Visual Hallucinations

Clinical Occurrence and Use in Differential Diagnosis

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Visual hallucinations occur in diverse clinical circumstances including ophthalmologic diseases, neurologic disorders, toxic and metabolic disorders and idiopathic psychiatric illnesses. Their content, duration and timing relate to their cause and provide useful differential diagnostic information. Hallucinations must be distinguished from delusions and confabulation. A systematic approach to differentiating among hallucinatory syndromes may improve diagnostic accuracy.

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Visual hallucinations are common clinical phenomena that occur in a wide variety of ophthalmologic, neurologic, medical and psychiatric disorders. As many as 75% of delirious patients manifest visual hallucinatory phenomena,[1] and when hallucinations dominate the clinical presentation, a patient may be misdiagnosed as suffering from an idiopathic psychiatric disorder. Hallucinations must be differentiated from confabulations and from delusions—syndromes that are also characterized by patient reports that conflict with the experience of clinicians. When hallucinatory syndromes are recognized and their distinguishing characteristics considered, hallucinations may provide considerable differential diagnostic information. In this report we review the definition of visual hallucinations, discuss the clinical significance of hallucinations, review the value of hallucinations in localizing central nervous system (CNS) disturbances and describe the pathogenetic mechanisms responsible for visual hallucinations. A systematic approach to the differential diagnosis of hallucination-producing conditions is presented.

Definition

A visual hallucination may be defined as a visual sensory perception without external stimulation[2] or, more operationally, as a behavioral syndrome in which a patient claims to see something or behaves as if he or she sees something that an observer cannot see.[3] In contrast, delusions are abnormal beliefs that are endorsed by patients as real, that persist in spite of evidence to the contrary and that are not part of a patient's culture or subculture.[2] Hallucinations per se (sometimes called "pseudohallucinations")[4] are recognized as false sensory phenomena, but when they coexist with delusions, a patient may endorse them as real. Hallucinations by themselves are not evidence of psychosis. Confabulation is yet another syndrome where a patient's report is at variance with an examiner's experience. Confabulation involves fabricating facts or events and occurs primarily in patients with memory disturbances.[2] Confabulations typically occur in response to questions probing a patient's orientation or history and do not involve ongoing sensory experience.

Differential Diagnosis

Visual hallucinations can be produced by ophthalmologic diseases, neurologic disorders, toxic and metabolic disturbances, psychiatric disorders and a variety of miscellaneous conditions. Table 1 provides a classification of the causes of hallucinations and presents a framework for the systematic analysis of hallucinatory syndromes.

Among ophthalmologic diseases, total or partial blindness is commonly associated with visual hallucinations. The blindness may be a product of injury to the globe or may be the result of cataract formation, macular degeneration or retinal disease.[3,5-7] Hallucinations associated with blindness have been called "phantom vision" to express their similarity to the phantom-like phenomena following amputation of an extremity. Hallucinations occurring with blindness may be either formed or unformed, but formed hallucinations predominate.[5-7] Unformed hallucinations of ocular origin occur with retinal traction and with glaucoma. Retinal traction gives rise to brief unformed flashes of light as a result of retinal detachment (Moore's lightning streaks) or rapid ocular movement (flick phosphenes).[3,8] Acute glaucoma with a sudden increase in intraocular pressure may result in the appearance of hallu-

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ABBREVIATIONS USED IN TEXT
CNS = central nervous system
REM = rapid-eye-movement

cinated rainbows surrounding objects. The sudden occurrence of visual hallucinations in the elderly is known as the Charles Bonnet syndrome. Formerly considered an idiopathic disorder, the syndrome has been found to be associated with an occult ocular pathologic disorder in most cases.[9,10] Hallucinatory images arising from ocular disease must be distinguished from entopic phenomena such as opacities in the vitreous humor or seeing elements of one's own retinal circulation (Scheerer's phenomenon).[3,11]

