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Mood Regulation, Dreaming and Nightmares: Evaluation of a Desensitization Function for REM Sleep

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This paper is an evaluation of the hypothesis that REM sleep and dreaming serve a mood regulatory function, in particular, that they desensitize affect. There is presently experimental evidence that daytime mood influences REM sleep and dreaming and that the latter, in turn, influence daytime mood. It is suggested that these interrelationships may be better understood using a modified behavioral perspective on dreaming. Specifically, it is proposed that anxious dream imagery may be desensitized during REM sleep by a process that is analogous to systematic desensitization therapy. This analogy attributes functional roles to both psychological (dreaming) and physiological (atonia) aspects of REM sleep. Abnormal REM sleep phenomena such as narcolepsy, REM sleep behavior disorder, and nightmares are evaluated from the behavioral-desensitization point of view, and the implications for future research on REM sleep, dreaming, and waking mood are discussed.

KEY WORDS: REM Sleep; Dreaming, Mood Regulation, Desensitization, Nightmare, Muscle Atonia, Sleep Function, Dream Function, Emotion

INTRODUCTION

The commonly reported correlations between anxiety and nightmares and between psychiatric disorders and abnormal REM sleep suggest that dreaming and waking affect are related. The nature of this relationship, however, is unclear. Among the various possibilities are that waking affect impinges on the quality and quantity of REM sleep and dreaming, that REM sleep and dreaming affect waking mood, or that both of these effects occur reciprocally. If dreaming and waking affect are related, then it is also possible that REM sleep and dreaming are somehow implicated in the regulation of waking mood. The present article is an evaluation

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of this possibility. Theoretical approaches to the question of how REM sleep and dreaming may affect waking mood are reviewed with special attention given to the hypothesis that dreaming may desensitize affect in a manner analogous to systematic desensitization therapy (SDT).

Dreaming, REM Sleep and Mood

There are three types of research that support the notion that disturbed mood affects both REM sleep physiology and dream content. First, there is evidence documenting the incidence of abnormal REM sleep phenomena in patients with psychiatric disorders. These findings include the tendency for a reduced REM latency in major depression, borderline disorder, and eating disorders, and the tendency for schizophrenics to not exhibit a REM rebound following REM deprivation (Reynolds, 1987; 1989; Zarcone, 1989; Benca, Obermeyer, Thisted, & Gillin, 1991; 1992).

Second, there is research concerning the dream content of subjects with psychiatric disorders. It had been found that clinical populations tend to exhibit different dream content profiles when compared to each other or when compared to normal subjects (Hall, 1966; Langs, 1966; Kramer, 1970; Kramer & Roth, 1979; Reimann, Wiegard, Majer-Trendel, Dippel, & Berger, 1988; Kramer, 1991b). For example, Kramer and Roth (1979), in an extensive review of dreams in psychopathology, describe the dreams of schizophrenics as more hostile, more affective, and more thought-disordered than the dreams of nonschizophrenics. Content differences have also been observed when dreams from the acute and remitted phases of psychiatric illness are compared (Kramer, Whitman, Baldridge, & Ornstein, 1968; Hauri, 1976; Reimann, Lauer, Wiegard, & Berger, 1991).

Third, there is substantial experimental evidence that pre-sleep anxiety and stress affect REM sleep and dream content. Although results have varied from study to study, the observed effects include incorporation of stressful elements into dream content, changes in dream affect, decreases in REM latency, and increases in REM density (Baekland, Koulack, & Lasky, 1968; Cartwright, Bernick, Borowitz, & Kling, 1969; Breger, Hunter, & Lane, 1971; De Koninck & Koulack, 1975; De Koninck & Brunette, 1991).

