

Auditory Hallucinations as a Separate Entity

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How do we normally hear voices?

An object that produces sound in space results in a vibrations which in turn moves the tympanic membrane (eardrum). Attached to the center of the tympanic membrane is the handle of the malleus connected to the incus and the stapes (ossicular system that conducts sound to the middle ear). The faceplate of the stapes lies against the membranous labyrinth where passes to the inner ear (cochlea) sound. The tensor tympani and stapedius muscles contract in opposing direction to reduce conduction in case of loud sounds. This function is important to remove share in background noise and allow concentration on communicable sounds. It also decreases one's hearing sensitivity to his own speech which is activated by collateral signals transmitted to these muscles the same time the brain activates voice mechanism.

The cochlea is a system of coiled tubes, the scala vestibuli separated from the scala media by the Reissner's membrane, with the basilar membrane lying between it and the scala tympani. On the surface of the basilar membrane lies the organ of corti which contains hair cells. The scala media is filled with endolymph (high K, low Na), while the scala vestibule and tympani are filled with perilymph (low K, high Na). The bodies of hair cells pass in the perilymph and their tops project in the endolymph. An electrical potential exists all the time to sensitize the hair cells in response to vibration of the basilar membrane. This in turn synapses at the cochlear nerve endings and neurotransmitter glutamate is released.

Normal auditory pathway in the central nervous system

The cochlear nerve endings relay on the spiral ganglia of Corti which send axons into the cochlear nerve that enter the dorsal and ventral cochlear nuclei in the upper medulla. At this point, all the fibres synapse, the second order fibres pass ipsilateral and opposite side to the superior olivary nucleus. Through the lateral lemniscus the fibres terminate on the inferior colliculus in the pons. Then the pathway synapse in the medial geniculate nucleus in the thalamus where it proceeds to the auditory cortex (primary auditory cortex in the superior gyrus of the temporal lobe and auditory association area).

How do we psychologically respond to loud voices?

Collateral fibres pass from the auditory tracts into the reticular activating system (activate arousal) to project upwards into the cerebral cortex and downwards to the spinal cord (associate motor action). Others pass to the cerebellum (coordinate verbal and motor activity). The importance of these tracts is to activate the nervous system in response to loud voice.

Can we perceive sounds in the absence of external stimulus?

A significant feature of the auditory pathway is that low rates of impulse firing continue even in the absence of sound all the way from the cochlear nerve fibres to the auditory cortex with the continuous movement of the basilar membrane.

How can we discriminate frequencies of sounds?

In the auditory cortex, specific neurons are excited by high frequency sounds posteriorly, while others are excited by low frequency sounds anteriorly. On stimulating the cochlea with one frequency, inhibition of signals caused by sound frequencies on either sides of the stimulated one occurs (phenomena of lateral inhibition). This occurs in different types of sensation transmission in the brain (hearing, vision).

How do we get the meaning of the sound?

The auditory association area in the parietal portion associates sound information to each other and with sensory information from other areas of the cortex.

How can we identify the source of sound and its direction?

We can determine the direction from which sound originates by two mechanisms:

1. The time lag between the entry of sound into one ear and the opposite one.
2. The difference in intensities of sounds between two ears.

Comparing signals in both cortices are required for localization. This function starts at the superior olivary nucleus to the primary auditory cortex.

The forebrain plays an important role in many aspects of sound localization behavior¹. The Auditory Archistraitum (AAR) and the area surrounding it are also essential for auditory spatial memory and for mediating changes in gaze to and guiding movements toward, remembered auditory stimuli. Consistent with their electrophysiological properties, behavioral experiments have demonstrated that both the auditory thalamus and the area surrounding including the AAR are involved in auditory orienting behavior². These behaviors are mediated, in part, by efferent projections of this area to midbrain and brain stem structures that are known to be involved in movement control³. Also the midbrain sound localization pathway has been well described in both birds and mammals⁴. The role of the forebrain and midbrain auditory space processing pathways can be differentiated based on the nature of the task. More specifically, it is proposed that the forebrain pathway primarily participates in voluntary shifts of gaze, such as those that require access to memory stores and that the midbrain pathway participates in all saccadic orienting movements to sounds and is particularly important for short latency, reflexive orienting movement. This division of function can be seen in behavioral studies in which various forebrain areas have been inactivated. For example, after ablation of the AAR and the area surrounding it or the auditory thalamus, barn owls can orient and fly toward auditory targets that are still present in the environment but cannot orient to a remembered target². Similarly, after ablation of the auditory cortex, mammals still can orient their gaze immediately toward auditory targets but cannot respond to a sound which no longer present in the environment⁵.

How can we neglect sounds and pay attention to others?

Inhibitory pathways go retrograde from the auditory cortex to the cochlea downwards. Stimulation of certain points in the olivary nucleus can inhibit specific areas of the organ of corti thus reducing their sound sensitivity to unwanted voices.

What controls our emotional reactions to sounds?

The lateral nucleus of the amygdala (LA), a key component of the fear conditioning circuitry, receives a rapid but relatively impoverished auditory input from the auditory thalamus and a slower but richer input from the auditory cortex. Synaptic transmission in both pathways depends on L-alpha-amino-3-hydroxy-5-methyl-4-isoxazole propionate (AMPA) receptors, whereas transmission in the thalamic pathway also depends on the involvement of N-methyl-D-aspartate (NMDA) receptors which allow the LA to integrate signals in the two pathways during the acquisition and expression of conditioned fear reactions during auditory perception⁶.

