REVIEW ARTICLE

Dreaming and Hypnosis as Altered States of the Brain-Mind

David Kahn, M.D. and J. Allan Hobson, M.D.

The primary purpose of this article is to put forth the state space concept of brain-mind function with special emphasis on the rapid eye movement (REM) state. We also consider other states such as lucid dreaming, sleep walking and hypnosis. The methodology correlates first person and third person information, such as the subjective experience of dreaming (first person) with the measurement of regional blood flow (PET and fMRI) during the REM state (third person). Our findings suggest that a continuum of brain-mind states exist ranging from the naturally occurring and stable states of waking, REM and non rapid eye movement (NREM) to induced and less stable states such as lucid dreaming and hypnosis. A heuristic three-dimensional model with which to visualize this continuum of brain-mind states is presented. This model is called AIM because it is based on the variables of brain region activation (A), the proportionate amount of external to internal information that is processed by the brain (I), and the proportionate amount of aminergic and cholinergic neuromodulation (M) of the brain. The underlying simplifications of the model are discussed as well as what details should be added in the future. The state space model is illustrative of how altered states of consciousness may be visualized as domains in brain-mind state space. **(Sleep and Hypnosis 2003;5(2):58-71)**

Key words: *sleep, dreaming, hypnosis, AIM, brain-mind, altered states, neuromodulation, activation*

INTRODUCTION

The study of dreams and its underlying neurophysiological mechanisms can provide important insights into how changes in the brain can account for changes in the conscious experience of waking and sleep. The study of the brain basis of dreaming is an important herald of what needs to be done to understand hypnosis at the level of the brain. In this paper we point out similarities and differences between sleep, dreaming and hypnosis but our main emphasis will be on the new science of dreaming.

The mental and emotional output of the brain during REM sleep differ considerably from the output of the brain during waking. To get at this difference we assess and measure what we call the formal properties of the mental content of the states waking and REM sleep (as well as sleep onset). In this way we attempt to articulate the distinguishing universal characteristics of dreaming as against the individual content of dreams. We are thus able to compare human brain regional activation

From the Laboratory of Neurophysiology, Department of Psychiatry, Harvard Medical School, Massachusetts Mental Health Center, Boston, MA, USA

Address reprint requests to: Dr. David Kahn, Neurophysiology Laboratory, Department of Psychiatry, Harvard Medical School
Mass Mental, 74 Fenwood Road, Boston, MA 02115 USA
Phone: 617 626 9481, Fax: 617 734 7851
e-mail: David_kahn@hms.harvard.edu

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pattern changes associated with dreaming and waking consciousness. With animals we are also able to compare brain chemistry changes in the wake and rapid eye movement (REM) state. Combining the evidence of regional activation and brain chemistry changes with changes in mental output, we hypothesize that changes in the mental output of the brain-mind during REM sleep dreaming are caused by changes in chemical and electrical activation patterns in the brain. In humans, the REM sleep stage is not the only sleep stage where dreaming occurs, but it is the stage from which dreams are most often recalled and for which dream reports are more vivid, longer, more bizarre and more emotional than from other sleep stages (1). To account for NREM dreaming we point out that the shift in activation, input-output and the modulation parameters is about half way between waking and REM. We show that the intensity and character of NREM dreaming matches these values.

We further hypothesize that differences in regional activation and neuromodulation may not be independent (2). According to this hypothesis, the shift in brain stem modulation from aminergic in waking to cholinergic in REM also directly affects regional cortical activation (3).

FORMAL PROPERTIES OF DREAMS

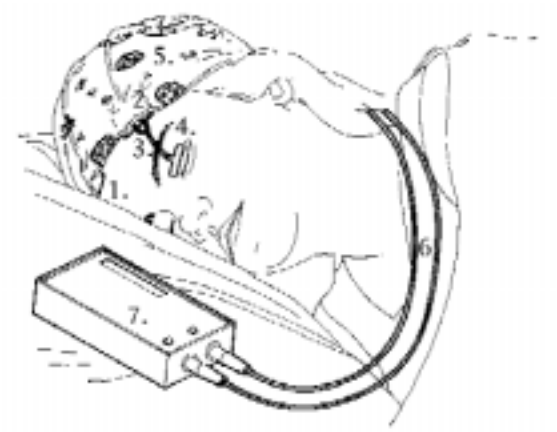
In this section we define five formal properties of dreams and assert that we can infer the nature of the dreaming state from a description and analysis of these formal properties. While the content of dreams may reveal emotionally salient concerns such as anxiety about not feeling prepared to carry out an assignment or feeling anxiety about missing travel connections, dream content alone cannot be used to define the dreaming state. For that we need to see what makes a dream a dream. We need to uncover the basic characteristics of dreams, what we have called the formal properties of dreams (3). To help do this a large database of mental activity during wake and sleep states was obtained from 16 subjects who provided 1600 reports of their conscious experience in five states: quiet waking; active waking; sleep onset (15, 30, 45, 60 and 75 seconds after falling asleep); NREM; and REM sleep. The subjects were instrumented to record sleep at home (4) as shown in Figure 1A.

Internally generated percepts (Hallucinations)

We hallucinate while dreaming. We see things also that are not there and we have the strong illusion of moving though dream space. We sometimes also hear, feel or smell in dreams. Hallucinations are a basic characteristic of dreaming, especially visuo-motor hallucinations. When subjects are asked to report on internally generated percepts we find that they are essentially non-existent in active waking, increase slightly in quiet waking, then increase progressively at sleep onset in NREM sleep and reach their peak in REM (5). Overall, there was a clear reciprocal relationship between thoughts and hallucinations as the data in Figure 1B shows.