There also exist at least three types of research which support the possibility that REM sleep physiology and/or dream content systematically affect waking mood. First, some studies suggest that REM deprivation in *normal subjects* increases the intensity of subsequent daytime feelings (Dement, 1960; Agnew, Webb, & Williams, 1967; Clemes & Dement, 1967). Although the magnitude of this effect is controversial (see Ellman, Spielman, Luck, Steiner, & Halperin, 1991 and Ellman, Spielman, & Lipschutz-Brach, 1991 for reviews), there is some indication that REM deprivation may produce anxiety and irritability (Dement, 1960), increased intensity of needs and feelings (Clemes & Dement, 1967), more feelings and wishes (Greenberg, Pearlman, Fingar, Kantrowitz, & Kawliche, 1970), and/or a reduced adaptability to stress (Greenberg, Pearlman, & Gampel, 1972

Second, there are studies that indicate that chronic REM sleep deprivation is related to mood improvements in *depressed individuals*. These studies have demonstrated that both behavioral and pharmacological methods of depriving subjects of REM sleep have antidepressant effects (Vogel, Vogel, McAbee, & Thurmond, 1980; Vogel, 1983; 1989).

Third, there are studies indicating that dream content is associated with changes in waking affect (Wasserman & Ballif, 1984; Cartwright, 1991; Kramer, 1991c). Wasserman and Ballif (1984) found that dreaming is perceived as a major influence on daytime mood; 46% of their subjects indicated, from among 10 possible causes, that dreams either moderately or substantially influence their daytime mood. Kramer and colleagues found, over the course of a series of studies, that the mood "unhappiness" is likely to decrease from evening to morning in normal subjects and that this change tends to be correlated with the number of characters which appear in dream content (for a review see Kramer, 1991c). Cartwright (1991) also found that "character incorporation" is associated with waking unhappiness. She found, in subjects undergoing divorce, that those who incorporated images of the ex-spouse into their dreams were better adjusted and less depressed at follow-up than those who did not.

Taken together, the above research strongly suggests that waking mood, REM sleep, and dreaming are interrelated phenomena. It is therefore plausible that dreaming during REM sleep may serve a mood regulating function.

Theories of Dream Function and Mood Regulation

There are several theoretical perspectives on how dreaming might accomplish mood regulation. Two of the most widely known are the psychoanalytic and cognitive perspectives; a less well-known is the behavioral perspective. These are summarized below and the behavioral perspective is elaborated in greater detail in the following sections.

The psychoanalytic perspective (Freud, 1900) suggests that dreaming allows for the safe expression of repressed ideation. The dream state is "safe" because unconscious ideas (wishes) may be expressed without consequences to either the ego or the real world. The transient satisfaction of the wish, in turn, allows for reduction in the strength of the wish and a temporary divesture of the psychical energies that maintain the repression during wakefulness. Were such a valve not present, or not functioning effectively, more psychical energy would be dedicated to repression mechanisms and less would be available for other, potentially more adaptive, means of coping. In short, dreaming is mood regulating from the psychoanalytic point of view because it accomplishes the homeostatic function of freeing up psychical resources for adaptive coping.

The cognitive perspective (e.g., Breger, 1969; Koulack, Prevost, & De Koninck, 1985; Cartwright, 1986; 1991) takes as its point of departure the association between dreaming and REM sleep. This association allows for dream function to be considered in relation to hypothesized functions of REM sleep. One such function is that REM sleep is related to the "off-line" processing of sensory information

and the consolidation of long-term memory (Empson & Clarke, 1970; Dewan, 1970; Crick & Mitchison, 1983; Winson, 1985; 1990). Since much of the material which is processed into memory is affectively charged, dreaming serves to facilitate the integration of affectively charged material into memory. It would appear that dreaming facilitates the process of integration because it is an automatic process, i.e., neither effortful nor subject to idiosyncratic coping strategies. Thus, from the cognitive perspective dreaming is mood regulating in that it is responsible for incorporation of affectively charged material which, when not integrated, may be inherently mood disturbing.

The behavioral approach, as first proposed by Beavers (1973), states that dream affect is regulated according to a desensitization process which is similar to that utilized in systematic desensitization therapy (Wolpe, 1985). More specifically, it was proposed that the coupling of anxiety-provoking stimuli in dreams with the somatic state of relaxation characterizing sleep produces desensitization, and that the symbolic features of dreaming mediate the anxiety reduction process. Implied in this model is that counter-conditioning effects acquired during dreaming transfer to wakefulness by the same mechanisms that are responsible for Pavlovian conditioning. Thus, from the behavioral perspective dreaming is mood regulating because it allows waking stressors to be desensitized through their dream image counterparts.

