

0732-118X(95)00023-2

REM SLEEP: A SOCIAL BONDING MECHANISM

PATRICK MCNAMARA

Vision House, 460 East Street, Tewksbury, MA 01876, U.S.A.

Abstract—It is proposed that REM sleep evolved, in part, to promote social bonding between (1) a mammalian infant and the mother and (2) sexual partners. The bonding hypothesis is consistent with the available evidence from psychobiologic studies of the attachment process in infants, with the known physiologic correlates of REM sleep, with the facts concerning the ontogeny and the phylogeny of REM sleep, and with phenomenological properties of dreams. Copyright © 1996 Elsevier Science Ltd.

INTRODUCTION

REM, or rapid eye movement sleep, occurs in almost all mammals (Zepelin, 1989). It is a cyclic phenomenon, occurring approximately every 90 min during sleep, with specially dedicated brain mechanisms that regulate its appearance and suppression (Aserinsky & Kleitman, 1953; Jouvet, 1960; Hartmann, 1973; Hobson, 1988). It may also be associated with a daytime 90-min daydreaming rhythm (Broughton, 1982). The duration of time spent in REM during a sleep episode varies with altriciality of the species—the greater the immaturity (at birth) of the species, the longer the time spent in REM (Zepelin, 1989). REM duration (percent of total sleep time) is greatest in infancy and declines with age (Roffwarg, Muzio & Dement, 1966). Total sleep deprivation produces a "rebound" or lengthening of time spent in non-REM, slow wave sleep (SWS) first and then a subsequent rebound in REM sleep. The SWS debt must be repaid first,* suggesting perhaps a more basic role (e.g. metabolic regulation) of SWS vs REM sleep in the biology of the organism. Deprivation of REM sleep over the course of a few nights produces a compensatory "REM rebound" (Dement, 1960; Vogel, 1975) and an increase in "drive-related behaviors" or appetitive behaviors (e.g. eating, inappropriate sexual interest) in cats and humans (Dement, 1965; Hirshkowitz, 1993)†. Sexual arousal (in the form of penile erections and clitoral

^{*}There is now good evidence that NREM sleep functions as a thermoregulatory mechanism. Prolonged disruption of NREM leads to thermoregulatory failure and death in rats. Thermoregulation does not occur in REM sleep (Zepelin, 1989). REM sleep alternates with NREM sleep in periodic cycles throughout the night. Thermoregulatory functions are intact in NREM but not in REM sleep. Endothermy (maintaining a high and constant body temperature, independent of environmental conditions) is biologically costly. Zepelin (1989) suggests that the sleep cycle (the REM-NREM alternation) may have evolved to help cope with that cost. Rats whose sleep cycles are experimentally disrupted gradually lose the ability to regulate body temperature and then die (Siegel, 1993). These facts suggest that the function of NREM sleep has something to do with thermoregulation, but they seem less relevant to possible functions of REM sleep. It seems unlikely that REM sleep would need specialized and dedicated brain mechanisms if it was simply an epiphenomenon of NREM sleep. With total sleep deprivation the SWS debt is repaid before the REM debt presumably because thermoregulatory failure would result in death, while attachment for the adult is not critical.

[†]Selective deprivation of REM sleep for any amount of time is followed by "REM rebound" where the organism spends an increased (relative to baseline) amount of time in REM for a few days thereafter (Vogel, 1993). It is as if there was need for REM that accumulates if one is deprived of it for any amount of time. REM deprivation studies, however, are plagued by the fact that it is exceedingly difficult to selectively deprive subjects of REM sleep. Most "REM-deprived" subjects are probably NREM-deprived as well. When REM deprivation studies were first conducted it was believed that REM deprivation was associated with a host of cognitive and emotional deficits. It was particularly argued that learning Footnote continued on next page

P. McNamara

engorgement) occurs with each REM episode (Hirshkowitz, 1993) and each ultradian daydreaming episode (Kripke & Sonnenschein, 1978). REM sleep is often, though not invariably, associated with vivid dreams (Dement & Kleitman, 1957; Foulkes, 1985) and is characterized by a complete absence of muscle tone in the anti-gravity muscles and by brain-wave activity like that in the waking state (McCarley, 1989). Many regions of the brain (especially limbic regions) sustain very high brain activation levels during REM sleep (Gillin, Buchsbaum & Wu, 1993). The time spent in REM progressively increases over the course of the night such that the morning hours are dominated by active (REM) sleep. In most species REM sleep is also associated with a characteristic electrical rhythm in the hippocampus called the theta rhythm-though such a rhythm has not yet been definitively associated with REM sleep in humans (Winson, 1985). McCarley (1989) has suggested that the phasic bursts of rapid eye movements are associated with pontine-geniculate-occipital waves (PGO waves). These are spikes that were originally associated with sites in the brainstem Pons, the lateral geniculate nucleus of the thalamus and the occipital cortex-all visual centers. Steriade (1989), however, has pointed out that PGO waves are recorded in most thalamic nuclei and therefore probably serve to activate much wider areas of cortex than the visual system during REM sleep, REM sleep, apparently, involves more than the visual experiences we call dreams.

