Visual Perceptual Abnormalities: Hallucinations and Illusions

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ABSTRACT

Visual perceptual abnormalities may be caused by diverse etiologies which span the fields of psychiatry and neurology. This article reviews the differential diagnosis of visual perceptual abnormalities from both a neurological and a psychiatric perspective. Psychiatric etiologies include mania, depression, substance dependence, and schizophrenia. Common neurological causes include migraine, epilepsy, delirium, dementia, tumor, and stroke. The phenomena of palinopsia, oscillopsia, dysmetropsia, and polyopia among others are also reviewed. A systematic approach to the many causes of illusions and hallucinations may help to achieve an accurate diagnosis, and a more focused evaluation and treatment plan for patients who develop visual perceptual abnormalities. This article provides the practicing neurologist with a practical understanding and approach to patients with these clinical symptoms.

Keywords: Illusion, hallucination, perceptual abnormalities, oscillopsia, polyopia, diplopia, palinopsia, dysmetropsia, visual allesthesia, visual synthesia, visual dysesthesia, sensation of environmental tilt, psychiatric, neurological

The topic of visual perceptual abnormalities, specifically hallucinations and illusions, spans many fields of medicine. The most prominent among these are neurology, ophthalmology, and psychiatry. A wide variety of pathological processes can lead to perceptual abnormalities. The purpose of this presentation is to review the neurological and psychiatric differential diagnoses of visual perceptual abnormalities. It is hoped that this will enable the clinician to understand the phenomenology while diagnosing and treating patients who present with these problems.

An *illusion* is the misperception of a stimulus that is present in the external environment.[1] An example is when an elderly demented individual interprets a chair in a poorly lit room as a person. A *hallucination* is a stimulus that is perceived, when in reality none is present.[2]

Objectives
On completion of this article the reader will be able to recognize the differential diagnosis, localization, and clinical significance of a variety of visual perceptual phenomena that may be encountered in clinical practice.

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The Parkinsonian patient who sees small children on his bed after increasing his carbidopa-L-dopa dose is experiencing a hallucination.

While a variety of psychiatric etiologies can cause perceptual abnormalities, several generalizations are important to consider. Primary psychiatric illness should not be accompanied by an altered level of arousal, myoclonus, asterixis, frontal lobe release signs, or focal neurological signs. Further, the patient should have no neurological signs of cognitive impairment such as aphasia, apraxia, significant amnesia, alexia, agraphia, or constructional apraxia.[3] Certain psychiatric illnesses such as schizophrenia can present with soft neurological signs.[4] As a rule, an electroencephalogram (EEG), spinal fluid examination, and imaging studies are within normal limits or have only nonspecific abnormalities in patients with primary psychiatric illness.[5]

It is commonly held that auditory hallucinations are most indicative of psychiatric illness whereas visual, tactile, olfactory, and gustatory hallucinations denote organic etiologies.[6] This is often but not always the case. Further, hallucinations and illusions that are incorporated into delusional systems are more consistent with primary psychiatric illness. Command hallucinations and multiple auditory hallucinations in a conversation about the patient are more common with psychiatric disease.[7] While illusions can occur with psychiatric illness, if combined with altered consciousness, they reflect a delirium.

PSYCHIATRIC ETIOLOGIES

Several psychiatric illnesses present with perceptual abnormalities. Major depression, especially in the elderly, can present with hallucinations, which tend to be consistent with depressive themes. These are most typically auditory or visual in nature but patients may at times describe olfactory or tactile illusions or hallucinations. The images can be simple or more complex, can be in black and white or color, often involve multiple modalities, and are often frightening to the patient. In addition, patients have vegetative symptoms including alterations in mood, motivation, energy, sleep, appetite, and libido. They may also experience suicidal ideation. This condition is termed depression with psychotic features. It can be effectively treated with a combination of an antidepressant and an antipsychotic or with electroconvulsive therapy (ECT).[8] Patients with bipolar illness in either the manic or the depressed phase can present with perceptual abnormalities.

