dysfunction in a network of affective processes that, during normal dreaming, are presumed to serve the adaptive function of fear

memory regulation and extinction. The underlying neurophysiology and biochemistry of REM sleep seems to be primed to activate these systems.[4,6] At the cognitive level, dreaming is proposed to facilitate fear memory extinction by 3 processes: memory element activation, memory element reactivation, and emotional expression. These processes are discussed in greater depth in the following sections.

Memory Element Activation

The first set of processes refers to the increased availability during dreaming of a wide range of memory elements. Memory elements, rather than complete memories, are emphasized, as complete episodic memories do not typically appear during dreaming.[39] Dreaming tends to express memory elements as though original memories had been reduced to more basic units.[40,41] Often, these appear as isolated features, such as an attribute of a familiar place or character. In other instances, several elements may appear together by virtue of their origin in a single past event or their grouping by some other form of organization, such as a script, or a semantic or phonological category. Although memory elements tend to obscure the relation of dreaming to daily experiences, the link is demonstrable by clinical and experimental observation. Clinically, Hartmann[42] has demonstrated that dreams often portray elements of a person's main emotional concerns (eg, stress, trauma) even if visual or auditory details of a specific memory are absent. Experimentally, memory elements have been detected as the day-residues of previous-day experiences.[43–45] Further, these residues may be temporally delayed by up to a week.[40,46] It remains unknown why normal dreaming disproportionately favors the partial activation of memory elements. One possibility is that it reflects a more general organizing principle of memory. For instance, declarative memories may be stored as multiple traces in which bits and pieces of a single experience are saved by structurally distinct memory systems.[47] In the waking state, episodic memories are then reconstituted when needed from the elements stored in these different systems.[48] In the dreaming state, memory elements may well be reconstituted in an alternative fashion, perhaps randomly,[6] perhaps linked metaphorically,[42] perhaps combined into composite context memories.[49] All of these possibilities may be true to some extent; elements may be activated as a function of emotional concerns[50] but with the possible introduction of some pseudo-random and incompatible associations. The net effect of this organization is to create novel, nonaversive contexts that facilitate fear extinction, particularly under conditions of heightened stress activation. In this way, the authors suggest that dreaming represents an endogenous form of self-correction or equilibration.[51]

An early conceptualization of fear memory organization referred to as fear memory structures[52,53] may help to understand the production of DD. Foa and Kozak[52] describe fear memory structures as networks of information that unite memory elements about (1) a feared stimulus situation (stimulus elements), (2) physiologic, verbal, and behavioral responses to that situation (response elements), and (3) the meaning of these stimuli and responses (meaning elements). During waking, fear memory structures bias the interpretation of new information by enhancing sensitivity and attention to a structure's stimulus elements, thus ensuring the allocation of more cognitive resources to the processing of this new information. Further, activation of these structures interfere with access to resources necessary for competing tasks, as exemplified by the response deficits shown by patients with PTSD on the emotional Stroop task.[54,55]

The construct of fear memory structures has been validated in some respects however, whereas the internal coherence of fear memory structures was believed to be modified or weakened by the introduction of incompatible elements, thus alleviating associated pathologic symptoms.[52] Recent work in experimental psychopathology suggests that these structures may be replaced by the more neurologically specific concept of extinction memories that inhibit fear memories.[56–58]

During dreaming, fear memories seem to vary in how completely and coherently their stimulus and response elements are expressed. Although minimal activation of these structures may trigger a mild anxiety dream in which a limited number of stimulus or response elements are activated, more extensive activation would result in a more intense, nontraumatic nightmare. Under conditions of minimal activation, a few fear memory elements may be expressed in relative isolation and in no coherent order, that is, as common residues. With extensive activation, more elements may be expressed and their order more veridical, rendering the form of the fear memory more easily identifiable from the nightmare's theme. From this model it is predicted that individuals with particularly problematic and coherent fear memories, including persons with specific phobias, ongoing interpersonal difficulties or other current sources of stress, should experience more recurrent dreams and dream themes.

When the level of waking stress reaches extreme levels as in trauma exposure, the fear memory elements may be activated globally and in a highly coherent manner resulting in a nightmare that reproduces the extreme memory with appropriate fear context, bodily reactions and, cognitive interpretations. This type of comprehensive activation is illustrated by PTSD nightmares that seem to replay large portions of the original trauma.[59] Between mild anxiety dreams and intense PTSD nightmares are various types of dysphoric dreams and nightmares that have fear memory elements either alone or in combination and with varying degrees of organization. Examples include dreams or nightmares with recurring objects, characters and themes,[60] typical dream themes,[46] and story- and script-like structures.[61] This ensemble of emotional imagery processes constituting fear memories most likely underlies a variety of clinical conditions characterized by anxiety and fear, such as panic disorder, phobia, and PTSD[57,62] as well as dysphoric dreams and nightmares.

