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# THE NEUROPSYCHOLOGY OF HALLUCINATIONS

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*Abstract* - Hallucinations are a psychopathological phenomenon with neuropsychological, neuroanatomical and pathophysiological correlates in specific brain areas. They can affect any of the senses, but auditory and visual hallucinations predominate. Verbal hallucinations reveal no gross organic lesions while visual hallucinations are connected to defined brain lesions. Functional neuroimaging shows impairments in modality specific sensory systems with the hyperactivity of the surrounding cerebral cortex. Disinhibition and expansion of the inner speech was noted with impaired internal monitoring in auditory verbal hallucinations. The subcortical areas and modal-specific associative cortex and cingulate cortex are essential for the occurrence of hallucinations.

*Key words*: Hallucinations, neuropsychology, cognition, brain, internal speech

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## INTRODUCTION

Hallucinations are perceptual experiences in the absence of external stimuli (Allen et al., 2008). Although they can affect any sense, auditory and visual hallucinations are usually present in psychopathology. Neuroimaging studies show that the lesions are located in the pathways of the senses in which hallucinations occur, and that the surrounding cerebral cortex is hyperactive due to attempts to compensate the deficit. In the relevant perceptual structures inhibitory mechanisms would be damaged, which then prevent the normal activation of sensory information. Thus, in somatic hallucinations, areas involved in processing tactile information are activated: the primary somatosensory cortex, posterior parietal cortex and the thalamus (Shergill et al., 2001). In the case of verbal hallucinations, activity is registered in the speech area and/or primary auditory cortex of the middle or upper temporal gyrus, while during visual hallucinations, activity of the secondary visual cortex is observed. Functional studies have revealed that the reality distortions (hallucinations and delusions) in psychoses are associated with activation in the temporal lobe and posterior cingulate cortex (PCC) (Allen et al., 2008). Positron emission tomography (PET) studies during auditory verbal hallucinations showed activation of the speech area, especially the Broca center, and to a lesser degree, of the PCC which participates in attention, and of the left temporal cortex which mediates the auditory perception and memory (McGuire et al., 1993). Other PET studies have demonstrated also the activation, during the occurrence of hallucinations, of subcortical areas (bilateral thalami, right putamen and caudate) and parahippocampal gyrus bilaterally, the right PCC and left orbitofrontal cortex (Silbersweig et al., 1995). Subcortical structures could be the origin or might modulate hallucinations, while cortical activity would give them specific content.

The current hypothesis is that subcortical areas and the modal-specific associative cortex are essential for the occurrence of hallucinations (Allen et

al., 2008). The role of the PCC in the development of hallucinations could be the impairment of speech monitoring and false attribution of internal imagination to an outside source.

## COGNITION AND HALLUCINATIONS

The simplest experimental paradigm is to apply sensory stimuli in the same modality in which hallucinations occur. It assumes a mechanism of competition between internal and external stimuli for the same neural apparatus. Studies have demonstrated that in the presence of actual hallucinations there is a reduced capacity of processing external stimuli in the same modality (Allen et al., 2008). A model of auditory verbal hallucinations is proposed that interprets them as badly remembered episodic memories of speech. The source of hallucinations is not clear, but it is assumed that they are externalized thoughts caused by disorders of inner speech monitoring mediated by a normal premotor cortex, cerebellar cortex, lentiform nucleus, thalamus, hippocampus, temporal cortex and some other structures. The role of the anterior cingulate cortex (ACC) would be to monitor whether the source is external or internal.