In mania, patients often have pressured speech, racing thoughts, irritability, hypersexuality, insomnia, and hallucinations consistent with grandiose themes. The hallucinations are often colorful, elaborate, and entertaining. Images may be in motion and often communicate with the patient, stating that the patient has great power, wealth, or beauty. The treatment in this situation involves a mood-stabilizing medication (e.g., lithium carbonate, valproic acid, carbamazepine, or gabapentin) along with an antipsychotic medication. The antipsychotic often can be tapered soon after the hallucinations have resolved, and only the mood stabilizer continued.[9]

Patients with schizophrenia often have complex, auditory hallucinations. These are accompanied by symptoms such as delusions (fixed false beliefs), abulia, apraxia, and poor interpersonal relationships. The hallucinations are often described as coming from outside of the patient's head and often are frightening. While auditory hallucinations are most common, patients may have visual hallucinations that talk to one another or to the patient directly. In one study visual and auditory hallucinations were equally as common in schizophrenia but this runs counter to common clinical experience. Olfactory and gustatory hallucinations are much less common and suggest the possibility of a primary neurological etiology. The illness begins in the second or third decade and there is usually a gradual deterioration in functioning.[10] Treatment involves long-term maintenance on antipsychotic medications. Given the side effects and long-term risk of tardive dyskinesia, the atypical neuroleptic agents, such as risperidone, olanzapine, and quetiapine, are preferred.[11]

At times patients with personality disorders can develop psychotic symptoms including hallucinations and illusions. These are termed micropsychotic breaks and are usually of short duration and related to an acute stressor. The hallucinations are often poorly described and may only be fragments of images that do not speak directly to the patient. Patients may describe shadows or their own voices telling them to hurt themselves or others. Auditory and visual modalities are by far the most common, and olfactory and gustatory symptoms may suggest neurological illness. One looks for a pattern of long-term dysfunction in interpersonal relationships, which is characteristic of personality disorders. The acute treatment involves the time-limited use of antipsychotic medications. Psychotherapy is the primary long-term treatment modality.[12]

Patients who have undergone traumatic experiences outside the realm of normal everyday experience can develop posttraumatic stress disorder. Such individuals can have illusions and/or hallucinations as well as delusional thoughts. Stimuli that are related to the original stressor can cause flashbacks to the trauma. Hallucinations can involve any sensory modality that was related to the original event. The hallucinations are vivid and terrifying to the individual. The patient can have dreams that may persist into the waking period. An individual exposed to a traumatic event on an ongoing or extended basis may learn to dissociate. In this situation, individuals describe leaving their bodies and looking down on what is happening to them. Patients have a high degree of concurrent depression, anxiety and suicidal ideation. Treatment is the judicious use of psychotropic medications, which may include antidepressants or anxiolytics, with long-term individual and group psychotherapy.[13]

As a general rule, patients with anxiety disorders do not become psychotic. However, patients during a panic attack can develop illusions with macropsia, micropsia, and misperceptions of auditory stimuli. Patients do not describe vivid auditory or visual hallucinations as seen in schizophrenia, and olfactory or gustatory misperceptions are distinctly unusual. Between attacks, perceptual

abnormalities are absent; when present they are accompanied by palpitations, diaphoresis, and intense fear.[14] A high degree of concurrent depression and suicide is found among these patients. The serotonin reuptake inhibitors and benzodiazepines are often used in treating this disorder and neuroleptics are avoided.

Patients with substance dependence often have perceptual abnormalities. This is especially common with stimulants, cocaine, and hallucinogenic drugs such as LSD, PCP, and marijuana. With hallucinogens, the hallucinations may begin with colored patterns, distortions, and geometric patterns often with fragmented images of animals or people. Insight into the nature of the hallucinations is usually present and the patient usually does not become delusional. The hallucinations lack the symbolism and idiosyncratic nature of those seen in schizophrenia and are most often visual in nature.[15]

The hallucinations induced by cocaine and the amphetamines are more often auditory and the patient may resemble a patient with paranoid schizophrenia. Patients are often fearful and will become aggressive to avoid the perceived threat.[16] Perceptual alterations can also occur with anticholinergic medications. Patients often describe visual and tactile hallucinations and may have a concurrent confusional state, constipation, dry membranes, and urinary retention.[17] In withdrawal, benzodiazepines and barbiturates can produce a picture identical to delirium tremens.[18] Opiates can also cause hallucinations and illusions in which the patient will describe tactile stimuli such as bugs crawling on them as well as fragmentary visual hallucinations. Confusion, miosis, constipation, and respiratory depression may also be present and give clues to the diagnosis.[19] Topical eye medications including atropinics and beta blockers also have the potential to cause formed and unformed visual hallucinations particularly in the elderly. Thus, a thorough and complete inventory of medications is needed in any patient who is having hallucinations or illusions.

