Rapid Eye Movement Sleep, Non-rapid Eye Movement Sleep, Dreams, and Hallucinations

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After the discovery of rapid eye movement (REM) sleep in 1953, oneiric activity was long thought to be associated uniquely with REM sleep. Subsequent evaluation of sleep in humans combining neurophysiologic, psychophysiologic, and, more recently, functional neuroimaging investigations, has instead shown that dreaming also occurs during non-REM (NREM) sleep. It has been documented that hallucinatory activity during sleep is a normal phenomenon that is not constant throughout the night but increases toward morning when it tends to become present to the same extent in REM and NREM sleep. The role of sleep mechanisms in the generation of visual hallucinations is well-recognized in narcolepsy in the case of hypnagogic hallucinations, which are thought to derive from a REM-dissociation state in which dream imagery intrudes into wakefulness. Similar mechanisms have been hypothesized to play a role in the physiopathogenesis of visual hallucinations in various neuropsychiatric disorders. Furthermore, a growing body of evidence indicates that not only REM but also NREM processes, such as arousal-related processes, may play a role in the physiopathogenesis of hallucinations in the aforementioned disorders. The role of these processes has been most extensively documented in visual hallucinations occurring in the context of delirium tremens and Parkinson's disease.

Introduction

The discovery of REM sleep [1,2] is justifiably regarded as a milestone in the evolution of neuroscience. It stimulated study of mental activity occurring during sleep and prompted neurologists and psychiatrists to include sleep evaluation in their investigations (clinical and research studies) of neuropsychiatric disorders. Just over 50 years ago, Aserinsky and Kleitman [3] thought, on the strength of reports of dreaming in subjects who had polysomnography (PSG) and who were experimentally awakened from REM sleep, that oneiric activity was unique to REM sleep. Therefore, dream activity was considered purely within the restricted neurophysiologic framework of this sleep stage. The authors continued to hold this opinion, and in later studies interpreted occasional reports of dreaming in normal subjects on awakening from non-REM (NREM) sleep as residual phenomena from a previous period of REM sleep [4].

Additional evaluation of sleep in humans, combining neurophysiologic and psychophysiologic approaches, showed that dream activity is not unique to REM sleep given that dream recall is possible during night, at sleep onset, and on awakening from NREM sleep preceding the first REM sleep [5,6], and during the day on awakening from a NREM nap [7]. However, qualitative differences between dream activity in REM and NREM sleep have been reported. Mental activity during NREM sleep has been reported to be (albeit not exclusively) thought-like compared with mental activity occurring during REM sleep, the latter instead being characterized by visuohallucinatory and bizarre content [5,8].

Reports of dreams occurring during NREM and REM sleep have challenged the notion that dreaming is uniquely associated with the neural mechanisms of REM sleep, namely the REM ponto-geniculo-occipital–like (PGO) processes in the brain stem. Furthermore, the disappearance of dreaming (but not of electrophysiologically identifiable REM sleep) in subjects with brain lesions sparing the brain stem indicated that the generation of dream activity is not uniquely linked to brain stem mechanisms [9,10].

The issue was additionally clouded by the emergence of several differences between the various methodological approaches used in the evaluation of mental activity during sleep [11].

It has been hypothesized that the dream activity in REM and in NREM sleep derives from two different generators, and here I refer to the so-called two-generator model, which also would account for the qualitative differences between dreaming in REM and in NREM sleep. Recently, a study based on electroencephalographic (EEG)

frequency spectral analysis in healthy volunteers [12•] indicated, through analysis of the relationship between dream occurrence and EEG frequencies, that the neural networks underlying dreaming during REM and NREM sleep are different; those underlying REM sleep are linked to the neural REM mechanisms and those underlying NREM sleep are linked to the intra-NREM sleep arousal processes. Dreaming during NREM sleep was hypothesized to be an effect of the recall of perceptual experiences previously incorporated into the memory during micro-arousals from NREM sleep—events of which the subject may be entirely unaware.

However, the question still is debated. According to the one-generator model, the generator of dream activity during REM and NREM is the same, and differences between reported dream activity in REM and NREM sleep can be explained by quantitative and qualitative differences between the memory processes occurring during the two different kinds of sleep. Additional hypotheses have been advanced, such as Nielsen's [13] "covert REM sleep hypothesis," according to which dream mentation during NREM sleep is attributable to the persistence, during this sleep stage, of some (non-electrophysiologically identifiable) processes of REM sleep. The suggestion is that their emergence leads the subject to report dream-like experiences on awakening from NREM sleep.