Memory Element Recombination

The second set of processes, responsible for the continuous assembly of memory elements into a constant flow of dream imagery, was termed "condensation"[1,3] and described as the merging of several separate (although motivationally linked) images into a single image. The authors propose that a similar type of reorganization that produces new image contexts during dreaming, much like the remapping of conjunctive representations under control of the hippocampus, occurs during the waking state. During dreaming, conjunctive representations are rendered into virtual simulations or here-and-now illusions[40] to maximize their effect on the amygdala, which favors perceptual, rather than imaginal, stimuli.[63] They are recombined or remapped to introduce elements that are incompatible with existing fear memories, thus facilitating (among other functions) the acquisition or maintenance of extinction memories. The latter inhibit fear memories and consequently, alleviate affect load. Recombinations of memory elements give dreams at once their alien and their familiar quality. Three features of recombination are especially pertinent to the pathology of DDs.

Unlikely combinations

The first is the de novo conjunctions of features, many of which produce dream experience that seems bizarre, incongruous, or incompatible with waking-life experience. Bizarreness is frequent in dreams[64–66] and dreams are significantly more bizarre than waking daydreams.[67] Although a widely accepted explanation for bizarreness is still lacking, 1 possibility is that bizarreness reflects the relative inactivity of dorsolateral prefrontal executive functioning during REM sleep.[6] Another possibility is that REM sleep selectively permits weakly associated (and thus possibly disjunctive) memory elements to become associated.[68] Regardless, the authors propose that the unlikely combinations of disparate memory elements facilitate acquisition and maintenance of conditioned fear memories and thus of fear responding. Bizarreness may be an inevitable consequence of this mechanism and we would expect to see higher levels of bizarreness in dreams of individuals with high affect load. As this question has not been directly investigated, it remains unknown whether nightmares are more or less bizarre than non-nightmare dreams in this specific sense of recombined elements. To the extent that nightmares replay fear memories or possess recurrent elements they would seem to be less, not more, organizationally bizarre. Further empirical investigation of the organizational coherence of nightmares and normal dreams among individuals suffering from frequent nightmares or from conditions marked by high fear coherence in waking states, such as PTSD or specific phobias, could help elucidate these mechanisms.

Fear memory templates

A second salient feature of recombination is the organizing influence of fear memory and other emotional memory structures. Although they are not usually expressed fully as memory replays, fear memories may act as organizing templates that structure dreams and within which other isolated and frequently incompatible memory elements are ordered and interrelated. As a result, fear-producing stimuli and their physiologic responses will be repeatedly paired with alternative, nonaversive contexts and thus extinguished gradually over time. Although the specific nature of such a mechanism remains speculative, phenomenological features of dream organization belie their presence. On the one hand, fear memories may assume a habitual easily recognized form and express a consistent emotional content in the dream, such as with themes of public nudity, being late, or being pursued.[69] Such themes recur frequently and are associated with diminished psychological well-being until they cease, at which time well-being is high.[12,30] On the other hand, fear memories may portray novel organizations in which a skeletal structure incorporates many unexpected elements, such as an interpersonal attack scenario that introduces many unanticipated characters and produces many unusual

consequences. The latter type of dream has been labeled as problem-solving and found to be associated with emotional adaptation.[7,70] Fear extinction is more likely to be associated with the latter type of dream and less likely to be associated with the former, although again, there is no research that addresses this issue directly. One useful line of investigation would be to compare organizational coherence of the dreams, nightmares, and daytime narratives of individuals who have frequent nightmares and report high or low levels of accompanying distress to determine whether memory organization corresponds to waking emotional reactivity. Because fear memories are purportedly responsible for the nonconscious detection of threat, it would also be informative to investigate whether individuals with high nightmare distress perform similar to individuals with anxiety disorders or PTSD on an affective backward masking paradigm or the emotional color-word Stroop test.

Reality simulation

A third important feature of dream imagery recombination is that the new image sequences consist, for the most part, of lifelike simulations of first-person reality. Memory elements are recombined on various levels of organization (eg, perceptual, schematic, thematic, symbolic) to produce coherent, continuous simulations of waking-life experience. Functionally, reconstituting disparate memory elements into virtual simulations may facilitate the creation or strengthening of new memory links and aid in the simulation of threats to species survival, thus optimizing off-line rehearsal of behavioral avoidance responses.[71,72] Thus, reality mimesis ensures that fear memories are processed in a phenomenological medium similar to that in which they were first formed. This process allows for the modification or integration of disturbing emotions during dreaming[8,42] in a fashion analogous to that induced by exposure therapy for waking-state fear-based disorders.[52] The finding that imagery rehearsal is highly effective in reducing recurrent nightmares in individuals with PTSD[73] is consistent with this formulation.