Alcohol can affect perception during intoxication, withdrawal, and even after the patient has been free of alcohol for an extended time.[20] During intoxication, patients can develop a confusional state where they may have illusions and occasional hallucinations. Wernicke Korsakoff syndrome with encephalopathy, ataxia, and ophthalmoplegia is not characteristically associated with hallucinations. Patients can be "talked into" confabulations about visual images that are not present (the "pick up the string that I'm holding" suggestion), but spontaneous hallucinations are rare.[21] During withdrawal patients can develop alcohol hallucinosis usually within the first 48 hours after they stop drinking. The patient may have continuous auditory hallucinations without an associated confusional state, but persistence of visual hallucinations is exceptional.[22]

With longer degrees of abstinence, patients can develop delirium tremens. In delirium tremens, patients have signs of sympathetic excess, encephalopathy, and can be extremely agitated. Patients may experience illusions and hallucinations in a number of modalities. Hallucinations often involve animals of normal size but lilliputian hallucinations may also occur. The patient often accepts the hallucinations as real but nonthreatening. Later, as the patient becomes more encephalopathic, the hallucinations may be quite disturbing to the patient. This condition is treated with thiamine, vitamins, and tapering doses of benzodiazepines. It is important to treat less severe degrees of withdrawal aggressively so that the full-blown condition will not develop, as this can be fatal in some 10 to 20% of patients.[23]

MEDICAL ETIOLOGIES

A variety of medical and neurological conditions can cause perceptual abnormalities. Patients who are in a delirium from any of a multitude of etiologies can experience hallucinations or illusions. The hallucinations and illusions may occur in a variety of modalities but most typically are visual and tactile. Often the images are fragmentary and frighten the patient. The hallucinations and illusions often occur in both the awake and drowsing state. Patients also present with alteration in level of consciousness; attention and concentration; disruption of the sleep–wake cycle; "sundowning," where the person becomes more confused at night; and agitation and fluctuation of symptoms over the course of a day.[24] Treating the cause of the delirium and using low doses of high potency neuroleptics, such as haloperidol, are usually effective in reversing the condition.[25]

Dementia from a multitude of etiologies can cause hallucinations and illusions.[26] Patients with Lewy Body dementia may be particularly prone to have visual hallucinations, but these can be seen in Alzheimer's disease (AD), Picks disease, HIV, Huntington's chorea, and multi-infarct states as well. The hallucinations seen in dementia can occur in a variety of modalities, but visual hallucinations may be the most common type. Patients often describe threatening vivid hallucinations that present paranoid themes. Illusions are quite common, especially in the evenings, and can lead to the patient becoming overtly psychotic. Patients may have a more difficult time remembering their hallucinations due to amnesia, or in communicating them due to aphasia. In demented patients who become psychotic, the atypical antipsychotics, such as risperidone, olanzapine and quetiapine, are suggested to minimize sedative, anticholinergic, and extrapyramidal side effects.[27]

In Lewy Body dementia the patient presents with Parkinsonian features and vivid visual hallucinations. These patients may have a higher serotonin to acetylcholine ratio and the hallucinations can worsen with serotonin reuptake inhibitors or anticholinergic medications.[28] Patients with AD tend to hallucinate less often than patients with Lewy Body dementia. Also, patients with AD who hallucinate have cortical Lewy Bodies. These patients have multiple neurotransmitter deficiencies but cholinergic neurons are the most severely affected. The hallucinations are usually auditory and less often visual. They can be fragmented and simple, or at times more complex. The hallucinations are often combined with delusions, which are often paranoid in nature. These may respond to acetylcholinesterase inhibitors, such as donepezil, as well as the atypical neuroleptics.[29]

Patients with Parkinson's disease frequently develop hallucinations. Parkinson's patients who hallucinate tend to be older and have more cognitive impairment than those who do not hallucinate. This can be due to cholinergic dysfunction, which, in part, explains the significant sensitivity that Parkinson's patients have to anticholinergic medications, which often produce visual hallucinations and confusional states.

