Perception is far from *perfection*: The role of the brain and mind in constructing realities

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Abstract: Dichotomizing perceptions, by those that have an objective reality and those that do not, is rejected. Perceptions are suggested to fall along a multidimensional continuum in which neither end is totally "pure." At the extreme ends, perceptions neither have an objective reality without some subjectivity, nor, at the other end, even as hallucinations, are they totally dissociated from reality.

Higher-level visual cognition is achieved through complex and dynamic interactions, both within the many processing components of the visual system and between the visual system and other brain areas and systems. Before considering the multiplicity of factors that play a role in determining what we see and experience, it is important to pull back and consider one's basic conceptualization and theoretical outlook. Such philosophical issues have (implicitly, if not explicitly) implications for how we approach and conduct research, and even for the very questions we ask and the type of answers we seek (Dror & Dascal 1997; Dror & Thomas 2005).

A naïve view of visual cognition deems that it normally provides perceptions and experiences that have an objective reality. Failures to see things correctly, such as in hallucinations, need to be investigated and explained as system malfunctions. This framework dichotomizes perceptions as either "normal" (i.e., having an objective reality) or as "failures" (i.e., lacking an objective reality). Other perceptual problems, such as illusions, misidentifications, distortions, and misperceptions, may be considered as a subcategory in which perceptions "fail" yet stem from an "objective reality" rather than being totally dissociated from it.

The nature of perception. An alternative view is suggested in which perceptions are seen to fall along a multidimensional continuum. Even at the far extremes of the continuum, perceptions are not entirely "pure." At the one extreme, perceptions never have a full and total "objective reality," and at the other extreme end, hallucinations and delusions are not totally dissociated from reality.

The underpinning of this framework is not its continuum nature, but that perception is never totally objective to begin with. The lack of objective reality across the plurality of perceptions results in a multidimensional continuum. If one adopts this view, then rather then asking what is wrong or wondering why the system is failing, one tries to understand what factors mediate perception and how they interact. Perhaps the former outlook derives from a more clinical approach and perspective whereby some people are considered "normal" and hence perceive the "objective reality," whereas patients and clinical populations have "disorders" because of system failures to have an "objective reality."

Understanding that *perception* is far from *perfection* provides a contextual framework for examining the visual cognitive system. The mind and the brain are dynamic systems that play active roles in how we perceive and construct realities. Our perceptions depend on a whole range of factors, which I will try to illustrate.

Mental states. Mental states play a critical role in how perceptual information is processed. For example, our hopes, fears, and expectations affect what we perceive. In a recent laboratory experiment, emotional states were shown to affect whether two visual patterns were perceived as the same or as being different (Dror et al. 2005). There are numerous phenomena that can further illustrate how the mind plays an active role in how we perceive and construct reality, such as motivation, wishful thinking, cognitive dissonance, self-fulfilling prophecies, and confirmation bias (e.g., Darley & Gross 1983; Festinger & Carlsmith 1959; Snyder et al. 1977).

Cognitive factors. Visual cognition is a set of complex and interactive processes (e.g., Grill-Spector et al. 1998). No cognitive system works on its very own. Each cognitive system is intertwined and interacts with a range of other cognitive systems. For example, how information is understood, processed, and collected depends on how it compares against information already stored in memory (e.g., Kosslyn et al. 1994). The influence of such processing further depends on how it is represented, available resources, goals of the system, context, and other factors (e.g., Eberhardt et al. 2004; Maier 1930; Reuter-Lorenz 2002; Smith & Dror 2001).

Perceptual mechanisms. Even the lower-level sensory mechanisms, which initially perceive and encode the input to the system, are not passive or isolated from a variety of factors. They try to make (impose) sense and consistency on the world around us, even when the input presents ambiguous or impossible information (e.g., Dror et al. 1997). Among other things, the perceptual mechanisms adjust and change sensitivity thresholds, segment and chunk information in a variety of ways, and perceive colour and lighting based on their own parameters settings (e.g., Land 1964; Prinzmetal 1995). Therefore, much of what we perceive, even at the lower-level mechanisms, is dependent on the perceiver rather than reflecting objective reality.