Emotional Expression

There is disagreement on whether emotions drive the selection of dream contents[42,74] or whether they arise later, in reaction to these contents.[75] Our view is that both occur in a progressive interactive expression of fear memories. Because stimulus and response elements are often encoded in a single fear memory,[76] activation of 1 type of element should activate the other. The notion of fear extinction implies a mechanism that produces a mimesis of the waking perception of emotional events, that is, in which stimulus elements preferentially lead to activation of response elements. This ordering maximizes the involvement of the amygdala, which responds preferentially to perceptual stimuli, and thus facilitates regulation of affect load. Emotional expression during dreaming is integral in dreaming's function of fear extinction. The emotions appearing in normal dreams are predominantly dysphoric (eg, two-thirds of normal dreams[60,77] with fear being by far the most prevalent[69]), which is consistent with this suggestion, as is the frequent occurrence of nondistressing bad dreams and nightmares. The authors consider normal dysphoric dreams, bad dreams, and nondistressing, nontraumatic nightmares to be related in this respect. The variable intensity of fear expression in these types of dreams may simply reflect variations in the strength or efficacy of the hypothesized fear extinction function, which is presumed to vary in step with an individual's day-to-day emotional requirements. In contrast, more severe nightmares, such as nontraumatic nightmares with high distress, PTSD nightmares, and extreme PTSD nightmares, are assumed to be shaped additionally by the presence of additional waking distress and/or previous trauma.

NIGHTMARES ARE PATHOLOGIC EXPRESSIONS OF FEAR MEMORIES

Pathogenic Changes Common to All Nightmares

Although fear memories are considered to be a normal phenomenon of human memory, they become pathologic when (1) they are highly coherent and resistant to extinction, and (2) they contain an excessive number of response elements.[52,78] The authors propose that individual vulnerability (ie, high levels of affect load and/or affect distress) interacts with the neurophysiologic state of REM sleep to favor the activation during nightmares of highly coherent fear memories, akin to those occurring in waking, fear-based pathologic conditions. Accordingly, the authors propose that nightmare-related fear memories are highly resistant to extinction, overly weighted with response elements (usually involving escape or avoidance) and, in more severe instances, corrupted by affect distress.

Increased resistance to extinction is reflected in several possible pathologic events. First, during dream formation there may be a marked bias to activate complete fear memories rather than isolated elements of fear memories. Traumatic

memories in particular preserve their structural coherence,[53] perhaps as a result of conditions of heightened arousal during encoding, and thereby enter dreams as apparent replays of the original trauma. This replay is accompanied by a sense of perceptual reinstatement and distressing emotions. Novel configurations, including incompatible elements, are thus less likely to be introduced and thus less able to permit acquisition of new extinction memories. Second, the fear memory may simply resist activation altogether. Because availability of a fear memory is a prerequisite for the successful acquisition of an extinction memory,[52,53] a lack of extinction may occur if the fear memory is not fully activated either during dreaming or later, during the waking state. Awakening from a nightmare may cut short fear memory activation and thereby prevent extinction. In addition, the awakening may actually strengthen the fear by serving as an avoidance response. Similarly, avoiding the recall of nightmares on awakening may prevent the eventual extinction of its underlying fear memory.

Increase in fear memory response elements is reflected in several indicators: an increase in the frequency and intensity of motor imagery in nightmares (eg, escaping, defending oneself, fighting, attempting to scream); increased activation of the sleep state as signaled by physiologic measures (eg, heightened autonomic arousal); and the overt expression of sleep behaviors, such as moving in bed, speaking, and emoting. These response elements are often the identifiable correlates of distress that individuals report experiencing during and following their nightmares. A preponderance of response elements may result from a failure of recombinatory processes to limit the number of response elements that are activated and introduced into the narrative.

The production of emotions during dreaming is compounded by the facilitating influence of high levels of waking affect distress. Affect distress elements may become incorporated into an individual's fear memories and other emotional structures such that, when a fear memory is activated, emotional responses will come to include expressions of distress as well as fear. An individual high in affect distress will therefore experience distress whenever certain fear memories are activated either during a nightmare or later in the day, when the nightmare is recalled. The distress experienced may even lead to further, similar nightmare episodes with recurrent themes. This cyclical process is consistent with the finding that intrusive imagery facilitates the release of stress hormones that heightens affect distress and potentiates further intrusive imagery.[79,80] Affect distress may thereby contribute to the pathologic portrait of an individual's nightmare disorder, including its cyclical nature.