Hallucinations can occur in 10–15% of Parkinson's patients who are treated with D1 and D2 receptor agonists including pergolide, bromocriptine, and amantidine, among others. This therapeutic complication was initially seen when L-dopa therapy was introduced before dopa-decarboxylase inhibitors were used. It was especially prominent at higher doses (4–6 g per day). At these high doses patients were often encephalopathic. The visual hallucinations typically begin some 10 years after levodopa therapy is initiated. Parkinsonian hallucinations tend to occur at the end of the day and associated sleep disturbances and vivid dreams are common, while insight is well preserved. The hallucinations are in color, are complex and may show motion and involve scenes of animals or people.[30]

The relationship between plasma drug levels and hallucinations is not straightforward. It has been noted with levo-dopa and every dopamine agonist that hallucinations are more prominent at higher plasma levels and may even resolve during off periods. A case was reported of a patient who developed hallucinations as part of a paranoid psychosis. The hallucinations did not resolve with a marked reduction in levo-dopa dose including a 3-day drug holiday. Thus, it seems that the hallucinations seen with dopamine agonist therapy involve more complex mechanisms than simply serum drug levels and may have distinct interindividual variation.[31]

Migraine is the most common single cause of visual hallucinations and illusions. Descriptions of migraine date back thousands of years. Migraine visual phenomena have been seen in every culture and every region in the world. This is a common disorder, which affects some 18% of women, 6% of men, and 4% of children in the United States.[32] The majority of migraine patients also have a positive family history. While the etiology of migraine is not known, it appears to involve median raphe activation of the trigeminovascular system. It may also involve blood flow changes, possibly related to a spreading depression of neuronal activity in the cortex usually moving from the occipital region anteriorly, but not respecting vascular territories.[33–35]

A number of visual phenomena have been reported. Elementary visual disturbances can include phosphenes (simple flashes), scotomata, specks, colored, or black and white geometrical forms or shimmering images. These may occur singly or in multiple forms. They often present in a hemi-field distribution but may occur directly in front or in an arc in front of the patient. Patients less often develop monocular visual loss, which has been termed ocular or retinal migraine. This is indistinguishable from vaso-occlusive amaurosis, but has no hallucinatory component. Areas of opaque translucent quality or sudden loss of all vision may be reported. The typical duration of these phenomena is 5 to 45 minutes but they may last much longer. The visual symptoms almost always occur before the headache but occasionally persist into the headache or begin to occur during the headache. Any deviation from the normal pattern needs to be evaluated by magnetic resonance imaging (MRI).[36]

The patient with migraine aura can also experience more complex visual phenomena. The most common of these is the teichopsia or fortification spectra. This visual event is quite suggestive of migraine, but has been seen in other conditions as well. The image can involve an arc of scintillating lines or lights which begins as a small moving spot in the center of the visual field and usually moves up to the upper part of the visual hemifield over several minutes. The leading edge can often present with zigzag or undulating characteristics and be in black and white or color. Patients describe images such as heat waves, water moving down a window, or like looking through water or cellophane. The vivid images initially may terrify the patient and may lead to significant anxiety and depression.[34]

Patients with migraine may also experience metamorphopsia, micropsia, macropsia (Alice in Wonderland syndrome), and other forms of geometric mosaic vision in which images split into fragments. In some patients complex visual images occur with multiple figures including animals, people, and elaborate scenes. They may have other concurrent events such as hallucinations of movement, paresthesias, déjà vu or j'amais vu, language disturbances, vertigo, photophobia, amnesia, dissociative states, or autonomic symptoms such as pallor, dizziness, or sweating.[37]

Older individuals especially can present with migraine accompaniments, so-called "migraine accompagne," where the visual hallucinations are not followed by a headache; this is also termed acephalgic migraine. However, in most young patients a headache follows the aura within minutes if not immediately. The usual headache is unilateral, throbbing, moderate-to-severe in intensity, and can be worsened by activity. It may begin gradually or abruptly and can remain unilateral or generalize. Headache duration can vary between several hours and several days.[38] Patients with repeated episodes of acephalgic migraine may be handicapped by not being able to drive. At least one otherwise healthy radiologist with frequent acephalgic migraine was unable to obtain malpractice insurance because of his frequent attacks. Dr. C. Miller Fisher described his personal 27-year experience with acephalgic migraines in a recent article. He noted spells of complex scintillating zigzags lasting from 5 to 30 minutes without obvious trigger, other than an association with close-up use of the eyes. He suggests that the brilliance of the visual display may reflect aberrant processing of ambient light, rather than only intrinsic abnormal activity in the visual cortex.[39] Treatment is usually a calcium channel blocker such as verapamil, or gabapentin. Attacks can be aborted with amyl nitrate or nitroglycerin. One should avoid the use of beta-blockers or any of the various triptan drugs.

