ing compared to controls and non-hallucinating PD patients. Poor source monitoring and perceptual processing, combined with intact internal image generating, may lead to visual hallucinations by confusion between images that were imagined with those that were actually seen. Collerton et al. point out that deficits in source monitoring and misidentification of internal images are unlikely to account for all RCVH, as many people with RCVH are aware that they are hallucinating. This is an important observation which serves to highlight the complexity of the processes involved and perhaps the need for a more precise terminology.

In the above case, "reality monitoring" is intact but the online appreciation of source may not be (i.e., is it a memory of an image, or a newly generated one?) and, more crucially, the process of attribution may be suspect. According to some models of reality monitoring, decisions about veridicality follow automatically from phenomenal characteristics. In short, if an image is vivid enough, it will be accepted as real. Such an algorithm may work well in most circumstances but could lead to "loss of insight" and hence false beliefs (see David & Howard 1994) if fairly low-level perceptual factors were enhanced (e.g., by neurotransmitter imbalance) or if supervisory processes were weakened (by general cognitive impairment). It may be that the underlying cognitive mechanisms in schizophrenia and PD psychosis are different from those in Charles Bonnet syndrome or sleep disorders because of such modulating factors.

Complex hallucinations in waking suggest mechanisms of dream construction

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Abstract: Waking hallucinations suggest mechanisms of dream initiation and maintenance. Visual association cortex activation, yielding poorly attended-to, visually ambiguous dream environments, suggests conditions favoring hallucinosis. Attentional and visual systems, coactivated during sleep, may generate imagery that is inserted into virtual environments. Internally consistent dreaming may evolve from successive, contextually evoked images. Fluctuating arousal and context-evoked imagery may help explain dream features.

Collerton et al. describe phenomenological and physiological differences between recurrent complex visual hallucinations (RCVH) and dream imagery. Nonetheless, highly complex visual hallucinations (albeit usually non-repetitive) do occur in dreams, and it is parsimonious to hypothesize some overlap in their mechanisms. Biological models of dreaming (e.g., Hobson et al. 2000) propose neural substrates for a fully expressed dream experience based, in part, upon recent positron emission tomography (PET) studies showing widespread cortical deactivation during sleep with selective reactivation of limbic areas during rapid eye movement (REM) sleep (Braun et al. 1997; 1998; Maquet et al. 1996; 1997; Nofzinger et al. 1997; 2002). Such global reorganization of brain activity differs from the more selective ventral stream and attentional system abnormalities superimposed upon waking activity in the PAD model of Collerton et al. However, when one considers how dreaming might be initiated and maintained, parallels become apparent.

Without retinal input, the dreamer cannot perceive veridical visual contexts that evoke RCVH in waking, according to Collerton et al. However, visual association cortices can be activated in REM (Braun et al. 1998) and NREM (non-rapid eye movement) sleep (Hofle et al. 1997; Kjaer et al. 2002). Ascending signals such as PGO (ponto-geniculo-occipital) waves may activate visual cortex during REM (Callaway et al. 1987) or the NREM-to-REM transition (Steriade 2000b). Visual cortex activation in NREM results from phasic activational processes, "covert REM" (Nielsen 2000), arising perhaps from activity in autonomic and limbic areas (Nofzinger et al. 2002; Rolls et al. 2003).

During sleep, therefore, visual association cortices may support ambiguous visual experiences – one prerequisite for RCVH in the PAD model. Simultaneously, ascending brainstem reticular activation may engage midline attentional structures, such as nonspecific thalamic nuclei and basal forebrain (Dringenberg & Olmstead 2003), and medial prefrontal cortex (Nofzinger et al. 1997), allowing some awareness of this fictive vision. Such partial awareness is deficient compared to normal waking – the other requirement for RCVH in the PAD model. If sufficient activation of visual association and midline attentional systems is achieved in sleep, a rudimentary visual context sufficient to evoke "proto-objects" may arise.

The dream might subsequently emerge by a "boot-strapping" process involving successive, contextually evoked visual images. Fictive "proto-representations" in other modalities (e.g., auditory) may emerge from regional activations of subcortical (e.g., motor), unimodal (e.g., somatosensory), heteromodal (e.g., memory), or limbic (e.g., emotion) areas. An image may elicit congruent representations in other modalities, achieving binding via long-range synchrony of high-frequency electrical activity (Kahn et al. 1997; Llinas & Ribary 1993). Specific memories may become woven into the emerging dream as their cortical representations are activated (Stickgold et al. 2001). Further aspects of dreaming can now be suggested.

