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## DISCUSSION COMMENTARIES

# COGNITIVE MODELS AND BIOLOGY OF AUDITORY HALLUCINATIONS

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Auditory verbal hallucinations (AVHs), the perception of external speech in the absence of a stimulus, are a cardinal feature of schizophrenia. In the past, the neural basis of these experiences was not amenable to direct investigation, and most research concentrated on developing a cognitive model that would help in explaining this puzzling phenomenon. The hope was to define these cognitive models in terms of the underlying brain regions. The advent of functional neuroimaging provided the ability to investigate the neural basis of these phenomena; both directly, by imaging while subjects were experiencing auditory hallucinations, and indirectly, by examining putative cognitive processes in both normal subjects and patients prone to hallucinations (see David, 1999, for review).

## Cognitive models

Various hypotheses have been proposed suggesting the cortical mechanisms relevant to the experience of auditory hallucinations (AH). 'Organic' conditions associated with auditory hallucinations tend to converge on the temporal lobes (Penfield and Jasper, 1954; Perez, Trimble, Murray and Reider, 1985). However, brain stimulation experiments have failed to provide a convincing model of 'functional' auditory hallucinations, as the complex auditory verbal hallucinations characteristic of schizophrenia are rarely evoked (Penfield and Perot, 1963). A study by Feinstein and Ron (1990) found no association between any particular site of brain pathology and psychotic experiences in patients seen at a specialist neurological hospital. Other theories have concentrated on inner speech. Frith and Done (1988) suggested that there are two types of stimuli: the first is external and has an impact via sensory organs, and the second is internally generated from planned or willed action. They believe that the internal monitoring of willed action is defective in psychotic illness, so that, in the case of AH, patients experience internally generated thoughts as externally generated. Similarly, David (1994) has hypothesized that AH may arise from a disturbance between the auditory analysis system ('inner ear') and phoneme output ('inner voice'). Bentall (1990) considers that the ability to discriminate between external and internal, or real and imaginary, sources is a metacognitive skill; he hypothesized that some individuals undergo a 'criterion shift' making them liable to interpret internal sources of stimulation as external. This notion is supported by evidence that hallucinating patients make hasty and overconfident judgements about the source of their perceptions (Heilbrun and Blum, 1984; Alpert, 1985), and that when their voice is distorted as part of a feedback process, they are more likely than normals to attribute the voice to another (Cahill, Silbersweig and

Frith, 1997). Slade and Bentall (1988) presented a five-factor theory of hallucinations, incorporating many of the ideas discussed above. They proposed that defective monitoring and reality discrimination may not be the sole factors in psychological explanations of AH, emphasizing the potential contribution of arousal, cognitive deficits, environmental stimulation, reinforcement and the patient's expectations. It seems that misattributions tend to be non-random and that it is the more unsavoury inner-speech acts which tend to be repudiated. Although somewhat neo-Freudian, this aspect of the affective content of hallucinations has been neglected by many hallucinations researchers and surprisingly does not draw much comment from Woody and Szechtman, despite their intention to bring affect into the centre of their theory.

## Imaging auditory hallucinations

Non-invasive neuroimaging has enabled the functional activity associated with AVHs to be examined, allowing the theories discussed above to be tested. McGuire, Shah and Murray (1993) first used single photon emission tomography (SPET) to study the areas activated during auditory hallucinations. Patients with schizophrenia were scanned twice, first while actively hallucinating and then at a later date when not hallucinating. The results showed increased blood flow in the left inferior frontal region (Broca's area) during hallucinations, thus implicating the language production areas in the experience of AH. Other studies, mainly using positron emission tomography (PET), implicated the anterior cingulate gyrus (Cleghorn et al., 1992), the left temporal cortex (Suzuki, Yuasa, Minabe, Murata and Kurachi, 1993), the left parahippocampal region (Liddle, Friston, Frith, Hirsch, Jones and Frackowiak, 1992) and the thalamus, striatum and cerebellum (Silbersweig et al., 1995). A striking feature of more recent functional magnetic resonance imaging (fMRI) studies of patients is that, while they were experiencing auditory hallucinations, they have shown activation in the left frontal and temporal cortex (David et al., 1996; Dierks et al., 1999; Shergill, Brammer, Williams, Murray and McGuire, 1999a), subcortical structures (Shergill et al., 1999a) and a prominent activation in the right temporal cortex (Woodruff et al., 1997; Dierks et al., 1999; Shergill et al., 1999a), suggesting that auditory verbal hallucinations involve several different neural regions. In sum, the temporal cortex seems to be more consistently implicated in AVHs than other candidate regions. Most of this work on patients does rely on them indicating accurately and reliably the onset of hallucinations in the absence of other objective criteria, a methodological weakness which is, at present, insurmountable and which also pertains to work using hypnosis.