Pathogenic Changes in Nontraumatic Nightmares

Nightmares that are nontraumatic and associated with low waking distress are produced by an intensification of the memory element activation/recombination mechanisms related to normal dreaming and modulated by levels of waking stress (affect load). The authors propose that affect load increases with short-term accumulations of interpersonal conflicts, current affective memory demands, and emotional reactions to transitory stressors. Affect load interacts primarily with the stimulus and contextual elements of fear memories such that high affect load may disrupt activation and recombination of dreamed stimuli, rather than responses. This may have the effect of producing recurrent, typical, bizarre, or macabre imagery with mild anxiety or fear, but not distress. Dreams with little emotional activation may be associated with greater fear memory resistance to extinction than more emotional dreams and nightmares.

Nontraumatic nightmares with high waking distress involve affect distress mechanisms in addition to fear memories and affect load mechanisms. As mentioned earlier, affect distress influences primarily the response elements of fear memories such that subjects high on this trait respond with more subjective upset during and following their nightmares. Thus, the activation of nightmare-related fear memories of individuals high in affect distress may have inadvertent effects during the waking state, such as the stimulation of various conditioned expectancies and biases for the recall and perception of fear-relevant stimuli. Although these processes have not been investigated directly in frequent nightmare individuals, there is abundant evidence that (1) negatively arousing memories are recalled with greater clarity than neutral ones, particularly for memories of high personal significance[81]; and (2) individuals with vivid imagistic abilities, a quality that characterizes frequent nightmare individuals, demonstrate heightened autonomic and emotional activation when presented with fear-relevant stimuli.[82]

Pathogenic Changes in Traumatic Nightmares

Trauma is hypothesized to cause an underlying fear memory to become firmly entrenched and highly resistant to extinction. This may mean that there is a diminution of recombinatory dream elements and thereby an increase of fear memory

coherence. Degree of fear memory coherence is believed to underlie the severity of PTSD symptoms.[62] The authors suggest that fear memory resistance to extinction is responsible for the finding that PTSD nightmare content seems to replicate the original trauma.[59] A similar process may underlie memory more generally. For example, highly specific autobiographical memories are often associated with higher levels of emotional distress.[53] Thus, in contrast to the variable progression of dysphoric imagery seen in nontraumatic nightmares, PTSD nightmares are more likely to be realistic and predictable as a result of the activation of structurally coherent fear memories.

Second, in the case of traumatic nightmares, the response elements of underlying fear memories may be especially salient and amplified by affect distress. This is suggested by the presence of several sleep-related hyperarousal symptoms, including increased awakenings, wake after sleep onset and insomnia, as well as nightmares in stages other than REM sleep and at times other than the habitual last third of the night; for example, stage 2 nightmares occurring early in the sleep episode.[83] It is also suggested by the expression of motor activity in sleep, including more frequent REM-related twitches in leg muscles, more periodic leg movements in sleep in all stages, more frequent gross body movements, and more REM-related motor activity and vocalizations.[84] That PTSD is comorbid with many cases of REM sleep behavior disorder,[85] which is characterized by motorically active dreams and diminished REM sleep muscle atonia, further implicates excessive response elements and distress in PTSD nightmare formation.

The spectrum of dysphoric dreams that includes nontraumatic and traumatic nightmares may be attributed to interactions between fear memories, short-term accumulations of affect load, and a pathogenic distress diathesis in vulnerable individuals. Fear memory coherence and resistance to extinction may be a factor common to all types of dysphoric dreaming, whereas affect distress distinguishes pathologic from nonpathologic nightmares. In addition to the pathologic sleep changes described earlier, it is also highly likely that these processes interact in various ways during waking states, and that sleep- and waking-state interactions among processes also occur. To illustrate, phobic individuals who selectively process phobic threat cues and focus narrowly on stimuli that activate their underlying phobia-relevant fear memories may apply the same acquired encoding biases to selectively scan their dream imagery for threats and to reflect on their recalled nightmares with a similar narrow focus. As a result, such individuals may experience nightmares as more threatening and distressing than do other types of individuals and may be more likely to misattribute their endogenous imagery to actual environmental threats; a type of source-monitoring deficit documented for normal dreaming.[86] Further, the physiologic conditions of REM sleep may facilitate this process. Thus, nightmares can be likened to false alarm responses, in a manner similar to the false alarm responses of panic disorder.[87]

