DREAMS and DREAMING

Dissociated Neurocognitive Processes in Dreaming Sleep

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The absence of the normal, i.e., waking, relationship between the overt behavioral state of the body and private, subjective experience is a fundamental characteristic of both hypnosis and dreaming sleep. Both states demonstrate a dissociation between mind/brain subprocesses that, in the waking state, are highly coordinated. Although the concept of dissociation has been central to theories of hypnosis for many years, it has not been developed in dream theory - despite the many shared features of the two states. This paper will briefly review the primary characteristics of dreaming that can be attributed to mind/brain dissociations and then examine in greater detail dissociations in the visual imagery-oculomotor system during dreaming. (Sleep and Hypnosis 1999;1:105-111)

Key words: dreaming, dissociation, eye movements

We may define a dissociation as a temporary loss of communication or interaction between the processes or states of two or more neurocognitive subsystems, relative to the their interaction in the normal waking state. For the purposes of this discussion, we may separate these dissociations into three classes: 1. sensory transduction and afferent pathways, 2. brain processes including both explicit and implicit mental processes, and 3. efferent processes, including motor behavior, which includes rapid eye movements (REMs). For the purpose of this discussion we assume that dreaming is the cognitive component of a neurocognitive process that is particularly characteristic of Stage 1 REM sleep, and is intensified in the last hours of sleep as the brain is activated by both the 90 min REM-NREM cycle but also by the rising edge of the 24 hr. diurnal rhythm that supports the waking state.

The dramatic dreams that most people remember tend to be the result of these two joint sources of activation (1). Sensory thresholds are generally elevated but also highly variable during REM sleep. When thresholds are high there is a marked dissociation between the sensory patterns that reach the sensory organs and the neurocognitive processes that take place in the association cortex. A variety of mechanisms account for this dissociation. Although eyelid closure accounts for part of the elevated threshold of visual perception, visual information is not transmitted even when the eyelids are taped open during dreaming sleep. Pompeiano (2) showed that the optic nerve transmitted little information, and Braun, Balkin, Wesensten, Carson, Varga, Baldwin, Selbie, Belenky, and Herscovitch (3), using H215O and PET to measure cerebral blood flow throughout the sleep-wake cycle, recently found that the activation of the visual projection region, the striate cortex, is actively diminished during REM sleep. In the auditory system, Pompeiano (2) also found that thresholds were increased by neural noise in the cochlea rather that high thresholds in the auditory nerve.

At the efferent end of the system, the motor cortex delivers motor commands, but spinal inhibition fortunately prevents them from being executed (4,5). The respiratory and oculomotor systems are the only skeletal muscle systems to escape this inhibition. The former is clearly essential for survival, but the function of the latter is not known. Between the afferent and efferent systems lie the brain regions that produce the cognitive and affective characteristics of the dream. With the publication of the recent study by Braun et al. (3) we have reached the point where the measured patterns of brain activation account rather well for the salient characteristics of dreaming sleep. The paradoxical concurrence of the active mental experience of dreaming and the wake-like brain activity (electroencephalograms EEG) on the one hand, coupled with a bodily state that resembles a coma, baffled sleep investigators throughout the 20 years following Aserinsky and Kleitman's discovery that dreaming sleep is associated

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Accepted February 23, 1999.

with Stage 1 REM EEG (6). Indeed, European investigators named the state paradoxical sleep. Clearly, the active dream reports suggested that some part of the brain was active enough to produce imagery and thought.

Moruzzi and Magoun (7) had previously shown that the widespread activation of the waking brain is controlled by the brain-stem reticular formation. But, it was not until McCarley and Hobson (5) showed that portions of the ascending reticular system in the brainstem provided widespread activation to portions of the cerebral cortex while another portion of the activating system inhibited the execution of motor commands generated in the cortex, that the origin of the wake-like EEG of REM sleep became clear. McCarley et al. showed that the sensory and motor responsiveness that accompany the active brain in the waking state were, in REM sleep, dissociated by an active pontine inhibitory process. But the association between an active cortex and active mental experience appeared to be largely intact - if a little odd!