False beliefs (Delusional)

All dreams are delusional. We are deluded while dreaming into believing we are awake even though we are asleep. We believe that we are perceiving a real outside world even though we create that world in our sleep, and we are unable to critically assess the contents of our delusion. Despite improbabilities of character and situation the dreamer is deluded into believing the wild tale of the dream as if it were occurring while awake, and not, in fact, while asleep. How can changes in the brain account for this? To explain dream delusion, we emphasize the increase in hallucinating autoactivation and the reciprocal impoverishment of memory and thinking (see



1. head movement sensor
2. eyelid movement sensor mount
3. eyelid sensor lead
4. eyelid sensor with adhesive backing
5. bandanna (worn "pirate style")
6. wires from sensors to Nightcap unit
7. Nightcap recording unit

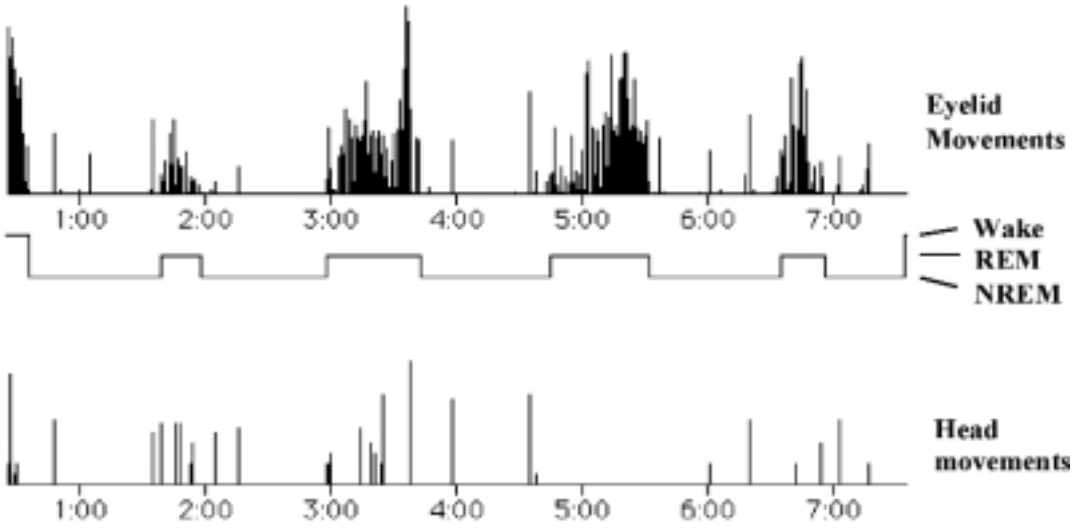


Figure 1A. The Nightcap (from Reference 3). Line drawing made from a photograph of a subject sleeping with the Nightcap. The Nightcap consists of the 7 elements listed in the figure next to the line drawing. The top trace is a histogram plot of eyelid movements. The trace immediately below that is a hypnogram of computer-scored Nightcap data. The bottom trace is a histogram plotting head movements. Time of night is indicated on the horizontal axis.

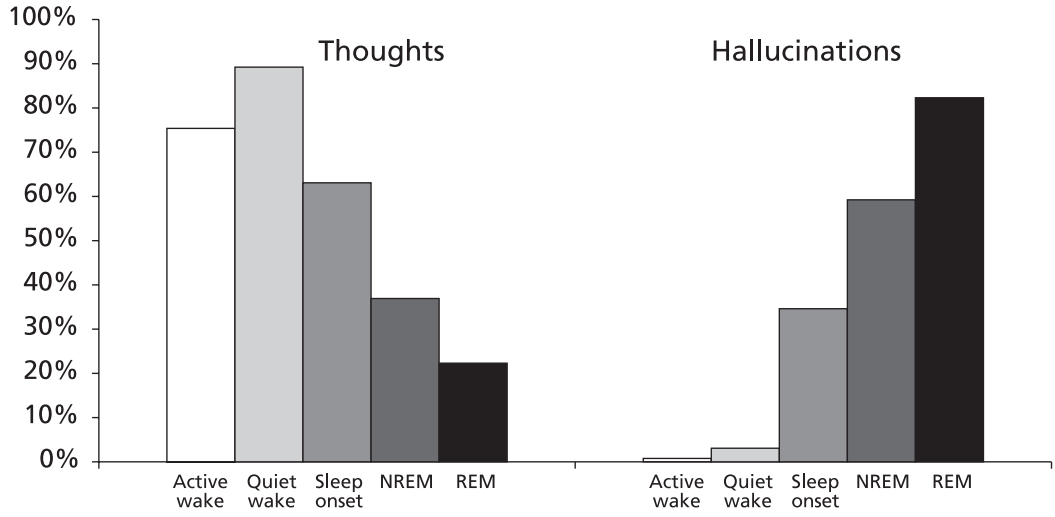


Figure 1B. Thoughts and Hallucinations (from Reference 5). The figure shows the percentage of reports with thoughts and percentage of reports with hallucinations in each wake-sleep state. We see a reciprocal relationship between thoughts and hallucinations across the wake and sleep states.

below). And in those relatively rare instances of lucid dreaming when one becomes aware that one is dreaming, can changes in the brain also account for this? In a later section we will suggest a state space diagram to see how both of these conditions may be visualized.

Cognition abnormalities (Changed and new associations)

Dreaming is characterized by the creation of stories which may contain unusual and new associations between characters and elements within the stories. The evidence that unusual associations are a byproduct of REM sleep dreaming comes from experiments which have shown that there is an enhancement of weak priming associations within the first few minutes of waking following REM sleep. Subjects retain some REM sleep neuromodulation and neurophysiology characteristics during the first few minutes of waking following REM sleep (6).

