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Dreaming and hallucinations – Continuity or discontinuity? Perspectives from dementia with Lewy bodies ☆

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ABSTRACT

Comparing the phenomenology, neurochemical pathology, and psychopharmacology of hallucinations and dreaming is limited by the available data. Evidence to date reveals no simple correspondence between the two states. Differences in the phenomenology of visual hallucinations and the visual component of dreams may reflect variations in visual context acting on the same underlying mechanism – the minimal visual input during dreaming contrasts with the more substantial perceived context in hallucinations. Variations in cholinergic, dopaminergic and serotonergic neurotransmitter function during sleep and during hallucinations in Lewy body dementias, together with relevant drug effects suggest that, on the whole, different, potentially opposite, changes characterise the two states. A similar analysis of other psychotic features in Lewy body dementia and other disorders suggests that, in contrast to hallucinations, there may be more convincing parallels between dreaming and delusional states.

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1. Introduction

Psychosis and dreaming have long been considered to be related phenomena. Both include bizarre non-veridical perceptions, distorted reasoning, and changes in alertness and arousal.

More recently, the discovery of the co-existence of disturbed dreaming (REM sleep behavioural disorder, RBD) and psychotic symptoms (particularly hallucinations) in up to a third of patients with dementia with Lewy bodies (Boeve, Silber, & Ferman, 2004), together with the identification of RBD as an early, sometimes prodromal, symptom in Parkinson's disease with lesions in sleep related nuclei (for example the locus subceruleus and amygdala e.g. Iranzo, Santamaria, & Tolosa, 2009), has lead to a renewed interest in the relationship between dreaming and psychosis. These findings have lead to the hypothesis that hallucinations, at least in these Lewy body disorders, reflect the intrusion of dreaming into the awake state (Arnulf et al., 2000; De Cock, Vidailhet, & Arnulf, 2008; Sinforiani et al., 2008).

Since psychosis and dreaming are complex, variable and multifaceted phenomena, aspects of both are highly likely to overlap. Hence comparing each, *in toto*, is not likely to be productive. We will therefore focus mainly on the relationships between just one aspect of both – visual hallucinations and the visual components of dreams – in the disorders which we are most familiar with: the dementias associated with Lewy bodies (Collerton, Perry, & McKeith, 2005; McKeith et al., 2005; Perry et al., 1991).

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2. How similar are the visual components of dreams and hallucinations?

There have been no direct comparisons between the visual phenomenology of dreams and hallucinations. However, despite their superficial similarity, there is indirect evidence of systematic differences between the visual component of dreams and the visual hallucinations of Lewy body disorders (for reviews, see Collerton et al., 2005; ffytche, 2010). Dreams are mutable, hallucinations are stereotyped. Dreams are multi sensory; hallucinations are in a single modality. Dreams are "full screen", while hallucinations in dementia with Lewy bodies are set against an existing background as one part of a perceived scene. To illustrate, one hallucination reported by a patient with dementia with Lewy bodies was *"Every night I would see a man and a young child standing in the corner of the room staring at me... it was really queer. They would move but not come any closer to me and didn't say anything...they both had on old fashioned clothing, like Victorian style with cloaks on."* Furthermore, dreams and hallucinations rarely have the same content (ffytche, 2010), and disturbed dreams and hallucinations more often exist within separate patients with the same disorder than they co-exist in the same patient (Gjerstad, Boeve, Wentzel-Larsen, Aarsland, & Larsen, 2008).

These differences suggest that classical dreams and the visual hallucinations in dementia with Lewy bodies may be overlapping but distinct phenomena. Resemblances between dreams and visual hallucinations may be greater in the peripheral stages of falling asleep and awakening. These are associated with a distinct type of hypnagogic and hypnopompic visual hallucinations – or dreams – that are, in their content (for example, people and animals, Ohayon, 2000; Ohayon, Priest, Caulet, & Guilleminault, 1996), not dissimilar to the hallucinations of dementia with Lewy bodies.