If we assume that REM sleep dreaming is produced by an active brain, it is reasonable to ask what is the input from which the brain constructs this dream. Ever since Aserinsky observed REMs everyone quite naturally assumed that the eye movements were tracking dream images. But sleep neurophysiologists knew that the REMs were associated in time with a ponto-geniculate-occipital (PGO) sequence. Because these spikes were dramatic in amplitude it was reasonable to entertain the assumption that they had a significant impact on the dreaming process. One hypothesis said that the PGO spikes disrupted ongoing dream mentation to give it the bizarre quality we associate with dreaming. But the evidence does not support this position (8).

Perhaps McCarley et al. (5) implicitly assumed that for the cerebral cortex to create a dream it must receive input from some external source. They proposed the pons as that input and the PGO spikes as the information. The proposal was a daring one inasmuch as we know very little about what information is carried by PGO spikes to the occipital cortex in either the waking or sleep state. In the final part of this paper we will present a critical review of the role of PGO spikes and REMs in dreaming sleep. Before that, however, we wish to make a case for the assumption that the cerebral cortex is entirely capable of generating the imagery and thought that has the unique characteristics of our dreams - without any concurrent external input.

The theoretical basis for this assumption is provided by the neural network models described in the two volume work on parallel distributed process, edited by Rumelhart and McClelland. In the chapter, Schemata and sequential thought processes in PDP models, by Rumelhart, Smolensky, McClelland and Hinton (9), the authors demonstrate how microcognitive units that represent characteristics of small clusters of neurons interconnected by positive or negative weights that represent the synapses of neuronal networks, can nicely simulate the characteristics of the macrocognitive concepts that Bartlett (10) called schemata. These schemata possess several characteristics of dream images and thoughts that have heretofore been difficult to conceptualize - such as the ability to represent a novel image schema and to generate such a schema from conceptual units that are themselves not novel. Secondly, an entire schema may be instantiated, i.e., become active, even though only a small part of its constituent units are active. This neural network model implies that activating a few features may be sufficient to result in the creation of an entire visual image schema.

Antrobus (11) demonstrated that, with a small modification, the Rumelhart et al. model could generate a complete schema - even when the only input to the neural network was noise, i.e., random neural activity. Antrobus made the additional assumption, that waking cognitive networks are learned, not in order to construct static representations of external events, but rather to anticipate future events (12). The schema representation of each moment in waking perceptual time includes representations or expectations of what might happen in successive moments. For example, if one imagines seeing a door open, one expects to then see a person enter. These neural network representations of the expected future may be the cognitive basis upon which dream mentation is projected through time (13). Antrobus trained a neural network to learn a sequence of events as they might occur in the waking state. Then in a simulated dream state, with internal neural noise, but no external input, the network generated a few novel schemata and then proceeded to produce a sequence of events similar to those learned in the waking state. This neural network model was not intended to simulate the full richness of the human dream, but simply to demonstrate that the novel as well as coherent character of dream sequences can be carried out without any external input to the system.

With the development of increasingly accuracy techniques of brain imaging in the past 15 years, we are developing increasingly accurate models of how different characteristics of perceptual, cognitive and imaginal processes are distributed across brain regions. For example, the texture, edges and color of an image many be created in the occipital cortex. But the object's shape, orientation and location are constructed in the parietal cortex, while the name is produced in the left temporal cortex, and the larger significance of the object is produced in the prefrontal cortex. Yet, while attention to a particular property of an object may require elevated activation of one specific region, the integrated schemata of the waking state may require that each brain region constrains the pattern of activation in the other. The integrated waking experience thus requires widespread activation of the cortex even though attention to

a particular feature of an object-event may be associated with increased activation in only one region of the brain.