NEURAL LEVEL EXPLANATION: A BRAIN NETWORK FOR FEAR IMAGERY

Recent research on the brain correlates of emotion, fear memory, PTSD, and human sleep and dreaming, has begun to clarify the nature of normal and posttraumatic emotional processing during sleep. Much of this work suggests that DDs may result from disturbances in a network of brain regions controlling the processing of fear and distress, namely a brain network of limbic, paralimbic, and prefrontal regions that constitutes the control center for several emotional processes, including the perception and representation of emotional stimuli and the expression and regulation of emotional responses. Although still preliminary, these structures likely include the amygdala and its medial prefrontal cortex extension, as well as the hippocampal complex and its anterior cingulate cortex extension.[88,89]

The authors suggest that the 4 designated brain regions operate synergistically as part of a larger emotional control structure, which in turn influences other perceptual, cognitive, memorial, and affective brain events. These conclusions are based on several anatomic and functional considerations. First, these regions are interconnected anatomically and functionally.[90] Amygdala, in particular, is massively connected to the other regions in a reciprocal fashion[91] and all 4 regions are functionally connected to sensory, motor, and autonomic brain regions, and are thus well-suited to mediate higher cognitive functions, behaviors, and affective responses. For example, the hippocampus and amygdala are now considered to be integral in basic dream production.[6,40] In addition, all 4 neural regions are crucial in the formation and regulation of normal emotions, fear in particular,[63] with the amygdala being central in this process, likely by virtue of its connections with hypothalamic structures.[91] The medial prefrontal cortex and hippocampus are critical for the acquisition and memory of conditioned fear and fear extinction, whereas the anterior

cingulated cortex (ACC) seems to be crucial for mediating affect distress.[92] These regions are associated with state and trait individual differences in emotional responding, thus allowing for future direct tests of cross-state continuity between waking functioning and neural and structural changes in the brain. Not surprisingly, these regions are also implicated in emotion-based disorders including, but not limited to, anxiety disorders (generalized anxiety, social anxiety, phobia, panic, obsessive-compulsive disorder), mood disorders (depression, bipolar disorder), personality disorders (borderline, psychopathy) and most importantly for our discussion, PTSD. Although the exact neurophysiologic mechanisms underlying emotion-based personality attributes and anxiety disorders remain a topic of intensive investigation, recent work in PTSD brain functioning is instructive for application to the pathogenesis of DD. One leading hypothesis for PTSD formation[89,93] is that symptoms result from a hyper-responsivity of the amygdala to threat stimuli, leading to exaggerated symptoms of arousal and distress, coupled with a failure of the other brain regions (hippocampus, medial prefrontal cortex, ACC) to adequately dampen this activation. A similar pathologic mechanism may explain the generation of DDs with the amygdala becoming increasingly responsive to fear-related memory elements portrayed in the dream, while its regulation by medial prefrontal cortex, hippocampus, and ACC is disturbed in some way. Severe and traumatic nightmares are particularly affected by disturbance of the ACC, which amplifies the intensity of the distress within the actual dream and on awakening from the dream. Imaging studies have also shown that activity levels in the 4 AMPHAC regions increase during REM sleep higher than levels seen in either wakefulness or non-REM sleep.[4,6,94,95]

SUMMARY

The AMPHAC/AND network is a vital component of the physiologic infrastructure of normal dreaming and likely influences the shaping of emotional imagery during normal and DD. By its endemic nature, dreaming is a naturally occurring self-regulatory process that may operate much like the emotional processing and habituation or desensitization that occurs during exposure therapy.[51,96] Thus, the nature and quality of REM sleep in particular likely interacts with these brain regions in the formation of dream imagery to facilitate the reduction or even elimination of fear-based memories in an ongoing attempt at achieving emotional homeostasis and optimize survival function.