3. Can we account for the overlap in the phenomenology of dreams and hallucinations?

We have previously suggested that hallucinations occur when spontaneous activity of the conscious visual perceptual system (an internal, sparse, functional, predictive, dynamic model of the visual input that the brain would receive if that model were correct) is *under constrained* by an impairment in attentional focus in combination with poor visual perception (Collerton et al., 2005). This allows the incorporation of a hallucinatory element into a scene, which is not disconfirmed by discrepant visual input. This PAD model has received empirical support in neurodegenerative disorders and in delirium (Barnes & Boubert, 2008; Brown et al., 2009; Ozer, Merai, Hanoglu, et al., 2007; Ramirez-Ruiz, Junque, Marti, et al., 2006) together with preliminary support in eye disease (Graham, Mosimann, Dudley, & Collerton, 2010).

Imaging and pathological data suggest that combined pathology in the ventral visual stream and dorsolateral frontal cortex is associated with visual hallucinations (reviewed in Collerton & Mosimann, 2010; Collerton et al., 2005). Functional imaging of Rapid Eye Movement (REM) sleep (which is more highly associated with dreaming than non REM sleep) shows analogous increased inferior temporal activity relative to decreased activity in frontal and primary visual cortex (Schwartz & Maquet, 2002). Thus, there is tenuous evidence that these common brain areas, critical for visual perception, may account for the similarities between the visual component of dreams and visual hallucinations. Direct comparisons of brain function during sleep and hallucinations would allow this hypothesis to be tested.

4. What then accounts for the phenomenological differences?

To answer this question, relevant information is available for another set of disorders associated with visual hallucinations – eye disease (ffytche, 2010). The Charles Bonnet syndrome of isolated visual hallucinations in blind people has two important features which are shared with the visual component of dreams but not the hallucinations of Lewy body disease. Both can fill the visual scene (though usually they do not: 44% of hallucinating patients with eye disease report panoramic, full scene hallucinations, versus 0% of patients with dementia; Makin et al., data in preparation), and both can be very bizarre.

To illustrate, we can compare a dream report "*I was in a hospital corridor, it was the department where I work as an intern and I saw many white gowns, a lot of people who were walking, then from the entrance an enormous person entered, really big – a gigantic person, who filled the whole doorway*." Cicogna, Occhionero, Natale, and Esposito (2007) with a report from the hallucinatory Charles Bonnet syndrome of blind people "*...there was a huge, vast canyon. I had to step over the edge to get to my seat. I have seen large churches with gravestones inside and everything was larger than life, but doors appeared shorter. The rooms just carried on, and I have to walk through things to get around – like a bath or a wall*" (Redman, Collerton, Mosimann, and Dudley, unpublished data). Both share scenic properties with size distortions. Contrast this with the prosaic figures of the dementia hallucination reported earlier.

One way to possibly account for these similarities is to take into account that restricted visual input is common to eye disease and sleep, but is not commonly seen in dementia. Lack of constraint from visual context may account for the phenomenological continuity between the visual hallucinations of eye disease and the visual component of dreams – panoramic and distorted – as well as their shared discontinuity from the hallucinations of dementia.

However, despite the phenomenological similarities, visual hallucinations in eye disease are rarely associated with other features of psychosis (or dreams) – strong emotions, behavioural acting out, loss of insight, or thought disorder – suggesting again that these are separable phenomena.

We can therefore speculate that we may be able to account for the similarities between dreams and visual hallucinations by the similarities in the internal systems that are engaged, and the differences by variations in external constraints. Direct comparisons of the character of dreams and hallucinations across different disorders together with imaging studies of common areas of interest will allow this possibility to be tested.

5. Dreaming, psychosis and neurotransmission

5.1. Neuropathological clues

We have argued that visual hallucinations in dementia with Lewy bodies and the visual components of dreams are not isomorphic, but that they reflect the activity of the same underlying cerebral visual structures. We can therefore ask: how are the key neurotransmitters which are involved in dreaming affected in dementia with Lewy bodies; with the supplementary question – how are these related to psychotic symptoms? The neurotransmitters that we will consider are acetylcholine (ACh), dopamine (DA), and serotonin (5-HT). Each is strongly implicated both in dreaming and in the pathology of Lewy body dementias. Thus, at least superficially, there appears to be an argument to be made that dreams and hallucinations reflect activity in the same neurochemical systems (Arnulf et al., 2000; De Cock et al., 2008; Sinforiani et al., 2008).

