Visual hallucinations, attention, and neural circuitry: Perspectives from schizophrenia research

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Abstract: We tested Collerton et al.'s model of visual hallucinations by re-examining a data set for correlations between visual hallucinations and measures of attentional function in schizophrenia patients. These data did not support their model. We suggest that cortical hyperexcitability plays an important role in hallucinations, and propose an alternative model that links evidence for cortical hyperexcitability with abnormal neural dynamics.

The study of hallucinations can shed light on many aspects of cognitive and brain function in healthy individuals and in individuals with neuropsychiatric disorders. Collerton et al. make a timely contribution to this area of inquiry with their Perception and Attention Deficit (PAD) model of recurrent complex visual hallucinations (RCVH). Collerton et al. have done an admirable job in distilling a unifying model of RCVH from the literatures on RCVH and visual attention. However, research into the neural substrates of schizophrenia has led to views of hallucinations that conflict with the PAD model. Here we discuss the implications of findings from our laboratory and others for the PAD model.

The necessity of attentional involvement in visual hallucinations. A dysfunction in attentional modulation of visual processing is an appealing hypothesis for RCVH. To test this hypothesis, we conducted an exploratory analysis of a data set of ours in which we found relationships between visual hallucinations and electrophysiological measures of visual perception in chronic schizophrenia patients (Spencer et al. 2004). Of the 20 patients in this sample, 7 had some history of visual hallucinations. We looked for relationships between visual hallucinations and attentional impairment by calculating correlations between the visual hallucinations scale of the Scale for the Assessment of Positive Symptoms (Andreasen 1984), the attention scales of the Scale for the Assessment of Negative Symptoms (social inattentiveness, inattentiveness during mental status testing, and global rating of attention) (Andreasen 1983), and the Positive and Negative Syndrome Scale (poor attention) (Kay et al. 1987). None of these correlations approached significance (all $r < 0.2$, $P > 0.42$). Looking further, we tested for relationships between performance measures (error rate and reaction time effects) and visual hallucinations, but found no correlations either (all $r < 0.33$, $P > 0.20$). Subgroup level t-tests (hallucinators vs. non-hallucinators) yielded negative findings also.

These results imply that attentional impairment is not necessarily linked to RCVH. However, we note that the poor impulse control scale of the PANSS did show a significant correlation with visual hallucinations ($r = 0.46$, $P < 0.05$). This finding suggests a possible involvement of deficient executive attention processes in visual hallucinations, rather than the attentional template/biasing processes proposed by Collerton et al.

The role of cortical excitability in hallucinations. We believe that Collerton et al. do not lend enough weight to the relationship between cortical excitability and RCVH, as there is substantial evidence that cortical excitability is related to hallucinations in schizophrenia. The main evidence for increased cortical excitability in schizophrenia comes from studies utilizing transcranial magnetic stimulation (TMS). For example, Hoffman and colleagues (2003) found that slow repetitive TMS, which decreases the excitability of the underlying cortex, reduced the incidence and severity of treatment-resistant auditory hallucinations (when applied to the left temporo-parietal region). Cortical hyperexcitability in schizophrenia would be consistent with the deficits in inhibitory neurotransmission that have been revealed by postmortem cellular studies (Lewis et al. 2005). In addition, there is evidence that N-methyl-D-aspartate (NMDA) antagonist ketamine increases cortical excitability (Di Lazzaro et al. 2003), which links the NMDA receptor hypofunction model of schizophrenia (Tsai & Coyle 2002) with cortical excitability. Finally, there is a report that slow repetitive TMS applied to the occipital lobe eliminated visual hallucinations in a non-schizophrenic patient (Merabet et al. 2003). Hence, we believe that there is in fact substantial evidence to support an important role for hyperexcitability of sensory cortex in hallucinations.

Neural dynamics and hallucinations. From our own research on visual perception and neural dynamics in schizophrenia, we have proposed a different (but not exclusive) account of RCVH. We recently reported that chronic schizophrenia patients show abnormalities in visual gamma-band oscillations (Spencer et al. 2003; 2004). These abnormalities include a response-locked oscillation (RLO) recorded over the occipital lobe, which we hypothesize reflects feature-binding processes in visual cortex. In schizophrenia patients, the degree of phase-locking of the RLO was positively correlated with their visual hallucination symptoms (Spencer et al. 2004). In contrast, the N1 visual evoked potential was reduced in amplitude for patients with visual hallucinations, compared to patients without. We proposed that this dissociation between a putatively endogenous process (increased RLO synchronization) and an exogenous response to sensory stimulation (decreased N1) reflected increased excitability in the visual cortex of schizophrenics with visual hallucinations. A similar pattern of increased endogenous activity and decreased responsiveness to external stimulation in visual hallucinators was found by ffytche et al. (1998).

If the neural substrate of a perceived object is an attractor state in a neural network (such as oscillatory synchronization among cells coding individual features of the object), then RCVH could result from an increased propensity for such a dysfunctional network to go into attractor states independently of other biasing influences, such as external input (sensory stimulation) or an attentional bias signal from prefrontal cortex. The main distinction between our account of hallucinations and the PAD model is that we emphasize dysfunction of the relevant sensory cortex, which is manifested by hyperexcitability. Although it is certainly plausible that attention deficits could exacerbate hallucinations, the lack of correlations between visual hallucinations and measures of attention suggests that attention is not a primary factor, at least in schizophrenia.

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