We assume that the distribution of perceptual-cognitive processes across brain regions is quasi-modular, as distinct from a strict modular arrangement in which each module operates independently of its neighbor. That is, we assume that there are not only rich connections among the neural units within a module, but there are also substantial connections between these units and those of neighboring modules. The interconnections among units within a module, however, are much denser than those between models. The inter-module connections allow a module to constrain the processes within the modules of its neighbors. Dream theorists have implicitly assumed a quasi-modular brain when they state that one imaginal process is enhanced while another is diminished during REM sleep. For example, it is generally assumed that, relative to waking imagery, visible features are enhanced while speech imagery - as distinguished from the meaning of speech - is reduced in REM sleep. But until the recent work of Braun et al. we have had no direct evidence to support the neurophysiological side of this assumption.

The spatial resolution of the Braun et al. (3,14) study is sufficiently accurate to delineate clear dissociations between activated and non-activated functional regions of the brain - during REM, as distinct from nonREM sleep stages and pre- and post-sleep waking control conditions. Although the extrastriate cortex is activated in REM sleep, the striate cortex, the projection region for the retina in wakefulness is, contrary to all expectations, relatively inactive! Since both the optic nerve and the striate cortex are inhibited in REM sleep, it is clear that the images of REM sleep dreaming cannot originate in these structures.

Therefore, the extrastriate cortex, which creates the more complex structures of waking visual perception, appears to be the region in which the most elementary visual features of dream imagery are initiated. And they must be created on the basis of the previously learned internal connections among its constituent neural units. Within the extrastriate cortex, for example, visual features such as color, edge and shape, are constrained by the information about what features and shapes are legitimate, i.e. previously learned. We start, then, with the working assumption that each perceptual and cognitive neural network module interprets its own internal noise together with whatever input or qualifications are imposed by its connections with neighboring modules - just as it does in the waking state. We then qualify this assumption with empirical evidence from dream reports and from neurocognitive data. We have long noted, for example, that dreamers report what their imagined characters are talking about but rarely report actual speech. Objects and people are often named only as the dreamer moves into wakefulness. The finding that the left temporal cortex - the language module - is inactive during REM sleep (3,14) accounts for this characteristic of REM dreaming. Note, that as the dreamer sleeps late into the morning hours, that more broad activation of cortical regions appears to support more language features in the dream (11).

Braun et al. (14) found that the activated extrastriate system in REM sleep is also associated with activation of the limbic-related projection areas and parahippocampal cortices that support emotional and short-term memory processes respectively. This pattern of activated areas supports affective responses to visual images and saves them in memory.

This extrastriate-limbic system is dissociated in REM sleep not only from the striate cortex, but also from the frontal heteromodal association regions of the brain that normally interpret the context and significance of visual objects and persons (3,14). Braun et al. suggest that because the activation of these regions may be necessary to critically evaluate the creations of the extrastriate-limbic modules, the lowered activation of these heteromodal regions may account for the dreamer's uncritical acceptance of bizarre dream events.

We assume that there are additional patterns of dissociation that the imaging procedures of Braun et al. (3,14) could not identify. Bizarre reports of knowing the identity of a dreamed person, while the visual features clearly do not fit [it was my brother but he was a girl], are quite rare in laboratory dream samples. Antrobus, et al., (11) have found them to increase in frequency as people have REM sleep past their normal waking time. The Braun et al. study was carried out on the initial REM period, and averaged over all sleepers so it was unlikely to identify rare dissociations.

It seems clear that brain-imaging technology has outstripped the ability to measure critical, but ephemeral, characteristics of dreaming. In particular, the traditional unstructured verbal report does not provide enough detailed information to evaluate some of the hypotheses about dream-image construction suggested by the Braun et al. (3,14) study. The dream report appears to be, in part, a product of the dreamer's attempt to make sense out of, that is, to complete the association of, a sequence of relatively dissociated imaginal events. The words themselves imply name and meaning associations that are not present in the dream itself. Reports of a dreamer's location (e.g., We were in my room.) are typically assumed to indicate that the visual features of the room were imaged, whereas they could simply be understood by the dreamer in a cortical region outside of the visual cortex. This class of dissociation suggests that the extrastriate cortex creates the experience of spatial location - even in the absence of imagined visual features. Analysis of classes of dissociation such as this may help to identify dissociations among cortical regions that have not yet been identified by PET scan procedures. But

such analyses will require substantial improvement in the measurement of dream imagery. Antrobus et al. (11) have employed color photographs scaled for brightness and clarity to measure the visual attributes of reported dream images. Future investigators must attempt to develop similar procedures for measuring spatial attributes.