FOREBRAIN SITES IMPORTANT FOR REM SLEEP

Orbito-frontal but not dorsolateral frontal lesions have been associated with suppression of REM sleep and the thalamic recruiting response, important for generation of sleep spindles, PGO waves, etc. (Skinner, Molnar & Harper, 1989; Villablanca, Marcus, & Olmstead, 1976; Jus. Jus. Villeneuve, Pires, Fortier, Lachance & Villeneuve, 1973). Lesions in the region of the cholinergic basal forebrain (which projects to orbito-frontal cortex) are also associated with REM sleep fragmentation (Skinner et al., 1989). Orbito-frontal lesions are known to be associated with impaired social and emotional behaviors in humans and may be associated with disruption to forebrain cholinergic circuits (Oscar-Berman, McNamara & Freedman, 1991). Finally, recent brain imaging studies (see review in Gillin et al., 1993) have revealed that limbic and frontal sites are particularly active during REM sleep and particularly affected after sleep deprivation. Interestingly, in the few mammalian species without REM sleep-the echnida (Allison, Van Twyver & Goff, 1972) and some species of dolphins and other sea mammals-orbito-frontal or insular cortices (both of which are densely interconnected with limbic system sites) have grown disproportionately large-even compared to humans (Winson, 1985; Morgane, Jacobs & McFarland, 1980). Thus, limbic/frontal sites in the echnida and in some sea mammals without REM sleep may handle functions normally mediated by REM sleep in all other mammalian

and memory deficits were prominent. Subsequent and more carefully controlled studies, however, could not document any psychiatric or cognitive deficits in humans (Vogel, 1993). One consistent finding, however, was an increase in driverelated or appetitive behaviors (e.g. eating, sexual interest) in cats and humans (Hirshkowitz, 1993; Vogel, 1993). In addition, REM deprivation has been shown to (at least temporarily) relieve depression (Vogel, Vogel, McAbee & Thurmond, 1980; Buysse, Reynolds & Kupfer, 1993). One reviewer concluded that "in humans RSD (REM sleep deprivation) does not cause psychological harm, improves endogenous depression. . . In animals RSD increases drive-related behaviors, may impair learning . . . and over several weeks is fatal" (Vogel, 1993, p. 180). Some (not all) antidepressant drugs, in fact, suppress REM sleep. Since several biogenic amine neurotransmitter levels decline during REM (Siegel, 1993), it is not surprising that delaying REM onset would be associated with a transiently improved mood. In any case the causes, concomitants and antecedents of depression are probably multiple (Barnet and Gotlib, 1988) and may include REM sleep changes.

species. For species who have REM sleep, limbic/frontal circuits appear to be selectively act vated during REM sleep.

As Crick and Mitchison (1986) point out, all of these facts concerning the psychobiology of REM sleep make it virtually certain that REM sleep has some important psychobiological function to perform. Although there have been numerous proposals concerning the functions of REM sleep (see, for example, the papers in Moffitt, Kramer & Hoffmann, 1993), it is generall accepted that none of these proposals can account for all of the relevant facts concerning REM sleep psychobiology (Crick & Mitchison, 1986; Moffitt et al., 1993; Siegel, 1993). In this paper I suggest that REM sleep probably has many functions but that one of the more important functions (for human psychology) is to promote filiative bonding between parent and child and between adult mating partners. This "bonding hypothesis" accounts for most of the facts concerning REM sleep in an unforced manner and leads to some testable predictions regarding REM sleep correlates.

In what follows I first lay out the bonding hypothesis in detail. I then review the evidence for that hypothesis. I conclude with some remarks on the implications of the hypothesis for a psy chology of dreams. (Some terminology: I am using the term "bonding" to describe filiative social attachment between two mammalian organisms. Although "bonding" usually refers to the mother's attachment to the child and "attachment" usually refers to the child's attachment to the mother, in this paper I use the terms interchangeably.)

HYPOTHESIS

REM sleep functions, in part, to promote, support or allow psychobiologic bonding of the organism with a significant conspecific. I conjecture that REM sleep promotes social bonding in two major ways:

- 1. Via activation of the biobehavioral systems important for social bonding or attachment. These systems include limbic-frontal systems, oxytocinergic limbic systems, circuits involving transmission of arginine vasotocin (a precursor to oxytocin), and circuitry involved in sexual and reproductive behaviors (see below for a review of the evidence supporting these assertions). It is important to note that the hypothesis implies activation of these systems not only every 90 min at night but every 90 min throughout the day as well. There is evidence that this activation does occur via the 90-min daydreaming cycle (see below).
- 2. Via synchrony. Attachment theorists have suggested that bonding is facilitated when the biological rhythms of, say, the child, are in phase with, or synchronous with, those of the mother REM sleep's inherent periodicity—i.e. the regular appearance of wave-like forms that resemble the waking state, and the rhythmic and selective activation of limbic brain systems—lends itsel to synchronistic interactions. This would be less true for SWS, since SWS involves a non-selective lowering of activation levels. REM sleep also represents a return to the fetal state: lack of thermoregulation, paralysis, twitching, respiratory irregularity, etc. The organism, in other words relies on, for example, parental body heat and respiratory cycles for thermoregulation and respiratory regulation. The REM state is intrinsically "other"-oriented.

In summary, I hypothesize that we are subject to periodic activation of the biobehavioral systems necessary for social bonding. When activation occurs nocturnally we call it REM sleep When activation occurs during the day we call it daydreaming.

It is important to note the following qualifications: I do not believe that social bonding is the only function of REM sleep, dreaming or daydreaming. All three of these behaviors involve much

38: P. McNamara

more than attachment. Social bonding, the hypothesis suggests, is merely one, albeit cri function of these brain states. The hypothesis, furthermore, does not require both organisa be in REM sleep simultaneously for social bonding to occur. REM sleep (and its associated dreaming rhythm) simply activates the circuits that incline the organism to bond to an appr ate object. On the other hand, co-regulation of the sleeping cycles of the two organisms w enhance social bonding and attachment prospects because there would be a greater chanc synchrony to develop between the two organisms. REM sleep would incline the infant to a by enhancing need states (e.g. for thermoregulation) or object-seeking behaviors: crying, coo babbling, etc. The increased number of arousals associated with active sleep/REM sleep in infant would also promote attachment because these arousals elicit caretaking behaviors nursing) from the mother. As the organism matures REM sleep would also be associated some kind of libidinal satisfaction of the object-seeking, drive state activated in the REM s The dreaming brain presumably builds a "search image" of the preferred object, which simulated the control of the preferred object. neously elicits and temporarily satisfies object-seeking states in the organism. With respect to mother's role, REM sleep would promote the inclination to bond with the baby by activa those brain systems (limbic/frontal) needed for caretaking and approach behaviors.