Bottom-up, top-down, and mental imagery. Perception and cognition, at all their levels, depend on bottom-up, data-driven processes and on top-down, conceptually driven processes (e.g., Humphreys et al. 1997). The top-down processes may be viewed as the source of subjectivity, individualization of perception, and distancing from the "objective reality." However, even the sensory mechanisms in a purely bottom-up mode do not reflect reality as it "really" is.

Mental imagery is a range of phenomena where perception and experience occur without direct perceptual input. Positron emission tomography (PET) and other studies have demonstrated that the same brain substrates are used to process imagined and perceived images, except that in imagery the input comes from other cognitive systems (Kosslyn et al. 1993; 1997). Furthermore, visual mental rotation, for example, shows that imagination follows the laws of physics and rotations in the physical world (e.g., Smith & Dror 2001). Hence, although all the processes involving imagery do not have direct input from the external world, the input and the way it is processed is not dissociated from normal perception.

Summary and conclusions. We are different people, with different experiences, different views, and different brains and sensory mechanisms. This entails that we have different perceptions. Most people share sufficient perceptual commonalities that allow labelling and communication within everyday life activities. Nevertheless, the perceptions across people are far from identical. Furthermore, even if we did perceive the exact same thing, that percept is not necessarily a true and accurate reflection of the "objective reality."

Perceptions fall along a multidimensional continuum and are subjective in nature. This individualization of perception derives from the active nature of cognition and the wide range of factors that affect what and how we perceive.

Two visual hallucinatory syndromes

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Abstract: When viewed from a distance, visual hallucinations fall into one of two symptom patterns, a dichotomy which poses a problem for theoretical models treating them as a single entity. Such models should be broadened to allow for two distinct but overlapping syndromes – one likely to relate to visual de-afferentation, the other to Perception and Attention Deficit (PAD) cholinergic pathology.

Are complex visual hallucinations a single pathophysiological entity? Collerton et al. would have us think so and have produced a

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compelling neurocognitive model – Perception and Attention Deficit (PAD) – with which to account for a range of their phenomenological, pathological, and clinical features. Yet the success of the model comes at an expense. For it to succeed, the authors have been forced to make it something of a Procrustean bed, stretching some parts of the visual hallucination evidence and amputating others. This is no more apparent than in their attempt to deal with the hallucinations associated with eye disease, the third-ranking pathological condition in their estimates of population-wide morbid load, exceeded only by delirium and the combined dementias.

As acknowledged by the authors, in eye disease (or, indeed, any visual-pathway lesion), simple hallucinations far outnumber complex ones, an observation which contrasts with core PAD disorders such as Lewy body dementia, Alzheimer's disease, and Parkinson's disease, in which complex hallucinations far outnumber simple ones. For eye and visual-pathway disease, complex hallucinations are only a small part of a much larger clinical picture, their importance needing to be stretched to match core PAD disorders. In fact, even after stretching, the match is an imperfect one. For example, complex hallucinations such as figures in patients with eye disease tend to be bizarre and unfamiliar, often wearing elaborate costumes and hats (Santhouse et al. 2000). In contrast, the figures hallucinated in Parkinson's disease and the dementias tend to be mundane and familiar (Fénelon et al. 2000). Yet, perhaps the most serious objection to including eye-related hallucinations in the PAD model is not the stretched importance of complex hallucinations but the amputation of their simple counterparts. All visual hallucinations, whether simple or complex, relate to phasic increases in activity within visual cortex, the difference between the two categories being the location of the activity increase. For example, activity increase in the human colour centre V4 will result in the hallucination of a "simple" formless coloured blob, whereas activity increase a few centimetres anterior to V4, in object-specialised cortex, will result in the hallucination of a "complex" object (ffytche et al. 1998). An important weakness of the PAD model is that it is forced to make an arbitrary distinction between these different cortical loci and their related hallucinations, amputating from its remit cortical areas underlying the simple hallucinations which typify those found in eye disease.