The study by Braun et al. (3,14) provides a compelling explanation for most of the characteristics of dream mentation: selected brain regions are active while neighboring regions that would normally both constrain and supplement the schemata in those regions, are inactive. The study is without question the most informative neurophysiological study of dreaming sleep since the original Aserinsky and Kleitman (6) discovery of REM sleep. Despite the high cost of this research and the difficulty of maintaining extended sleep in PET scan apparatus, it is hoped that they will continue their work and extend it from the first to later REM periods-which we assume are characterized by a broader pattern of cortical activation and correspondingly richer dream schemata sequences (11).

In passing, it should be apparent to students of hypnosis that some of the dissociation processes of REM sleep are shared by certain hypnotic patterns. For a further description of dissociations that appear to distinguish sleep from waking, that is, dissociations that are common to both REM and nonREM sleep, the reader is referred to Braun et al. (3). We return now to the role of REMs in REM sleep dreaming and the PGO spikes with which they are closely associated in time. It is quite clear from the work of Braun et al., from our neural network simulation of how pattern sequences can be created without external input, and from the empirical evidence that dreaming occurs in the absence of concurrent REMs, that neither REMs nor PGO activity are necessary to dreaming. Because Hobson continues to support the original McCarley et al. (5) hypothesis that dreaming is the synthesis of the information that PGO waves provide the occipital cortex about the direction of REMs, we feel that the advance of dream theory will be adumbrated if we show how the data not only fail to support, but actually contradict, the synthesis side of the theory (See also, 8,15,16). First, we will review some of the functions that have been attributed to REMs-together with their supporting evidence. Aserinsky and Kleitman's (6) initial assumption, upon observing the REMs of Stage 1 EEG sleep, was that the dreamer was engaged in some form of scanning the dream images. This scanning model prompted Roffwarg, Dement, Muzio, and Fisher (17) to see if the actual right-to-left direction of the EMs (measured by electro-oculograms, EOG) is correlated with judges' prediction of EM direction, as inferred from the dreamer's recall of imagined eye and head movement orientation with respect to visual images. Judges then compared the EOG pattern with the predicted EM direction on a report by report basis and rated how well the two patterns agreed. It was not a blind rating, unfortunately! Therefore, although the authors reported a strong association between predicted and actual EM patterns, no conclusions could be drawn from the basic analysis. A secondary finding, that the rated agreement between EOG and predicted EMs improved with judges' confidence in their predictions of EM direction, did however, provide indirect support for the model.

If the dream image is scanned, where is the image? The original hypotheses were that the image originated from random neural activity in the striate cortex, or possibly on the retina itself. To test the latter assumption, Rechtschaffen and Foulkes (18) taped open the eyes of a sleeper and, when the sleeper entered REM sleep, they presented a number of illuminated visual objects. Awakening the dreamers several seconds later, they found no evidence of object recognition. The conclusions, subsequently confirmed by Pompeiano (2), established that no visual information is transmitted from the retina to the cortex during REM sleep. By default, it was assumed that dream images must originate in the brain, most likely the striate cortex. But Rechtschaffen (19), in a review of EM-scanning research up to 1973, concluded that there was only modest empirical support for the scanning model.

Despite the limited support from this strong test of the scanning model, the model was broadly supported by a weaker test. Dement in 1964 compiled evidence from a number of studies that showed a significant association between EM density and some measures of dream intensity such as rated visual imagery. Furthermore, most investigators were of the opinion that individuals are not sufficiently aware of their EMs in either waking or sleeping states to produce data that can provide a fair strong test of the dream-EM scanning model. For these two reasons, they were reluctant to discard the model until a better test could be provided.