How do auditory hallucinations occur?

Auditory Hallucination (AH) occurs with a lifetime prevalence of 10% to 15% in persons without neuropsychiatric diseases⁷. In 1838, Esquirol was the first to formulate the

concept of a brain-based origin of hallucinations. In 1900, Wernicke hypothesized that a pathologic activation of the primary acoustic cortex was the basis of the experience of external sensory stimulation during AH⁸. In 1919, Kraepelin had postulated that AH was a result of temporal lobe abnormalities⁹. This hypothesis was supported by severe abnormalities in the left temporal lobe in the brains of patients with schizophrenia, found post mortem. The present hypotheses of the generation of AH, considering recent neuroimaging findings, propose that AH derive from inner speech misidentified as external by means of defective self-monitoring¹⁰. Imaging studies suggest that the superior temporal lobe is altered in patients with Auditory Hallucination, associating dysfunctions within brain regions that are important for language and auditory processing¹¹.

Why do we hear hallucinations in the mother language?

Functional imaging studies in schizophrenic AH revealed involvement of the frontal motor and premotor speech areas (the Broca area and supplementary motor area) and temporo-parietal speech areas (the Wernicke area) that are necessary for the decoding and encoding of language¹⁰. In right-handed individuals, the speech-relevant areas are predominantly located in the left hemisphere, which may be related to the fact that the left hemisphere also appears to be more functionally involved in the generation of AH than the right hemisphere¹².

Anatomical connection between the language and auditory areas

The arcuate fasciculus is divided into

1. A medial part that contains longer fibers connecting the lateral frontal cortex with the dorsolateral parietal and temporal cortex.
2. A lateral part, with shorter U-shaped fibers connecting the frontoparietal, parietooccipital and parietotemporal cortex¹³; fibers originate in the prefrontal and premotor gyri (mainly the Broca area) and project among others posteriorly to the Wernicke area. The lateral part of the arcuate fasciculus provides a pathway by which frontal speech-production areas can influence auditory and speech perception areas during overt and inner speech. The importance of the arcuate fasciculus in language is underlined by results from neurological findings in aphasia research. A disruption of the arcuate fasciculus leads to a disturbance of the neuronal connections from the frontal Broca area to the temporal Wernicke area, which results in a disturbance of the stream of speech¹⁴.

One link between AH and inner speech is the common clinical observation that the content of AH is often closely related to the content of the patient's own thought and sometimes is even reported as thoughts becoming loud. It can be hypothesized that high white matter directionality in the lateral part of the arcuate fasciculus in AH is associated with

high connectivity (increased activation) between distributed language and auditory areas. These alterations may have a developmental origin and may contribute to an understanding of how internally generated language is perceived to be generated externally. The aberrant connections may lead to abnormal activation in regions that normally process external acoustical and language stimuli. That accounts for these patients' inability to distinguish self-generated thoughts from external stimulation¹⁵.

Functional MRI study, demonstrated an increase of neuronal activity in the primary auditory cortex and language-related areas during hallucinations in patients with schizophrenia¹⁶.

Auditory hallucinations involve activation of the person's own memory that is influenced by his education and culture:

Electrophysiological recordings in human¹⁷ indicate that neuronal activity in the temporal cortex is powerfully modulated by vocalisation. This modulation can precede articulation by hundreds of milliseconds, suggesting that it is related to the intention to speak (rather than articulation per se) and mediated by the direct anatomical connections linking areas that generate and perceive speech, within the left inferior frontal and bilateral temporal cortices respectively. Contemporary cognitive models propose that auditory verbal hallucinations are derived from inner speech that the person has misidentified as 'alien' through defective self-monitoring¹⁸.

Auditory verbal imagery – imagining another person's speech – implicitly involves both the generation and monitoring of inner speech and in healthy volunteers is associated with activation in the left inferior frontal cortex and the temporal, parahippocampal and cerebellar cortex¹⁹. A positron emission tomography (PET) study of auditory verbal imagery in participants with schizophrenia who were prone to auditory hallucinations revealed normal activation of the left inferior frontal gyrus, but differential activation of the left temporal cortex, compared with both people with schizophrenia but no history of hallucinations and healthy volunteers²⁰. A more recent functional magnetic resonance imaging (fMRI) study using a similar paradigm in another group of hallucination-prone participants again demonstrated normal activation of the left inferior frontal gyrus and attenuated activation of the right temporal cortex²¹. In addition, there was relatively attenuated activation in the parahippocampal and posterior cerebellar cortex bilaterally. A common feature of lateral temporal, parahippocampal and cerebellar cortex is that these regions are all implicated in cognitive self-monitoring. Lesion and neuroimaging studies suggest that the cerebellum acts as a 'comparator' in both motor and verbal tasks, comparing intended with actual performance and modulating cerebral cortical activity appropriately²². The hippocampus has also been proposed as the 'comparator' in experimental models of cognitive self-monitoring²³, whereas the lateral temporal cortex has been more specifically implicated in the monitoring of inner speech¹⁰.

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