REFERENCES

1. Freud S. The interpretation of dreams. New York: Basic Books; 1955.
2. Gilboa A, Shalev AY, Laor L, et al. Functional connectivity of the prefrontal cortex and the amygdala in posttraumatic stress disorder. Biol Psychiatry 2004;55:263–72.
3. Jung CG. Dreams: the collected works of C.G. Jung. Princeton (NJ): Princeton University Press; 1974.
4. Maquet P, Péters J-M, Aerts J, et al. Functional neuroanatomy of human rapid-eye-movement sleep and dreaming. J Cogn Neurosci 1996;383: 163–6.
5. McGaugh JL. The amygdala modulates the consolidation of memories of emotionally arousing experiences. Annu Rev Neurosci 2004;27:1–28.
6. Hobson JA, Pace-Schott E, Stickgold R. Dreaming and the brain: towards a cognitive neuroscience of conscious states. Behav Brain Sci 2000;23: 793–842.
7. Kramer M. The selective mood regulatory function of dreaming: an update and revision. In: Moffitt A, Kramer M, Hoffmann R, editors. The functions of dreaming. Albany (NY): State University of New York; 1993. p. 139–96.
8. Cartwright R. Dreaming as a mood regulation system. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 565–72.
9. Cartwright RD. Affect and dream work from an information processing point of view. J Mind Behav 1986; 7:411–27.
10. Vogel GW. A review of REM sleep deprivation. Arch Gen Psychiatry 1975;32:749–61.
11. Cartwright RD, Kravitz HM, Eastman CI, et al. REM latency and the recovery from depression: getting over divorce. Am J Psychiatry 1991;148: 1530–5.
12. Brown RJ, Donderi DC. Dream content and self-reported well-being among recurrent dreamers, past-recurrent dreamers, and nonrecurrent dreamers. J Pers Soc Psychol 1986;50:612–23.
13. Cartwright RD. The nature and function of repetitive dreams: a survey and speculation. Psychiatry 1979; 42:131–7.
14. Zadra A, Desjardins S, Marcotte E. Evolutionary function of dreams: a test of the threat simulation theory in recurrent dreams. Conscious Cogn 2006; 15:450–63.
15. Bremner JD, Vythilingam M, Vermetten E, et al. Neural correlates of declarative memory for emotionally valenced words in women with posttraumatic

stress disorder related to early childhood sexual abuse. Biol Psychiatry 2003;53:879–89.
16. Breslau N, Chilcoat HD, Kessler RC, et al. Previous exposure to trauma and PTSD effects of subsequent trauma: results from the Detroit area survey of trauma. Am J Psychiatry 1999;156:902–7.
17. Breslau N, Roth T, Rosenthal L, et al. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. Biol Psychiatry 1996;39:411–8.
18. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders: an opportunity for prevention? JAMA 1989;262:1479–84.
19. Walker MP, Stickhold R. Sleep, memory and plasticity. Annu Rev Psychol 2006;57:139–66.
20. Levin R, Nielsen T. Disturbed dreaming, posttraumatic stress disorder, and affect distress: a review and neurocognitive model. Psychol Bull 2007;133: 482–528.
21. Levin R, Nielsen T. Nightmares, bad dreams, and emotion dysregulation. Curr Dir Psychol Sci 2009; 18:84–8.
22. Liberzon I, Phan KL. Brain-imaging studies of posttraumatic stress disorder. CNS Spectr 2003;8:641–50.
23. Nielsen T, Levin R. Nightmares: a new neurocognitive model. Sleep Med Rev 2007;11:295–310.
24. Levin R. Sleep and dreaming characteristics of frequent nightmare subjects in a university population. Dreaming 1994;4:127–37.
25. Berquier A, Ashton R. Characteristics of the frequent nightmare sufferer. J Abnorm Psychol 1992;101: 246–50.
26. Blagrove M, Farmer L, Williams E. The relationship of nightmare frequency and nightmare distress to well-being. J Sleep Res 2004;13:129–36.
27. Hartmann E, Russ D, Oldfield M, et al. Who has nightmares? The personality of the lifelong nightmare sufferer. Arch Gen Psychiatry 1987;44:49–56.
28. Levin R, Fireman G. Nightmare prevalence, nightmare distress, and self-reported psychological disturbance. Sleep 2002;25:205–12.
29. Nielsen T, Levin R. The dimensional nature of disturbed dreaming: reply to Weiss (2007). Psychol Bull 2007;133:533–4.
30. Zadra A, Donderi D. Nightmares and bad dreams: their prevalence and relationship to well-being. J Abnorm Psychol 2000;109:273–81.
31. Nielsen TA, Laberge L, Tremblay R, et al. Development of disturbing dreams during adolescence and their relationship to anxiety symptoms. Sleep 2000;23:727–36.
32. Beauchemin K, Hays P. Prevailing mood, mood changes and dreams in bipolar disorder. J Affect Disord 1995;35:41–9.
33. Belicki K. Nightmare frequency versus nightmare distress: relations to psychopathology and cognitive style. J Abnorm Psychol 1992;101:592–7.
34. Levin R. Ego boundary impairment and thought disorder in frequent nightmare sufferers. Psychoanal Psychol 1990;7:529–43.
35. Mellman TA, David D, Kulick-Bell R, et al. Sleep disturbance and its relationship to psychiatric morbidity after hurricane Andrew. Am J Psychiatry 1995;152:1659–63.
36. Woodward SH, Arsenault NJ, Murray C, et al. Laboratory sleep correlates of nightmare complaint in PTSD inpatients. Biol Psychiatry 2000;48:1081–7.
37. Wood JM, Bootzin RR, Rosenhan D, et al. Effects of the 1989 San Francisco earthquake on frequency and content of nightmares. J Abnorm Psychol 1992;101:219–24.
38. Levin R, Fireman G, Pope A. Disturbed Dreaming. Front Neurosci 2009;3:448–9.
39. Fosse MJ, Fosse R, Hobson JA, et al. Dreaming and episodic memory: a functional dissociation? J Cogn Neurosci 2003;15:1–9.
40. Nielsen TA, Stenstrom P. What are the memory sources of dreaming? Nature 2005;437:34–8.
41. Nielsen TA, Zadra AL. Nightmares and other common dream disturbances. In: Kryger M, Roth N, Dement WC, editors. Principles and practice of sleep medicine. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 926–35.
42. Hartmann E. Dreams and nightmares: the new theory on the origin and meaning of dreams. New York: Plenum; 1998.
43. Cipolli C, Bolzani R, Tuozzi G, et al. Active processing of declarative knowledge during REM-sleep dreaming. J Sleep Res 2001;10:277–84.
44. Hoelscher TJ, Klinger E, Barta SG. Incorporation of concern and nonconcern-related verbal stimuli into dream content. J Abnorm Psychol 1981;90:88–91.
45. Saredi R, Baylor GW, Meier B, et al. Current concerns and REM-dreams: a laboratory study of dream incubation. Dreaming 1997;7:195–208.
46. Nielsen TA, Kuiken D, Alain G, et al. Immediate and delayed incorporations of events into dreams: further replication and implications for dream function. J Sleep Res 2004;13:327–36.
47. Schacter DL, Tulving E. Memory systems 1994. Cambridge (MA): MIT Press; 1994.
48. Nadel L, Moscovitch M. Hippocampal contributions to cortical plasticity. Neuropharmacology 1998;37:431–9.
49. Johnson JD. REM sleep and the development of context memory. Med Hypotheses 2005;64:499–504.
50. Klinger E. Daydreaming. Using waking fantasy and imagery for self-knowledge and creativity. Los Angeles (CA): Jeremy P. Tarcher; 1990.
51. Walker MP, van der Helm E. Overnight therapy? The role of sleep in emotional brain processing. Psychol Bull 2009;135:731–48.
52. Foa EB, Kozak MJ. Emotional processing of fear: exposure to corrective information. Psychol Bull 1986;99:20–35.