Then, in 1977, the status of the scanning hypothesis took on new significance with the publication of the PGO-REM synthesis proposal of McCarley and Hobson (2). They proposed that when the cortex is activated by the PGO waves it creates a story - the dream - consistent with the associated EM pattern. In claiming that the direction of EMs was determined in the pons, they explicitly ruled out cortical, and therefore, any higher cognitive process in determination of EM direction. Perhaps, spurred by this challenge, Herman et al., (20) attempted to replicate the Roffwarg et al. (17) 1962 study - but this time they used the appropriate blind procedure for analyzing their data. The results supported a more modest version of the scanning model, and again showed that the degree of EOG matching correlated positively with judges' confidence in their predictions of EM direction. Herman et al. (21) and Herman (22) concluded that some, but by no means all, REMs are associated with the direction of the dreamer's apparent looking response.

Although the weak test - that REMs were concurrent with dreaming had been presented as support for the EM-scanning model, it was also valid as a weak test of the McCarley et al. (5) EM-synthesis hypothesis. Both models assume that visual imagery is concurrent with REMs. A strong test of the synthesis hypothesis requires that dreaming is confined to the vicinity of PGO-REM activity, the phasic portion of REM sleep. An extensive review of this literature by Pivik (16) shows that the model clearly fails this test. Dreaming is sustained throughout the REM period regardless of PGO or REM activity. Further, in the active PGO interval in NREM sleep just prior to the onset of a REM interval there is no associated increment in dreaming.

Even if the synthesis hypothesis were modified so that PGO activity elicited only the visual features of dreaming, the Pivik review finds that visual imagery does not differentiate between 'tonic' and 'phasic' reports. although 'phasic' reports are associated with greater incidents of both kinds of imagery (16, p 236). The recent study of Hong, Potkin, Antrobus, Dow, Callaghan, and Gillin (23,24) suggests that improved experimental procedures and scaling of visual imagery may yet demonstrate a strong association in time between visual imagery and PGO activity. Using multiple awakenings with unfortunately only one subject, they found that visual imagery was prominent in the one or two min. interval after an eye movement burst following which the less visual part of the dream continued. In conclusion, while the McCarley et al. (5) hypothesis that the dream is created in response to PGO input to the cortex is clearly without support, a modified synthesis hypothesis and the scanning model both receive weak support from the modest association in time of phasic events and visual imagery.

We turn next to neurophysiological evidence that bears on the issue, including some that McCarley and Hobson have cited as support for the synthesis process. It is well established that, in REM sleep, REM bursts occur together with PGO spike bursts (25) that spread to many areas of the brain including the oculomotor nuclei (26). Subcortical regions may also become activated in response to the PGO waves.

Burst cells which generate PGO waves have been located in the brachium conjunctivum (5) as well as pontine giant cells that project to the oculomotor and vestibular neurons (2). The brachium conjunctivum and dorsal ponto-mesencephalic junction have been localized through lesion studies as the probable neuronal output generators for PGO waves (28-30). The brachium conjunctivum, also called the superior cerebellar peduncle, is located dorsolaterally to the locus coeruleus and connects the pons to the cerebellum. One possible function of these neurons is to carry fine tuning of motor commands, issued by cortical centers, from the cerebellum to the brainstem.

In the waking state, PGO spikes occur *after* the EM is complete - which also implies after head or body movement are complete. And neither PGO spikes, lateral geniculate or occipital cortex activity are lateralized with respect to EM direction. In REM sleep, by contrast, PGO spikes, and lateral geniculate body and visual cortex single unit activity occur ipsilateral to EM direction. Commenting on the relatively large amplitude of PGO spikes in REM sleep, the authors speculate that the increase may be due to disinhibition resulting from the arrest of firing of diffusely projecting aminergic inhibitory neurons of the dorsal raphe and locus coeruleus (30).

In contrast to the Monaco et al. study, McCarley, et al., (28) found that REM direction was contralateral to occipital activity as measured by scalp electrodes. Possibly the scalp electrodes recorded dipole projections from the contralateral hemispheres. They also showed that the onset of occipital activation preceded REM onset by 8 ms. and the occipital peak occurred 4 ms. after REM onset.

