LESSONS LEARNED AT THE INTERFACE OF MEDICINE AND PSYCHIATRY

The Psychiatric Consultation Service at Massachusetts General Hospital (MGH) sees medical and surgical inpatients with comorbid psychiatric symptoms and conditions. Such consultations require the integration of medical and psychiatric knowledge. During their thrice-weekly rounds, Dr. Stern and other members of the Psychiatric Consultation Service discuss the diagnosis and management of conditions confronted. These discussions have given rise to rounds reports that will prove useful for clinicians practicing at the interface of medicine and psychiatry.

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Visual Hallucinations: Differential Diagnosis and Treatment

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Have you ever encountered a patient who reported isolated visual hallucinations but did not have any other symptoms of delirium or psychosis? Have you wondered which medical and neurologic illnesses may present with visual hallucinations? Have you deliberated about how best to work up and treat patients with visual hallucinations?

If you have, then the following questions and answers should serve to frame the differential diagnosis of visual hallucinations and to explore the available options for diagnostic testing and treatment.

What Are Visual Hallucinations?

Hallucinations, defined as the perception of an object or event (in any of the 5 senses) in the absence of an external stimulus, are experienced by patients with conditions that span several fields (e.g., psychiatry, neurology, and ophthalmology). When noted by nonpsychiatrists, visual hallucinations, one type of sensory misperception, often trigger requests for psychiatric consultation, although visual hallucinations are not pathognomonic of a primary psychiatric illness.

Visual hallucinations have numerous etiologies. Here, we discuss possible mechanisms and offer a differential diagnosis of visual hallucinations, with an emphasis placed on conditions that arise in the context of medical and surgical illness. Treatment typically rests on the underlying etiology, so timely recognition and an understanding of causative mechanisms are crucial.

What Causes Visual Hallucinations?

Numerous hypotheses have been suggested to explain the genesis of visual hallucinations. These have been summarized and categorized by Asaad and Shapiro[1]: psychophysiologic (i.e., as a disturbance of brain structure), psychobiochemical (as a disturbance of neurotransmitters), and psychodynamic (as an emergence of the unconscious into consciousness). Visual hallucinations can be the result of all 3 processes, given the interplay among disturbances of brain anatomy, brain chemistry, prior experiences, and psychodynamic meaning.

To date, no single neural mechanism has explained all types of visual hallucinations; however, the similarity of visual hallucinations that are associated with seemingly diverse conditions suggests a final common pathway. Manford and Andermann[2] summarized 3 pathophysiologic mechanisms thought to account for complex visual hallucinations.

The first mechanism involves irritation (e.g., seizure activity) of cortical centers responsible for visual processing. Irritation of the primary visual cortex (Brodmann's area 17) causes simple elementary visual hallucinations, while irritation of the visual association cortices (Brodmann's areas 18 and 19) causes more complex visual hallucinations.[3] These data

are supported by both electroencephalographic (EEG) recordings and direct stimulation experiments.[2]

Lesions that cause deafferentation of the visual system may lead to cortical release phenomenon, including visual hallucinations.[4] Normal inputs are thought to be under the control of inhibitory processes that are effectively removed by deafferentation. It has been further suggested that deafferented neurons undergo specific biochemical and molecular changes that lead to an overall increase in excitability (similar to the denervation hypersensitivity seen in phantom limb syndrome experienced by amputees).[5]

A multitude of lesions can cause this loss of input and inhibit other cognitive functions.[6] Of note, visual hallucinations may be induced by prolonged visual deprivation. One study reported visual hallucinations in 10 of 13 healthy subjects blindfolded for a period of 5 days; this finding lends strong support to the idea that the simple loss of normal visual input is sufficient to cause visual hallucinations.[7]

Finally, due to its role in the maintenance of arousal, the reticular activating system has been implicated in the genesis of visual hallucinations. Lesions of the brainstem have led to visual hallucinations (as in peduncular hallucinosis). Further, visual hallucinations are common in those with certain sleep disorders, and occur more frequently in those who are drowsy. The observation that visual hallucinations occur more frequently in those who are drowsy (even in the absence of frank sleep pathology) suggests that the reticular activating system plays a role in visual hallucinations, although the precise mechanism has not yet been established.

Which Conditions Can Present With Visual Hallucinations?

