Hallucinations are involuntary sensory experiences that are perceived as emanating from the external environment, in the absence of stimulation of relevant sensory receptors. Hallucinations can occur in a variety of contexts but are perhaps most striking and debilitating in the context of schizophrenia, in which they are generally experienced as real and emotionally significant, are related to concurrent delusions, and represent a manifestation of psychosis. Hallucinations can occur in any sensory modality and can involve multiple modalities. Auditory hallucinations are the most common in schizophrenia and other illnesses that are traditionally termed psychiatric, and visual hallucinations are the most common in illnesses termed neurological. Hallucinations can be described at multiple levels of analysis, including cognitive, neurochemical, computational, and social/psychological. This article presents a functional neuroanatomic approach to hallucinations describing and analyzing them in terms of disorders of sensory input and subcortical (mindbrain/thalamus) and higher brain regions, including cortical sensory, limbic, and frontal regions. It touches also on treatment considerations.

Disorders of Sensory Input Associated with Hallucinations

Hallucinations produced by disorders of the peripheral sensory system appear to result from ongoing cortical sensory processing in the setting of degraded or absent sensory input. In this setting, perception may be dominated by the cortically generated expectations (top-down processing) that interact with peripheral input (bottom-up processing) in the generation of normal perception. Hallucinations of this sort are frequently seen in the visual system, in which case they are termed the Charles Bonnet Syndrome. These are usually vivid, colorful representations of people, animals, trees, and so on that appear smaller than normal (Lilliputian) and are often engaged in activities. Notably, the individuals experiencing these hallucinations are aware that they do not represent reality, and generally they have no strong emotional reaction to them. Similar hallucinations can occur in conditions such as stroke that involve destruction of primary visual cortex, as this region provides input to unimodal association areas involved in the generation of complex hallucinations. When lesions are limited to one hemisphere, hallucinations may occur only in the affected contralateral visual field.

In the somatosensory system, a striking example of hallucinations caused by disordered sensory input occurs in the phantom limb syndrome, in which an amputated limb continues to be experienced as present, able to move in space, and able to feel pain or tingling. In the auditory system, individuals with peripheral dysfunction (including deafness) can develop complex hallucinations such as music or voices, or simple hallucinations such as ringing, buzzing, or isolated tones.
Mindbrain/Thalamic Disorders Associated with Hallucinations

Hallucinations similar to those produced by peripheral lesions can occur with lesions of the upper mindbrain and adjacent thalamus. Originally attributed to a lesion in the mindbrain peduncular region, they remain known as peduncular hallucinations. Like Charles Bonnet hallucinations, they are usually vivid visual hallucinations, frequently of people or animals, sometimes Lilliputian, often with activities. Unlike those produced by peripheral lesions, peduncular hallucinations are generally associated with disturbances in sleep and arousal and may at times be interpreted as real.

These disturbances in sleep and arousal provide clues to the mechanisms by which hallucinations are generated by midbrain and thalamic lesions. Frequency-specific oscillations in thalamocortical circuits have been associated with the temporal binding of perception and with dreaming—a normal condition involving the absence of external stimuli. During the awake state, thalamic relay nuclei faithfully transmit inputs to the cortex; during dreaming, they do not. Neurotransmitters, notably acetylcholine and serotonin, play an important role in initiating this switch in relay mode. Abnormalities of cholinergic and serotonergic transmission brought on by disease, medication, or drug use are frequently accompanied by hallucinations. Similarly, transitions between states of sleep and wakefulness are associated with hallucinations, usually in the setting of sleep disorders. These are generally multimodal, vivid, and emotionally charged. Common examples are the feeling of being about to fall into an abyss or be attacked, of being caught in fire, or of sensing a presence in the room. Hallucinations in the settings of delirium and sedative drug withdrawal are also associated with disturbances in sleep and arousal. Such hallucinations should be distinguished from illusions, which are misinterpretations of actual sensory stimuli.

Disorders of Higher Brain Regions Associated with Hallucinations

Hallucinations, such as those that occur in migraine, epilepsy, and schizophrenia, may also be associated with primary pathology at higher levels of the brain. In recent years, studies employing functional neuroimaging techniques have implicated a number of higher brain regions in the generation of hallucinations, corresponding to their form, content, and setting.

Cortical Sensory Activity Associated with Hallucinations

Regardless of the mechanism by which they are generated, hallucinations appear to be associated with activity in cortical sensory regions corresponding to their modality and complexity. The hallucinations previously described may be categorized as complex or formed. Noncomplex hallucinations are referred to interchangeably as simple, unformed, or crude. In the visual system, these are known as photopsias. These occur most frequently with migraines, and they may also be seen at the onset of partial seizures, for the first few days following an infarction of the central visual system, and with disorders of visual input. Photopsias may consist of colored or colorless glittering spots or of black and white zigzag patterns known as fortification lines. They often occur unilaterally, but they may fill the entire visual field. Simple hallucinations are believed to reflect activity in primary sensory or adjacent early unimodal association areas and to correspond, in form, to the area’s functional specialization. For example, colored photopsias would be associated with activity in occipital subregions involved in color processing.