53. Lang PJ. Presidential address, 1978. A bio-informational theory of emotional imagery. Psychophysiology 1979;16:495–512.
54. Foa EB, Feske U, Murdock TB, et al. Processing of threat-related information in rape victims. J Abnorm Psychol 1991;100:156–62.
55. McNally RJ. Experimental approaches to cognitive abnormality in posttraumatic stress disorder. Clin Psychol Rev 1998;18:971–82.
56. Lang PJ, Davis M, Ohman A. Fear and anxiety: animal models and human cognitive psychophysiology. J Affect Disord 2000;61:137–59.
57. Mineka S, Zimbarg R. A contemporary learning theory perspective on the etiology of anxiety disorders: it's not what you thought it was. Am Psychol 2006;61:10–26.
58. Ohman A, Mineka S. Fears, phobias and preparedness: towards an evolved module of fear and fear learning. Psychol Rev 2001;108: 483–522.
59. Mellman TA, Pigeon WR. Dreams and nightmares in posttraumatic stress disorder. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine. 4th edition. Philadelphia: Elsevier Saunders; 2005. p. 573–8.
60. Domhoff GW. Finding meaning in dreams. A quantitative approach. New York: Plenum; 1996.
61. Cipolli C, Bolzani R, Tuozzi G. Story-like organization of dream experience in different periods of REM sleep. J Sleep Res 1998;7:13–9.
62. Foa EB, Meadows EA. Psychosocial treatments for post-traumatic stress disorder: a critical review. Annu Rev Psychol 1997;48:449–80.
63. Phan KL, Wager T, Taylor SF, et al. Functional neuroanatomy of emotion: a meta-analysis of emotion activation studies in PET and fMRI. Neuroimage 2002; 16:331–48.
64. Levin R, Livingston G. Concordance between two measures of dream bizarreness. Percept Mot Skills 1991;72:837–8.
65. Revonsuo A, Salmivalli C. A content analysis of bizarre elements in dreams. Dreaming 1995;5:169–87.
66. Rudy JW, Matus-Amat P. The ventral hippocampus supports a memory representation of context and contextual fear conditioning: implications for a unitary function of the hippocampus. Behav Neurosci 2005;119:154–63.
67. Kunzendorf RG, Hartmann E, Cohen R, et al. Bizarreness of the dreams and daydreams reported by individuals with thin and thick boundaries. Dreaming 1997;7:265–71.
68. Stickgold R, Scott L, Rittenhouse C, et al. Sleep-induced changes in associative memory. J Cogn Neurosci 1999;11:182–93.
69. Nielsen TA, Zadra AL, Simard V, et al. The typical dreams of Canadian university students. Dreaming 2003;13:211–35.
70. Cartwright RD. Dreams that work: the relation of dream incorporation to adaptation to stressful events. Dreaming 1991;1:3–9.
71. Stickgold R, Hobson JA, Fosse R, et al. Sleep, learning, and dreams: off-line memory reprocessing. Science 2001;294:1052–7.
72. Revonsuo A. The reinterpretation of dreams: an evolutionary hypothesis of the function of dreaming. Behav Brain Sci 2000;23:877–901.
73. Krakow B, Zadra A. Clinical management of chronic nightmares: imagery rehearsal therapy. Behav Sleep Med 2006;4:45–70.
74. Newell PT, Cartwright RD. Affect and cognition in dreams: a critique of the cognitive role in adaptive dream functioning and support for associative models. Psychiatry 2000;63:34–44.
75. Foulkes D. Dreaming: a cognitive-psychological analysis. Hillsdale (NJ): Lawrence Erlbaum Associates; 1985.
76. Rudy JW, Huff NC, Matus-Amat P. Understanding contextual fear conditioning: insights from a two-process model. Neurosci Biobehav Rev 2004;28: 675–85.
77. Hartmann E. We do not dream of the 3 R's: implications for the nature of dreaming mentation. Dreaming 2000;10:103–10.
78. Vrana SR, Cuthbert BN, Lang PJ. Fear imagery and text processing. Psychophysiology 1986;23: 247–53.
79. Pitman RK, Orr SP. Psychophysiology of emotional memory networks in posttraumatic stress disorder. In: McGaugh JL, Weinberger NM, Lynch G, editors. Brain and memory: modulation and mediation of neuroplasticity. New York: Oxford University Press; 1995. p. 75–83.
80. Rauch SL, Shin LM, Segal E, et al. Selectively reduced regional cortical volumes in post-traumatic stress disorder. Neuroreport 2003;14: 913–6.
81. Ochsner KN. Are affective events richly recollected or simply familiar? The experience and process of recognizing feelings past. J Exp Psychol Gen 2000;129:242–61.
82. Lang PJ, Greenwald MK, Bradley MM, et al. Looking at pictures: affective, facial, visceral, and behavioral reactions. Psychophysiology 1993;30: 261–73.
83. van der Kolk B, Blitz R, Burr W, et al. Nightmares and trauma: a comparison of nightmares after combat with lifelong nightmares in veterans. Am J Psychiatry 1984;141:187–90.
84. Lavie P. Sleep disturbances in the wake of traumatic events. N Engl J Med 2001;345:1825–32.
85. Husain AM, Miller PP, Carwile ST. REM sleep behavior disorder: potential relationship to post-traumatic stress disorder. J Clin Neurophysiol 2001;18: 148–57.

