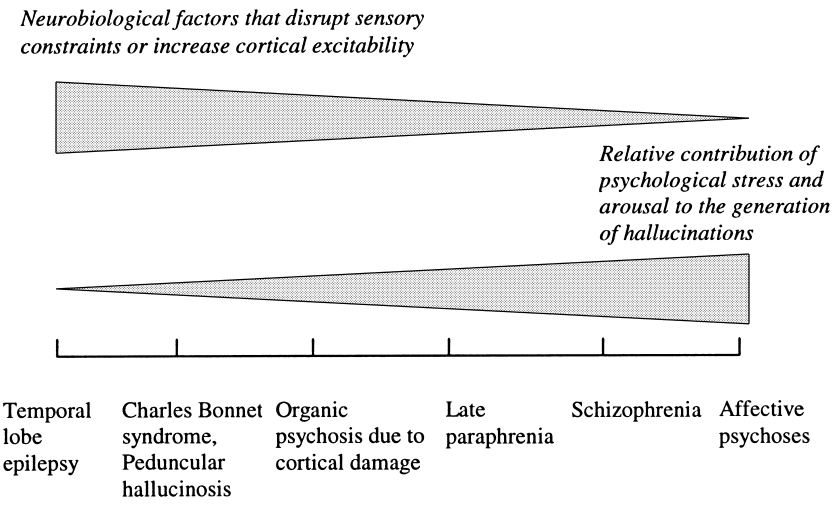

Figure 5. Disorders that feature hallucinations could be placed along a continuum, depending on the balance between neurobiological and psychological factors that contribute to the generation of hallucinations. Mediated by general arousal or attentional mechanisms, psychological factors may contribute to pathological activation of thalamocortical circuits alongside any disruption of sensory constraints. Alternatively, or in addition, pathological lesions may cause cortical hyperexcitability and increase the cortical drive on the thalamus. Psychological concerns are unimportant in the generation of hallucinations in temporal lobe epilepsy and Charles Bonnet syndrome, although attentional factors may shape their content. In schizophrenia and late paraphrenia, increased attention and arousal under psychological stress may be crucial for the generation of hallucinations, with a relatively moderate biological vulnerability given in the form of reticular thalamic nucleus dysfunction or peripheral hearing impairment. In affective psychoses, there may be a minimal biological predisposition to hallucinations.

hallucinations, the Charles Bonnet syndrome, late paraphrenia and schizophrenia, and perhaps plays a role in Parkinson's disease and Alzheimer's dementia. A disorder of serotonergic raphe nuclei, as may be the case in peduncular hallucinosis, may also cause global disinhibition in specific thalamic nuclei.

Neurobiological and psychological aspects are essential and complementary in understanding the nature of hallucinations, particularly in mental illness. In patients with a predisposition to underconstrained perception, *verbal* hallucinations may develop at times of heightened arousal and increased attention to *social* stimuli. In musical hallucinations and the Charles Bonnet syndrome, psychological disturbances are less important, although – in the absence of effective sensory constraints – attentional mechanisms may still shape thalamocortical self-organisation and determine the content of the perceptual experience. In hallucinations resulting from cortical lesions, intrinsic cortical hyperexcitability may complement attentional mechanisms in generating corticofugal input to thalamic relay cells. In hallucinations resulting from temporal lobe seizures, attentional mechanisms are irrelevant (Fig. 5).

In conclusion, the notion that normal perception and hallucinations are essentially equivalent, both being manifestations of intrinsic thalamocortical resonance in sensory areas, and the consequential reappraisal of the relationship between sensory input and physiological processes underlying perception, may provide a framework for the integration of neurobiological and clinical findings relating to hallucinations in schizophrenia and other disorders.

Open Peer Commentary

Underconstrained perception or underconstrained theory?

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Abstract: Although the evidence remains tentative at best, the conception of hallucinations in schizophrenia as being underconstrained perception resulting from intrinsic thalamocortical resonance in sensory areas might complement current models of hallucination. However, in itself, the approach falls short of comprehensively explaining the neurogenesis of hallucinations in schizophrenia, as it neglects the role of external attributional biases, mental imagery, and a disconnection between frontal and temporal areas.

According to the theory proposed by Behrendt & Young (B&Y), hallucinations and normal perception are essentially equivalent, both being manifestations of intrinsic thalamocortical resonance in sensory areas. Their approach is presented as an alternative to existing cognitive models of hallucination, which are criticized because they seem to suggest that the absence of a correspondence to external reality (which is thought to characterize self-generated mental events) has to be complemented by a conviction of their external origin to yield hallucinations. However, as the authors rightly point out, it is doubtful that thoughts, inner speech, or retrieved

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memories can be transformed into experiences with perceptual qualities just by virtue of their misattribution to an external origin.

Unfortunately, in their critique, the authors neglect a large body of evidence in favor of the external attribution bias hypothesis (for reviews, see Bentall 1990 and Seal et al. 2004). Any comprehensive account of the neurocognitive basis of hallucinations in schizophrenia should incorporate these findings. Nonetheless, we agree with B&Y that an external attribution bias cannot explain the sensory characteristics of hallucinations. However, attempts to address this issue have been extensively reported in the literature. Therefore, it is surprising that the authors seem to be unaware of the *imagery hypothesis* of hallucination. This is arguably the first scientific hypothesis of hallucinations: Sir Francis Galton proposed, more than a hundred years ago, that hallucinations result from an increased vividness of mental imagery (Galton 1883). He also devised introspective questionnaires to test his hypothesis and concluded that the evidence was in favor of his hypothesis. Indeed, an approach that focuses on mental imagery may have considerable explanatory power, as mental imagery has been shown to share important characteristics with bottom-up perception (Kosslyn 1994).