It is also important to note when evaluating the evidence for the hypothesis that modern hur child-rearing or reproductive practices need to be considered along with phylogenetically ancichild-rearing and reproductive behaviors. The bonding hypothesis is concerned with the evo tionary origins of human social bonding behaviors. It argues that REM sleep physiology v recruited to support these bonding behaviors. Human attachment behaviors, and (by implication) REM sleep's role in attachment, evolved hundreds of thousands of years ago when humans liv in small hunter-gatherer groups; when human sexual bonding was erratic, unstable and even degerous; when human mothers had a child about once every four years; when mother and ch invariably co-slept; when the average life expectancy for a human being was 40 years old, e REM sleep-associated social bonding mechanisms, in other words, were designed for a pers who was born helpless and dependent on an other (mother); whose early survival depended that mother; who sexually-reproduced at adolescence; and then intensely invested in the child f about four years (until the next pregnancy); and then died by 40 years of age. REM sleep indic correlate with the life-cycle (or with the attachment needs) of a human being who lived in the "Environment of Evolutionary Adaptation" or the so-called EEA. The formal content of dream therefore, should partially reflect conditions in the EEA.

I turn now to the evidence in support of the hypothesis.

PHYLOGENY AND ONTOGENY OF REM SLEEP AND ATTACHMENT

REM sleep seems to have evolved in mammalian species only (Winson, 1985; Zepelin, 1989) although some species of birds experience REM-like episodes (Kaufman, 1993). Avian REM sleep, however, constitutes a lower percentage (about 5%) of total sleep time than that seen in mammals (15-30%). Why did the mammalian order "need" REM sleep? Mammalian young (and most species of birds) tend to be born in a state of dependency and immaturity. Without parenta or adult assistance the infant may not survive. By hypothesis, REM sleep promoted social-physiologic bonds between infant and mother, thus inclining the mother to nurture the child. The hypothesis, therefore, predicts a strong relation between the degree of immaturity (or altriciality) of a given species and percentage of time spent in REM sleep during development. Opossums and ferrets are born in an extremely immature state. These animals devote about 30% of their total sleep time to REM (Siegel, 1993). In terms of altriciality humans are intermediate between opossums/ferrets and horses/elephants. Not surprisingly, therefore, adult humans spend a little less time (24% of total sleep time) in REM sleep relative to opossums/ferrets, and a little more time in REM than horses/elephants (who spend approximately 22% of their total sleep time in REM).

REM sleep, therefore, is essentially a fetal adaptation. REM sleep durations are highest in the fetus. Human neonates spend about half of their sleep time in REM, Percentage of time spent in REM declines with age until the adult state is reached. REM sleep, therefore, must be critical for some aspect of CNS development. Roffwarg et al. (1966) argue that REM sleep serves to promote CNS development by providing periodic bursts of stimulation to the system. This theory, however, does not explain (1) why REM sleep is associated with selective activation of limbic and visual association areas; (2) why REM sleep is associated with erections/clitoral engorgement; (3) why REM sleep is associated with neurobiologic attachment mechanisms (see below); (4) why REM deprivation leads to heightened libidinal states; and (5) why REM sleep persists into the adult state. Can the bonding hypothesis account for these objections? Yes: limbic areas are activated in REM sleep because bonding is an emotional process and limbic structures "handle" socio-emotional functions. With respect to the persistence of REM into the adult state it seems likely that REM sleep would support similar functions in the infant and the adult, although REM sleep's function would be more critical for the infant, Bonding in infancy is critical for survival. In the mature state bonding with another is no longer critical for individual survival, but it is critical for reproduction of the species. Finally, with respect to the issue of sexual arousal, if REM involves activation of brain circuits important for bonding, then it would not be surprising to find, as we in fact do, that REM sleep is coupled to sexual arousal throughout the night. REM deprivation, therefore, should involve some disruption of sexual and social bonding abilities and some compensatory attempt to engage in those behaviors when they are being blocked. Thus, the reports of hypersexuality after REM deprivation. The social bonding/attachment hypothesis, therefore, straightforwardly accounts for many of the developmental correlates of REM. It is now necessary to look more closely at attachment phenomena.

Attachment

Reite and Capitanio (1985, p. 224) have defined attachment as "a neurobiologically based and mediated biobehavioral system, one of whose major functions is to promote the development and regulation (or modulation) or psychobiological synchrony between organisms". By "synchrony" these authors mean the attunement of biological rhythms in one organism to those in another. Field (1985), Reite and Capitanio and others (e.g. Hofer, 1987; Stern, 1985) have argued that attachment can be fruitfully understood as psychobiological attunement or synchrony between individuals. From this perspective attachment is synchrony of behavioral and biological rhythms between individuals. Field cites a long list of behavioral and biologic variables that have been documented to display synchrony of one kind or another in organisms undergoing attachment. Among these variables are sleep states and EEG waveforms. Synchrony and attunement of physiological variables such as autonomic indices and EEG waveforms may help each member of the attached pair to better regulate their own internal physiological systems. The importance of attachment for the normal development and well-being of a human being has been amply documented in the past two decades (see Bowlby, 1988; papers in Reite & Field, 1985; Belsky & Nezworski, 1988; Kraemer, 1992). Insecure or disrupted attachments in infancy and early childhood are predictive of later psychological disturbances, physiological disorganization, poor health and cognitive deficits. Insecure attachments in the mature state are also associated with poor health and depression. Attachment is therefore central to the well-being of our species.