REM SLEEP AS A SYSTEMATIC DESENSITIZATION SYSTEM

The behavioral perspective on REM sleep and dream function (Beavers, 1973, Hayes & Mooney, 1975), although not incompatible with the more popular theories, has been largely overlooked by theorists and researchers in the sleep and dreams domain. This is somewhat surprising when one considers how consistent the model is with some of the first psychophysiological observations regarding nightmares:

> One of the most striking findings is that the latter [nightmares as opposed to night terrors] may show what we have called a desomatization of the anxiety response, that is, an absence of the physiological concomitants of anxiety even when the reported anxiety is quite severe. There seems to be some sort of mechanism in the REM state for controlling, modulating and muting this response . . . The REM dream does not appear to tolerate very great autonomic change before an awakening occurs. The postulated desomatization process may assist in mastering anxiety, and may help keep the person asleep, even though he is having an anxiety dream, by diminishing its intensity (Fisher, Kahn, Edwards, & Davis, 1973).

Within the last four years there has been a renewed interest in the behavioral perspective. Several investigators have suggested that certain components of REM sleep may be responsible for the putative desomatization or desensitization process. Shapiro (1989) suggested that the eye movements of REM sleep may serve to desensitize affect in a way which is similar to eye movement desensitization/reprocessing therapy. Nielsen, Kuiken and McGregor (1989) and Nielsen (1991) proposed that the atonia of REM sleep may produce a desensitization effect by repeatedly blocking kinesthetic feedback during traumatic dream imagery so as to produce extinction of its somatic correlates. In the following section we re-examine the systematic desensitization therapy (SDT) analogy.

Systematic Desensitization Therapy

SDT is a waking state strategy for breaking down "neurotic anxiety response habits" (Wolpe, 1985). It requires that an individual be exposed to anxiety-producing stimuli while in a physiological state that is inhibitory to anxiety. According to the reciprocal inhibition model of SDT (Wolpe, 1954; 1958), repeated pairing of anxiety-producing stimuli with a physiological state that is inhibitory to anxiety results in counter-conditioning, i.e., conditioned non-arousal responses to aversive or arousing stimuli. Exposure may be accomplished by having the client confront either imaginal (in vitro) or actual (in vivo) anxiety-provoking stimuli. The anxiety-provoking stimuli themselves may consist of feared objects, persons or situations. The physiological state paired with the anxiety-producing stimuli is usually that of deep muscle relaxation.

The systematic component of SDT requires that stimulus intensity be controlled in one of two ways: through hierarchical presentation of stimuli or through the regulation of exposure time. In the case of the former, subjects are exposed first to their least anxiety-provoking images and then, over the course of successive trials, to images which are progressively more anxiety-provoking. In the case of the latter, subjects are exposed to anxiety-provoking images for periods of time which increase over successive trials. Both methods are thought to facilitate desensitization because they allow clients to acquire non-arousal responses in a graded fashion.

The SDT Analogy of Mood Regulation During Dreaming

During SDT, clients are exposed to either imaginal (in vitro) or actual (in vivo) anxiety-provoking experiences. Since waking stressors appear to be readily incorporated into dream content (e.g., Domhoff, 1992; Wood, Bootzin, Rosenhan, Nolen-Hoeksema, & Jourden, 1992), the dream images of these stressors are, at least, comparable to in vitro exposure and, given the hallucinatory quality of dreaming, probably more comparable to in vivo exposure. During SDT, either in vitro or in vivo experience is paired with muscular relaxation so as to diminish sympathetic reactivity. During REM sleep, dream images of waking stressors are experienced while in a state of muscular atonia which is induced by motor inhibition. As with the process of SDT, it is hypothesized that the pairing of dream imagery and atonia may serve to diminish sympathetic reactivity. If the two conditions are sufficiently analogous, one would expect a "desomatization" or desensitization effect. It is further hypothesized that the counter-conditioning effects acquired during REM dreaming may transfer to wakefulness by the same mechanisms which underlie Pavlovian conditioning. Implicit in this model is that the dream need not be remembered in order for waking affects to be desensitized.