## Imaging cognitive processes

There has also been an increased interest in neuroimaging of cognitive processes that may be involved in auditory hallucinations. Auditory verbal imagery, the process of imagining speech, bears some resemblance to auditory hallucinations in that the subject experiences another person's speech in the absence of an external stimulus. However, while normal inner speech seems to be experienced mainly in the first person (Hulbert, Happe and Frith, 1994), auditory hallucinations in schizophrenia are usually in the second or third person (Nayani and David, 1996). The reason for this apparent disparity between the grammatical forms of inner speech and auditory

hallucinations is unclear. McGuire and colleagues compared neural activation during auditory verbal imagery in patients with schizophrenia and normal controls, involving sentences with an affective content similar to true AVHs (for example, 'you are stupid'). While all subjects activated the left inferior frontal region, patients with a predisposition to verbal hallucinations showed less activation in the left temporal cortex and the supplementary motor area compared with both patients with no history of AVHs and with controls (McGuire et al., 1995, 1996a). Thus, this supports the notion that auditory hallucinations are related to defective monitoring, as opposed to generation, of inner speech (McGuire et al., 1995). Furthermore, the pattern of the activation observed when healthy volunteers imagine another person talking to them is remarkably similar to that seen during auditory hallucinations, with activation in the inferior frontal, anterior cingulate, premotor, and lateral temporal cortex (McGuire, Silbersweig, Murray, David, Frackowiak and Frith, 1996b, Shergill, Bullmore, Simmons, Brammer, Murray and McGuire, 1999b).

## Hypnotically induced auditory hallucinations

The article by Woody and Szechtman (2000) describes an experiment to use hypnosis to study the nature of hallucinations and test the results within two different cognitive models, and speculates about the links between hypnosis and reality distortions in mental illness. From the description of the aims of the study it is difficult to know why the authors did not carry out a study to examine auditory hallucinations per se, rather than through hypnosis. This approach merely serves to include additional ill-defined confounding variables such as the level of hypnosis, as well as differences in characteristics between the hallucinating and non-hallucinating subjects. Nevertheless, given the complexity of hallucinations and their evanescent properties it is perfectly valid and indeed desirable that different approaches are used to study them. Hopefully, convergence from distinct methodologies will ultimately provide real answers as to the origins of AVHs. For example, the study of pharmacologically induced hallucinations may provide insights into their biochemical origins. However, we assume that the authors do not think that hypnosis and hallucinations share a common (cognitive) basis (unlike say, conversion disorders). Hallucination-like experiences may in fact have widely disparate origins.

These issues aside, Woody and Szechtman found that the right anterior cingulate gyrus was active in hypnotized hallucinating, compared with hypnotized non-hallucinating, subjects when they were hearing a repeated sentence aloud and when they believed they were hearing the same. They interpret the data as demonstrating that this region is critical in experiencing hallucinations. They report that non-hallucinating subjects showed greater activation of the right temporal cortex during hearing and 'hallucinating' conditions, compared with baseline and imagery, and differed significantly from the hallucinating group. They suggest that activation of this area is not a sufficient basis for hallucinations. Further data demonstrate more extensive temporal cortical and right anterior cingulate gyrus activation in the hallucinating, compared with the non-hallucinating, group during the hearing condition.

The anterior cingulate gyrus has been implicated in two previous studies of auditory hallucinations, although, as the authors mention, in a more dorsal part than in their study. The studies investigating auditory hallucinations both reported that there were also other significant regions of activation. The most parsimonious interpretation

of their finding is likely to be due to the focusing of attention externally in response to the command to listen to speech. The more one attends to an auditory cue, the more activation one would expect in both the attentional system and in the auditory cortex; both these regions are more prominently engaged during the hearing task in the hallucinating group. The argument that the anterior cingulate region may be involved in emotional valence is weakened by the fact that the sentence that subjects hallucinated was not emotionally salient.

## Familiarity and knowing versus perception and reality

The further elaboration of how one could develop a 'sense of knowing' and the links with philosophy and hypnosis are interesting but scientifically tenuous. There are few data addressing this question directly, but the awareness of knowledge is largely a characteristic of one's memory. A recent study of recognition memory that addresses a similar issue – that of familiarity in recall – suggests that there is a dissociation between recognizing paired objects through remembering their pairing, and recognition of paired objects through a sense of familiarity, in patients with schizophrenia (Danion, Rizzo and Bruant, 1999). In fact, the recollection of a conscious experience led to a faulty memory of items being tested, contrary to the performance of normal subjects, while the sense of familiarity or knowing without conscious recollection led to improved recognition of items. It is possible that this familiarity may be a common feature in allowing an individual to accept 'impossible' events such as hallucinations and delusions. However, with regard to hallucinations and delusional beliefs, there is a cogent argument that can be made for these delusional beliefs developing in response to hallucinatory experiences rather than vice versa.

There are limitations in our ability to scientifically evaluate awareness, and Woody and Szechtman make an attempt towards improving that understanding. It would be fascinating to repeat their study with patients with schizophrenia who are in remission, using hypnosis to induce symptoms during neuroimaging. Would hypnosis induce the same neural network as evident in the acute illness period?

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