As with phenomenology though; once we look more closely, more differences than similarities arise. In the awake state, ACh, 5-HT and DA are all active. In REM sleep (80% of which is occupied with "classic" bizarre, colourful dreaming, though this has been questioned recently by Hodoba, Krmpotić, and Kujundzić-Tiljak (2008) and others), there is high ACh, low 5-HT, and steady DA, while in NREM sleep, in which dreams are less frequent, and possibly less "bizarre", there is low ACh, low 5-HT, and steady DA (Christodoulou et al., 2006; Hobson & Pace-Schott, 2002; Kalia, 2006; Monti & Monti, 2007; Murillo-Rodríguez, Arias-Carrión, Sanguino-Rodríguez, González-Arias, & Haro, 2009; Perry et al., 1991; Siegel, 2009; Stahl, Markowitz, Papadopoulos, & Sadik, 2004). This suggests that it is the balance between different neurochemical systems, particularly the cholinergic system, that influences dreaming, not the absolute activity of one.

Comparing this with the pathology which underlies visual hallucinations in Lewy body dementias throws up challenges to the idea that similar neurochemical changes underlie dreaming and hallucinations. Thus, low ACh in the neocortex correlates with visual hallucinations in dementia with Lewy bodies (Perry et al., 1991) – the opposite to the increase in ACh resulting from increased firing of basal forebrain cholinergic neurons seen in REM sleep. These basal forebrain nuclei additionally innervate the thalamus. The absence in dementia with Lewy bodies of a correlation between visual hallucinations and cholinergic activity in the thalamus (Ziabreva, Perry et al., in preparation) indicates a key role in hallucinogenesis for the basal forebrain innervation of the cortex, as opposed to sleep-related thalamic and brainstem projections. In addition, pathology occurring in sleep-related brainstem cholinergic nuclei in Progressive Supranuclear Palsy, which is a disorder which is not associated with visual hallucinations, similarly suggests these nuclei are not key in hallucinogenesis.

If the data suggests no similar changes in cholinergic activity in hallucinations and REM sleep – the best proxy that we currently have for the dreaming state – what of other neurotransmitters? In contrast to the steady dopaminergic function associated with sleep, hallucinations in dementia with Lewy bodies have been proposed to be associated with dopaminergic over activity; primarily because of the induction of hallucinations by l-dopa treatment of the motor symptoms of Lewy body dementias. However, there is no compelling pathological evidence linked to psychosis in Lewy body dementias (in contrast to schizophrenia), and imaging of striatal dopamine transporter correlates, not with hallucinations, but with apathy in Lewy body dementia (David et al., 2008). In a more recent report, decreased dopamine transporter (123-I-FP-CIT SPECT imaging) correlated with visual hallucinations (Roselli et al., 2009), again suggesting that high dopaminergic function is not associated with hallucinations. In contrast to these findings from dementia with Lewy bodies, in schizophrenia hyperactivity in dopamine neurotransmission is directly related to hallucinations and delusions (Van Os & Kapur, 2009). With respect to 5-HT, there is evidence of relative hyperactivity of this system associated with visual hallucinations in Lewy body disorders (Perry et al., 1990, 1993, Cheng et al., 1991), again the reverse of the diminished activity in sleep.

5.2. Do drug induced psychoses inform the dreaming psychosis issue?

If pathological comparisons suggest different patterns of neurotransmission in dreams and hallucinations, pharmacological studies paint an analogous picture. Thus, evidence suggests that there are opposite effects of cholinergic drugs on hallucinations in the waking state and dreaming in the sleep state. Anticholinergics such as scopolamine and atropine induce visions of people and animals in the awake subject *"There were animals looking at me keenly with contorted grimaces and staring and terrified eyes. At the same time I experienced an intoxicating sensation of flying."* (Gustav Schenk; a German pharmacologist experimenting with henbane, Perry, 2002) but, in contrast, reduce dreams (Toscano, Pancaro, & Peduto, 2007). As a corollary, there are increased abnormal dreams and nightmares as a side effect of cholinesterase inhibitors in people with dementia, even though these drugs reduce visual hallucinations in the awake patient, and are more effective in treating hallucinating compared to non hallucinating patients (Cummings, Aarsland, & Dronamraju, 2010; Dunn, Pearce, & Shakir, 2000; McKeith, Wesnes, Perry, & Ferrara, 2004; Stahl et al., 2004; Zahodne & Fernandez, 2008).