Optic nerve disease gives rise to unformed visual hallucinations when the nerve is inflamed. Patients with optic neuritis frequently experience bright, transient flashes of light when moving their eyes horizontally.[12] This is particularly likely to occur or to be noticed by a patient in a darkened environment or when the eyes are closed. Patients with optic neuritis, optic nerve tumors or arteritis involving the optic nerves may occasionally experience unformed or semiformed hallucinations when startled by sound.[13]

Brain-stem lesions produce a unique type of visual hallucination syndrome known as peduncular hallucinosis. The hallucinations are a product of vascular, neoplastic or other structural involvement of the pons or midbrain and are usually accompanied by disturbances of the sleep-wake cycle and by cranial nerve palsies. The hallucinations typically occur in the evening and consist of geometric patterns, more complex kaleidoscopic scenes of landscapes, flowers, birds, animals or people or visions of miniature animals and beings. The patient may be amused or astonished by the hallucinations and may look forward to their occurrence.[14,15]

Hemispheric lesions may cause ictal hallucinations during the course of a seizure or they may produce "release" hallucinations associated with a visual field defect.[16] Release hallucinations occur with hemispheric infarctions, tumors or other destructive lesions of the geniculocalcarine pathways. The images are often complex regardless of the lesion location, consisting of complex visual patterns or identifiable images of objects, animals or people. They are usually located within the visual field defect, and they may be influenced by environmental factors such as opening, closing or moving the eyes. They are typically novel visual experiences rather than visual memories.[17,18] Release hallucinations are more common with right-sided than with left-sided lesions,[3] and the inciting lesion is often an infarction in the distribution of the right posterior cerebral artery.[17] In the acute stage, release hallucinations persist for as long as several hours per day; they become less frequent and less enduring in the resolution phase of the injury.[19] Figure 1 shows semiformed hallucinations described by a patient who sustained a right occipital infarction (Figure 2).

Ictal hallucinations may be difficult to distinguish from release hallucinations, but a few clinical features help to differentiate the two (Table 2). Ictal hallucinations tend to be brief, stereotyped visual experiences that are not necessarily associated with a visual defect and are frequently not lateralized within the visual field. Ictal hallucinations tend to be unformed when associated with occipital lesions and formed when associated with temporal lobe lesions. The hallucinations produced by temporal lobe foci occasionally consist of visual memories.[20,21] Consciousness may be altered during an ictal hallucination, and some patients have forced head and eye deviation at the time of the hallucination. Visual distortions—metamorphopsia, macropsia, micropsia—sometimes accompany ictal visual hallucinations of temporal lobe origin.[21]

Migraine is a well-known cause of visual hallucinations. Fortification spectra (zig-zag lines, often with an associated scotoma) are the most frequent type of hallucination, but patients who have migraines may experience every variety of hallucinatory image from simple unformed lines and spots to highly complex, formed scenes.[22] Visual distortions, including macropsia and micropsia, may also occur. Such sen-

TABLE 1.—*Causes of Visual Hallucinations*

- Ophthalmologic Diseases
  - Enucleation
  - Cataract formation
  - Retinal disease
  - Choroidal disorder
  - Macular abnormalities
  - Glaucoma
- Neurologic Disorders
  - Optic nerve disorders
  - Brain-stem lesions (peduncular hallucinosis)
  - Hemispheric lesions
  - Epilepsy
  - Migraine
  - Narcolepsy
- Toxic and Metabolic Conditions
  - Toxic-metabolic encephalopathies
  - Drug and alcohol withdrawal syndromes
  - Hallucinogenic agents
- Psychiatric Disorders
  - Schizophrenia
  - Affective disorders
  - Conversion reactions
- Miscellaneous Conditions
  - Dreams
  - Hypnagogic hallucinations
  - Childhood (imaginary companions)
  - Eidetic images
  - Sensory deprivation
  - Sleep deprivation
  - Hypnosis
  - Intense emotional experiences

TABLE 2.—*Features Useful in Distinguishing Ictal and Release Hallucinations Associated With Hemispheric Lesions*