Complex hallucinations are associated with activity in sensory association areas, with or without involvement of primary sensory cortex. As with simple hallucinations, their form and content correspond to the location of activity. For example, in a functional neuroimaging study of an individual experiencing ongoing auditory-visual hallucinations while schizophrenic, we detected activations in association

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cortices mediating higher-order visual perception, speech perception, and intermodal processing.

### Limbic/Paralimbic Activity Associated with Hallucinations

The study just cited included other subjects with schizophrenia, all of whom experienced frequent auditory hallucinations. Although each had a somewhat different pattern of sensory cortical activation, perhaps reflecting differences in the form and content of their hallucinations, group analysis revealed a significant pattern of common activations in thalamic (see earlier discussion), limbic, and paralimbic areas—regions involved in the processing of emotion and memory and their integration with sensory information. Just as abnormal activity in sensory cortex is correlated with the form and content of hallucinations, it is likely that aberrant activity in limbic/paralimbic regions gives rise to marked emotional significances of hallucinations in persons with schizophrenia.

Further evidence of a role for thalamic and limbic system dysfunction in the generation of schizophrenia symptoms is provided by postmortem, neuropsychological, electrophysiologic, and neuroimaging studies that reveal structural and functional abnormalities of thalamic and limbic regions in individuals with schizophrenia, including hyperactivity of temporal regions, left greater than right, associated with psychosis. Additionally, activity of the limbic system is closely interconnected with that of dopamine, a neurotransmitter implicated in the generation of hallucinations and delusions in schizophrenia, medication toxicity, and drug abuse. Recently, dysfunction in the glutamatergic excitatory transmitter system has also been implicated. Hallucinations that arise in the context of severe emotional stress may also involve abnormal limbic activity.

Temporal lobe structures also play a role in the generation of hallucinations associated with epilepsy. The onset of partial seizures can be accompanied by simple hallucinations in any modality, reflecting ictal discharges in primary sensory areas, or by complex hallucinations reflecting discharges in limbic and sensory association areas. Olfactory hallucinations can also be seen in association with epilepsy. These complex hallucinations most often involve temporolimbic regions, including hippocampus and amygdala, which have the lowest seizure thresholds of all brain structures, as well as sensory association areas. Like the hallucinations seen in schizophrenia, these are often emotionally charged. Unlike those seen in schizophrenia, they are more often visual than auditory and are not usually believed by the person experiencing them to represent reality. Individuals who suffer from epilepsy over prolonged periods may also develop hallucinations between seizure episodes. These may resemble more closely those seen in schizophrenia, because they are frequently emotionally charged, accompanied by delusions, and believed to represent reality, and they are as often auditory as visual. As in schizophrenia, they appear to be associated with temporal lobe abnormalities, left more often than right.

### Frontal/Executive Activity Associated with Hallucinations

The lack of awareness that hallucinatory experiences do not correspond to reality is a striking feature of schizophrenia. In addition to temporal lobe abnormalities, numerous studies have revealed frontal dysfunction and abnormal frontotemporal connectivity associated with schizophrenia. The frontal lobes, in concert with interconnected regions, mediate the higher, more complex aspects of cognition, termed executive functions, that include judgment, insight, and self-monitoring. Although relevant studies have produced mixed results, there is evidence to suggest that frontal dysfunction may contribute to the inability of individuals with schizophrenia to identify the internal origin of their hallucinatory experience and its relation to their illness. Temporal lobe epilepsy may also be accompanied by executive as well as other forms of cognitive dysfunction, and by abnormalities of frontal activity.

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Treatment of Hallucinations

For hallucinations accompanying schizophrenia or other primary psychiatric disorders, medications that alter transmission of dopamine and related neurotransmitters (such as serotonin), termed antipsychotics, are the mainstay of treatment. In other contexts, the first step in the treatment of hallucinations is to address the condition that underlies their existence. Where this is impossible or ineffective, antipsychotic medications may be tried. However, these tend to be less effective in conditions that do not involve limbic, striatal, or dopaminergic pathology. Fortunately, hallucinations in the setting of sensory input disorders, where antipsychotics are least effective, are often less disturbing to those experiencing them, as previously described. Such hallucinations sometimes respond to carbamazepine, a medication used to treat a variety of neuropsychiatric conditions. When hallucinations are distressing and unresponsive to medication, psychological treatments, including cognitive-behavioral and supportive therapies, may be helpful. Future developments in the treatment of hallucinations are likely to be guided by the functional neuroanatomic approach, altering neurotransmission (via medications) or cortical activity (via techniques such as transcranial magnetic stimulation) in specific cerebral regions.

See also

Brain; Epilepsy; Schizophrenia.

Suggested Readings