40 P. McNamara

The periodicity inherent in the REM state makes it a suitable candidate for synchrono entrainment with other rhythmic phenomena. SWS cycles are also inherently periodic, but bra activation levels decline in SWS. Since attachment presumably requires elevated activation levels (e.g. to promote approach tendencies), SWS would not be a suitable candidate for the attachment mechanism. More importantly, those brain systems (limbic, frontal, etc.) that are periodically activated in REM (and not activated in SWS or Stage 2 NREM) may be critical for attachment. It is not surprising, therefore, that disruption of the maternal—infant bond would be associated with REM sleep fragmentation in all species studied. Beebe, Gertsman and Carso (1982), Bernieri, Reznick and Rosenthal (1988) and Belsky and Nezoworski (1988), amore others, have documented the role of synchrony in the attachment process (in humans) between mothers and infants. Poor attachment is reliably associated with disruption in the norm synchronous interaction between mother and infant.

Hofer (1987) has reviewed the psychobiology of early attachment in mammals. Matern deprivation (prolonged separation of the infant from the mother) is associated with disrupted and decreased REM sleep in the "despair phase" of the separation (protest then despair) process. SW is unaffected. Disruption of REM sleep after mother—infant separation is also known to occur is humans and in most primate species (see Kalin & Carnes, 1984; Reite & Capitanio, 1985, for recent reviews).

Hofer and his colleagues have also shown that nursing (the paradigmatic attachment behave ior) in the rat is controlled by an elaborate set of cues all embedded in sleep. Infants sleep while they are nursing and suck while they are asleep. Their sucking induces milk ejections (via oxy tocin release) to occur in the mother. However, oxytocin release in the mother cannot occur unless she is in slow wave sleep (SWS). Presumably when oxytocin release occurs in SWS, lev els will peak with the onset of REM. Interestingly, oxytocin levels in humans peak in the earl hours of the morning (see below) when REM duration is greatest. The infant is apparently i active sleep while nursing, since it undergoes periodic 30-second awakenings while at the nip ple. In other species suckling and milk ejection is associated with desynchronized (REM-like electrocorticographic activity in the frontal lobes (Neve, Paisley & Summerlee, 1982). Thus, bot mother and infant can nurse only when cues are passed from one to the other during the sleet state. Hofer following Stern (1985) calls these exquisitely timed sleep interactions between mother and infant "attunement behaviors", or examples of synchrony between the mother and infant. Other attunement behaviors that support attachment have been found to be tied more specifically to REM sleep. Emde and Koenig (1969), for example, found that the earliest smile of the infant arise out of transient REM states. Smiling, in turn, in the infant is known to elici caretaking responses in the mother. In addition, active sleep is associated with greater motor and grasping activity, with greater autonomic nervous system variability and therefore greate amounts of crying, cooing and babbling. All of these infant behaviors reliably elicit caretaking behavior in the mother.

These sleep-related attachment processes would work more effectively if the infant slept near the mother. Interestingly, co-sleeping (where child and parent sleep in close proximity) apparently is a near universal mammalian practice. Co-sleeping is also practiced in virtually every human culture (McKenna, 1993). The sole exception is 20th century industrialized cultures. Sagi van Ijzendoorn, Aviezer, Donnell and Mayseless (1994) hypothesized that communal sleeping arrangements (where the infant sleeps away from the mother) deviates markedly from the child rearing associated with the environment of evolutionary adaptedness (EEA) for humans. The EEA is best approximated by infant—mother co-sleeping, and this co-sleeping promoted attachment, Sagi et al. found that only 48% of communal (away from mother) sleeping infants were

securely attached to their mothers, while 80% of the home-based (with or near mother) infants were securely attached.* As the bonding hypothesis predicts, sleeping patterns do make a difference for infant-mother attachment. Finally, Vladimirova (1983) has found that REM sleep is important for social adaptation beyond the infancy stage. She observed children's sleep-wake patterns (mean age two years, three months) while the children were socially adapting to day nursery school and found that social adaptation was associated with REM sleep intensity and not with slow wave sleep variables.

Attachment processes are mediated by specialized neurohormonal systems. Neuro-hypophysial hormones like oxytocin and vasopressin, as well as arginine vasotocin, seem to be especially important for attachment and other social and sexual behaviors (see papers in Pedersen, Caldwell, Kirikowski and Insel, 1992). The chemical composition of the three hormones differ in only a single amino acid. All three hormones promote sexual and social behaviors in a vast array of species. Oxytocin, for example, like REM sleep, is found exclusively in mammals. It is synthesized in the hypothalamus and is released from the neurohypophysis to induce uterine contraction during labor and milk ejection during nursing. Oxytocin also functions as a neurotransmitter. Its synthesizing cells in the hypothalamus send efferents to widespread areas of the limbic system and frontal lobes making contact with specific receptors for oxytocin. When injected centrally it induces several of the behaviors associated with social bond formation in mammals (e.g. contact with young, caretaking behaviors, and species-typical reproductive behaviors) (Insel, 1992). Oxytocin plays a central role in the expression of maternal behavior, sexual behavior, social bond formation, grooming, memory and learning, autonomic regulation, feeding and yawning (Argiolas and Gessa, 1991). This last function (yawning) is particularly interesting given its relation to sleep. When yawning is induced in rats via centrally administered oxytocin it is invariably associated with penile erection (Melis, Stancampiano & Argiolas, 1992). Penile erections occur exclusively in the REM state during sleep. Oxytocin-induced erections can be prevented by depletion of oxytocin in the hypothalamic-hippocampal oxytocinergic pathway (Melis et al., 1992). This pathway, in turn, may play a role in regulation of the theta rhythm associated with REM sleep (Bohus, Urban, Van-Wimersma-Greidanus & de Wied, 1978). Finally, oxytocin appears to display a sleep-sensitive pattern of release in humans with peak levels occurring at about 4 o'clock in the morning (when REM sleep begins to predominate over NREM) (Forsling, 1993).