86. Johnson MK, Kahan TL, Raye CL. Dreams and reality monitoring. J Exp Psychol Gen 1984;113: 329–44.
87. Clark DM. A cognitive model of panic attacks. In: Rachman S, Maser JD, editors. Panic: psychological perspectives. Hillsdale (NJ): Erlbaum; 1988. p. 71–89.
88. Hull AM. Neuroimaging findings in post-traumatic stress disorder. Systematic review. Br J Psychiatry 2002;181:102–10.
89. Nutt DJ, Malizia AL. Structural and functional brain changes in posttraumatic stress disorder. J Clin Psychiatry 2004;65(Suppl 1):11–7.
90. Morgane PJ, Galler JR, Mokler DJ. A review of systems and networks of the limbic forebrain/limbic midbrain. Prog Neurobiol 2005;75:143–60.
91. LeDoux JE. Emotion circuits in the brain. Annu Rev Neurosci 2000;23:155–84.
92. Eisenberger NI, Lieberman MD. Why rejection hurts: a common neural alarm system for physical and social pain. Trends Cogn Sci 2004;8:294–300.
93. Rauchs G, Bertran F, Guillery-Girard B, et al. Consolidation of strictly episodic memories mainly requires rapid eye movement sleep. Sleep 2004; 27:395–401.
94. Braun AR, Balkin TJ, Wesensten NJ, et al. Dissociated pattern of activity in visual cortices and their projections during human rapid eye movement sleep. Science 1998;279:91–5.
95. Nofzinger EA. What can neuroimaging findings tell us about sleep disorders? Sleep Med 2004;5(Suppl 1):S16–22.
96. Hartmann E. Making connections in a safe place: is dreaming psychotherapy? Dreaming 1995;5: 213–28.