A special area of study is the lateralization of the brain function in people with schizophrenia. An interesting hypothesis is posed by Julian Jaynes (1976) on the "bicameral brain" where one hemisphere (right) gives orders and the other (left) listens and executes orders. This Latin term means "consisting of two chambers" and is used in contemporary language for two legislative or parliamentary chambers. According to the bicameral theory, up to around 1000 BC, humans used the right hemisphere to process the supernatural voices of "gods" and "demons" (actually hallucinations), and the left one to produce speech. People of that era were not self-conscious and could not, for example, distinguish between the living and the dead, hence the cult of ancestors, embalming and the like. The collapse of bicameralism occurred during the second millennium BC, due to the great migrations and growing social complexity that required changes in the human mind towards a greater flexibility, introspection and abstraction. Societies that did not evolve in this direction disappeared (the ancient Maya, the Middle Kingdom of ancient Egypt and others). In schizophrenia, the brain processes are again bicameral. These patients experience auditory hallucinations as commands in the same way the ancient people, according to the theory of bicameralism, received "God's" commandments. Studies have shown that patients with schizophrenia indeed have reduced cerebral lateralization. Results are still controversial. The latest studies have shown bilateral activation of Broca's area instead of only left side activation that brings new life to the theory of mind bicameralism (Weiss et al., 2006). The reduction of lateralization even correlated with the intensity of hallucinations. Another assumption is that in patients lacking adequate ACC-left upper temporal lobe connection there is an erroneous impression that inner speech has an external source.

Allen and colleagues proposed a neuroanatomical model of hallucinations (Allen et al., 2008). According to this model, there are abnormalities in the gray and white matter in the areas that mediate conscious perception. Hyperactivation of secondary sensory areas is in the center of the disorder. In auditory hallucinations the left secondary and primary auditory cortex is activated during erroneous perception. The term over-perceptualization signifies the assumption that there are exaggerated perceptions based on an increase in the spontaneous activity of the perceptual cortex. In addition, significantly larger areas of the brain are included, of which some have a monitoring role: reduced gray matter of temporal cortex, increased activation of the subcortical centers, reduced control by the dorsolateral prefrontal cortex, impaired activation of the centers involved in emotional attention (ACC), reduced activity of the supplementary motor cortex and cerebellum that are also thought to be involved in the process of monitoring and control (Allen et al., 2008). This is a "top-down" neural network, which is impaired in every type of hallucination and thus creates the false experience that internal stimuli are external, that they have a commanding character which is against the patient's will, and where hallucinations are also emotionally charged. Disor-

der in hallucinations has the opposite, bottom-up flow. Excessive activity in the secondary and possibly primary sensory areas of the cortex leads to perception in the absence of the object of perception, which is magnified by inadequate monitoring of the ventral ACC, prefrontal, premotor and cerebellar cortex that all contribute to the false feeling that the source of perception comes from the outside. Strengthened links between, for example, the cingulate cortex and sensory areas may lead to their abnormal activation. These processes are closely linked to emotional components mediated by parahippocampal gyrus, amygdala, insula, cingulate and orbitofrontal cortex.

## AUDITORY VERBAL HALLUCINATIONS

In psychiatric disorders with auditory verbal hallucinations no gross structural disorders can be detected. Patients with schizophrenia and auditory verbal hallucinations show, in some studies, a reduced volume of the auditory cortex in the upper temporal gyrus in magnetic resonance imaging (MRI), proportional to the hallucination's intensity and enlarged ventricles (Barta et al., 1990). Disinhibition of the inner speech was noted with impaired internal monitoring. Related to this is the atrophy of the right prefrontal cortex, which undermines the fronto-temporal control mechanisms. Homotopic regions of the brain are linked with reciprocal inhibitory pathways, so a disorder on one side can lead to disinhibition of the opposite side (Allen et al., 2008). Voxel MR volumetry showed that the decrease of left Heschl's gyrus was associated with hallucinations, while reduction of the left planum temporale was associated with delusions (Sumich et al., 2005).

Other functional MRI (fMRI) studies have shown that larger areas of the brain may be more active during auditory hallucinations (Shergill et al., 2000): the lower frontal and insular cortex, cingulate cortex, bilateral temporal cortex, right thalamus and the lower colliculus, left hippocampus and parahippocampal cortex. Activation of the left lower frontal cortex and right middle temporal gyrus was observed 6-9 seconds before the patients gave a sign that he was hallucinating, when the activity became primarily bitemporal and at the left insula. This sequence suggests that the inner speech area is activated first, following by the speech perception area.