There are other features of visual hallucinations which require amputation for eye and visual pathway disease to fit the PAD model. In eye and visual-pathway disease, visual hallucinations, whether simple or complex, tend to resolve over time, with 60% of patients with visual hallucinations related to eye disease being hallucination-free at 18 months (Holroyd & Rabins 1996) and almost all patients with visual hallucinations related to visual-pathway infarcts being hallucination free within weeks (Kölmel 1985). Such patients do not develop elaborate delusional explanations for the experiences and typically gain insight into their hallucinatory nature even if, initially, some believe them to be real. These visual hallucinations are invariably silent and are not interspersed with hallucinations in other sense modalities. This overall clinical picture is sufficiently characteristic that exceptions to it point to the presence of other, non-ophthalmic causes for the hallucinations. Contrast this with the clinical picture found in the dementias, Parkinson's disease and schizophrenia. Here the visual hallucinations tend to persist or progress with time (see, for example, Goetz et al. 2001b in the context of Parkinson's disease) and are typically associated with insightless, delusional explanations. The visual hallucinations in these conditions tend to be associated with other sense modalities, either simultaneously (e.g., seeing and hearing the hallucination) or on different occasions (e.g., visual hallucinations interspersed with auditory hallucinations). Indeed, it is something of a psychiatric axiom that visual hallucinations in schizophrenia never occur without auditory hallucinations, either as separate hallucination events or as simultaneous, multimodality hallucinations. The PAD model is forced to ignore these striking clinical differences to allow the visual hallucinations of one set of conditions to sit comfortably with those of another.

Without stretching and amputation, what seems to emerge from the visual hallucination evidence taken as a whole is two distinct, overlapping syndromes.[1] One syndrome consists of predominantly simple hallucinations which resolve with time, occur with insight and without delusions, and are purely visual. The second consists of predominantly complex hallucinations which persist over time, occur with delusions and without insight, and cross sensory modalities. Setting aside those conditions in which the visual cortex is stimulated directly (e.g., migraine and epilepsy), to a first approximation all clinical conditions in which visual hallucinations occur are associated with one or other of these syndromes: eye and visual-pathway disease to the first, core PAD conditions to the second.

The existence of two distinctive syndromes poses a significant challenge to PAD and other models that treat visual hallucinations as a single pathophysiological entity. It seems unlikely that two such very different symptom profiles could emerge from the same disordered mechanism.

Perhaps the time for unitary models of visual hallucinations has passed. If there are two syndromes of visual hallucinations rather than one, we need to broaden our explanatory accounts to allow for this dichotomy. One approach would be to include two distinct but interacting pathophysiological mechanisms into our models, each related to one of the two syndromes. Obvious candidates would be visual de-afferentation as underlying the first syndrome and PAD cholinergic dysfunction the second syndrome (ffytche 2004; 2005). If correct, such expanded, bipartite pathophysiological models have important implications. In the clinic, they suggest, unlike their unitary counterparts, that different types of visual hallucinations need different treatment strategies (ffytche 2004). In the laboratory, and perhaps more significantly, by providing a comprehensive account of the neural mechanisms of disordered conscious vision, such extended models take us a step closer to a neural account of visual consciousness.

NOTES

1. These syndromes are unrelated to those associated with eye disease described in Santhouse et al. (2000), which would be considered sub-syndromes of the first, predominantly simple hallucination syndrome described here.

Hallucinations and perceptual inference

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Abstract: This commentary takes a closer look at how "constructive models of subjective perception," referred to by Collerton et al. (sect. 2), might contribute to the Perception and Attention Deficit (PAD) model. It focuses on the neuronal mechanisms that could mediate hallucinations, or false inference – in particular, the role of cholinergic systems in encoding uncertainty in the context of hierarchical Bayesian models of perceptual inference (Friston 2002b; Yu & Dayan 2002).

Collerton et al. provide a compelling synthesis implicating cholinergic dysfunction in the aetiology of recurrent complex visual hallucinations (RCVH). Furthermore, they observe "that both sensory release and top-down activation are necessary, but neither in itself is sufficient to cause high rates of RCVH" (sect. 6.3, para. 3). This fits very comfortably with models of perceptual inference based on hierarchical Bayes, in which cholinergic mechanisms may balance bottom-up sensory evidence and top-down priors by encoding their relative uncertainty or precision. In short, cholinergic dysfunction may result in a failure to properly integrate sensory information and prior expectations. In what follows, I try to explain how this might happen.

Perceptual inference is the same as statistical inference and

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