In a semantic priming task a list of words is shown in one column while a second column may contain real words or may contain non-words. The real words may be strongly associated with the word in the first column, for example, if the word in the first column is dog, a strongly associated word would be cat. A word that is not strongly associated with dog would be raccoon. A non-word might be dgo. The subject is asked to indicate whether the word in the second column is a real word. When the word in the second column is strongly associated with the word in the first column, normally (in the waking state) the subject is fastest at indicating that the letter string is a word (cat would be very quickly recognized as a word when paired with dog). When the target word is only weakly associated with the prime word in the first column, (say, raccoon paired with dog) the subject takes longer to indicate that the string is a word. However, when subjects are awakened from REM sleep, they recognize weakly primed words more quickly than strongly primed ones (raccoon paired with dog is more quickly recognized as a real word than is cat paired with dog). The interpretation of these results is that non-common associations are made more easily than the common ones during REM sleep dreaming. This would lead to the often creative, and sometimes bizarre images within a dream narrative (6).

Other experiments have shown that people are creative and more cognitively flexible within the first 15 minutes of waking following REM sleep awakening (7). This study compared the performance on anagram word puzzles following REM and NREM awakenings and waking performance during the day. REM awakenings produced a significant advantage in the number of anagrams solved compared with NREM awakenings and was equal to that of wake time trials. It was hypothesized that the data indicate that the neurophysiology of REM sleep represents a brain state more amenable to flexible cognitive processing than NREM.

Cognition abnormalities (Thinking in dreams)

References to thinking decline sharply across the sleep states in reports as references to internally generated percepts increase. This reciprocal process reaches its peak in REM which is percept rich and thought poor (5) as shown in Figure 1B.

Thinking in dreams is at times as logical as it is in waking but at other times it is uncritical and illogical. This is illustrated by an excerpt from a dream report of one of us with comments inserted.

"I am in a pool with the blue fish and my friend Alan S. and he is sort of taking care of them, teaching them. At one point many of the blue fish are outside the pool, sort of on the edge. I wonder how come they can breathe so long outside of the water. (Notice how some thinking is preserved, e.g., knowing that blue fish cannot breathe out of the water, while other thinking is absent, e.g., not realizing that going

from the ocean to a pool and having my friend teach blue fish was strange). Then I see they have tubes that are in their mouths that go into the water. I am flabbergasted at how clever they are using the equivalent of human snorkels to breathe but in their case, breathe oxygen when the tube is submerged into the water (Again, notice how some thinking is sharp, devising reverse snorkels so the fish can breathe is creative, but uncritical in believing that the fish themselves devised the reverse snorkels!)."

In addition, it is noteworthy that within the dream, implausible aspects often go unrecognized until we have awakened. Thus, while implausibility is sometimes discerned within the dream, more often it is not (8). This leads to the conclusion that reflective consciousness is diminished during dreaming as evidenced by improbable, unlikely or implausible events going unnoticed as such while dreaming.

Other cognition abnormalities of dreams, e.g., bizarreness, emotionality and amnesia

Dream bizarreness has been shown to be present in improbable and sometimes impossible situations (called incongruity) and in plot shifts and character transformations (called discontinuity) (9). Specific events that occurred during waking hours are rarely replayed exactly during dreaming indicating that episodic memory is impoverished (10). Dreams are not easily recalled upon awakening despite often being highly emotional with elation, anxiety and anger being particularly strong.

Mixtures of states

At the edges of sleep are hypnagogic hallucinations (sleep onset), hypnopompic hallucinations (continuation of REM sleep dreaming into waking), and sleep paralysis (continuation of REM sleep atonia after awakening); within sleep are sleep walking, sleep talking, and lucid dreaming. In these conditions consciousness has features characteristic of one state mixed with features characteristic of another. Hypnosis is also a dissociated state of waking into which many of the features of sleep have been inserted (3). The ability to be hypnotized, (hypnotizability), is correlated with the ability to imagine and the ability to focus concentration internally and become extremely absorbed in imaginative activity even to the point of hallucination (11, 12). As shown in Table 1, this is a hallmark of dreaming, as well. This suggests that the study of sleep may provide landmarks for improving our scientific understanding of hypnosis.

Some of the Phenomenological factors in hypnosis and dreaming are noted below and summarized in Table 1.

Responsiveness to external stimuli

Awareness of the outside world diminishes and internal awareness increases when a subject enters the hypnotic state just as it does in sleep. The difference is that in hypnosis subjects can maintain contact with the external world whereas this is virtually impossible in dreaming.

Hallucination

The increased internal awareness in both sleep and hypnosis can proceed to hallucinosis, directly and easily in the case of dreaming sleep, and with more difficulty in the deep stages of trance in hypnosis. Importantly, in both the waking and dreaming states, hallucination is seen to be a normal propensity of the brain-mind (3).

Movement and Volition

Voluntary movement is actively suppressed in both REM sleep dreams and in deep hypnotic trance. Though the mechanisms of suppression of movement are different, in both states there is a loss of the ability to voluntarily control movement.

Orientation and Memory

Recent memory suffers within the hypnotic and dreaming states, while long past memories may find their way into the brain-mind. It is thus not surprising that orientation to time, place, and person is impaired in dreaming and in hypnosis. Amnesia is also a major feature of both states: dreamers have a tough time recalling the dream, and hypnotic subjects on emerging from their trance have great difficulty recalling their experience (3).

the limbic areas including the amygdala, the hippocampal formation, and the deep subcortical frontal zones thought to mediate emotion. There is also selective activation of the anterior cingulate cortex, the visual extrastriate cortex and the pontine brain stem (13). Thus, the circuit consisting of the DLPFC and parts of the parietal cortex are selectively deactivated during REM sleep while the emotional and visual circuits are selectively activated.