Psychosis (schizophrenia/schizoaffective disorder). *The Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition (DSM-IV) lists hallucinations as a primary diagnostic criterion for various psychotic disorders (including schizophrenia and schizoaffective disorder).[8] Hallucinations may also be a feature of other psychiatric illnesses (including major depressive disorder and bipolar disorder) when they present with psychotic features. While the majority of hallucinations reported in primary psychotic disorders are auditory, they may also be visual, olfactory, tactile, or gustatory. Visual hallucinations have been reported in 16%–72% of patients with schizophrenia and schizoaffective disorder.[9] Mueser and colleagues[9] reported a prevalence of 16%, but then went on to examine the relationship between clinical variables and the type of hallucination. They found that the global severity of illness was significantly higher in patients with schizophrenia and visual hallucinations as compared to those without visual hallucinations. This finding may help explain widely varying estimates of prevalence of visual hallucinations in those with schizophrenia, as patients with more severe illness (e.g., those requiring long-term hospitalization) might be expected to experience more visual hallucinations than those with less severe illness.

Visual hallucinations in those with schizophrenia tend to involve vivid scenes with family members, religious figures, and animals.[10] Reactions to these visions can vary and include fear, pleasure, or indifference. The hallucinations are usually described as colorful and involving normal-sized people and objects; however, visual hallucinations of giants have also been reported.[11]

Delirium. Delirium, a syndrome that involves an acute disturbance of consciousness as well as a diminished ability to sustain attention, is caused by myriad medical conditions, metabolic disturbances, infections, drug effects, and intracranial processes. It is often manifest by symptoms (e.g., hallucinations and delusions) that are suggestive of a primary psychotic illness. Indeed, several studies have shown that in general hospitals, many patients seen by psychiatric consultants for sensory misperceptions were initially referred for evaluation of a primary psychiatric disorder (most often depression).[12,13] Among those with delirium, visual hallucinations are the most common type of hallucination.[14] In fact, Webster and Holroyd[14] reported psychotic symptoms in 43% and visual hallucinations in 27% of such patients. They also noted a strong positive correlation between visual hallucinations and the number of active somatic diagnoses.[14]

Delirium from alcohol withdrawal (i.e., delirium tremens) or stimulant intoxication (e.g., with cocaine or methamphetamine) is typically accompanied by visual hallucinations. Patients with these conditions often report seeing crawling insects, perhaps as a result of contemporaneous tactile disturbances.[15] A comparative study of the hallucinatory content of patients with schizophrenia and those with cocaine abuse found visual hallucinations of insects to be more frequently associated with cocaine intoxication.[16] Hallucinations due to drug intoxication or drug withdrawal vary in duration from being brief to being continuous; such experiences often contribute to agitation.[2]

Dementia. Dementia with Lewy bodies (DLB) is the second most common form of dementia after dementia of the Alzheimer's type.[17] Prominent symptoms include parkinsonism, visual hallucinations, and cognitive fluctuations. Visual hallucinations occur in more than 20% of patients diagnosed with DLB.[18] The visual hallucinations in DLB involve seeing objects move when they are actually still and seeing complex scenarios of people and items that are not present. Patients may have insight into their hallucinatory content. Visual hallucinations are an important clinical clue indicating that dementia may be associated with Lewy bodies rather than with another

subtype of dementia. Tiraboschi et al.[19] reported an 83% positive predictive value of visual hallucinations in distinguishing DLB from dementia of the Alzheimer's type. Harding and colleagues[20] reported a strong correlation between Lewy bodies located in the temporal structures (specifically in the amygdala and the parahippocampus) and well-formed visual hallucinations.

Visual hallucinations also occur in up to half of patients with Parkinson's disease.[21] These hallucinations are similar to those in patients with DLB, and can range from seeing a person or animal to seeing more complex, formed, and mobile people, animals, or objects.[22] Lippa and colleagues[23] agreed that DLB and dementia associated with Parkinson's disease are "more similar than different" and noted that the major difference is in the timing of symptom onset (with motor symptoms preceding cognitive decline in Parkinson's disease and vice-versa in DLB).