In general, migraineurs are aware that the images they see are not real and tend not to incorporate the hallucinations into delusional systems. They do not experience symptoms such as thought withdrawal or broad-

casting as do patients with psychotic disorders. There is, however, a high co-morbidity between mood disorders and migraine. Patients have a three- to six-fold increased risk of depression, mania, and anxiety disorders compared to the general population.[40] Thus, migraine must be considered in psychiatrically ill patients who present with visual hallucinations.

Posterior cerebral infarctions can also lead to hallucinations. The lesions involve the occipital lobes and often the thalamus as well. The hallucinations are most often restricted to the side with the abnormal visual field. The pathology is usually ischemic infarction and may be delayed days to weeks after the infarct before the hallucinations begin. Most often they last several days to weeks, but may persist longer. A residue of intact visual cortex is required for the hallucinations to develop, which supports the hypothesis that these are release phenomena.[41]

Peduncular hallucinosis consists of vivid, usually formed, colorful hallucinations of people, animals, and complex scenes with motion. They occur more frequently in patients who are sedated. (Lhermitte initially described this condition in the context of rostral midbrain lesions. The anatomical localization is usually the anterior red nucleus) (Fig. 1). The hallucinations often begin several days after the lesion develops and persist for weeks to years. Individual hallucinations last minutes to hours and are less prominent in the day and more appreciated in the evening hours. Insight is usually preserved even though the patient may have concurrent auditory or tactile hallucinations. The lesions responsible are most often ischemic. The reticular activating system or its connections in the thalamus are likely involved, as isolated thalamic lesions rarely produce the same clinical picture. Depending on the extent of involvement of the reticular activating system, the patient may be less alert and drowsy.[42,43]

Narcolepsy can cause visual hallucinations, even as a presenting complaint. Narcolepsy is a genetically based neurological condition that often begins between the ages of 10 and 35. While the cause is unknown, it is believed to relate to brain stem dysfunction that allows REM sleep to persist into the waking state. The classic tetrad includes cataplexy, sleep paralysis, sleep attacks, and hypnogogic and hypnopompic hallucinations. All four elements are present in only 10 to 20% of patients. Sleep paralysis involves an inability to move when the patient first wakes up, which may be quite frightening. It involves persistence of REM-related atonia into wakefulness. A sleep attack consists of an overwhelming sense of fatigue causing the patient to fall asleep for 10 to 30 minutes. The patient may often awake feeling refreshed. Patients may also develop automatic behavior consisting of memory lapses and repetitive disorganized behaviors. Cataplexy involves the transient loss of muscle tone often precipitated by strong emotion in the patient.[44,45] We will concentrate on the hallucinations present in this condition.

Narcoleptic hallucinations are termed *hypnogogic* (entering sleep) and *hypnopompic* (awakening). Hallucinations in this disorder last several minutes and are present in some 30% of narcoleptics. The hallucinations