Further, during REM there is an uncoupling of gamma frequency activity between the

Table 1. Parallel interplay of phenomenological factors in hypnosis and dreaming (from Reference (3))

	Hypnosis	Dreaming
Responsiveness to external stimuli	Diminished (in light trance) to anesthesia (in deep trance)	Diminished (in sleep onset dreaming) to hypoanesthesia (in REM dreams)
Hallucination	Enhanced in deep trance Propensity in waking defines hynotizability	Enhanced in sleep onset dreams to markedly enhanced in REM sleep dreams
Movement	Disfacilitated to paralyzed in deep trance	Disfacilitated to paralyzed in REM dreams
Orientation	Sometimes impaired	Poor for times, places, and person
Volition	Voluntarily suspended	Diminished (at sleep onset); Lost in REM sleep
Memory	Amnesia for state; enhanced memory for remote events	Amnesia for state; enhanced memory for remote events

NEUROPHYSIOLOGY AND NEUROCHEMISTRY CHANGES IN REM SLEEP

Dissociation between selective brain regions in REM sleep

From imaging studies we have learned that the dorsal lateral prefrontal cortex (DLPFC) and parts of the parietal cortex (the precuneus) are less active during the REM sleep stage than during waking or slow wave sleep. (13). Since the DLPFC is generally considered to be the seat of executive cognition functions we might expect it to be altered also in hypnosis. In REM sleep dreaming there is a selective activation of

frontal and perceptual regions (frontal-parietal, frontal occipital and frontal temporal) and between prefrontal-parietal and prefrontal-temporal regions. On the other hand, there is an increase in gamma frequency activity among perceptual regions themselves (parietal-occipital, parietal-temporal and occipital-temporal) (14). Synchronized firing of neurons in the cortex in the gamma frequency range has been associated with cognition and consciousness (15). These findings suggest that cognition and consciousness during REM will be altered as a result of a diminished neuronal communication between frontal and perceptual brain areas.

We will later discuss how this changed

activation pattern can produce the formal features of dreaming (3). Here we simply note that this selective activation pattern of REM sleep provides the brain with internally generated imagery and emotion while depressing the cognitive control provided by the DLPFC.

Changes in brain regional activation during hypnosis

The use of imaging and other techniques have begun to elucidate how hypnosis can change the state of the brain by selectively changing brain activity (16-19). In one study subjects were asked to imagine color and gray objects (16). Positron emission topography (PET) images of the subjects were taken. The right hemisphere registered what people were told to see independently of what they actually saw whether or not they were hypnotized. During hypnosis both the left and right hemispheres became activated. This study thus showed that the left hemisphere color area was activated when people were told to see color only when they were hypnotized. When not under hypnosis only the right hemisphere was activated during the task, that is, the right hemisphere responded to imagery per se but the left responded to imagery only under hypnosis (16). There are no lateralization differences during REM dreaming where color is readily hallucinated. This is similar to the above finding that in the hypnotic state both hemispheres are activated when color is imagined.

Neurochemistry changes during REM

Moving from neuronal activation to neurochemistry, experiments in cats and rodents revealed that the chemistry of the brain changes from a state of high aminergic and high cholinergic neuromodulation in waking to zero aminergic but still high cholinergic neuromodulation in REM (9). Thus, the cholinergic to aminergic ratio reaches extremely high values during REM. This ratio will become the highly variable M dimension in our 3-D model. In slow wave sleep aminergic and cholinergic values are intermediate between waking and REM values (9). It is known that local neural circuit operations may be changed in a dynamic way by aminergic and cholinergic neuromodulatory influences (20). While direct neurochemical probing of the human brain is not morally defensible, there are strong reasons to assume that the same neurochemical changes that have been observed in animals hold for humans. One is that nature is conservative in her use of basic mechanisms of body functions. The other is through the experimentation with drugs that are agonists and antagonists of molecules such as the cholinergic agonist carbachol, which increases REM sleep. Similar studies have been done with the aminergic agonists which produced wakefulness at the expense of sleep (for a review see Ref. 1).

Significantly less is known about neurochemistry changes during hypnosis. This is because pharmacological research has been limited to examining the agents that induce hypnotic sedation, anesthesia and analgesia such as the benzodiazepines, barbiturates and anti histamines and little research has attempted to discover how brain chemistry changes while under hypnosis (21). Further research is needed before we can make meaningful pharmacological comparisons between the hypnotic and REM states.

Non-pharmacological animal models of hypnosis are also limited since they have concentrated on procedures for the hypnotic induction of animals. These have included restraining the movement of the animal, inversion of the animal and the administration of stimuli that trigger an inhibitory reflex in motor areas of the cortex. These procedures often induce hypnotic-like behavior in the animal that actually resembles the muscle atonia of REM sleep.

Increased and decreased activation of brain regions during REM

Table 2 provides a summary of increased (↑) and decreased activation (↓) of brain regions during REM. Importantly, there is decreased neural activity between frontal and perceptual cortical brain areas during REM. This has been shown for neural activity in the gamma frequency range. It remains to be shown for other frequency bands.

Table 2. Summary of increased (↑) and decreased (↓) activation of brain regions during REM compared to waking

Brain Region	Activation
DLPFC	↓
Parietal (especially precuneus)	↓
Other frontal and prefrontal cortical brain regions	↑
Temporal cortex	↑
Occipital cortex	↑
Limbic, e.g., anterior cingulate, amygdala	↑

Functions of the different regions

The DLPFC is involved in spatial and conceptual reasoning processes. It is also involved in executive functioning that includes goal selection, the development of an action plan, initiation, monitoring outcome and the inhibition of distracting or completion of a complex task. It is involved in the monitoring and manipulation of working memory information held on-line. The ventral prefrontal cortex (VPFC), on the other hand, is intimately connected with limbic nuclei involved in emotional processing. It is involved in behavioral self-regulation. The VPFC helps define human individuality and social behavior (22). It is significant for understanding dreaming that the VPFC is activated during REM sleep but the DLPFC is not. It may be no coincidence that while our dreams are rich in social behavior, our reasoning about it is impaired.