It has been documented that sleep-related hallucinatory activity is not constant throughout the night but increases toward morning when it tends to become present to the same extent in REM and NREM sleep [14]. It has been hypothesized that the increase in hallucinatory activity during REM sleep in the latter part of the night is triggered by increased PGO-like process intensity attributable to increased activation of the cholinergic pontine brain stem [15]. However, decreased intensity of synchronization processes, as reflected in decreased power of "slow wave activity" and decreased frequency of posterior cortical spindle frequency during late-night NREM sleep [16], would account for increased hallucinatory activity during NREM sleep in the latter part of the night.

The combined use of functional neuroimaging and/or neuropsychologic or neurophysiologic investigation is leading to a better, more in-depth understanding of the mechanisms underlying perceptual phenomena during REM sleep [17].

By means of positron emission tomography and functional magnetic resonance imaging studies, regions of the brain involved in perception and attention during the awake state and which became deactivated during NREM sleep, have been shown to become reactivated but aminergically unmodulated during REM sleep, except for the dorsolateral prefrontal cortex, which remains deactivated as in NREM sleep. It has been hypothesized that hallucinations during REM sleep derive from the integrated function of limbic and paralimbic areas, brain stem PGO system, and unimodal association areas in the visual and auditory cortex [18].

The available neuroimaging findings reinforce one of the most comprehensive hypotheses about dreaming, hallucinatory activity, thinking, and memory during sleep: the "activation-synthesis model of dreaming" advanced by Hobson in 1977. It was later reformulated in a broader framework as the AIM model (Activation, Input source, Modulation), details of which are given in a specific review [19].

The existence of hallucinatory activity during sleep has long stimulated the interest of philosophers, neurologists, and psychiatrists. The occurrence of a sleep-related mental experience characterized by visual hallucinations and bizarre content in some ways reminiscent of some pathologic episodes during sleep (oneiricisms) and some psychiatric, particularly psychotic, disorders. This might have led philosophers such as Kant and Schopenauer to write "the madman is a waking dreamer" and "dreams are a brief madness and madness a long dream" [20] and might have led psychiatrists think that investigating physiologic and pathologic mental experiences occurring during sleep (dreams and altered dreams) would lead to a better understanding of the physiopathology of psychiatric disorders [20]. Recent clinical and instrumental data have reinforced this belief and encouraged this approach for clinical and for research purposes.

Accordingly, combined neuroimaging and/or neuropsychologic evaluation of dreams has documented the presence of several bizarre features similar to those encountered in some neuropsychologic syndromes characterized by delusional misidentifications of faces and places, and led to the suggestion of a common anatomic-functional background [21••].

A revised and expanded version of the Iowa Sleep Experiences Survey Scale was used to investigate various sleep- and dream-related experiences. Preliminary findings in a sample of college students suggested that sleep experiences may be related to schizotype personality traits independent of the potential underlying influence of other major personality traits such as neuroticism and extraversion [22]. Individuals who tended to experience vivid and unusual psychotic-like feelings during the daytime seemed more likely to have frequent sleep- and dream-related experiences-at night, such as hypnagogic hallucinations, nightmares, and vivid dreams. On the basis of these data, it has been hypothesized that schizotype and sleep-related phenomena are interrelated and reflect a kind of unusual cognitive and perceptual state that has been termed "transliminalitis" [23], "the tendency to pass readily from reality-based mentation to a more fantasy-based mentation."

It has been suggested that the vigilance state will likely play a role in this state and its related experiences. In one study, psychotic experiences were reportedly triggered

by brief intrusions of stage 1 NREM sleep into waking consciousness [24].

Physiopathogenesis of Visual Hallucinations: The Role of Sleep-Wake Mechanisms

Hallucinations during sleep-wake transitions

Hallucinations strictly linked to sleep onset are defined as hypnagogic, whereas those strictly linked to awakening from sleep are defined as hypnopompic. From a physiopathogenetic point of view, hypnagogic hallucinations are thought to constitute a REM-dissociation state in which dream imagery breaks into wakefulness. These hallucinations may involve many perceptual modalities including the visual one. Their visual manifestations range from simple (sometimes vividly colored) spots of light to complex images. Insight is preserved and the emotional reaction generally is pleasant, although at times the content of the hallucination can be terrifying, threatening, or may alter the subject's behavior [25].