Arginine vasotocin (AVT) is considered to be the evolutionary precursor to vasopressin (Pedersen et al., 1992). Both vasopressin and oxytocin promote various types of bonding behaviors in a range of mammalian species. Vasotocin promotes sexual behaviors in various non-mammalian species. Subcutaneous injection of AVT to human subjects causes a selective increase in REM sleep. Intranasal administration causes a dramatic enhancement of REM sleep indices. Lumbar CSF levels of AVT vary with REM sleep indices, and AVT levels are abnormally high during REM sleep in narcoleptics (Popoviciu, Corfariu, Foldes, Farkas, Goldstein & Pavel, 1979). Finally, prolactin and testosterone levels vary with the stages of sleep. Testosterone levels in males, for example, are highest at the transition from NREM stages to REM (see Borbely & Tobler, 1989, for review and critique of the literature on REM sleep-associated endogenous substances).

Brain systems in attachment

In their review of the neuroanatomic structures that mediate attachment, Steklis and Kling (1985) concluded that limbic system sites (especially the amygdala), the anterior temporal cortex and the orbital frontal cortex were critical for attachment. Lesioning any of these three sites

^{*} I thank an anonymous reviewer for bringing this study to my attention.

42 P. McNamara

produces profound disruption in attachment processes. As Steklis and Kling point out, anteritemporal, orbito-frontal and amygdaloid nuclei all synthesize sensory information (multimod sensory information) with emotional information. Stimulation of any of these sites also produce a variety of autonomic changes. Thus, these forebrain regions seem to play a special role in regulating homeostasis, adaptive behaviors and emotional behaviors—all important processes development of attachment. As discussed earlier, these limbic/frontal forebrain sites that are scritical for attachment are also the sites activated in REM sleep and the sites that are enlarge (relative to body weight) in species without REM.

Summary of evidence

REM sleep appears to involve activation of attachment-related systems: limbic/frontal systems; oxytocinergic systems; AVT systems, and sexual and reproductive systems. Across species REM sleep indices (e.g. duration) vary with altriciality, and within a species REM sleep indice vary with attachment needs.

DREAMS AND ATTACHMENT

In the mature state dreams are predominantly visual phenomena. Reports of smell, taste of pain sensations are very rare (Hobson, 1988; Symons, 1993). Although we are hypermness within the dream state (many memories become available during a dream), we are often amnetic for dreams upon awakening (Crick & Mitchison, 1986; Hobson, 1988). Extraordinary even in dreams tend to be uncritically accepted (Hobson, 1988). Dream images often function as symbols or metaphors for emotionally significant events in the dreamer's life. Finally, dreams tend to have a story-like or narrative quality (see Foulkes, 1985; Hobson, 1988 and Symons, 1993, for discussions of the phenomenological properties of dreams).

If dreaming is often about attachment, why should dreams have just those phenomenological properties? We are predominantly visual creatures, so the visual sense "leads". Pain and unplease ant tastes or smells would interfere with the approach tendencies the REM state is designed to promote. Oxytocin is known to suppress olfactory functioning in order to promote maternate behaviors in rats (Pedersen et al., 1992). It (oxytocin) is also known to promote forgetting in wide range of species (see references in Pedersen et al., 1992). The REM-associated rise in oxytocin and AVT levels might help to explain poor recall of dreams. It is, furthermore, not necessary to consciously remember moments of synchrony in order for bonding to occur. All that inneeded is repeated and periodic activations of the systems designed to orient an organism towar fusing with another. This "fusing with another" may explain the vaguely threatening character of some dreams. Unconscious symbiotic fantasies may in some sense be threatening, since fusion represents dissolution of self.

To the extent that REM sleep allows for expression of libidinal and sexual wishes, dreaming should be intrinsically gratifying (or "drive dampening"). However, since REM sleep also involves activation of object search strategies (again in service to attachment), dreaming must also involve activation of a need state ("drive heightening"). In short, REM sleep appears to allow for the expression of desire of another, and it can therefore be both pleasant and unpleasant. Since attachment, furthermore, involves some degree of synchrony and symbiosis with another, the dream-ego may be threatened with dissolution. Thus, dreaming [as Hartmann (1991) has pointed out] may be particularly unpleasant for those individuals with thin or developing boundaries (e.g. children and subjects who suffer nightmares).

We have seen that attachment theory requires construction of an internalized object or searchimage that represents the desired-for attachment object. The object is literally a visual image that
elicits libidinal strivings in the dreamer. Freud asserted that dream content needs to be disguised
because it represents unacceptable (to the ego) wishes and desires. Thus, we turn these wishes
into elaborate symbol systems and then repress all memory of the dream upon awakening. But
there may be no need to invoke disguise and suppression in order to explain poor dream recall.
The REM-associated rise in oxytocin and AVT levels might help to explain poor recall of dreams.
In addition, attachment is too important a function to put into the hands of a conscious ego. It
must proceed automatically and unconsciously.

Dream content

If REM sleep is involved in attachment processes, then we would expect that some significant proportion of REM-associated dreams would reflect "bonding themes" (especially in subjects not currently attached). Content analyses of large numbers of dreams are consistent with this prediction (Hall, 1966). We tend to dream mostly of family members, love or sexual objects and friends. Curiously, many dreams are rated as unpleasant experiences by both children and adults. Perhaps these ratings reflect the basically needy fetal state we are all subject to during REM. In order to explain other facts concerning dream content (e.g. the large number of aggressive acts committed by males) it is necessary to view dreaming within an evolutionary psychology framework.