In a recent experiment (23) it was found that reappraisal of negative emotion requires that the lateral prefrontal cortex (LPFC) and the medial prefrontal cortex (MPFC) be active. These two areas are essential for working memory, cognitive control and self-monitoring. Successful reappraisal was accompanied by an increase in LPFC and MPFC activity and by a decrease in amygdala and medial orbital frontal cortex (MOFC) activity (23). In other words, reappraisal of negative emotion involved both an increase in activity in cognitive self-monitoring circuits (LPFC and MPFC) and a decrease in activity in affect and appetitive circuits (MOFC).

An implication of this study for REM sleep dreams is that the reason that feelings in REM dreams persist and do not undergo cognitive scrutiny is because the DLPFC is functionally disconnected from the amygdala and the medial orbital frontal cortex. We dwell on these results because of their obvious relevance to hypnosis. An imaging study of hypnotized subjects found (17) increased activation in the left inferior parietal area (Broadman area (BA) 40) and the left VLPFC (BA 45) and in the right anterior cingulate cortex compared to normal alertness. Decreased activation was found in the precuneus (BA 7) and in the MPFC (BA 8). In another study (18) significant hypnosis-related increases were similarly found in the left lateral frontal cortex (BA 44, BA 45) and in the right anterior cingulate cortex. Decreased hypnosis-related activation was also similarly found in the precuneus. In summary, the similarities between the two states include the activation of the anterior cingulate and the deactivation of the precuneus; the differences include increased limbic activation in REM and no increased limbic activation in hypnosis. There is also a different pattern of prefrontal cortical activation and deactivation in the two states. These imaging data thus reflect both the similarities and the differences between the REM and hypnotic states:

Prefrontal parietal circuit

Performance of a dual task that required executive decision to carry out two tasks together activated both the DLPFC and the

precuneus and intraparietal sulcus (24). In this study participants had to perform concurrent tasks in which two stimuli were presented that caused interference. One task was a reaction time task on the pitch of an auditory stimulus, and the other a three-choice reaction task on the position of a visual stimulus. The results of this fMRI study supported the conclusion that the dorsolateral prefrontal and superior parietal cortices were involved in the coordination of concurrent and interfering task processing. Thus, executive functions are mediated by a network of brain areas including prefrontal and parietal cortices (24). The results of this study provide an explanation for the lack of executive control over the dream images that occur in REM sleep because the DLPFC and the precuneus have become dissociated. Similar lack of executive control exists in high hypnotizable subjects in deep trance (see Table 1).

Clinical studies

Clinical studies have also focussed on these questions. In one such study of 350 neurological patients Solms (25) found that changes to the DLPFC has no effect on dreaming but that lesions in or near the parieto-temporo-occipital junction of the inferior parietal region (supramarginal gyrus) caused a total cessation of dreaming. Lesions of the white matter connecting limbic areas with the frontal cortex in the ventro-mesial quadrant of the frontal lobe also caused cessation of dreaming (26). These findings are in agreement with imaging studies discussed above and reinforce the conclusion that dreaming is brain-based and that the DLPFC is not necessary for dreaming. On the other hand, the parieto-temporal-occipital junction which is part of the dorsal stream (the "where" for processing spatial information) is crucial for dreaming.

State space

We have discussed the formal properties of dreams on the one hand and the neurophysiology and neurochemistry of the REM sleep brain on the other. For the neurophysiology and neurochemistry, we then discussed what is known about the relative changes in neuromodulation and in activation that accompany a change from waking to REM sleep. Where existing evidence existed, we did the same for hypnosis. Now we frame both hypnosis and the different stages of sleep within a heuristic state space model of the brain-mind.

Concept of state

In physics a state is well defined, for example, the gaseous, liquid, and solid states. As in sleep, these states are often called phases. The transition between states is known to be dependent on temperature, pressure and volume. It is important to note that these definitions of state are strictly true only under equilibrium conditions. In physics, equilibrium exists when a system is isolated from outside influences so that any inhomogenities in temperature, for example, are quickly equilibrated. Under non-equilibrium conditions other kinds of transitions to other kinds of "states" become possible. Examples are the Benard instability which occurs when a pan of water is heated from below and heat is applied so as to increase the temperature difference between the top and the bottom. Thermal convection occurs as the fluid itself begins to move. Fluctuations then occur in the form of convection cells which become amplified as the temperature gradient increases. This continues until a critical value (instability) is reached at which point the system self-organizes into a new state which consists of a pattern of hexagonally shaped cells made up of individual water molecules (27). The point is that under non-equilibrium conditions new states become possible. Since states of the human brain-mind are never observed under equilibrium conditions, an infinite number of states is possible.

AIM Model

In biology the canonical sleep and wake states are clearly defined. Within sleep other well-defined states are the REM and NREM states. The activation, input, modulation (AIM) model (3) is a heuristic model which posits that sleep-wake states are particularly sensitive to three kinds of changes in brain function: overall activation level (A), input-output gating (I), and chemical modulation (M). The resulting AIM model captures some essential features of state space by purposefully ignoring underlying complexity such as, for example, the role of the posterior and anterior hypothalamus in influencing sleep and wake states, respectively, and the role of such neurotransmitters as hypocretin, orexin and histamine in influencing REM and NREM states. But any model is preferable to no model and our new four dimensional (time included) model is clearly better than the traditional two dimensional model derived from human sleep lab data.

First, we must define how we conceive of equilibrium conditions in the context of the AIM model. We assume that equilibrium conditions are most nearly achieved when the state space is clearly divided into REM and NREM sleep states. The rationale for this definition is that the REM and NREM and waking states robustly re-occur which we move into and out of on a consistent and periodic basis. Within the AIM model these states are dependent upon the values taken by the three variables of the model, activation, input and modulation (AIM).