Hypnagogic hallucinations are one of the hallmarks of narcolepsy [25] and can occasionally lead a misdiagnosis of psychosis, especially when hallucinations are terrifying and behavior altering [26]. Furthermore, they also can occur in a kind of paraphysiologic condition, called parahypnagogia, which is characterized by other sleep-related experiences such as vivid waking dreams and intrusive thoughts [27].

Outside the context of these conditions, hypnagogic and hypnopompic hallucinations frequently are encountered in the general population. They were reported in approximately 33% of normal people in a questionnaire-based survey of 13,057 adult subjects in three European countries [28]. In more than 50% of cases, sleep-related hallucinations were not found to be related to a neuropsychiatric pathology. However, hallucinations of this kind have been found to be more prevalent in subjects affected by daytime hallucinations than in subjects who are not.

Furthermore, hypnagogic and/or hypnopompic visual hallucinations have recently been reported to account for the hallucinatory predisposition (measured by a modified version of the Launay-Slade Hallucinations Scale) in general population, thereby providing evidence that they contribute to the multidimensionality of hallucinatory predisposition [29•].

In conclusion, hypnagogic and/or hypnopompic hallucinations, when not part of a narcoleptic picture, are a physiologic phenomenon. However, evidence of their role in determining the hallucinatory predisposition encountered in the general population, together with their high prevalence in subjects having daytime hallucinations, suggests that sleep mechanisms, namely REM dissociation that underlies hypnagogic hallucinations, is likely to play a role also in hallucinations that do not occur in strict relation with sleep.

Visual hallucinations in neuropsychiatric disorders

Data on the prevalence of hallucinations in the general population are scanty. According to the aforementioned questionnaire-based survey of a huge sample of the general population in three European countries [28], hallucinations occur as regular phenomena (more than once per week) in 2% of adults. Mental disorders (particularly psychoses), organic diseases, and use of psychoactive substances (such as drugs, benzodiazepines and antidepressants) were reported to account for the occurrence of hallucinations. Auditory and visual hallucinations were found to prevail in psychotic patients.

In psychotic individuals, hallucinations are perceived as real and thereby influence thoughts and behavior. Although hallucinations occur frequently in psychoses, the exact prevalence of the phenomenon has not been defined.

In schizophrenia, hallucinations mainly are auditory, and tend to occur during the day and in conditions of hyperarousal. Of the nonschizophrenic psychoses (schizoaffective disorder, delusional disorder, brief psychotic disorder, shared psychotic disorder [folie à deux], psychotic disorder attributable to a general medical condition, substance-induced psychotic disorder; and psychotic disorder not otherwise specified), hallucinations are most prevalent in psychotic disorders attributable to a general medical condition and in substance-induced psychotic disorders, which also exist in variant "with hallucinations" forms. In these psychoses, hallucinations mainly are visual [30].

Rapid eye movement sleep mechanisms have been hypothesized to play a role in the physiopathogenesis of visual hallucinations in some neuropsychiatric disorders. A particular role of sleep mechanisms in the physiopathogenesis of visual hallucinations has been documented in delirium tremens. In 1881, Lasegue [31] hypothesized on clinical grounds that dream activity and hallucinatory phenomena were closely linked in delirium tremens. Modern studies done using EEG have had conflicting results [32,33]. In 2002, Plazzi *et al*. [34•] provided definitive video-PSG evidence that visual hallucinations in delirium tremens can occur in the context of REM dissociation with dream imagery intruding into wakefulness. Patients with this disorder responded well to low doses of clonazepam.

The role of disordered sleep in the physiopathogenesis of visual hallucinations in Parkinson's disease (PD) has been suggested. A few studies, mainly clinical, cross-sectional investigations, indicate that disordered REM sleep and an altered level of daytime vigilance may play a role in the physiopathogenesis of some visual hallucinations in PD [35]. This would encourage the use of drugs improving sleep, particularly REM sleep abnormalities, in approaching the treatment of these patients [35].

The presence of REM behavior disorder in a PD patient series has been reported to be significantly related to the development of visual hallucinations [36]. However, it was

recently reported that sleep disorders are not predictive of subsequent occurrence of hallucinations in patients with PD in a long-term, prospective follow-up study [37].