Studies in people with schizophrenia have shown that during the occurrence of verbal hallucinations there is increased active of the primary auditory cortex, Broca's area and the middle temporal lobe, while in healthy individuals there is no activation of the primary auditory cortex during normal inner speech (Allen et al., 2008). The auditory verbal hallucinations are most frequently connected with disturbances of the perisylvian speech area and ACC that has a monitoring role (Allen et al., 2008). A possible explanation is that abnormal activation of the primary auditory cortex interferes with internal speech, giving the impression that this is a real external sound. Some other studies did not confirm the primary auditory cortex activity during hallucinations. A problem arises with brain activity caused by MR sound and the activity of pressing the button with which the patient indicates the occurrence of hallucinations. Also, brain activity changes with the anticipation of hallucinations.

According to modern concepts, based on the research of inner speech and methods of brain activation, auditory verbal hallucinations are caused by impairment of the transition from condensed to extended inner dialog (Jones and Fernyhough, 2007). People usually hear two or three different voices, and 96% perceive that the voices speak to them (Leudar et al., 1997). The voices can advise the person, require or forbid certain actions. The inner speech in healthy subjects activates the left inferior frontal lobe and right temporal cortex. Auditory verbal hallucinations are also connected with left lower frontal lobe activity (Jones and Fernyhough, 2007).

Cognitive neuropsychology considers verbal hallucinations in schizophrenia a disorder of the monitoring of inner speech where one's own thoughts are experienced as other people's. Inner speech activates the left inferior frontal cortex, but also the parietal cortex and the supplementary speech area, partly the analogous areas of the right hemisphere, which

is probably due to the processing of prosody. It is assumed that the verbal motor cortex sends signals to Wernicke's area through the fasciculus arcuatus so that the speech to follow is produced by the person herself in order to alleviate the response of the verbal auditory cortex. It was found that the lateral temporal cortex is active when a person imagines he hears someone else's speech. People with impairment of the lateral temporal cortex have difficulty in imagining someone else's speech (Zatorre and Haplern, 1993). In patients in remission of schizophrenia there is a possible defect in these areas but the findings are not consistent (Jones and Fernyhough, 2007). The pathological activation of these areas may be caused by a disruption of normal ACC control activity. The cingulate cortex is activated during tasks that require a special mental effort, including the presumed consequences of actions and regulation of consciously perceived conflicts.

Inner speech can be defined as a subjective phenomenon when a person talks to him- or herself, creating an auditory-articulatory image of speech without vocal production (Levine et al., 1982). Close to inner speech is verbal thinking. For experimental purposes subvocal repetition of given sentences is used. From the developmental point of view this fits into the theory of Vygotsky (Vygotsky, 1934/1987), according to which inner speech represents the endpoint in which, during the early development, external speech gradually internalizes and finally forms verbal thinking. The transitional phase is the so-called "private speech" when the child talks to him or herself in the form of dialogue (for which there is experimental evidence). Private speech often has a dialog form of mutual exchange of information (Jones and Fernyhough, 2007). Vygotsky recognized the social origin of higher mental processes. According to him, every mental function appears twice in development: first as interpsychological, i.e. as the communication between two or more persons, and secondly as intrapsychological, or an internalized version of the original function (Vygotsky, 1931/1997). For example, an interview between child and adult becomes the basis of the later inner speech that has a regulatory function as verbal thinking. This regulatory function is related to behavior and cognition. Children often tell themselves what they have to do using their own private speech (Luria, 1961).