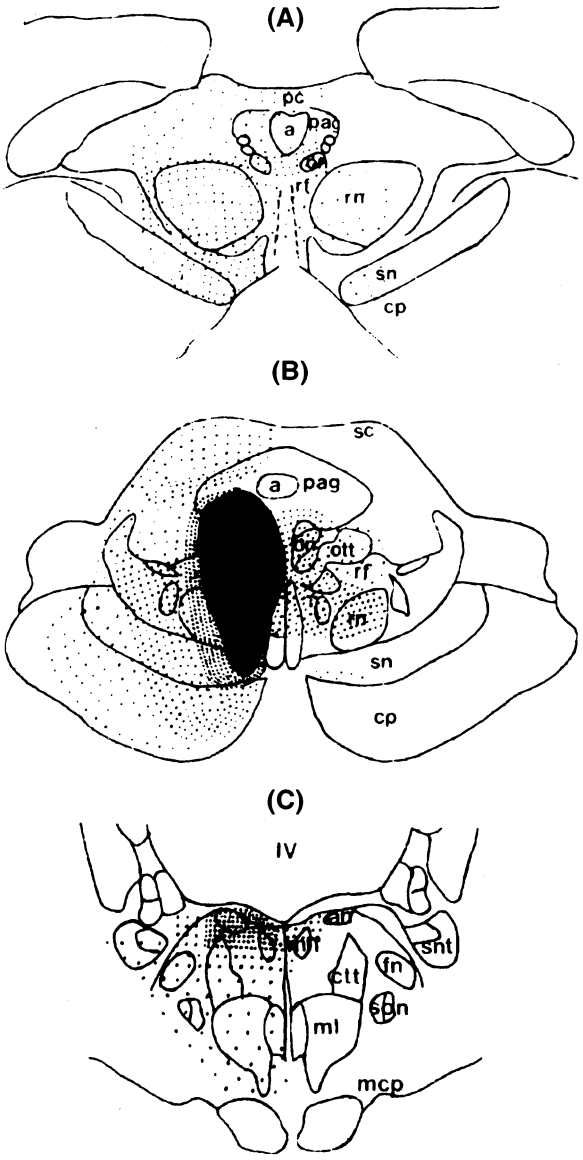

Figure 1. Composite illustration of the location of lesions found in patients with peduncular hallucinosis reported with neuroimaging or pathology. Note that the bulk of the lesions involve the mesencephalic paramedian reticular formation at the level of the rostral red nucleus. The extent of the lesions is pretectal to pontine levels. The denser the dots, the more frequently that area was involved. a = aqueduct; an = abducens nucleus; cp = cerebral peduncle; ctt = central tegmental tract; fn = facial nucleus; IV = fourth ventricle; ml = medial lemniscus; mlf = medial longitudinal fasciculus; mcp = middle cerebellar peduncle; on = oculomotor nucleus; pag = periaqueductal grey matter (includes raphe nuclei); pc = posterior commissure; rf = reticular formation; rn = red nucleus; sc = superior colliculus; son = superior olivary nucleus; sn = substantia nigra; snt = spinal nucleus and tract of trigeminal nerve. (From Manford M, Andermann F. Complex visual hallucinations: clinical and neurobiological insights. Brain 1998;121:1823, with permission of the authors and Oxford University Press.)

can be visual, auditory or tactile, but typically not olfactory. They may consist of colorful images, which may involve people, animals, and panoramic scenes. Patients often have a vague sense that someone else is in the room with them, which is often frightening.[46] Unlike dreams, there is no clear thematic content present in the hallucinations. Patients realize that what they are experiencing is not a dream and that they are awake. When the images are vivid the patients may have a difficult time believing that they are not based in reality. On occasion the narcoleptic patient may develop a delusional system about the experience. The hallucinations are not typically accompanied by psychotic features such as thought insertion, thought withdrawal, or voices that comment on the patient or converse with each other. Treatment involves the use of stimulants, such as dextroamphetamine sulfate.

Patients with epilepsy may also experience perceptual abnormalities. These occur most often during partial simple or complex seizures with or without secondary generalization. There are several cerebral localizations that produce the typical range of epileptic or seizure-related illusions or hallucinations. These can occur in visual, auditory, olfactory, and gustatory modalities. Patients may present with single or multiple types of hallucinations or illusions during any seizure. The hallucinations are usually brief, stereotyped, and fragmentary and may be in black and white or color. Rarely, hallucinations may be more complex and resemble those seen in narcolepsy. If the patient is in nonconvulsive status epilepticus, the hallucinations may be more prolonged. The etiology of epileptic hallucinations involves stimulation of the visual association cortex, which must be present for these to occur. Patients have variable insight into the validity of their hallucinations. At times, they are incorporated into delusional systems. They may be perceived as unreal and produce anxiety. In the individual patient with a seizure focus in the temporal lobe, elements of the epileptic personality such as hypergraphia, hyposexuality, and hyperreligiosity may be noted. In addition, patients may display alterations in mood, including manic and depressive symptoms.[47,48]