In contrast to the opposite direction of effects of pharmacologically manipulated cholinergic activity on dreams and hallucinations, there are more similar dopaminergic drug effects in the two states. For example, cocaine users report

hallucinations in several modalities (visual and auditory) as well as strange hallucinatory dreams. In Lewy body dementias, dopaminergic medication not only induces hallucinations as noted earlier, but also is associated with increased dreaming (Dooley & Markham, 1998; Goldman, Goetz, Brandabur, Sanfilippo, & Stebbins, 2008; Iseki, Marui, Nihashi, & Kosaka, 2002; Papapetropoulos & Mash, 2005). As an instance, 27/83 (30.7%) of Parkinsonian patients on chronic dopaminergic agonist therapy developed drug-related dream phenomena, including vivid dreams, night terrors and nightmares, which were correlated to the duration of therapy (Sharf, Moskovitz, Lupton, & Klawans, 1978). Again, as a corollary, neuroleptics reduce both psychotic symptoms and dreaming (Scarone et al., in preparation).

5HT drug effects on hallucinations are well established, with 5-HT 2A agonists inducing complex visual hallucinations and perceptual changes, but their effects on dreaming are unclear with a sparse and inconsistent literature (Pace-Schott et al., 2001).

Thus, in summary, the available data, though sparse and at times inconsistent, suggests that cholinergic changes seem opposite in dreaming and hallucinations, dopaminergic changes are similar, and there is, as yet, no clear pattern with 5-HT transmission.

6. The dreaming hypothesis of psychosis may apply more to other symptoms other than visual hallucinations

While the psychological, pathological, and pharmacological evidence above does not obviously suggest parallels between dreaming and visual hallucinations in Lewy body disorders, there is some evidence of a link to delusional symptomatology. Cholinergic activity is higher in thalamic nuclei (e.g. centromedian) in dementia with Lewy bodies patients with delusions than in those without (Ziabreva, Perry et al., in preparation). Increased M1 receptor density in temporal cortex (Ballard et al., 2000) and increased M2 receptor density in cingulate cortex (Teaktong et al., 2005) have both been linked to delusions in dementia with Lewy bodies. Relative cholinergic hyperactivity of both basal forebrain and brainstem cholinergic nuclei would then be a common feature of both dreaming and delusional states.

In addition to hallucinations and delusions, fluctuating cognition or conscious awareness is another key feature of dementia with Lewy bodies that might appropriately be considered for its relation to dreaming; given that dreaming itself normally involves variations in awareness – at least to judge by recall of only some dreaming experiences. Neurochemical correlates of such fluctuations in dementia with Lewy bodies include higher nicotinic (Teaktong et al., 2005), muscarinic (Pimlott et al., 2006) and dopamine D2 receptors (Piggott, Perry et al., in preparation). Higher muscarinic, nicotinic and dopamine receptors may amplify small variations in transmission leading to instability in the mechanisms underpinning consciousness and attention. Not only do such features of dementia with Lewy bodies contribute to the dreaming – psychosis debate, as discussed in this review, but similar symptoms occurring in other conditions such as schizophrenia (associated with hyper dopaminergic function and decreased muscarinic and serotonergic receptors) could also be examined in this context.

7. Conclusions

Freud described dreaming as the royal road to the unconscious. From the perspective of trying to understand psychosis, it seems to us to be more of an unmapped royal road system with numerous side routes and cul de sacs.

There appears to be no simple relationship between dreams and hallucinations. Depending on the type of evidence, similarities are greater or lesser. Functional imaging suggests a close anatomical relationship between the two; phenomenology, a partial overlap; and neurochemistry, both common and opposite changes in activity.

How these types of evidence relate to each other is not clear. Neither hallucinations nor dreams are unitary phenomenon; each has shared aspects and unique features.

Direct comparisons of the phenomenology and neurophysiology of the two experiences across normal and pathological states, within a common model of how the mind and brain function, are likely to illuminate both states.

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