The present conceptualization of desensitization differs from Beavers' (1973) initial formulation in that the putative desensitization process in explicitly linked to REM sleep rather than to sleep in general. The present model links desensitization not to the passive postural relaxation of sleep but to the active atonia mechanisms of REM sleep. Although it is true that most stages of sleep are characterized

by muscular relaxation relative to quiet wakefulness, the REM sleep state in particular is characterized by profound muscular inactivity. This muscular inactivity is 1) more global and more pervasive than that in either NREM sleep or wakefulness (Tauber, Coleman, & Weitzman, 1977; Hobson, 1988; Chase & Morales, 1989), and 2) unlikely to be interrupted by sustained bouts of motor activity (Chase & Morales, 1989; 1990). There do occur occasional muscle jerks and twitches in association with supraspinal pyramidal discharges during REM sleep (Mouret, Delorme, & Jouvet, 1964; Baldridge, Whitman, & Kramer, 1965; Gardner, Grossman, Roffwarg, & Weiner, 1975; Evarts, 1964), but these are muted, peripheral, and normally phasic in nature (Pompeiano, 1976; Chase & Morales, 1989; 1990). Thus, when potentially anxious dream content occurs during REM sleep, the state of profound muscle relaxation associated with REM sleep is particularly well-suited to oppose sympathetic responding.

The present conceptualization of desensitization differs from that of Beavers (1973) in a second important respect as well. Beavers stated that the systematic component of dream desensitization was mediated by symbolic distortion in the dreamed representation of waking stressors. More specifically, he proposed that "anxiety-provoking dream content is initially presented in the form of distorted symbols so as to minimize potential anxiety responses and, as dreaming becomes less aversive by desensitization, these symbols become less distorted" (p. 31). While this possibility certainly merits further exploration, it is also possible that 1) there is no systematic component, i.e., desensitization occurs in a single trial or, 2) the systematic component of desensitization during dreaming may be related to the patterning of dream content over time.

One possibility is that dream images of waking stressors may systematically vary within individual REM sleep periods. For example, it may be that particularly stressful material is usually preceded and followed by less emotionally charged dream contents. Dream content patterning of this type would serve to both "regulate exposure time" and restrict the amount of time during which an anxiety response could occur should the desensitization process be unsuccessful. This notion is consistent with Kramer's (1991a) description of dream content patterning within the individual REM period.

> The periodicity of the development of dream content intensity is similar to the periodicity of the density of eye movements seen during REM sleep. The intensity of the dream increases rapidly to peak at 10 to 20 minutes then declines and starts up again. (p. 278).

A second possibility is that dream images of waking stressors may be differentially incorporated across successive REM periods. For example, it may be that recent and/or novel events may only be incorporated into the earlier REM cycles and, as a result, exposure time to the dream images of these events would be restricted to the shorter duration REM periods. This possibility is supported by Roffwarg, Herman, Bowe-Anders, and Tauber's (1978) description of the recency phenomenon. As described by the authors, " . . . recent perceptions tend to be dreamed in the first REM periods of the night, and with increasing longevity of the input, their expression in dreaming penetrates into the later REM periods" (p. 319).

A third possibility is that exposure to dream images of waking stressors may vary as a function of how significant events are incorporated into different dreams across successive nights (Powell, Nielsen, & Cheung, 1993; Nielsen & Powell, 1989; 1992) and/or in how often specific dream contents recur over time (e.g., Domhoff, 1992). For example, Powell, Nielsen and Cheung (1992) observed a tendency for an emotionally charged event to enter dream content for the first three days following the event and then again after a delay of from 6-7 days following the event (a dream-lag effect). Domhoff (1992) has observed that stress-related dream contents may recur over periods of time lasting from days to years.

Each of the above perspectives, with the possible exception of the first, imply that the dream image of the waking stressor remains constant over time. If it is the case that dream images of waking stressors are repeated over time, it is not necessary to assume that the dream images remain static. It is entirely plausible that there are multiple representations for each individual waking stressor. The multiple representations may derive naturally from components of the actual stressful scenario (e.g., dentist, drill, pick, surgical lamp, etc.) or from the organization of episodic memory itself.