Brevity of NREM reports. In sleep, episodic and working memory are deficient (Fell et al. 2003; Fosse et al. 2003; Hobson et al. 2000; Pace-Schott et al. 1997). Without this mnemonic "glue" that ensures continuity of our waking experience across attentional lapses, a developing dream, sustained only by elicitation of successive proto-representations, may be disrupted by any hiatus in conscious awareness. Awareness may, in turn, require continued ascending activation, preventing emergence of the endogenous synchronous thalamocortical and corticocortical oscillations of NREM sleep (Steriade 2000b). Sustained activation is present in REM but may be discontinuous in NREM, leading to brief, relatively unrelated NREM dream episodes. By contrast, the common experience of resuming the same dream following brief arousal is possible because further activation enables sufficient memory to span a semi-waking hiatus. Such continuity may be unavailable when the dream hiatus consists of deepened NREM sleep with resumption of intrinsic oscillatory activity. Forebrain activation may even become insufficient to support consciousness, resulting in cessation of the dream experience.

Internal consistency. The remarkable internal consistency of dream plots may arise because the evolving dream context itself determines which proto-representations will next be evoked. Such self-organization of dreams (Kahn & Hobson 1993), utilizing successively evoked proto-representations, may also explain how coherent plots can emerge despite deficient episodic and working memory (Fosse et al. 2003; Hobson et al. 2000).

Bizarreness. Prototypical forms of dream bizarreness – discontinuities, incongruities, and uncertainties (Hobson 1988) – may arise from interaction between fluctuating arousal and context-generated imagery. Lapses of attention may account for discontinuities such as abrupt scene shifts (Sutton et al. 1994). Dream incongruities may similarly be explained by evocation of contextually semi-congruent but illogical proto-representations. Uncertain recall, to a degree that appears bizarre by waking standards, may be inherent in such ad hoc constructions, especially if context-evoked proto-representations do not fully resolve into fictive percepts before subsequent representations arise. The importance of visual context in spanning attentional lapses is apparent in object transformations – discontinuities that are explicable by visual similarity between the original image and its transform but not by their semantic relatedness (Rittenhouse et al. 1994).

Global dream cessation. Damage to inferior parietal heteromodal association areas (BA 39 and 40) can alone result in global dream cessation (Doricchi & Violani 1992; Solms 1997), caused

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by, perhaps, the dreamer's inability to perceive a virtual environment. Maintenance of a dream environment may rely preferentially upon visuo-spatial processing subserved by these areas (Mesulam 2000). Lateralization of visuo-spatial function may account for the greater likelihood of dream cessation following right versus left inferior parietal damage (Solms 1997). Dependency on fictive vision for dreaming may be analogous to cessation of RCVH with total blindness and their dependence on dorsal stream integrity, as proposed by Collerton et al. However, dream cessation following left parietal damage and non-visual dreaming following bilateral extrastriate damage (Solms 1997) suggest that fictive dream environments can be based, at least in part, upon other modalities.

Dream visions and RCVH occur in globally differing brain states and cannot be equated. For example, whereas cholinergic deficits often underlie RCVH (Collerton et al. target article), REM sleep shows cholinergic activity equal to, or greater than, in waking within cholinergic projection neurons (Dringenberg & Olmstead 2003) and their terminal fields in the thalamus (e.g., Williams et al. 1994), basal forebrain (e.g., Vazquez & Baghdoyan 2001), and cortex (e.g., Marrosu et al. 1995). Nonetheless, comparison of dreaming with waking pathologies can provide fresh insights into the neural bases of both conditions (Pace-Schott 2005; Schwartz & Maquet 2002).

Hallucinating objects versus hallucinating subjects

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Abstract: Collerton et al. propose that one and the same mechanism (PAD) underlies recurrent complex visual hallucinations (RCVH) in various disorders, including schizophrenia, dementia, and eye disease. The present commentary offers an alternative account of RCVH and other recurrent complex hallucinations specific to schizophrenia and related disorders only. The proposed account is consistent with the bias of schizophrenic RCVH contents toward animate, socially active entities.

The variety of sensory hallucinatory phenomena is characterized by a dichotomy that may be easy to notice and hard to understand. Considering the visual modality separately, Collerton et al. in the target article point at a double dissociation of recurrent complex visual hallucinations (RCVH) versus simple hallucinations including dots, lines, flashes, amorphous shapes, and panoramic landscapes. If a measure of visual complexity is indeed the best separator of the two dissociated categories, then all RCVH are likely to originate from one general mechanism that is distinct from mechanisms underlying other types of hallucinations. Following this logic, Collerton et al. introduce PAD as a general model of RCVH applicable to all cases in which RCVH are observed, including dementias, delirium, schizophrenia, eye disease, and others.