Characteristics	*Ictal Hallucinations*	*Release Hallucinations*
Duration	Brief (usually seconds to minutes)	Persistent (hours)
Variability	Stereotyped content	May be stereotyped, variable or change slowly over time
Visual field defect	Visual field defect may or may not be present	Visual field defect usually present
Lateralization	May or may not be lateralized	Usually lateralized to side of visual field defect
Environmental influence	Little or no response to environmental influences	Frequently influenced by environmental factors, such as opening, moving or closing eyes
Content	May consist of a visual memory	Usually novel
	Tend to be unformed with posterior lesions and formed with temporal	May be formed regardless of lesion location
Associated findings	Consciousness often altered during or after ictal event; head and eye deviation common during hallucination	No associated ictal behaviors or alteration of consciousness

sory distortions have been called the "Alice-in-Wonderland" syndrome, after the tale by Lewis Carroll who called on his own migraine experiences to describe Alice's dramatic changes in size.[23]

Narcolepsy may produce visual hallucinations that are confined to those instants just as one is falling asleep (hypnagogic) or just as one is awakening (hypnopompic). A patient's level of arousal is usually depressed and the hallucinations have a dreamlike quality. The patients may see geometric patterns, landscapes, faces or figures, and there may be associated visual distortions. Hypnagogic hallucinations occur in 15% to 50% of persons with narcolepsy, are often accompanied by sleep paralysis and are a result of intrusions of the dreams of rapid-eye-movement (REM) sleep into wakefulness.[24]

Toxic and metabolic disorders are among the most common causes of visual hallucinations. The hallucinations may be the sole manifestation of the toxic encephalopathy or they may be one expression of a complex delirious state. Visual hallucinations are present in 40% to 75% of patients in metabolic encephalopathies associated with cardiopulmonary insufficiency, uremia, hepatic disease, endocrine disturbances, vitamin deficiency states and inflammatory and infectious diseases.[1] Hallucinations have also been induced by many drugs and toxins including stimulants, antiparkinsonian agents, antidepressants, analgesics and nonsteroidal anti-inflammatory agents, anticonvulsants, cardiovascular agents, antibiotics, hormonal agents and a wide variety of other compounds (Table 3).[1,25-55] In addition to the hallucinations induced during a period of drug ingestion, hallucinations may also be prominent in withdrawal syndromes when the use of alcohol, opiates or sedative-hypnotics is abruptly discontinued.[1] Hallucinogenic compounds routinely capable of producing hallucinations in the absence of delirium include LSD, psilocybin, psilocin, mescaline, harmine, amphetamines, phencyclidine hydrochloride and *Cannabis* derivatives.[25]

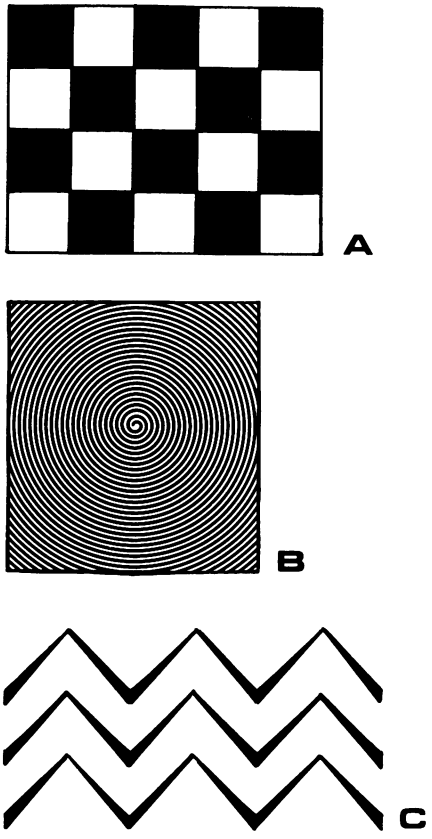

Figure 1.—Visual hallucinations described by a patient with right occipital infarction (computed tomographic scan shown in Figure 2). **A, B** and **C** images appeared sequentially during the first several weeks following the stroke.