The evolutionary context

Human attachment behaviors and (by implication) REM sleep's role in attachment evolved hundreds of thousands of years ago when humans lived in small hunter—gatherer groups situated in the wild. REM sleep attachment systems, in other words, were designed for a person who was born helpless but prepared to meet and to attach to an other (mother); whose survival depended on that mother; who grew up prepared to bond with a mate and (if male) to compete with other males for access to women; who sexually reproduced at adolescence; and then intensely invested in a child for about four years (until the next pregnancy); and then died by 40 years of age, REM sleep phenomena, in other words, correlate with the life-cycle (or with the attachment needs) not of modern humans but of a human being who lived in the "Environment of Evolutionary Adaptation" or the so-called EEA. REM sleep "expects", for example, that an infant will co-sleep with the mother and that pair-bonded adults will also co-sleep. The emotional content of dreams, therefore, should partially reflect social bonding needs and social conditions of the EEA. Take the case of sex differences in dream content. Men's dreams are more aggressive than women's. Why? The attachment hypothesis may throw some light on the matter.

Since the female egg is biologically more expensive to produce than male sperm, females in the EEA represented the limiting resource in human reproductive biology. Thus, males had to compete for access to the females, and aggression between males was common. In order for males to successfully pair-bond with a female they had to win the competition with other males. Object-seeking for males, therefore, requires dealing aggressively with other males, and these facts are reflected in their attachment-oriented dreams. Object-seeking for females, on the other hand, involved no comparable aggressive competition with other females; rather, females merely had to choose a suitable mate and then get him to provide regular resources to her and the child. These sex differences in object-seeking strategies appear to be reflected in the dreams of men and women. Two-thirds of the characters in male dreams are males (with whom they have aggressive interactions), while females have approximately equal male—female ratios.

44

Whether or not the attachment hypothesis throws any new light on the phenomenologic properties of dreams, it seems reasonable to suggest that it may be consistent with an old view of the interpretation of dreams—namely Freud's. Freud argued that the latent content of modreams represented a revival of infantile libidinal strivings. If REM sleep is designed to promos social bonding, then one strategy would be to reactivate the systems utilized by infants to attact to a care-giver. Some current theories of love and adult pair-bonding conceptualize romanti bonding as an extension of basic attachment processes (Shaver, Hazan & Bradshaw, 1988). Thus it seems reasonable to pursue some of Freud's insights concerning the revival of infantile object seeking strivings during dreams.

LIMITATIONS OF THE HYPOTHESIS

There are some correlates of REM sleep that are not so readily explained with the bonding hypothesis. Why, for example, is REM associated with paralysis of the anti-gravity muscles. Why, for that matter, are there rapid eye movements? What about non-altricial or precocia species? How important is REM for bonding in those species? Why does the duration of a REM episode increase across the night, i.e. why does REM occur mostly towards the end of the night. Finally, there is the problem of dream content. While many dreams might be interpreted as wishfullfillment/object-seeking scenarios, many more cannot be so interpreted. I will not attempt to answer any of these important objections to the bonding hypothesis, except to point out again that REM sleep probably involves several functions beyond those associated with social bonding.

PREDICTIONS

I have already mentioned several predictions that flow logically from the hypothesis. I will add a few more here.

- 1. The most obvious prediction is that REM deprivation should be associated with impaired attachment or social bonding. REM-deprived rats, for example, should show poor attachment, while SWS rats should not. Parsing out side-effects of sleep deprivation on behavior is, of course, always a problem in these experiments. REM-deprived human subjects should perform poorly on tests that measure ability to emotionally connect with another. Conversely, infants showing insecure attachment patterns should also show some evidence of REM sleep disruption.
- Percent of time spent in REM should be greater in altricial species than in precocial species.Properties of REM sleep should vary with the social bonding patterns of a given species.
- REM sleep should be disrupted in disorders associated with social bonding deficits. These
 disorders include autism, Asperger's syndrome, some forms of schizophrenia, sociopathy,
 endogenous depression and possibly anorexia.
- 4. The brain anatomy of REM sleep and that of social bonding should overlap to a significant extent. Limbic/frontal lesions should disrupt REM sleep and social bonding. Lesions elsewhere should not.

In summary, REM sleep is associated with selective activation of brain regions (limbic and frontal) that are known to be critical for attachment. Although the evidence suggests that attachment is one, perhaps crucial, function associated with REM sleep, I do not wish to argue that attachment is the only function of REM sleep. Our dreams seem to reflect all kinds of themes and concerns beyond those of attachment. Anthropological studies of the role of dreams in culture

suggest that dreams can unify whole tribes around certain goals, etc. REM sleep seems to support a host of other physiological functions from thermoregulation to memory consolidation to healing. Any single explanation of *The* function of REM sleep and dreaming will probably not be able to capture the fascinating richness of the dream experience (see papers in Moffit *et al.*, 1993). Nevertheless, the attachment hypothesis is consistent with a large range of the evidence concerning the possible psychobiologic functions of REM sleep. The hypothesis, therefore, deserves further study.

ACKNOWLEDGEMENTS

I would like to thank Dr Brad Lown, Howard Reid, Michael Zborowski, David Kemmerer, Ernest Hartmann and Virginia Wyly for comments on earlier drafts of this paper.

REFERENCES

Allison, T., Van Twyver, H. & Goff, W. (1972). Electrophysiological studies of the echnida Tachyglossus aculeatus: I. Waking and sleeping. Archives of Italian Biology, 110, 145–184.