Occipital lobe seizures in adults most commonly present with simple visual phenomena. Patients may experience negative symptoms such as scotomata, amaurosis, or hemianopsia or more commonly unformed positive symptoms such as phosphenes. These may be described as sparklers or pinwheels. While they move, they do not usually have the stereotyped movement progression from center to periphery of visual field seen in migraine. If the focus is in the temporo-parieto-occipital region, patients may describe illusions in which objects appear to be distorted. This can involve changes in size including micropsia or macropsia, change in shape (metamorphopsia), colorful scenes of varying complexity, and the illusion that the patient is outside his or her own body (autoscopy). Most often these visual events are perceived in the contralateral hemi-field, but can at times generalize to the whole visual field. Patients concurrently describe the sensation of eye pulling, and are seen to have rapid eyelid fluttering and eye blinking or epileptic nystagmus.[49] Occipital epilepsy in children can take on several forms. Early onset cases present nocturnally with eye deviation, nausea, and vomiting. Later onset cases display visual phenomena such as hemianopsia, temporary blindness or migraine-like features such as scotomata, photopsia, and fortification spectra.[50]

Several features help to separate occipital lobe seizures from migraine headaches even though at times this can be quite difficult. In occipital lobe epilepsy, the visual hallucinations are stereotyped for each patient and last 5–30 seconds rarely up to 1 minute. There are no consistent precipitating factors present. Occipital lobe seizures tend to occur daily and consist mainly of multiple, bright-colored circular images that are most often seen in the temporal field and move contralaterally but may begin centrally. Occipital lobe seizures may be followed by a headache that is indistinguishable from a migraine. The headache can be unilateral, pounding, and associated with nausea and vomiting. The headaches usually begin within 3–15 minutes after the hallucinations have stopped and last one-half to 3 hours.

By contrast, migraine patients more often have a family history of headaches, the hallucination (aura) occurs several times a week to month and not daily, and the headaches last hours to days. One can often define specific precipitants that are associated with these headaches. As has been mentioned migraine often starts with predominantly flickering achromatic or black and white linear and zigzag patterns in the center of the visual field, gradually expanding over minutes into the periphery of one hemi-field, which often leaves a scotoma. The headaches in these two conditions, however, are quite similar. Treatment is sadly not effective for occipital epilepsy, which responds neither to anticonvulsants nor to standard migraine prophylactic medications.[51]

Parietal lobe seizures may produce somato-sensory and visual hallucinations either together or separately. Patients may experience paresthesias, dysesthesias, hypesthesias, or a desire to move a body part. Nausea or a sensation of intra-abdominal sinking may occur. Visual perceptions include formed visual hallucinations or photopsias when the dominant hemisphere is involved. With nondominant parietal lobe foci, the patients may experience metamorphopsia, which involves distortions of objects or body parts.[52]

During the post-ictal state from complex partial or generalized tonic clonic seizures, patients may experience perceptual abnormalities. These are similar to the illusions and hallucinations seen in a delirium of many causes. The patient is often somnolent, inattentive and will be at least partially amnestic for what occurs during this period. Thus, epilepsy must be in the differential diagnosis of a variety of perceptual abnormalities that may arise in the settings of either clear or clouded sensorium. This makes the electroencephalogram (EEG) an important diagnostic tool in the evaluation of these hallucinatory states.[53]

OPHTHALMOLOGIC ETIOLOGIES

The Charles Bonnet syndrome is characterized by complex visual hallucinations present when the patient has ocular pathology resulting in visual deterioration, most often due to macular degeneration. The patient is

clear and conscious in the absence of psychosis, with no focal neurological illness, substance abuse, or acute eye disease. This syndrome is present in some 10% of patients with severe visual loss and is more common in the elderly. The hallucinations are not usually stereotyped and involve vivid scenes of animals, flowers, and people. They may be black and white or in color, static or dynamic, and may be of normal proportions or altered in size. The hallucinations may be episodic, periodic, or continuous. The hallucinations are more common in the evening, and the patient has both eyes open. Most patients realize the hallucinations are not real, and their emotional responses range from indifference to amazement. Many individuals can stop the hallucinations by opening or closing their eyes or by deviating their gaze in another direction. The hallucinations are not believed to be related to visual input or low lighting levels and occur more commonly in patients with ocular pathology who have concurrent age-related degenerative CNS lesions.[54–56]