The McCarley and Hobson assumption that PGO spike provide EM information to the occipital cortex must be regarded with skepticism. Since no one claims to know exactly what the PGO waves tell the brain in the waking state, a strong claim cannot be made about the function of PGO in sleep. Since PGO spikes do not send lateralized EM information to the brain during the waking state it seems unlikely that the brain in REM sleep could interpret lateralized occipital activity as information about EM direction. The same objection applies to the difference in the waking and REM sleep timing of EMs and PGO spikes. Waking PGOs follow EMs by about 40 mss. (30) whereas REM PGOs are concurrent with EMs. If PGO spikes tell the waking brain that head and EMs are complete, then it is difficult to imagine that the same brain in REM sleep would interpret the same spike activity as evidence of concurrent EM motion and direction!

Braun et al. (3) pointed out that the ventral branch of the ascending reticular activating system is part of a widespread cholinergic system that is centered, and may indeed originate, in the pontine structure that initiates PGO spikes. The high cholinergic activation of these pontine neurons and/or their disinhibition as a result of reduced activity of the aminergic inhibitory neurons of the dorsal raphe and locus coeruleus (25,30) appear to cause the pontine spikes to fire prematurely, i.e., before EMs terminate. At best, then, any information that they provide the occipital cortex is misinformation - the eyes are still moving.

In the waking state, this misinformation would be disastrous because the striate cortex would be receiving a smeared moving image. The waking PGO spike may, indeed, tell the cortex that the time is ripe for producing a visual percept. But, of course, the visual cortex in REM sleep receives nothing from the retina, so no harm is done. What the PGO may provide the extrastriate cortex is a surge in widespread activation that enables the occipital and parietal

regions to construct a vivid and coherent image. What role might the EMs themselves play in this scenario? Certainly the eyes cannot scan the image because it does not exist in a two-dimensional space on the retina or the cortex. But the dreamer might attempt to sharpen an image or to look at a different location on the current image. In the waking state, this intention would produce an EM that places the intended image on the fovea and relocates it in the parietal cortex (31). The parietal cortex actually starts to reconstruct the extrafoveal image in the center region before the EM is complete. We assume, as mentioned earlier, that the function of the waking PGO spike is to signal the completion of the EM. This reconstruction is one of the steps of scanning a waking scene, but unlike a waking scan, it cannot foveate the image so it cannot improve the accuracy of the dream image unless, perhaps, the center parietal region provides a more dense neural working space for image construction.

To the extent that the EM fails to elicit a sharper foveal image in REM sleep, the visual system may interpret the EM as a failure. Antrobus has speculated elsewhere (in press) that the failure to obtain the anticipated visual feedback may cause the frontal eye fields to reissue the oculomotor command - repeatedly, thereby producing the pattern of EM bursts that are so common in REM sleep.

Because EMs are an integral part of waking visual perception and are the most dramatic observable characteristic of dreaming sleep, they have been assigned many roles in the production of dream imagery. Careful examination of research evidence, however, fails to support any of the hypothesized functions of EMs in the dreaming process. The visual imagery of dreaming is the production of an extrastriate - parietal cortex activated by a cholinergic system controlled by pontine structures. There is no evidence that REMs modify this image construction process in any way. They may indicate the time at which some cortical module attempts to modify the image, but there is no reliable evidence to support this assumption. The PGO waves may provide widespread activation to the cortical, image-constructing regions, but there is no evidence that they provide EM or other specific information that can be synthesized into the dream.

In conclusion, we have elaborated on the position described by Braun et al. that dreaming is the production of modular brain whose image-producing modules are flexibly integrated in the waking state as a function of the intentions and environmental demands of the individual, but are selectively activated during REM sleep as a function of pathways determined in the pons. This pattern of cortical activation, together with the dissociation from regions with which the active modules normally interact appears to account for the characteristics that distinguish dream from waking mentation. No pontine information to the cortex is necessary to account for dream imagery and thought.

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