Commentary on: "Why people see things that are not there: A novel Perception and Attention Deficit model for recurrent complex visual hallucinations" by Daniel Collerton, Elaine Perry, and Ian McKeith

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Believing is Seeing in Schizophrenia: The role of top-down processing

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Abstract:

The etiology of visual hallucinations is largely undetermined in schizophrenia. The PAD model partly concurs with what we know about neurocognition in schizophrenia but we need to specify the types of perceptual and attentional abnormalities that are implicated in RCVH. Available data suggest that abnormal attentional control and top-down processing play a larger role than the ventral stream deficits.

Hallucinations are core features of schizophrenia. While cognitive and neuroanatomical origins of auditory hallucinations are beginning to be elucidated (David, 1999), the underlying etiology of visual hallucinations remains undetermined. Therefore, the PAD model of RCVH, which generates testable hypotheses, is both timely and valuable. The broad outline of this model concurs with what is known about neurocognition in schizophrenia. Significant proportions of schizophrenic patients hallucinate, show deficits in a wide variety of attention tasks, and have prefrontal and temporal abnormalities (Shenton et al 2001; Mitchell et al, 2001). However, to move beyond these surface similarities, we need to specify the types of perceptual and attentional deficits that may result in RCVH and test them empirically.

Attention is a multifaceted concept that can be parsed into distinct multiple systems, implemented by overlapping but separable neural circuits. Schizophrenic patients have problems in orienting, deploying, focusing, shifting and sustaining attention in space. What aspects of attentional abnormalities in schizophrenia might contribute to RCVH? The PAD model predicts that RCVH are located at the focus of attention and unlikely to be experienced at the periphery of the visual field. Schizophrenic patients have difficulties in shifting spatial attention (Posner et al, 1988) and this deficit is associated with positivey symptoms such as hallucinations (DiGirolamo & Posner, 1996). Specifically, they show deficits in shifting attention to the right visual field (RVF) but not to the left visual field (LVF). This suggests that they may neglect the RVF. Interestingly, schizophrenic patients tend to experience visual hallucinations more in the LVF (Bracha et al, 1985). A question arises here for the PAD model. If subjects have difficulty shifting focal attention to RVF, they are unlikely to “see things that are not there” in the RVF. Therefore, visual search tasks or Posner’s covert orienting task may yield a pattern of greater accuracy in the LVF, coupled with greater incidences of RCVH in the LVF. In contrast, the same patients might make more

perceptual errors in the RVF. However, it is unclear whether such a relationship has been reliably observed. Following this line of reasoning (i.e., RCVH appear where attention goes), it might be possible to develop strategies for extinguishing RCVH by increasing external visual signals at the location of the RCVH or by re-directing attention.

Control of attention is mediated partly by expectancies or top-down processes. It has been observed that schizophrenic patients give greater weight to top-down expectations on perception than normal controls do (Aleman et al, 2003). This provides a clear test of the PAD model. If top-down expectancies guided by stereotypical scene representation play a stronger role in hallucinators' perception, then, when presented with a familiar scene, they should detect context-congruent objects more quickly and at a lower threshold than non-hallucinators. With unfamiliar scenes, the weight of top-down processing may increase further in hallucinators to make the novel input fit their “theory”. Top-down processing may also increase if hallucinators are presented with visually-degraded or ambiguous scenes (e.g. blurred, low intensity) to make sense of the visual noise (e.g. seeing faces in the clouds or Rorschach inkblots). So unfamiliar or ambiguous scenes may trigger more top-down processing and lead to RCVH. This possibility seems incongruent with the PAD model.

In addition to attentional deficits, the PAD model postulates the existence of concurrent perceptual deficits within the ventral visual processing stream. Schizophrenia patients, indeed, exhibit a variety of visual abnormalities. The majority of reported deficits, however, are confined to dorsal stream such as motion perception (Chen at al, 1999) and backward masking (Slaghuis & Bakker, 1995). For example, schizophrenia patients are impaired in a visual backward masking task when required to detect target locations but not when asked to identify masked letters (Cadenhead et al, 1998). These findings are corroborated by VEP studies that report abnormal P1 component over dorsal visual areas, but normal P1 over ventral regions.The N1 component (generated by early ventral stream structures) is also normal in schizophrenia (Foxe et al, 2001; 2005).

Lack of deficits in early ventral processing does not preclude the existence of “high-level’ abnormalities of semantic and object categories. Indeed, evidence of temporal lobe abnormalities with behavioral consequences in schizophrenia is rather striking (Shenton et al, 2001; Mitchell et al, 2001) have behavioral consequences. Although schizophrenic patients show no deficits on simple object perception tasks, they are impaired in higher-level ventral tasks such as recognition of atypical objects (Gabrovska et al 2002). But the PAD model does not make an explicit distinction between low and high-level ventral stream functions; it suggests the deficits are in the generation of proto-objects, which would arguably involve low to mid-level ventral processes. The PAD model should identify the levels of ventral stream processing involved in RCVH and provide converging evidence for the ventral stream deficits in schizophrenia.

In our opinion, top-down processing seems to be the main driving force behind experiencing hallucinations. Ventral stream deficits may not be necessary for RCVH, at least in schizophrenic subjects, as suggested by the lack of strong evidence for ventral abnormalities. However, perceptual deficits can facilitate generation of RCVH. Generally speaking, all visual defects can be construed as increasing the noise at the expense of "veridical" visual signals. Impoverished visual representations are more susceptible to misinterpretation. Only when combined with abnormal top-down attentional and semantic processes, the misinterpretation of visual input can lead to RCVH. Namely, the role of perceptual deficits in RCVH is to simply increase the noise, whereas the actual generation of RCVH lies within faulty higher-level processes. This account of RCVH does not require localization of deficits within the ventral stream and it accords with the increased frequency of RCVH in situations when the visual input is degraded by external factors (e.g. dim lighting). In schizophrenia, widespread dorsal system deficits coupled with structural abnormalities of the primary visual area (Selemon et al, 1995) may be enough to degrade early visual representation, thus making it vulnerable to faulty top-down processes.

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