Most previous studies that investigated the imagery hypothesis of hallucinations (e.g., Mintz & Alpert 1972; Roman & Landis 1945; Starker & Jolin 1982) relied on subjective and introspective scales of imagery vividness, which may explain the inconsistent results reported in those studies. However, a number of recent studies have assessed the relationship between mental imagery and hallucination with objective, behavioral measures (Böcker et al. 2000; Evans et al. 2000;). Evans et al. (2000) used only auditory measures and failed to find differences between 12 hallucinating and 7 nonhallucinating patients. Böcker et al. (2000) assessed imagery and perception in the auditory and visual modalities in 13 hallucinating and 19 nonhallucinating patients. Consistent with Evans et al. (2000), no between-group differences were observed. However, regarding within-group comparisons, Böcker et al. observed a stronger influence of imagery on perception in the auditory compared to the visual modality in patients with auditory hallucinations. That is, imagining a stimulus enhanced subsequent detection of that stimulus to a larger extent in an auditory task than in an analog visual task. This significant difference was not evident for the nonhallucinating group. Moreover, in a recent cognitive neuropsychiatric case study (Aleman et al. 2002), we observed a modality-specific imbalance between imagery and perception in a patient with continuous auditory hallucinations, compared to five nonhallucinating patients. Specifically, whereas control subjects always performed better on a perception condition than on an imagery condition of the same task (the perception-superiority effect), this patient showed the reverse effect (imagery > perception) in the auditory but not the visual modality.

More recently, we investigated performance of 77 subjects on multiple behavioral measures of auditory and visual mental imagery and perception, and also on a measure of reality monitoring (Aleman et al. 2003). Our approach to the behavioral assessment of imagery-perception relations started from the assumption that, in normal conditions, mental images are less vivid; that is, they have fewer sensory and contextual characteristics than percepts. Kosslyn et al. (1999) and Aleman et al. (2000) recently provided evidence for this assumption in nonpsychiatric samples. According to the theory of Johnson and Raye (1981), increased vividness of images will make them less distinctive from percepts, which may lead to reality-monitoring errors. To test the imagery hypothesis, we assumed that a reduced distinctiveness of mental images with respect to bottom-up generated percepts would be observable differences in performance on tasks of imagery-perception relations. Therefore, we investigated whether patients with hallucinations would show smaller differences in performance on imagery and perception conditions of the same task, after controlling for nonspecific attentional variables. Comparisons were made between performance of schizophrenia patients with ($N = 22$) and without ($N = 35$) hallucinations and matched normal comparison subjects ($N = 20$), after controlling for attentional factors. No differences emerged on any of the mental imagery measures or on reality monitoring accuracy. This suggests that there is no stable disposition toward abnormal mental imagery associated with hallucinations. However, for patients with active hallucinations ($N = 12$), hallucination severity correlated positively with a measure of imagery-perception interaction in the auditory modality. Although preliminary, this finding is consistent with recent theoretical proposals in which hallucinations have been suggested to result from a larger influence of top-down sensory expectations (imagery) on conscious perception, and is therefore consistent with the approach taken by B&Y.

Top-down generated images can be confused with bottom-up percepts when there is a disconnection between prefrontal areas (involved in consciously generating an image) and temporal areas (involved in decoding the image, e.g., "image inspection" or "listening to the inner voice"). Evidence that such a disconnection is present in schizophrenia, and moreover, is associated with the occurrence of hallucinations, is accumulating at a rapid rate (Hubl et al. 2004; Lawrie et al. 2002; Shergill et al. 2003; Silbersweig & Stern 1996). In contrast, there is a lack of empirical data from patients with schizophrenia supporting the thalamocortical model proposed by B&Y. For example, a study by McKay et al. (2000) failed to find basic sensory impairment in patients with hallucinations as compared to patients without hallucinations, whereas deficits in perception of complex auditory stimuli were associated with hallucinatory status.

Nevertheless, we concur with B&Y that subcortical mechanisms may play an important role in the neurogenesis of hallucinations, as is apparent from studies reporting brain activation patterns during hallucinations (Shergill et al. 2000). Therefore, although the evidence remains tentative at best, the conception of hallucinations in schizophrenia as being underconstrained perception resulting from intrinsic thalamocortical resonance in sensory areas might complement current models of hallucination. However, in itself, the approach falls short of comprehensively explaining the neurogenesis of hallucinations in schizophrenia.

Underconstraint and overconstraint in psychiatry

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Abstract: Hallucination lies at an intriguing border between psychiatry and philosophy. Although Behrendt & Young (B&Y) tie their proposal to Kantian transcendental idealism, other philosophical positions are equally consistent. Cognition is underconstrained by reality not only in hallucination but also in autism and dreaming. Sensory underconstraint is insufficient to encompass schizophrenia. There is also a breakdown in integrative capacity on the cognitive side. From a wider clinical perspective than schizophrenia, there can be underconstraint or overconstraint in sensory and cognitive functionalities.

The problematic of hallucination brings psychiatry and philosophy up close. What adamantly *is* for the hallucinating patient, does not appear to the observer. This is a bona fide Being in itself, despite being hallucinated. What appears, the phenomena that onrushing experience discloses, is no basis for ontology, given the honest conviction of the hallucinator, who finds himself already thrown amidst a world in which, for example, voices may emanate from the shut-off television. And what if the non-schizophrenic world, too, is *maya*, illusion? Could non-schizophrenics in some deep ontological sense be hallucinators too? After all, Descartes meditating, seated by the fire, could find no certain truth that he was not dreaming it.

Behrendt & Young (B&Y) explicitly tie their theory of brain functioning to Kantian "transcendental idealism." "The world that we see around us is internally created and a fundamentally sub-

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