Visual hallucinations have been documented to occur in relation to REM sleep in nocturnal PSG recordings and during sleep-onset REM periods at Multiple Sleep Latency Test during the daytime in nondemented patients with PD. On the basis of these data the authors hypothesized that REM dream imagery may underlie visual hallucinations in nondemented individuals with PD [38••].

In a clinical and 24-hour PSG study in PD patients, we documented that visual hallucinations were related to nighttime REM sleep and, during daytime, to rapid shifts from wake to sleep and vice versa, in approximately 30% of the hallucinations we recorded in 20 patients with PD [39].

Rapid eye movement dissociation it is not the only sleep mechanism that may underlie visual hallucinations. In 1999, Manford and Andermann [40] suggested that sleep mechanisms other than REM dissociation, namely those underlying arousal during NREM sleep, could play an important role. The authors interpreted the physiopathogenesis of visual hallucinations in different neurologic, toxic, and psychiatric disorders (Charles Bonnet syndrome, alcohol withdrawal, barbiturate and benzodiazepine withdrawal, PD, and psychoses). These data have been reinforced by neurophysiologic (EEG spectral analysis-based) investigations indicating that physiologic hallucinatory activity during NREM sleep in healthy people is related to arousal processes [12•].

The networks through which visual inputs pass from the retina to the visual cerebral cortex are, in part, the same ones involved in sleep-wake control. Vigilance may act on one of the mechanisms possibly underlying visual hallucinations. An example is the release from inhibition of the visual cortex. In particular, the arousal and/or de-arousal mechanisms (neural processes of desynchronization and/or synchronization) would facilitate this inhibition release process. The dorsolateral geniculate nucleus and lateral pulvinar are known to play a fundamental role in the processing of visual inputs because they are an intermediate station that receives inputs from the retina and brain stem and sends inputs to the visual cortical areas. Reticular formation stimulation at level of dorsal lateral geniculate nucleus cells has (via a complex interaction between cholinergic and serotoninergic inputs) important electrophysiologic effects: a release from inhibition and release from the control of retinal afferents. This explains why, as stated by Munford and Andermann in their article, a lesion of the raphe nuclei may alter the fidelity of reticulogeniculate-cortex transmission of visual inputs creating ideal background for the generation of hallucinations.

Therefore, the level of arousal may be particularly important in determining the occurrence of hallucinations.

The previous hypothesis also is supported by data concerning vigilance and the occurrence of daytime visual hallucinations in patients with PD [39].

Conclusions

Visual hallucinations are dysperceptive phenomena encountered in various conditions. According to classic neurophysiologic interpretations, they can derive from irritative cortical (epileptic) processes, from defects in visual input (visual hallucinations in the blind), or from defective cortical processes of otherwise normal visual inputs.

However, it is known that sleep mechanisms underlie hallucinations occurring at sleep onset (hypnagogic hallucinations) in narcolepsy and occur as sporadic phenomena in healthy people.

Several lines of experimental and clinical research indicate that mechanisms underlying vigilance and the sleep-wake cycle also are likely to play a role in the physiopathogenesis of visual hallucinations associated with pathologic conditions of a different etiology, several of which are neurologic and psychiatric. REM dissociation with dream imagery intruding into wakefulness is one of the most well recognized sleep mechanisms involved in the physiopathogenesis of visual hallucinations. However, the arousal processes occurring during NREM sleep and wakefulness also have been indicated as potential modulators of visual hallucinations in various neurologic and psychiatric conditions.

Additional clinical and PSG monitoring of psychiatric patients with visual hallucinations is needed to further clarify the complex inter-relationship among arousal, NREM, REM sleep, and the occurrence of dysperceptive phenomena. Greater use should be made of portable PSG systems that make it possible to monitor a subject's nocturnal sleep or entire nyctohemeral period in his or her own environment. The preliminary findings available in the literature are already enough to alert clinicians to the importance of doing sleep evaluation in psychiatric patients.

Sleepiness, sleep fragmentation, and sleep alterations, particularly disordered REM sleep, should be carefully considered. Pharmacologic and nonpharmacologic (sleep hygiene) strategies to improve sleep dysfunctions should be considered when approaching psychiatric patients with dysperceptive phenomena.

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