On the contrary, it may seem reasonable to account for RCVH in schizophrenia and, for example, in eye disease based on different mechanisms, if, instead of complexity, another cognitive dimension specific to schizophrenia underlies the dichotomy. Indeed, most hallucinations in schizophrenic states involve various forms of agents engaged in social interactions with the subject (Frith et al. 1998; Mellors 1970; Silbersweig et al. 1995). This happens regardless of the perceptual modality. For example, the following types of auditory hallucinations are characteristic of schizophrenia (Cahill & Frith 1996): voices arguing, voices commenting on one's action, audible thoughts (voices repeat verbatim or comment on subject's thoughts), and voices that command the subject. On the other hand, auditory hallucinations after deafness may include noises and melodies along with singing or talking voices that do not engage in social interactions (Hammeke et al. 1983). The situation is similar with RCVH in non-schizophrenic cases reviewed by Collerton et al. (reviewed in support of PAD). These include RCVH in visual impairment cases described by Charles Bonnet: faces that never smile (Santhouse et al. 2000), RCVH induced by electrical stimulation of the brain (Penfield & Perot 1963), experienced after stroke, in Parkinson's disease (Manford & Andermann 1998), caused by drugs (Hoffmann 1983; Huxley 1959), and so forth. Generally, all non-schizophrenic hallucinations lack a certain degree of animacy and interactive social activity that are typical for schizophrenic hallucinations.

Therefore: (1) Of the following two statements, (a) one and the same mechanism is responsible for RCVH and recurrent complex auditory hallucinations in schizophrenia, and (b) one and the same mechanism is responsible for RCVH in schizophrenia, in dementia and in eye disease, (a) appears to be more credible than (b). (2) The mechanism underlying recurrent complex hallucinations in schizophrenia probably has to do with the concepts of agency, animacy, social interactions, and more generally, the self and its representation in the brain.

A theory based on the latter idea (2), and supported by analysis of clinical and introspective data, was recently proposed by Samsonovich and Nadel (2005). According to this theory, under normal conditions, discrete instances of the subject's own self (labeled *I-Now, I-Previous, I-Next*, etc.) and of the self of any currently perceived external subject, together with all subjective experiences attributed to those instances, are represented in working memory as separate units (*mental states*) that are processed in parallel and interact with one another, obeying a set of hardwired rules (*self axioms*). From this point of view, schizophrenia is a condition in which identities and normal relations among mental states determined by self axioms become lost or altered (Samsonovich & Nadel 2005). As a result, malfunctioning mental states become independent agents and start creating new memories (delusions), engage in dialogues (voices), independently perform imagery (thereby producing hallucinations), or take control of actions. From this point of view, the visual appearance of a socially active RCVH is secondary with respect to its simulated subject, which is a malfunctioning mental state.

Alternatively, one may assume that in schizophrenic RCVH the step of creating a theory-of-mind (ToM) representation of an imaginary character (i.e., "hallucinating a subject") is secondary with respect to developing a sensory hallucination of a face, a body or a voice ("hallucinating an object"). In this case, it would be difficult to understand the nature of the bias toward animate, socially active RCVH in schizophrenia: starting from this point of view, one should expect an opposite bias, toward inanimate or socially inert RCVH, given that ToM abilities are specifically impaired in schizophrenia (Corcoran et al. 1995; 1997; Doody et al. 1998; Frith & Corcoran 1996; Langdon et al. 1997; Sarfati & Hardy-Baylé 1999). It is not clear why the well-known ToM deficit that is characteristic of schizophrenia in general should be reversed in hallucinatory cognitive activity, unless an opposite assumption is made: that in schizophrenia and related disorders, hallucinating a subject (i.e., having a "lost" or misattributed mental state in working memory) causally underlies the hallucination of the related object (face, body, voice). Stated differently, both well-known attributes of schizophrenia – the general ToM impairment and ToM-biased hallucinations – may have one and the same common origin: malfunctioning of the system of mental states (Samsonovich & Nadel 2005).

The PAD model of Collerton et al. has at least several problems; however, the present commentary is focused on one of them: PAD does not account for the specificity of contents of RCVH in schizophrenia and in fact suggests an opposite specificity, as explained above. Although the combined attentional and visual perceptual impairments interacting with internal scene representations could in principle result in a particular schizophrenic RCVH, it is not clear from the point of view of Collerton et al. why there should be a bias toward elaboration rather than simplification of agency and social activity of hallucinated entities. The above analysis suggests that the

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