Various idiopathic psychiatric disorders can produce visual hallucinations. Hallucinations associated with schizophrenia are primarily auditory in nature, but visual hallucinations occur as a minor symptom in from 24% to 72% of patients at some time during the course of their illness.[56,57] Similarly, auditory hallucinations are the principal type of hallucinatory experience reported by patients with affective disorders, but visual hallucinations are present in 10% of manic-depressive patients at the time of first admission and occur during the course of the illness in as many as 70%.[56,57] Hallucinations occurring with major affective disorders are usually mood congruent, reflecting either the guilty hopelessness of a depressed patient or the expansive grandiosity of mania. Visual hallucinations may also occur as a conversion symptom in patients with conversion reactions.[56,58]

Hallucinations are usually indicative of ocular, neurologic, metabolic or psychiatric disorders, but in some situations they may occur in normal persons; they are not inevitably the harbinger of a disease process. Visual hallucinations are not uncommon in children. Eidetic imagery—the ability to recall visual images with hallucinatory clarity—occurs in some children and less frequently in adults. Children may also have imaginary play objects and companions that they appear to visualize.[59] Dreams are common to all and are visual expe-

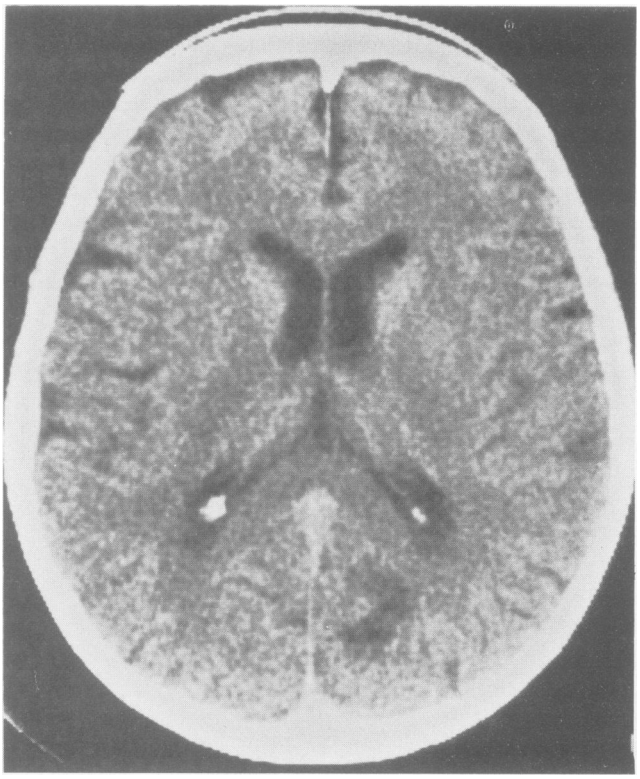

Figure 2.—A computed tomographic scan of a patient with release-type visual hallucinations following a right occipital infarction.

riences included in some definitions of hallucinations. In adults, visual hallucinations may occur during periods of sleep deprivation and sensory isolation or as hypnagogic phenomena unassociated with narcolepsy.[60-62] Hallucinations may also occur as a product of hypnotic suggestion and during the course of intense emotional experiences such as grief reactions. Some cultural groups appear to be more prone than others to hallucinate in stressful circumstances.[63,64] Historically, hallucinations have also occurred in a number of exceptional persons including Socrates, Joan of Arc, Mohammed, Luther, Pascal, William Blake, Bunyan, Napoleon, Raphael and Goethe.[65]

Phenomenology

Distinct varieties of hallucinations have been described and attempts made to correlate them with specific causes or localized regions of brain dysfunction. The most common generalization of this type is to associate unformed hallucinations with occipital lesions and formed hallucinations with temporal lobe injury. As noted above, this rule is most applicable to ictal hallucinations but has little validity for release hallucinations. The latter may be formed images regardless of the location of the lesion. Thus, the characteristics of hallucinations occurring as part of an epileptic seizure may provide important localizing information, whereas other clinical features must be used for localizing lesions associated with release hallucinations.