Posterior cortical atrophy is another neurodegenerative syndrome associated with visual hallucinations and parkinsonian symptoms. The chief pathologic features of posterior cortical atrophy are neurofibrillary tangles and senile plaques similar to those seen in dementia of the Alzheimer's type but with a distribution limited to the occipital and parietal lobes. Patients with posterior cortical atrophy may present with visual agnosia, anomia, apraxia, and features of Gerstmann syndrome.[24] Neuroimaging typically reveals atrophy of the bilateral occipital, parietal, and posterior temporal lobes.[25]

Charles Bonnet syndrome. The Charles Bonnet syndrome (CBS) involves the occurrence of visual hallucinations in the visually impaired. Any cause of visual impairment (including macular degeneration, glaucoma, cataracts, cerebrovascular disease, and tumors) may be associated with CBS. Charles Bonnet syndrome has been thought of as a nonpsychiatric cause of visual hallucinations; while this remains true, there is growing evidence that preexisting dementia or cognitive impairment may predispose a patient to the development of CBS.[26] Patients with CBS may avoid reporting their hallucinations for fear of being stigmatized as being mentally ill.

The visual hallucinations in CBS have been described as clear and detailed; they often involve people, faces, animals, and inanimate objects. While patients may initially be unaware that these images are indeed hallucinations, one of the hallmarks of CBS is the eventual development of insight regarding their visual hallucinations.[26] The visual hallucinations of CBS are thought to be a cortical release phenomenon. The strongest risk factors for CBS include bilateral visual impairment, declining visual acuity, cerebral damage, cognitive defects, social isolation, and sensory deprivation.[26] Not surprisingly, the best treatment for CBS is the improvement of vision. Antipsychotics and selective serotonin reuptake inhibitors have been used to treat CBS, but there is no clear evidence to support this approach.[27–29]

Anton's syndrome. Anton's syndrome is a rare condition in which patients with cortical blindness deny that they have visual loss. This presentation has been described as a combination of anosognosia and confabulation.[30] It may take some days before others around the patient realize that the patient has become blind. The problem may only be discovered when the patient is found running into or falling over objects while reporting odd or fantastical reasons why they did not see the object with which they collided.

The mechanism that underlies Anton's syndrome remains unclear, but it is thought to be related to infarction of the primary visual cortex (Brodmann's area 17) with preserved function of the visual association cortices. These patients may have alterations in emotional reactivity that predispose them to denial.[31] The prevalence of this syndrome is not known, but a study of 50 patients with cortical blindness showed that only 3 patients denied their blindness.[32] These patients were shown to have memory impairment and blindness, raising the possibility that they may have been unable to remember that they were blind. Another hypothesis suggests that there may be a synesthetic translation of the remaining senses into mental images that are perceived by the patient as vision.[33]

Seizures. Visual hallucinations caused by seizures have often been described as simple, brief, and consistent for each patient; they usually consist of small, brightly colored spots or shapes that flash.[34,35] Content of the hallucination may be distorted in size or it may suddenly change shape,[36] moving from a lateral field toward the center of the field of vision. Those that remain isolated in 1 visual field suggest seizure activity that originates on the contralateral side. Complex visual hallucinations due to seizures are thought to require the involvement of the visual association cortex.[2] Panayiotopoulos[35] reported a 4.6% prevalence of occipital seizures in patients with epilepsy, with nearly all occipital seizures involving visual manifestations.

Since occipital seizures are frequently accompanied by a postictal headache, they may be difficult to distinguish from migraines, creating confusion and the delay of appropriate treatment. Nonetheless, Panayiotopoulos reported that the symptoms of occipital seizures "are entirely different from migraine visual aura in their clustering of color, shape, size, location, movement, duration, and development."[35(p539)]

While neurologic literature indicates that visual hallucinations associated with seizures are simple, there is a growing body of work that describes complex, formed visual hallucinations as ictal, peri-ictal, and intra-ictal phenomena.[37,38] These symptoms may occur as part of a broader ictal psychosis that may feature delusions and

paranoia, and may be indistinguishable from a primary psychotic disorder, especially if the seizures are of the complex partial type. The occurrence of complex visual hallucinations as a symptom of epilepsy can be a point of disagreement between neurologists and psychiatrists.[39]

Migraines. Visual hallucinations associated with migraine headaches can be a classic aura of migraine as well as a less common manifestation (such as migraine coma and familial hemiplegic migraine). The prevalence of migraines in the general population has been reported as between 15%–29%.[40] Up to 31% of those with migraine have an aura,[41] and nearly all (99%) of those with an aura have visual symptoms.[42] The classic visual aura starts as a flickering, uncolored, unilateral zig-zag line in the center of the visual field that gradually progresses toward the periphery, often leaving a scotoma, that lasts less than 30 minutes (and almost always lasts less than 60 minutes). Variations of this classic picture (such as colored patterns) also occur. The simple visual hallucinations described above are the most common, but more complex hallucinations can occur in migraine coma and familial hemiplegic migraine.