In sum, we are suggesting that REM sleep dreaming may serve a mood regulating function and that this process may work in a way which is analogous to systematic desensitization therapy. The desensitization component depends on the naturally occurring state of atonia to attenuate physiological arousal during dreaming and to promote counter-conditioning. The systematic component of the process, if it exists, may be related to either the temporal structure of REM sleep and/or to the patterning of dream content over time.

EVIDENCE FOR A LINKAGE BETWEEN ATONIA AFFECT AND DESENSITIZATION

Abnormalities of REM Sleep and Desensitization

Certain abnormalities of REM sleep are consistent with the suggested link between atonia and affect. One such abnormality is the cataplexy component of narcolepsy. Narcolepsy is considered to be a state-disorder syndrome where components of REM sleep inappropriately emerge during wakefulness (e.g., Guilleminault, Wilson, & Dement, 1974; Mahowald & Schenck, 1989). The cataplexy component of this disorder is usually precipitated by the experience of strong emotion (Guilleminault, 1989). This association between the experience of strong emotion and the engagement of atonia suggests that these components may be similarly engaged during normal REM sleep, i.e., atonia may also be engaged or enhanced when the dreamer experiences images that are associated with strong waking emotions. Evidence for this possibility derives from the related findings that 1) the most intense dreams are experienced during the rapid eye movement periods of REM sleep (e.g., Foulkes, 1962; Antrobus, 1991), and 2) motor inhibition is at its most intense during these same periods (Chase & Morales, 1990; Perlis, Drummond, Fleming, & Bootzin, 1991).

A second type of REM sleep abnormality linking REM sleep atonia to emotion is the episodes of motor activation associated with emotionally intense dreaming. Such episodes occur in a particularly marked form in REM sleep behavior disorder (RBD). Individuals with RBD often emit vigorous, sometimes violent, motor activity in association with nightmarish dreams (Schenck, Bundlie, Ettinger, & Mahowald, 1986; Mahowald & Schenck, 1989). Motor activation during REM sleep has also been measured electromyographically from the expressive facial muscles of vivid dreamers and nightmare sufferers (Perlis, Wright & Bootzin, 1990; Perlis, Drummond, & Bootzin, 1991) and in the peripheral muscles of PTSD subjects (Ross, et al., 1990). These phenomena suggest that increased motor activation during REM is related to increased emotionality during dreaming.

A third REM sleep abnormality is the side-effects of L-dopa on REM sleep and dreaming. When used to treat Parkinson's disease, L-dopa often produces insomnia and/or nightmares (Klawans, Moskovitz, Lupton & Scharf, 1978; Sharf, Moskovitz, Lupton & Klawans, 1978). The nightmare effect is also evident when the dopamine precursor is given in sub-clinical doses to subjects who are prone to nightmares; in these subjects, L-dopa produces more vivid, emotionally intense, and nightmarish dreams (Hartmann, Scoff, Russ & Oldfield, 1978; Hartmann, 1984). Since dopamine is associated with the regulation of movement and muscle tone, it is possible that L-dopa blocks or attenuates the atonia of REM sleep and thus allows for increased emotional intensity during dreaming.

IMPLICATIONS FOR FUTURE RESEARCH

Nightmare and Desensitization—The REM Sleep Nightmare

According to the desensitization hypothesis, REM sleep dream content is normally experienced as less emotional and less physiologically arousing than waking experience because of atonia and reciprocal inhibition. Since the nightmare is experienced as physiologically arousing and results in an awakening, it may be that the putative desensitization process has at this time failed because atonia could not be maintained during cognitively unpleasant or anxiety-provoking dreams. On the other hand, it is also possible that nightmare imagery represents a natural augmentation of REM sleep desensitization activity which is evoked by conflict, trauma or other daytime stressors. To differentiate these possibilities, it may be useful to consider how much emotional arousal and motor activity can be tolerated during REM sleep before the proposed desensitization process fails. One line of thought suggests that any motor activation during REM is maladaptive and incompatible with reciprocal inhibition. Another line of thought suggests that the maintenance of absolute atonia is not necessary to produce desensitization. In the latter case, the individual may not remain totally atonic during dreaming, but as long as the level of motor activation is less than that which would produce an awakening habituation (if not counter-conditioning) may still occur. This possibility may be thought of as analogous to the behavioral techniques of implosion or flooding (Stampfl & Levis, 1967).