Not all hallucinations can be adequately described as either formed or unformed. Flashes of light or color are unformed, and complex figures such as animals, people and plants are formed, but many hallucinations have patterned features such as lattices, cones and spirals that are of intermediate complexity. Preliminary cortical stimulation studies suggest that unformed hallucinations are associated with activity of area 17, the primary occipital striate cortex; more patterned hallucinations are associated with dysfunction of area 18, the peristriate visual association cortex; bizarre apparitions such as disengaged objects and people occur with abnormalities of area 19, the more advanced visual association area, and complex remembered visual experiences correlate with medial temporal lobe disturbances.[66]

Lilliputian hallucinations—named after the experiences of Jonathan Swift's character, Gulliver, with the little people of the island of Lilliput[67]—are frequently given special attention in descriptive reports. The patients report seeing tiny but otherwise normally shaped people, animals and objects. Such diminutive figures, however, have been described in a variety of metabolic, neurologic, drug-induced and psychiatric conditions and appear to have no specific localizing or etiologic significance.[18,68,69]

Like Lilliputian hallucinations, autoscopy—the experience of seeing oneself as an hallucinated image—although particularly striking, is without specific focal or etiologic correlates. It has been reported with epilepsy, migraine, cerebral tumors, head trauma, infectious diseases, drug intoxication, schizophrenia and depression.[70,71] Autoscopy figured prominently in several well-known literary works including Edgar Allen Poe's *William Wilson*, Oscar Wilde's *The Portrait of Dorian Gray* and Dostoyevsky's *The Double*.[71]

Geometric "psychedelic" images consisting of checkerboards, spirals and funnels are characteristic of the hallucinations induced by hallucinogenic drugs but are also reported in cases of sensory deprivation and with structural insults to the brain.[60,72,73]

Palinopsia refers to the persistence or recurrence of visual images after the exciting stimulus has been removed.[74] A patient views an object, and the image is retained for 20 to 60 seconds after the gaze is redirected. The image may spontaneously recur for a period of up to several hours after the initial experience. Palinopsia is a special variant of release hallucination, occurring primarily in patients with visual field defects and right-sided posterior hemispheric lesions.[75]

Distinctions are usually made between hallucinations, where the perception is independent of a stimulus object, and illusions, where there is a distortion of an existing environmental stimulus. Clinically, however, the two phenomena frequently co-occur and have been reported together in pa-

TABLE 3.—*Drugs Associated With Visual Hallucinations*

- Hallucinogens
  - Dimethyltryptamine
  - Harmine
  - Ketamine hydrochloride
  - LSD
  - Mescaline
  - Nitrous oxide
  - Phencyclidine hydrochloride (PCP)
  - Psilocybin
  - Tetrahydrocannabinol
- Stimulants
  - Amphetamine
  - Cocaine
  - Methylphenidate
- Antiparkinsonian Agents
  - Amantadine hydrochloride
  - Anticholinergic drugs
  - Bromocriptine
  - Levodopa
  - Lisuride
  - Mesulergine
  - Pergolide mesylate
- Antidepressants
  - Amitriptyline hydrochloride
  - Amoxapine
  - Bupropion hydrochloride
  - Doxepin hydrochloride
  - Imipramine hydrochloride
  - Lithium carbonate
  - Phenelzine sulfate
- Anticonvulsants
  - Ethosuximide
  - Phenobarbital
  - Phenytoin
  - Primidone
- Cardiovascular Agents
  - Digitalis
  - Disopyramide
  - Methyldopa
  - Propranolol hydrochloride
  - Quinidine
  - Reserpine
  - Timolol
- Antibiotics
  - Antimalarial agents
  - Cycloserine
  - Isoniazid
  - Podophyllum resin
  - Procaine penicillin
  - Sulfonamides
  - Tetracycline
- Hormonal Agents
  - Levothyroxine sodium
  - Steroidal agents
- Analgesics and Nonsteroidal Anti-inflammatory Agents
  - Indomethacin
  - Nalorphine
  - Narcotic agents
  - Pentazocine
  - Phenacetin
  - Salicylates
- Miscellaneous Agents
  - Baclofen
  - Bromide
  - Cimetidine
  - Clonazepam
  - Diethylpropion hydrochloride
  - Disulfiram
  - Ephedrine
  - Heavy metals
  - Hexamethylamine
  - Metrizamide
  - Phenylephrine hydrochloride
  - Promethazine hydrochloride
  - Ranitidine
  - Solvents
  - Vincristine
  - Volatile hydrocarbons