Hadjikhani and colleagues[43] used functional magnetic resonance imaging (fMRI) to show that migraine aura is likely caused by spreading cortical depression. This phenomenon involves a brief period of hyperperfusion followed by a slow spreading wave of hypoperfusion. These events are thought to be due to a neuronal dysfunction rather than to a primary vascular event.[41]

Peduncular hallucinosis. Peduncular hallucinosis is characterized by visual hallucinations that arise following an infarct of the midbrain. It was originally described in 1922 by Lhermitte and has since been the topic of many case reports. The mechanism has been difficult to pinpoint since the literature features a wide variety of lesions. Lesions may involve the cerebral peduncles, but most often involve the reticular formation or its targets.[2,44] The hallucinations are thought to be a release phenomenon and often involve colorful, vivid scenes with people, animals, and other patterns.[44] The visual hallucinations usually start within a few days of the initial insult and resolve within a few weeks, but they may last for years. Each hallucination may last from minutes to hours, often occurring in the evening. Patients eventually develop insight into their hallucinations, and they may find them interesting or amusing.[2]

Sleep disturbances. Hypnagogic hallucinations are visual and auditory perceptions that occur during sleep onset, while hypnopompic hallucinations occur on awakening. They are usually visual and may be bizarre and dreamlike, but with some preservation of consciousness.[45] Ohayon and colleagues[46] reported an overall prevalence of 37% for hypnagogic hallucinations and 12.5% for hypnopompic hallucinations. It was also shown that patients with insomnia, excessive daytime sleepiness, or mental disorders were more likely to experience hallucinations. Hypnagogic and hypnopompic hallucinations are frequently associated with narcolepsy and are included in the diagnostic criteria for the disorder (although they are only reported by 25%–30% of narcoleptics).[47]

Drug effects. Many drugs are labeled as hallucinogens because they alter perceptions,[48] although true hallucinations are perceptions in the absence of any actual stimulus. Hallucinogenic drugs (including mescaline, psilocybin, and lysergic acid diethylamide [LSD]) are agonists of serotonin 5-HT_{2A} receptors; they do not always produce true hallucinations unless they are used at high doses. The effects also depend to some degree on the mood of the user and the situation in which the drug is used. Other drugs often considered to be hallucinogenic include phencyclidine (PCP), ecstasy, atropine, and dopamine agonists.

Tumors. Tumors that lie along, or compress, the optic path may cause visual hallucinations. In one case series, 13 of 59 patients with temporal lobe tumors experienced visual hallucinations.[49] These hallucinations are described as complex and may involve vivid scenes (including people carrying out mundane tasks). The majority of these visual hallucinations are thought to be related to seizure activity caused by the tumor. Another case series found that only 15% of people with occipital tumors have visual hallucinations.[50] These hallucinations more closely resemble those related to occipital seizures, and consist mostly of unformed spots or shapes of light, leading to the conclusion that complex formed hallucinations are not produced by occipital tumors.

Inborn errors of metabolism. A handful of inborn errors of metabolism may cause visual hallucinations. While these are quite rare, they are nonetheless important to consider because patients with inborn errors of metabolism may present with hallucinations at a time when their disease is treatable and when serious neurologic damage has not yet occurred.[51] Specific inborn errors of metabolism that may present with visual hallucinations include homocysteine remethylation defects, urea cycle defects, GM_2 gangliosidosis, Neimann-Pick disease type C, and α-mannosidosis.

Creutzfeldt-Jakob disease. Creutzfeldt-Jakob disease (CJD) is a fatal progressive neurodegenerative illness caused by central nervous system prion infection.[52] Prominent symptoms typically include fatigue, anxiety, and personality change, with progression to dementia, ataxia, and myoclonus in the later stages. Symptoms of CJD may also include visual hallucinations, especially in the Heidenhain variant of the illness. Visual effects may include color changes, visual field defects, visual agnosia, cortical blindness, metamorphopsia, and micropsia that progresses to the frank visual hallucinations

Table 1. Features of Visual Hallucinations Indicative of Etiology

Features of Visual Hallucination	Most Likely Etiologies
Simple patterns, spots, shapes, or lines; unilateral distribution; associated with headache	Migraine, seizure, tumor
Macropsia, micropsia, metamorphopsia	Seizure, Creutzfeldt-Jakob disease
Associated with going to, or waking from, sleep	Hypnagogic or hypnopompic hallucinations
Confabulation of all vision	Anton's syndrome
Frightening content	Psychotic disorder, delirium, hallucinogenic drug
Good insight	Charles Bonnet syndrome, migraine, peduncular hallucinosis

characteristic of the Heidenhain variant.[53] Electroencephalographic studies in cases of Heidenhain variant CJD have shown characteristic periodic non-generalizing complexes over the occipital region.[54]

How Can the Etiology of Visual Hallucinations Be Determined?