Argiolas, A. & Gessa, G. (1991). Central functions of oxytocin. Neuroscience and Biobehavioral Reviews, 15, 217–231.
Aserinsky, E. & Kleitman, N. (1953). Regularly occurring periods of eye motility and concomitant phenomena during sleep. Science, 118, 273–274.

Barnet, P. A. & Gotlib, I. H. (1988). Psychosocial functioning and depression: Distinguishing among antecedents, concomitants, and consequences. Psychological Bulletin, 104, 97–126.

Beebe, B., Gertsman, L. & Carson, B. (1982). Rhythmic communication in the mother-infant dyad. In M. Davis (Ed.), Interaction Rhythms: Periodicity in Communicative Behavior, New York: Human Sciences Press.

Belsky, J. & Nezoworski, T. (1988). Clinical Implications of Attachment. Hillsdale, NJ: Lawrence Erlbaum Associates. Bernieri, F. J., Reznick, S. J. & Rosenthal, R. (1988). Synchrony, pseudo synchrony, and dissynchrony: Measuring the entrainment process in mother-infant interactions. Journal of Personality and Social Psychology, 54, 243-253.

Bohus, B., Urban, I., Van-Wimersma-Greidanus, T. & De Wied, D. (1978). Opposite effects of oxytocin and vasopressin on avoidance behavior and hippocampal theta rhythm in the rat. Neuropharmacology, 17, 239–247.

Borbely, A. A. & Tobler, I. (1989). Endogenous sleep promoting substances and sleep regulation. Physiological Reviews, 69, 605–670.

Bowlby, J. (1988). A Secure Base. New York: Basic Books.

Broughton, R. (1982). Human consciousness and sleep/wake rhythms: A review and some neuropsychological considerations. *Journal of Clinical Neuropsychology*, 4, 193–218.

Buysse, D. J., Reynolds, C. F. & Kupfer, D. J. (1993). Depression. In M. A. Carskadon (Editor-in-Chief), Encyclopedia of Sleep and Dreaming (pp. 169–175). New York: Macmillan Publishing.

Crick, F. & Mitchison, G. (1986), REM sleep and neural nets. The Journal of Mind and Behavior, 7, 229(99)–250(120).
Dement, W. (1960). The effect of dream deprivation. Science, 131, 1705–1707.

Dement, W. (1965). Recent studies on the biological role of rapid eye movement sleep. American Journal of Psychiatry, 122, 404–408.

Dement, W. & Kleitman, N. (1957). The relation of eye movements during sleep to dream activity: An objective method for the study of dreaming. *Journal of Experimental Psychology*, 53, 339–346.

Emde, R. N. & Koenig, K. L. (1969). Neonatal smiling and rapid eye movement states. Journal of the American Academy of Child Psychiatry, 8, 57–67.

Field, T. (1985). Attachment as psychobiological attunement. Being on the same wavelength In M. Reite & T. Field (Eds.), The Psychobiology of Attachment and Separation (pp. 415-454). New York: Academic Press.

Forsling, M. (1993). Neurohypophysial hormones and circadian rhythm. In W. North, A. Moses & L. Share (Eds.), The Neurohypophysis: A Window on Brain Function: (Vol. 689, pp. 382–395). New York: The New York Academy of Sciences.

Foulkes, D. (1985). Dreaming: A Cognitive-Psychological Analysis. Hillsdale: Lawrence Eribaum Associates.

Gillin, J. C., Buchsbaum, M. S. & Wu, J. C. (1993). Cerebral metabolism. In M. A. Carskadon (Editor-in-Chief), Encyclopedia of Sleep and Dreaming (pp. 96–98). New York: Macmillan Publishing.

Hall, C. S. (1966). The Meaning of Dreams. New York: McGraw-Hill.

Hartmann, E. (1973). The Functions of Sleep. New Haven: Yale University Press.

Hartmann, E. (1991). Boundaries in the Mind: A New Psychology of Personality. New York: Basic Books.

Hirshkowitz, M. (1993). Sex and sleep. In M. A. Carskadon (Editor-in-Chief), Encyclopedia of Sleep and Dreaming (pp. 535–537). New York: Macmillan Publishing.

Hobson, J. A. (1988). The Dreaming Brain. New York: Basic Books.