Inner speech may have two forms, expanded and condensed (Jones and Fernyhough, 2007). The expanded form retains the characteristics of an external dialogue, while the condensed inner speech loses linguistic characteristics and becomes thinking in pure meanings (Vygotsky, 1934/1987). According to the Fernyhough model (2004), the internalization process takes place in four stages: the first level is the external dialogue; the second (private speech) as a child's speech to himself (still as an form of external speech); the third is internalized expanded speech (the initial phase of internalized speech), and the fourth stage is condensed inner speech generated by a syntactic and semantic reducing of the speech of the previous phase. According to Vygotsky, inner speech and thinking are neither identical nor completely separated but can be represented as two circles that partially intersect (Vygotsky, 1934/1987). Expanded inner speech may be delusionally externalized in patients with auditory verbal hallucinations. The similarities are obvious: the most common is the form of dialogue and the voices dictate what is to be done.

In fMRI activation experiments, the inner speech is not always caused by activation of the same areas. This might be a reflection of different requirements, namely, expanded inner speech necessitates a broader cortical activation than condensed inner speech. The occurrence of hallucinations could be understood as a re-expansion of condensed inner speech (Fernyhough, 2004).

## COMPLEX VISUAL HALLUCINATIONS

Lesions associated with complex visual hallucinations are located in the visual pathways (especially the occipital cortex) and the ascending reticular activating system, including the brain stem and thalamus. Serotoninergic receptors are supposed to be of the utmost importance.

Complex visual hallucinations are not only a pathological phenomenon but may occur in some healthy individuals during the initial stage of sleep. Hallucinations during drowsiness before sleep are called hypnagogic hallucinations, and can occur in one-third of normal individuals.

Unlike the auditory hallucinations that are common in functional psychiatric disorders, complex visual hallucinations are usually caused by organic disorders of the brain in conditions such as narcolepsy, delirium tremens, peduncular hallucinosis, Parkinson's disease, Lewy body dementia, migraine coma, Charles Bonnet syndrome (visual hallucinations of the blind), schizophrenia, epilepsy and states induced by hallucinogenic substances (Manford and Andermann, 1998). Epilepsy cases are caused by direct irritation of the cerebral cortex in areas that integrate complex visual information. Findings point to the posterior parietal cortex and temporal association areas. Disturbances of the visual pathways impair the information input so that the brain produces hallucinations as a form of cortical release phenomena.

Damage to the brain stem origin of visual hallucinations is most probably related to cholinergic and serotonergic systems and is associated also with sleep disorders. Peduncular hallucinations were first described by Lhermitte in 1922 in patients with infarction of the upper brain stem. Lesions of this type usually involve reticular formation and the parts of the thalamus associated with it and isolated pulvinar lesions. Seemingly isolated midbrain peduncle lesions are often not really isolated, affecting other structures as well, such as the reticular nucleus of the thalamus and substantia nigra pars reticulata. Examinations of patients with Alzheimer's disease using MRI showed the presence of visual hallucinations in geniculate and calcarine lesions with the preservation of white matter tracts (Lin et al., 2006).

Visual hallucinations can last several hours; most of them remit after several weeks but can persist for years. They occur mostly at night, but they can persist during the day. Sometimes visual hallucinations are accompanied by hallucinations of a tactile and acoustic type. These hallucinations can last from several seconds to more than 15 minutes. The content is diverse, pleasant or unpleasant, so the patients can experience fear, and they can be of different degrees of complexity. Patients can see people and animals, sometimes diminished, so called Lilliputian, hallucinations. Images can be static or moving. Rarely sound phenomena occur also. Neuropsychological testing may not show any deficits.

Patients with Charles Bonnet syndrome show activation of the visual cortex including the ventral occipital lobe on fMRI (Ffytche et al., 1998). The content and form of hallucination match the active areas. Thus in the case of activation of the color center in V4, hallucinations are in color and if they do not include this center they are black and white.

## CONCLUSION

Hallucinations are an important psychopathological phenomenon with clear neuropsychological, neuroanatomical and pathophysiological correlates in specific brain areas. Functional neuroimaging can help discern brain areas and functional systems and their role in the phenomenon of hallucinations. These findings can give new insights in brain function de norma and in pathology.

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