Given the broad variety of potential etiologies of visual hallucinations outlined previously, it is clear that an accurate diagnosis is required before effective treatment can be initiated. A thorough history and clinical examination are the most vital elements of a workup for visual hallucinations. Associated symptoms and characteristics of the visual hallucinations themselves may help direct diagnosis (Table 1). The elicitation of signs or symptoms of psychosis, inattention, parkinsonism, impaired vision, or headache will narrow the diagnosis and prompt further diagnostic studies. An EEG is potentially the most revealing diagnostic study, since it can not only highlight seizure activity, but also detect delirium (with theta-delta slowing), delirium tremens (with rapid beta activity), and CJD (with occipital periodic non-generalizing complexes).[54,55] An MRI of the brain can uncover tumors or infarcts that may be responsible for Anton's syndrome or peduncular hallucinosis, and may also show the characteristic "pulvinar sign" associated with CJD.

How Are Visual Hallucinations Treated?

Since effective treatment of visual hallucinations (see Table 2) is entirely dependent on the underlying cause, care should be taken to ensure diagnostic accuracy, especially since treatments that may be beneficial for one cause of visual hallucinations may exacerbate another. For example, benzodiazepines are the treatment of choice for delirium tremens, but they will almost certainly worsen a delirium due to any other cause.

Neuroleptic medications (i.e., dopamine antagonists) are the mainstay of treatment for visual hallucinations due to primary psychotic illness. These medications are also

Table 2. Diagnostic Findings for Causes of Visual Hallucinations and Indicated Treatments

	Schizophrenia	Delirium	Delirium Tremens	Dementia With Lewy Bodies or Dementia Associated With Parkinson's Disease	Posterior Cortical Atrophy	Charles Bonnet Syndrome	Seizures	Creutzfeldt-Jakob Disease
Diagnostic hallmark	Other symptoms of psychosis	Inattention; waxing/waning course; theta-delta slowing on EEG	Tremor, diaphoresis, autonomic instability; rapid beta activity on EEG	Other symptoms of parkinsonism	Atrophy of occipital, parietal, and posterior temporal lobes	Visual impairment	Epileptiform activity on EEG; hyperperfusion or hypoperfusion on SPECT	Occipital, periodic, non-generalizing complexes on EEG; "pulvinar sign" on MRI
Treatment	Neuroleptic medication	Neuroleptic medication (most evidence for intravenous haloperidol)	Benzodiazepines	Quetiapine or clozapine (other dopamine antagonists may exacerbate parkinsonism)	Cholinesterase inhibitors	Optimization of vision, neuroleptic medication, SSRIs	Anticonvulsant medication	None

Abbreviations: EEG = electroencephalogram, MRI = magnetic resonance imaging, SPECT = single-photon emission computed tomography, SSRI = selective serotonin reuptake inhibitor.

beneficial for the management of delirium (in which hallucinations are thought to be due to release of endogenous dopamine), with intravenous haloperidol having the most evidence for safety and efficacy.[55]

Unfortunately, due to their dopamine-blocking activity, most neuroleptics will significantly exacerbate parkinsonian symptoms in patients with DLB or dementia associated with Parkinson's disease. Quetiapine and clozapine have a niche role in the treatment of these patients, since their very low affinity for dopamine receptors renders them less likely to cause this serious adverse effect.[55] As with other forms of Alzheimer's dementia, cholinesterase inhibitors may have some benefit in posterior cortical atrophy.

More focal causes of visual hallucinations may require more focal treatment. Seizures may be treated with anticonvulsants, tumors with surgery and radiation, and migraines with triptans or β-blockers. Unfortunately, some causes of visual hallucinations (e.g., CJD) have no definitive treatment. For these patients, neuroleptics may minimize visual hallucinations and distress. Most patients with visual hallucinations, regardless of cause, will benefit from the reassurance of their caregivers. Some may also benefit from more formal psychotherapeutic interventions (e.g., cognitive behavioral therapy) directed to improve insight.

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