These two possibilities lead to very different views on the adaptiveness of awakenings that terminate nightmares. In the case of the former, absolute atonia perspective, a nightmare awakening may be viewed as functional because it allows for conscious appreciation of the source of the anxiety experience and the realization that "it was only a dream". While such awakenings may lead to dream interruption insomnia (Greenberg, 1967) or nightmare distress (cf. Belicki, 1992), they may also prevent a maladaptive sensitization process from occurring, i.e., the automatic pairing of physiological arousal with dysphoric dream imagery. This type of pairing and its consequent sensitization might be expected to be related to waking mood disturbances such as anxiety disorders or depression. In the case of the latter, relative atonia perspective, awakenings from nightmares may best be considered as maladaptive in that the exposure process is interrupted and thus further desensitization efforts are aborted (Hayes & Mooney, 1975).

Both the absolute and relative desensitization perspectives on nightmare function allow for new conceptualizations of the etiology of vivid dreams, lifelong nightmares and trauma-related nightmares. For example, vivid dreamers and lifelong nightmare sufferers may be more neurophysiologically predisposed than others to express motor activation during REM sleep. This predisposition may allow dream content to become more physiologically arousing and may increase the probability that the individual will awaken from REM sleep and process the dream into long term memory.

Trauma-related nightmare sufferers may also be predisposed to motor activation but for different reasons: 1) the affective charge of their dream content—because it relates to specific traumatic memories—may be too great to be inhibited by REM sleep atonia, and/or 2) the traumatic experience may have altered the individuals' ability, in both wakefulness and sleep, to inhibit motor activity and/or sympathetic arousal. The latter possibility is consistent with Kramer, Schoen, & Kinney's (1984) suggestion that intense physio/skeletal activity in nightmare sufferers may influence their emotional responsivity to dream experience.

Nightmares and Desensitization—the NREM Nightmare

Desensitization hypothesis may also contribute to our understanding of Stage 2 or NREM sleep nightmares (e.g., Schlosberg & Benjamin, 1978; Hartmann, 1984; Van der Kolk, Blitz, Burr, Sherry, & Hartmann, 1984; Woodward, Arsenault, Bliwise, & Gusman, 1989; Kramer, 1991a). It has been hypothesized that the Stage 2 or NREM nightmare is a type of state-disorder syndrome such as narcolepsy or RBD (Kramer, 1991a). During the NREM nightmare it is thought that the cognitive aspect of REM sleep dislocates or overflows into the early night NREM sleep periods. The desensitization analogy adds to this the suggestion that NREM dreams are emotionally intense and experienced as nightmares precisely because they occur during a state in which there is less motor inhibition to block emotional arousal. It is thus unlikely that desensitization of dream imagery will be achieved during this stage.

Possible Research Paradigms

Because the present work suggests that dream desensitization depends upon REM sleep atonia, empirical exploration of atonia's relation to emotional experience during dreaming and daytime mood are called for.

Experimental approaches to the question of desensitization could manipulate either the intensity of dream content or the state of REM sleep atonia and then observe whether the consequences of these manipulations are consistent with the desensitization model. To alter the intensity of dream content, one might utilize the dream intensification effect of REM deprivation (Pivik & Foulkes, 1966; Weinstein, Schwartz, & Ellman, 1991). The desensitization analogy would predict that as REM pressure increases, rebound REM sleep would contain increased amounts of motor activity and that this activity would be positively correlated with the intensity of REM mentation. The feasibility of this suggestion is shown by the fact that, in rabbits, REM deprivation leads to motor activation (Pivik, Bylsma, & Cooper, 1987).