Equilibrium conditions

Neuromodulation M: Equilibrium is defined when the aminegic to cholinergic ratio, M >> 1 in the wake state; M<< 1 in REM; and M is approximately =1 in NREM.

Input Output Gating I: The processing of external to internal information, I>>1 in the wake state; I <<1 in REM; and I <<1 in NREM.

Global activation A: The overall activation level A of the brain is high in the wake state; and it remains high in the REM state.

Table 3 summarizes this data.

Table 3. Equilibrium conditions

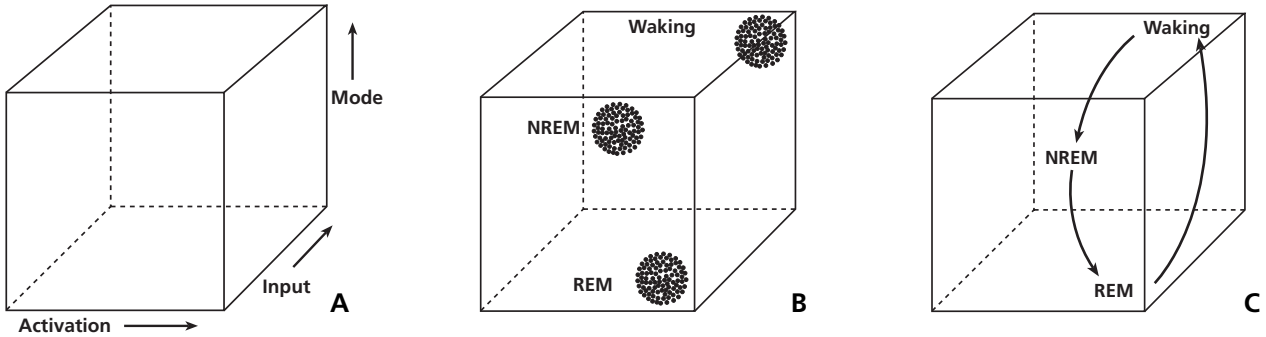
A, I, and M	Wake State	REM	NREM
Neuromodulation, M	M >> 1	M << 1	M ≅ 1
Input Output Gating, I	I >> 1	I << 1	I << 1
Global Activation, A	A high	A high	A low

Trajectories

Time is the fourth dimension of the AIM model. AIM state space (Figure 2A) is traversed in time as changes occur in the values of A, I and M. For example, as subjects progress through a sleep cycle of waking, NREM and REM (Figure 2B) successive areas in the AIM state space are traversed. A normal sleep cycle follows an elliptical trajectory as shown in Figure 2C.

Lucid Dreaming

When one or more of the conditions shown in Table 3 do not hold interesting things are possible, that is, we can go into novel "states." For example, non-equilibrium conditions can lead to lucid dreaming within the REM state. Lucid dreaming which occurs in the REM state has a low aminergic to cholinergic ratio M<< 1 (but it may be higher than in normal REM). The processing of external to internal information is low, I<< 1 (again as in normal REM, but as La Berge (28) has shown, lucid dreamers can signal out). The global activation is high as in normal REM but the DLPFC-Precuneus circuit has perhaps become partially reactivated from its non-activated state in normal REM. Lucid dreaming is not a stable state and has a tendency to go either to the REM equilibrium state of M<<1, I<<1 and A>>1, or the waking state. The lucidity domain is shown in Figure 3A.



Figures 2. The AIM model (from Reference 3). 2A. The A-axis is the global activation level of the brain. As one goes further to the right on this axis the greater is the global activation. The I-axis measures the level of external and internal inputs. Internal input is high in the plane of the paper and external input increases as one goes into the plane of the paper. The M-axis is the ratio of aminergic to cholinergic modulation. Aminergic modulation increases from bottom to top. 2B. The REM state with high A but low I and low M is shown as an area in the bottom right hand corner of state space. Additionally, the REM state is characterized by the low activation level of the dorsal lateral prefrontal cortex (DLPFC) and precuneus in the parietal lobe. The NREM state with intermediate levels of A, I and M is shown in the middle of the AIM state space. The wake state with high A, I and M is shown as an area in the upper right hand corner of AIM space. The DLPFC remains highly activated. 2C. This figure shows a normal elliptical trajectory of a sleeping cycle. The shape of the ellipse changes throughout the night since subjects descend less and less deeply into NREM and more and more deeply into the REM domains.

Sleep walking (Motor output in sleep)

Non-equilibrium conditions can also lead to sleep walking and sleep talking in the NREM state. In sleep walking and sleep talking there is an incomplete arousal from NREM sleep with a resulting dissociation. For example, in sleep walking the cortex may remain inactivated while the subcortex may become activated allowing automatic behaviors such as sleep walking. This state too is unstable and will go over to the wake or NREM state unless kept at non-equilibrium by continued dissociation between the motor and cognitive systems.

REM Sleep Behavior Disorder (REM without muscle atonia)

In REM sleep behavior disorder patients enact their dreams through movement. The motor inhibition and muscle atonia of REM sleep become weaker than the normal movement commands of REM sleep. This condition can be caused by a malfunctioning inhibitory brainstem medulla which normally provides sufficient muscle atonia to prevent enactment of movement commands occurring during dreaming. Or it can be caused by a greater than normal excitation of the REM sleep generator circuits that project to the motor cortex (9).

Hypnotic state

In the hypnotic state awareness of the outside world diminishes with the exception of the suggestions of the hypnotist. In terms of the AIM model, which cannot exactly measure this selective attention during waking, the measure of external to internal input I decreases, as it does in both the REM and NREM stages of sleep. In that sense there is a correspondence between sleep and the trance state of hypnosis in the states' reduced receptivity to external stimuli.