- Hofer, M. A. (1987). Early social relationships: A psychobiologist's view. Child Development, 58, 633–647.
- Insel, T. (1992). Oxytocin and the neurobiology of attachment. Behavioral and Brain Sciences, 15, 515-516.
- Jouvet, M. (1960). Telencephalic and rhombencephalic sleep in the cat. In G. E. W. Wolstenholme & M. O'Connor (Ed CIBA Foundation Symposium on the Nature of Sleep (pp. 188-208). Boston: Little Brown.
- Jus, A., Jus, K., Villeneuve, A., Pires, A., Lachance, R., Fortier, J. & Villeneuve, R. (1973). Studies on dream recal chronic schizophrenics after prefrontal lobotomy. Biological Psychiatry, 6, 275.
- Kalin, N. & Carnes, M. (1984). Biological correlates of attachment bond disruption in humans and nonhuman prima Pragress in Neuro-Psychopharmacology and Biological Psychiatry, 8, 459-469.
- Kaufman, K. (1993). Birds. In M. A. Carskadon (Editor-in-Chief), Encyclopedia of Sleep and Dreaming (pp. 76-New York: Macmillan Publishing.
- Kraemer, G. (1992). A psychobiological theory of attachment. Behavioral and Brain Sciences, 15, 493–541.
- Kripke, D. F. & Sonnenschein, D. (1978). A biologic rhythm in waking fantasy. In K. S. Pope and J. L. Singer (Ed The Stream of Consciousness: Scientific Investigations into the Flow of Human Experience. New York: Plenum Pro
- McCarley, R. W. (1989). The biology of dreaming sleep, In M. H. Kryger, T. Roth & W. C. Dement (Eds.), Princip and Practice of Sleep Medicine (pp. 173-183). Philadelphia: Saunders.
- McKenna, J. J. (1993). Co-sleeping. In M. A. Carskadon (Editor-in-Chief), Encyclopedia of Sleep and Dreaming (143–148). New York: Macmillan Publishing.
- Melis, M., Stancampiano, R. & Argiolas, A. (1992). Hippocampal oxytocin mediates apomorphine induced penile er tion and yawning, Pharmacology, Biochemistry and Behavior, 42, 61-66.
- Moffitt, A., Kramer, M. & Hoffman, R. (1993). The Functions of Dreaming. Albany: State University of New York Pre Morgane, P. J., Jacobs, M. S. & McFarland, W. L. (1980). The anatomy of the brain of the bottlenose dolphin (Tursie truncatus). Surface configurations of the telencephalon of the bottlenose dolphin with comparative anatomical obs vations in four other cetacean species, Brain Research Bulletin, 5, 1-107,
- Neve, H., Paisley, A. & Summerlee, A. (1982). Arousal: A prerequisite for suckling in the conscious rabbit? Physiole and Behavior, 28, 213-217,
- Oscar-Berman, M., McNamara, P. & Freedman, M. (1991). Delayed response tasks: Parallels between experimental ab tion studies and findings in patients with frontal lesions. In H. S. Levin, H. M. Eisenberg & A. L. Benton (Eds.), Fron Lobe Function and Injury (pp. 230-255). New York: Oxford University Press.
- Pedersen, C. A., Caldwell, J. D., Jirikowski, G. F. & Insel, T. R. (1992). Oxytocin in Maternal, Sexual and Soc Behaviors (Vol. 652). New York: Annals of the New York Academy of Sciences.
- Popoviciu, L., Corfariu, O., Foldes, A., Farkas, E., Goldstein, R. & Pavel, S. (1979). REM sleep dependent release vasotocin into cerebrospinal fluid of narcoleptics. Waking and Sleeping, 3, 341-346.
- Reite, M. & Capitanio, J. (1985). On the nature of social separation and social attachment. In M. Reite & T. Field (Ed The Psychobiology of Attachment and Separation. New York: Academic Press.
- Reite, M. & Field, T. (1985). The Psychobiology of Attachment and Separation. New York: Academic Press.
- Roffwarg, H. P., Muzio, J. N. & Dement, W. C. (1966). Ontogenetic development of the human sleep-dream cyc Science, 152, 604-619.
- Sagi, A., van Ijzendoom, M. H., Aviezer, O., Donnell, F. & Mayseless, O. (1994). Sleeping out of home in a kibbutz cor munal arrangement: It makes a difference for infant-mother attachment. Child Development, 65, 992-1004.
- Shaver, P., Hazan, C. & Bradshaw, D. (1988). Love as attachment: The integration of three behavioral systems. In R. Sternberg and M. L. Barnes (Eds.), The Psychology of Love (pp. 68-99). New Haven: Yale University Press.
- Siegel, J. (1993). Function of REM sleep. In M. A. Carskadon (Editor-in-Chief), Encyclopedia of Sleep and Dreamin (pp. 507-510). New York: Macmillan Publishing.
- Skinner, J., Molnar, M. & Harper, R. (1989). Higher cerebral regulation of cardiovascular and respiratory functions. In M. Kryger, T. Roth, & W. C. Dement (Eds.), Principles and practice of sleep medicine (pp. 231-251). Philadelphia: Saunder
- Steklis, H. & Kling, A. (1985). Neurobiology of affiliative behavior in nonhuman primates. In M. Reite, & T. Fields (Eds.) The Psychobiology of Attachment and Separation (pp. 93-134), New York: Academic Press.
- Steriade, M. (1989). Brain electrical activity and sensory processing during waking and sleep states. In M. H. Kryge T. Roth & W. C. Dement (Eds.), Principles and Practice of Sleep Medicine (pp. 86-103). Philadelphia: Saunders.
- Stem, D. N. (1985). The Interpersonal World of the Infant. New York: Basic Books.
- Symons, D. (1993). The stuff that dreams aren't made of: Why wake-state and dream-state sensory experiences diffe Cognition, 47, 181-217, Villablanca, J., Marcus, R. & Olmstead, C. (1976). Effects of caudate nuclei ablation or frontal cortex ablations in cat-
- II. Sleep-wakefulness, EEG, and motor activity. Experimental Neurology, 53, 31–50.
- Vladimirova, G. (1983). Polygraphic characteristics of daytime sleep during social adaptation of normal children les than three years of age. Activitas Nervosa Superior, 25, 59-61.
- Vogel, G. W. (1975). A review of REM sleep deprivation. Archives of General Psychiatry, 32, 749–761.
- Vogel, G. (1993). Selective deprivation. In M. A. Carskadon (Editor-in-Chief), Encyclopedia of Sleep and Dreaming (pp 178-180). New York: Macmillan Publishing.
- Vogel, G. W., Vogel., F., McAbee, R. S. & Thurmond, A. J. (1980). Improvement of depression by REM sleep depriva tion. Archives of General psychiatry, 37, 247-253.
- Winson, J. (1985). Brain and Psyche: The Biology of the Unconscious, New York: Doubleday.
- Zepelin, H. (1989). Mammalian sleep. In M. Kryger, T. Roth & W. C. Dement (Eds.), Principles and Practice of Slee, Medicine (pp. 30-49). Philadelphia: Saunders,