Alternatively, one might attempt to alter dream vividness and emotionality by expanding upon Hartmann et al.'s experiment with L-dopa (1978). Here the desensitization model would predict that observed increases in dream vividness and emotionality should be associated with increased motor activation during REM sleep.

Finally, in order to induce muscular activation during REM sleep, low-level electrical stimulation could be applied to the skeletal and/or facial musculature. Here the desensitization analogy would predict that 1) activation that is not terminated by a spontaneous or elicited awakening will systematically affect subsequent daytime measures of anxiety and depression; the greater the activation during REM sleep the larger the mood effects, and 2) dream reports following activation will be more vivid and will contain more emotion than control awakenings from undisturbed REM sleep.

Correlational approaches to the question of dream desensitization could evaluate the proposed model by demonstrating that the relationships between atonia, physiological arousal and dream emotion do indeed exist. For example, if nightmares and vivid dreams result from the failure to maintain atonia during REM sleep, it would be expected that subjects with such dreams should exhibit significantly more motor activity during REM sleep than subjects who find their dreams to be unremarkable. Presently, there is some evidence to suggest that subjects who are prone to exceptionally emotional dreams do exhibit motor activation during REM sleep (Ross, et al., 1990; Perlis, Wright, & Bootzin, 1990; Perlis, Drummond, & Bootzin, 1991).

The correlational approach could also be used to evaluate the possibility that motor activation during uninterrupted REM sleep may result in waking mood disturbances (e.g., anxiety disorders and depression). If this is the case, subjects with endogenous mood disorders should exhibit both unusual motor activation during REM sleep and normal REM sleep efficiency. The severity of the waking emotional disturbance should be positively correlated with the amount of motor activation present during uninterrupted REM sleep. There is some limited evidence to suggest that measures of waking depression are correlated with the amount of facial muscle activity present during REM sleep (Perlis, Drummond, & Bootzin, 1991). There is also evidence that movement inhibition during dreaming is related to waking emotional variables such as daytime mood and a feeling of weakness during emotions (Kuiken, Nielsen, & Chahley, 1988). Finally, the correlational approach could be used to distinguish between the absolute and relative atonia perspectives on desensitization. If the awakening which terminates a nightmare is maladaptive, it would be expected that nightmare subjects should exhibit more mood disturbance on days following nightmare experiences than on days with normal or no dream recall.

CONCLUSION

We have proposed that dreaming during REM sleep may allow for mood regulation by a process which is analogous to systematic desensitization therapy. It should be stressed, however, that this model is presented only as an analogy. The present model of dream desensitization is largely based upon SDT's use in the treatment of anxiety disorders. The therapy, however, has also been used in the treatment of arousal disorders in general. Thus, although this paper focuses on the dream desensitization of waking stressors, it is in keeping with the SDT analogy that all dream content, positive or negative, may be desensitized during REM sleep.

It should also be noted that there is significant debate concerning the active mechanism of desensitization therapy. Although it is accepted that SDT is an effective therapy, research over the last twenty years has failed to show precisely which components of the therapy (e.g., exposure, systematic exposure, non-arousal during exposure) are responsible for treatment success (see Lick & Bootzin, 1975, for review). Concerns have also been raised about the Pavlovian concept of classical conditioning (e.g., Rescorla, 1967, Kamin, 1969).

Finally, the dream desensitization model does not take into account that during REM sleep there is not only motor inhibition induced atonia, but also an active inhibition of some branches of the sympathetic nervous system. In the present conceptualization of the desensitization theory, atonia is thought to be the critical component in the inhibition of physiological reactivity during dreaming. It may be the case, however, that motor activation during REM sleep is simply an index of the failure of the central mechanism(s) to contain sympathetic responding. In this eventuality, what remains important is that REM sleep is still uniquely suited for desensitization, i.e., the state provides a condition in which the individual may be exposed to dream images of waking stressors while experiencing a diminution of the physiological concomitants of anxiety.

Despite these caveats, the desensitization model has the heuristic value of implicating functional roles for both psychological (dreaming) and physiological (atonia) aspects of REM sleep. It may thus serve to bring into closer rapport current theories about the adaptive functions of dreaming and knowledge of REM sleep physiology.

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