As the subject becomes more and more hypnotized, the activation pattern of the brain changes. In the hypnotic state we predict that the DLPFC region of the brain which serves executive and volitional functions becomes less activated. Alternatively, executive functions may remain high but are subjected to the will of he hypnotist. Thus, a subject in the hypnotic state has a reduced ability to act willfully. This is similar to what happens in the REM stage of dreaming. In terms of the AIM model the global activation A remains high, but unlike in lucid dreaming, the activation of the DLPFC remains

low, as in normal REM.

As the hypnotic state becomes established the neuromodulatory balance M shifts from the high aminergic environment of the non-hypnotic wake state to a larger cholinergic presence. This too is what happens in the REM stage of dreaming. In terms of the AIM model M decreases as the hypnotic state is reached. See Figure 3B, below.

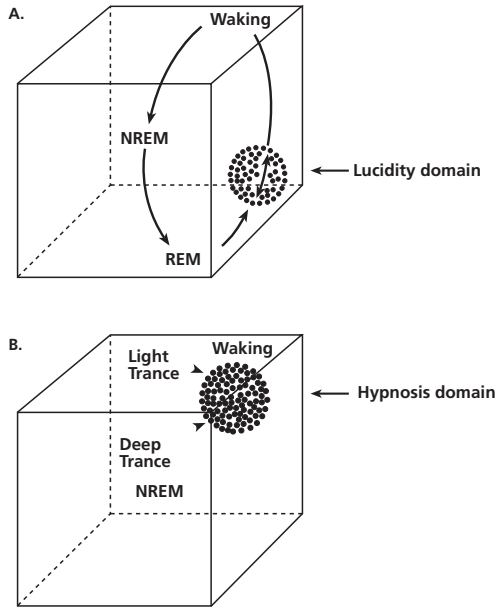


Figure 3. Lucid Dreaming and Hypnosis (from Reference 3). 3A. In lucid dreaming subjects while in REM sleep become aware that they are dreaming and thus regain some aspects of waking consciousness. The lucid dreaming domain is an unstable state that will tend to go back fully into REM or move into full waking. 3B. The hypnotic state with high global activation, low to intermediate values of I, and intermediate to high values of M is shown as the area marked hypnosis domain in the state space diagram. As for selective regional activation, the hypnotic state is believed to be have low dorsal lateral prefrontal (DLPFC) and precuneus activation similar to what exists in the REM state. There are probably other regional differences between these two states, for example, the emotional limbic circuits that include the amygdala are especially highly activated in REM. These emotional circuits may not be especially highly activated in the hypnotized state with the exception of the anterior cingulate which is highly activated in both states.

Understanding hypnosis by studying the brain basis of dreaming

In hypnosis the hypnotist tries to create a relaxed state in the subject to help prepare the subject to pay attention only to the hypnotist's instructions and not to what is going on elsewhere. When successful, and upon receiving an appropriate cue, the subject can be stuck with a pin and not feel the pain, can get up and walk and talk to a person across the room, and so on. Once the suggestions have been made by the hypnotist and have been incorporated by the subject, the subject carries these out upon receiving the requisite cues.

We have suggested that the brain basis for this hypnotic state is both similar and different from REM stage dreaming. Both states can be seen as areas in state space defined by values of brain chemistry neuromodulation, (M), the relative proportion of external and internal information processing (I) and the global activation of the brain (A) with regional activation patterns differentiated.

The REM stage of dreaming is similar to the hypnotic state in that global activation in both states is high. This high activation level allows information to be processed, as opposed to non-dreaming sleep or coma where the activation level of the brain is low and information is minimally processed. In REM as in the hypnotic state the executive function is altered so that willful action is diminished. This may be manifested in the brain by a shutting down of the dorsal lateral pre frontal cortex, so that connections between it and other brain area are short-circuited.

The REM stage of dreaming is similar to the hypnotic state in that awareness of external stimuli is diminished. However, in REM there is almost complete reliance on internal input (e.g., memories) even though these may come without temporal order and be loosely associated. In the hypnotic state there is increased awareness of internal memories but the initial external input of the hypnotist is never lost and will be retrieved when the pre-programmed cue is received externally. In the hypnotic state there is a disconnect from the outside world while awake and not while asleep. This allows verbal suggestions made by the hypnotist to be heard and processed.

The REM stage of dreaming is similar to the hypnotic state in that the neuromodulation shifts from aminergic to cholinergic. However, in REM the ratio of cholinergic to aminergic is extremely high owing to the near zero level of the aminergics. In the hypnotic state the subject is awake and hence his aminergic system is up and running. However, the subject, because profoundly relaxed, has a different aminergic to cholinergic firing ratio than in normal waking. We speculate that in the hypnotic state both the aminergic and cholinergic concentrations will go down maintaining a ratio close to unity (Figure 3B).

Caveats

It is clear that our AIM model is an already obsolete oversimplification. We know that the wake and sleep states are sensitive to selective brain region activation and not just overall activation level. For example, selective brain regions are activated during the performance of particular kinds of tasks in the wake state (29). In the REM state selective brain regions are activated while others are deactivated compared to both NREM and to the wake state. A more complete model would take account not only of the dependence of state on chemical modulation; input-output gating and overall activation level, but also the dependence of state change on changes in the activation level of specific brain regions. We acknowledge that there are two different kinds of brain activation, one in waking and one in REM. The AIM model should thus be considered as only a starting point for a conceptual understanding of state space under equilibrium and non-equilibrium conditions.

Further, we did not address the equally interesting neuronal population level of description of how changes of state occur from interacting excitatory, inhibitory and modulatory neuronal populations that self-organize into macroscopic neuronal firing patterns. This level of description has been discussed elsewhere (27,30,31,32).

CONCLUSIONS

We have theorized that the unique properties of brain-mind states, such as the wake, REM, NREM and hypnotic states, can be described by a limited set of variables. These are the overall brain activation level (A), the regional brain region activation levels and the level of communication between regions, the neuromodulatory environment (M), and the proportion of external to internal information that is processed (I). The AIM model was used to help visualize such brain–mind state space.