tients with epilepsy, migraine, narcolepsy and in toxic and metabolic conditions.[23,24,64,72] Their occurrence in the same clinical disorders suggests that they may arise from similar neurophysiologic alterations.

Mechanisms of Hallucinations

Despite the wide variety of clinical circumstances in which visual hallucinations may occur, most cases can be attributed to a few basic pathogenetic mechanisms. A common situation in which hallucinations emerge involves reduced visual input. Thus, hallucinations occur with sensory deprivation, enucleation, cataract formation, retinal disease, choroidal disorder or macular degeneration and with optic nerve lesions. Reduced visual stimulation is also present in the release hallucinations associated with hemispheric damage involving the geniculocalcarine pathways and producing homonymous visual field defects. Deprivation of visual input produces visual hallucinations with similar characteristics, and West proposed a "perceptual release theory" suggesting that diminished sensory input results in a release of spontaneous activity within CNS structures normally responsible for mediating perceptual phenomena.[76] The disinhibited sensory activity induces hallucinations within the appropriate sensory sphere. This basic mechanism might also account for hallucinations associated with diminished arousal in narcolepsy, hypnotic and trance states, confusional disorders and some idiopathic psychiatric disturbances as well as those associated with clinically unmistakable visual-sensory loss.

Abnormalities of sleep and dreaming also account for many cases of visual hallucinations. Hypnagogic and hypnopompic hallucinations of narcolepsy occur when the dreams of REM sleep intrude into wakefulness.[24] Dream intrusion also appears to account for the visual hallucinations associated with withdrawal from alcohol and sedative agents, the long-term use of which suppresses REM sleep and produces a rebound of excessive REM activity during withdrawal periods.[77] A diurnal schedule of peduncular hallucinosis suggests that the syndrome may also be related to sleep and dream mechanisms.[14,15]

Ictal hallucinations represent a distinct pathophysiologic variety of visual hallucinations. Spontaneous or iatrogenic stimulation of the occipital cortex may produce unformed hallucinations and stimulation of the temporal cortex often produces formed hallucinatory images. The tendency for both ictal and release hallucinations to arise from lesions in the right hemisphere correlates with the nonverbal visuoperceptual specialization of the right brain.

Several mechanisms have been proposed to explain the hallucinations induced by hallucinogenic agents.[25] LSD is among the most powerful of hallucinogens and has effects on serotonergic and limbic system structures. It has both agonist and antagonist properties in serotonergic systems and may interfere with serotonin-dependent raphe nuclei that regulate REM sleep.[78] A role for the limbic system in LSD-induced hallucinations is suggested by the observation that LSD phenomenology is diminished following temporal lobectomy.[79] Noradrenergic and dopaminergic agents causing hallucinations also appear to exert their actions on limbic and brainstem structures, whereas hallucinations associated with taking anticholinergic agents and overdoses of other drugs are usually part of a delirious state.

Thus, most hallucinations can be correlated with a few basic mechanisms—perceptual release, ictal discharges, dream intrusion or neurochemical effects on brain-stem and limbic system structures.

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