

Auditory verbal hallucinations and dysfunction of the neural substrates of speech

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Abstract

Objective: to evaluate the neural substrate of auditory verbal hallucinations (AVH), the correlation between AVH and subvocal speech (hereafter SVS), and the relationship between speech and AVH. **Method:** we reviewed the papers found by an electronic literature search on hallucinations and speech. The review was extended to the papers cited in these publications and to classical works. **Results:** there is no conclusive evidence of structural abnormality of the speech perception area in hallucinating schizophrenic patients. However there is evidence of electrophysiological abnormalities of the auditory and speech perception cortices. Functional imaging data are inconsistent, yet point to the left superior temporal gyrus as one of the neural substrates for AVH. There is also evidence that SVS could accompany the experience of AVH. **Conclusion:** there is evidence that dysfunction of brain areas responsible for speech generation is a fundamental mechanism for generating AVH in schizophrenia. It results in a secondary activation of Wernicke's area (speech perception) and Broca's area (speech expression). The first leading to the experience of hallucinations, and the second, eventually, gives rise to a variable degree of vocal muscle activity detectable by EMG, and/or faint vocalizations detectable by sensitive microphones placed at proximity of the larynx. Direct stimulation or disease of Wernicke's area produces AVH without SVS. © 2001 Elsevier Science B.V. All rights reserved.

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1. Introduction

Auditory verbal hallucinations (AVH) refer to the experience of perceiving speech in the auditory modality without corresponding external stimuli. Up to 70% of schizophrenic and a variety of psychiatric and neurological patients suffer from AVH. Under-

standing the neural correlates of AVH is important for the clarification of the pathophysiology of schizophrenia and related illnesses and an important insight into brain function. Some advocate that AVH reflect an auditory dysfunction (Mckay et al., 2000), but most relate this type of hallucination to a speech disorder (Frith and Done, 1988; Hoffman, 1986). Accumulating evidence from lesion and functional imaging studies indicate that speech generation is supported by a distributed neural network involving areas such as the supplementary motor area (SMA), left premotor

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area, left dorsal prefrontal area, thalamus, Broca's and Wernicke's areas (Sadato et al., 1998), and left inferior frontal gyrus, left superior and posterior middle temporal gyri (Herholz et al., 1996). The roles of each of these brain regions are not clear but it is established that Broca's and Wernicke's areas subserve speech motor execution and perception, respectively.

In this study we examine the evidence correlating AVH with the abnormality of the speech perception area and with the motor component of speech or subvocal speech (SVS). Contrary to the theory suggesting that hallucinating patients are virtually hearing their SVS, which is activating the auditory system just as external speech (Gould, 1950; Beck and Kinsbourne, 1987; Frith and Done, 1988), we suggest that both the motor (SVS) and perceptive (AVH) components are related to a central process, most likely speech generation. This hypothesis is consistent with current neuropsychiatric theories implicating inner speech in the pathogenesis of hallucinations and draws support from multiple methodologies.

2. Method

Extensive searches of the world literature were carried out using Medline (between 1965 and 2000) and PsychInfo (between 1972 and 2000). Publications in English and French were included. The search strategy included papers cross-referenced for the following combinations of key words: 'speech' and 'hallucinations', 'hallucinations' and 'temporal lobe', 'hallucinations' and 'temporal lobe and MRI', and 'hallucinations' and 'emission computed tomography'. The review was extended to relevant cited papers and to relevant classical works such as those of Penfield, Piaget, and Vygotsky.

Papers included in the review were: (1) theoretical papers about the relation between speech and verbal hallucinations and about the mechanisms of auditory hallucinations; (2) postmortem studies in schizophrenia; (3) structural imaging studies in schizophrenia when hallucinations are correlated to brain regions; (4) functional imaging studies of hallucinations including PET, SPECT, and fMRI; (5) electrophysiological studies including EMG, EEG, and stimulation

studies; and (6) single case reports. Statistical significance criteria and sample size were specified for all the structural and functional imaging studies.

Papers discussing other types of hallucinations and reviews were excluded. To limit the focus on speech and onward processes, theoretical and experimental papers discussing primary auditory processes were not included.

A total of 118 publications were selected. The review focused on the neural substrates of AVH, the correlation between AVH and SVS, and theoretical relationship between speech and auditory hallucinations.

3. Results

3.1. Neural correlates of VH

3.1.1. Postmortem studies

A recent review article (Heckers, 1997) of postmortem studies in schizophrenia, where AVH is common, showed that many studies report cytoarchitectural abnormalities in the prefrontal cortex, the anterior cingulate gyrus and the superior temporal gyrus (STG). Other brain regions such as the hippocampus and entorhinal cortex were implicated as well (Waddington 1993; Longson et al., 1996; Benes et al., 1998). In one study (Falkai et al., 1995), the volume, anterior–posterior diameter, and the surface area of the planum temporale (posterior part of the superior temporal gyrus) were measured. A smaller left–right asymmetry coefficient (left larger than right in normals) with respect to the first two measures was found in patients with schizophrenia.

Molecular biological abnormalities have been reported in the left superior temporal gyrus. A study (Yang et al., 1998) showed down regulation of G protein isotype q-alpha as measured by immunoreactivity. The authors attributed the finding to chronic stimulation of G_q alpha-coupled receptors. Other biological abnormalities were found in the temporal lobe but not limited to the speech area. These included a reduction in the concentrations of D2 receptors in the supragranular layers and elevation in the granular layer (Goldsmith et al., 1997), deficits in neuropeptide Y and cholecystokinin concentration (Gabriel et al., 1996), decrease in cholecystokinin (CCK) mRNA

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