REFERENCES

1. *Hobson JA, Pace-Schott EF, Stickgold R. Dreaming and the brain: toward a cognitive neuroscience of conscious states. Behavioral and Brain Sciences 2000;23:793-842.*
2. *Mazur A, Pace-Schott EF, Hobson JA. The prefrontal cortex in sleep. Trends in Cognitive Sciences 2002;6:475-481.*
3. *Hobson JA. Dream Drugstore. MIT University Press, 2001.*
4. *Stickgold R, Scott L, Malia A, Maher E, Bennett D, Hobson JA. Longitudinal collection of mentation reports over wake-sleep states. Sleep 1998;21(Suppl):280.*
5. *Fosse R, Stickgold R, Hobson JA. Brain-Mind states: Reciprocal variation in thoughts and hallucinations. Psychological Science 2001;12:30-36.*
6. *Stickgold R, Scott L, Rittenhous C, Hobson JA. Sleep-induced changes in associative memory. J Cogn Neurosci 1999;11:182-193.*
7. *Walker MP, Liston C, Hobson JA, Stickgold R. Cognitive flexibility across the sleep-wake cycle: REM-sleep enhancement of anagram problem solving. Cognitive Brain Research 2002;14:317-324.*
8. *Kahn D, Hobson JA. State dependence of character perception: implausibility differences in dreaming and waking consciousness. Journal of Consciousness Studies 2003;10:57-68*
9. *Hobson JA. The Dreaming Brain. Basic Books, 1988.*
10. *Fosse MM, Fosse R, Hobson JA, Stickgold R. Dreaming and episodic memory: a functional dissociation? Journal of Cognitive Neuroscience 2003;15:1-9.*

11. *Tellegen A, Atkinson G. Openness to absorbing and self-altering experiences (absorption), a trait related to hypnotic susceptibility. J Abnorm Psychol 1974;268-277.*

12. *Wilson SC, Barber TX. The fantasy-prone personality: Implications for understanding imagery, hypnosis and parapsychological phenomena. In: Sheikh AA, ed. Imagery: Current Theory, Research and Application. New York: Wiley, 1982;340-387.*

13. *Maquet P. Functional neuroimaging of normal human sleep by positron emission tomography. J Sleep Res 2000;9:207-231.*

14. *Perez-Garci, del-Rio-Portilla, Guevara, Arce, Corsi-Cabrera, Paradoxical sleep is characterized by uncoupled gamma activity between frontal and perceptual cortical regions. Sleep 2001;24:118-126.*

15. *Desmedt JE, Tomberg C. Transient phase locking of 40 Hz electrical oscillations in prefrontal and parietal human cortex reflects the proces of conscious somatic perception. Neuroscience Letters 1994;168:126-129.*

16. *Kosslyn SM, Thompson WL, Costantini-Ferrando MF, Alpert NM, Spiegel D. Hypnotic visual illusion alters color processing in the brain. Am J Psychiatry 2000;157:1279-1284.*

17. *Maquet P, Faymonville MF, Degueldre C, Delfiore G, Franck G, Luxen A, Lamy M. Functional neuroanatomy of hypnotic state. Biol Psychiatry 1999;45:327-333.*

18. *Rainville P, Hofbauer RK, Paus T, Duncan GH, Bushnell MC, Price DD. Cerebral mechanisms of hypnotic induction and suggestion. J Cogn Neurosci 1999;11:110-125.*

19. *Szechtman H, Woody E, Bowers KS, Nahmias C. Where the imaginal appears real: a positron emission tomography study of auditory hallucinations. Proc Natl Acad Sci USA 1998;95:1956-1960.*

20. *Mountcastle VB. Perceptual Neuroscience. The cerebral cortex. Harvard University Press, 1998.*

21. *Kumar V, Mad-Y NK, Rana RD, Garg BD. Effect of isoproturon on barbiturate induced hypnosis in mice. Indian Journal of Pharmacology 1996;28:152-154.*

22. *Gaviria M, Franco J. Frontal lobes and neuropsychiatric illness. www.psych.uic.edu/education/courses/brain/garviria. 2002.*

23. *Ochsner N, Bunge SA, Gross JJ, Gabrieli JDE. Rethinking feelings: An fMRI study of the cognitive regulation of emotion. Journal of Cognitive Neuroscience 2002;14:1215-1229.*

24. *Szameitat AJ, Schubert T, Müller K, Yves von Cramon D. Localization of executive functions in dual-task performance with fMRI. Journal of Cognitive Neuroscience 2002;14:1184-1199.*

25. *Solms M. The neuropsychology of dreams: A clinico-anatomical study. Erlbaum 1997.*

26. *Solms M. Dreaming and REM sleep are controlled by different brain mechanisms. Behavioral and Brain Sciences 2000;23:843-850.*

27. *Kahn D, Hobson JA. The self-organization theory of dreaming. Dreaming 1993; 3:151-178.*

28. *La Berge SP, Nagel LE, Dement WC, Zarcone VP. Lucid dreaming verified by volitional communication during REM sleep. Perceptual and Motor Skills 1981;52:727-732.*

29. *Rypma B, Berger JS, D'esposito M. The influence of working-memory demand and subject performance on prefrontal cortical activity. Journal of Cognitive Neuroscience 2002;14:721-731.*

30. *Freeman WJ. How brains make up their minds. Weidenfeld and Nicolson, 1999.*

31. *Kahn D, Krippner S, Combs A. Dreaming as a function of chaos-like stochastic processes in the self-organizing brain. NonLinear Dynamics, Psychology, and Life Sciences 2002;6:311-321.*

32. *Kahn D, Krippner S, Combs A. Dreaming and the self-organizing brain. Journal of Consciousness Studies 2000;vol. 7 number 7 July.*