Charles Bonnet syndrome can occur with bilateral lesions located in the occipital cortex. This is termed cortical blindness and presents with loss of vision, normal light reflexes, and the absence of opto-kinetic nystagmus. Acutely, patients may also be disoriented, agitated, and present similar to an acute confusional state. The hallucinations experienced by these patients may be simple or more complex if the visual association cortex is involved. Lesions that also extend into the medial temporal lobe can cause amnesia and patients may not be aware of their visual loss. This latter condition is termed visual anosognosia or Anton's syndrome. Patients may still be able to direct their gaze to auditory stimuli, as subcortical areas, the optic tracts, and lateral geniculate bodies may mediate this function.[57,58]

Oscillopsia is an illusion of movement of the environment. It is classified as: (1) due to an abnormality of VOR (vestibulo-ocular reflex) caused by damage to the peripheral vestibular apparatus, or to central vestibular dysfunction; (2) due to nystagmus; or (3) due to central lesions producing seizures. Rarely, noise-induced peripheral vestibular pertubations (Tullio phenomenon) will produce oscilloptic movement of the environment. The sensation of oscillopsia can be appreciated if you close one eye and gently jiggle the globe of the open eye.[59]

Brief "buzzing" rotatory diplopia and oscillopsia occur with superior oblique myokymia. Attacks are seconds long and the sound of muscle contraction like a motorbike acceleration can be appreciated by auscultation of the orbit during the spells.[60] Rarely, fractures through the roof of the orbit with development of an encephalocele will produce a vertical synchronous oscillopsia in one eye.

Polyopia or diplopia of cerebral origin is a rare manifestation of occipital cortex or central visual pathway lesions. It has been described in conditions such as multiple sclerosis, trauma, encephalitis, seizure, and migraine. The number of images that can be seen ranges from $1\frac{1}{2}$ to hundreds. Patients usually describe that the true and false images are quite similar, but the false images may differ in intensity, color, or size. Head movement, ocular fixation, or movement of the object can precipitate polyopia. Usually the latent period for polyopia is several seconds after fixation on the object occurs, but can occur even more rapidly. The images usually only persist as long as the stimulus is present but can persist (palinopsia) and are usually evenly spaced in relation to one another. There is often an associated homonymous hemianopsia on the involved side and the polyopia is located in the area of macular sparing. One type of polyopia is termed entomopia. This involves the experience of seeing multiple copies of an image in a grid-like pattern. The lesion causing polyopia is most likely within the extrastriate cortex.[61,62]

Palinopsia is a continuation of visual sensations after cessation of light stimuli or as episodic, intermittent, or paroxysmal reappearance of images without immediate visual stimulation. It is usually a transient process, but can persist. The images are brief and recur periodically in the impaired visual field. This is thought to be a release phenomenon involving lesions of the parietal and occipital lobes most often in the nondominant hemisphere.[63] A number of drugs and medications are reported to produce these images. Several cases have been described in which epileptic phenomena may be involved in the production of palinopsia, but this is felt to be less prominent than release phenomena.[64] In one report a patient with schizophrenia developed palinopsia that persisted for 5 years. There are conflicting reports as to whether anticonvulsants are effective in treating palinopsia.[65] Table 1 is a list of causes and associations of palinopsia. The illustrations in Figures 2A–F were made by a commercial artist who had a parasagittal parieto-occipital meningioma removed. Before surgery he had a macula-sparing right homonymous hemianopsia. Prior to surgery, he had

Table 1. Causes and Associations of Palinopsia

Drugs
Marijuana
Mescaline
Lysergic acid diethylamide
3,4-methylenedioxymethamphetamine
Interleukin-2
Trazodone
Clomiphene citrate
Nefazodone
Seizures
Temporal
Occipital
Periodic lateralized epileptiform discharges
Focal cerebral lesions
Trauma
Parasite
Abscess
Stroke
Tumor
Arteriovenous malformation
Jakob–Creutzfeldt disease
Multiple Sclerosis
Carbon monoxide poisoning
Non-ketotic hyperglycemia
Migraine
Psychiatric disease
Schizophrenia
Psychotic depression

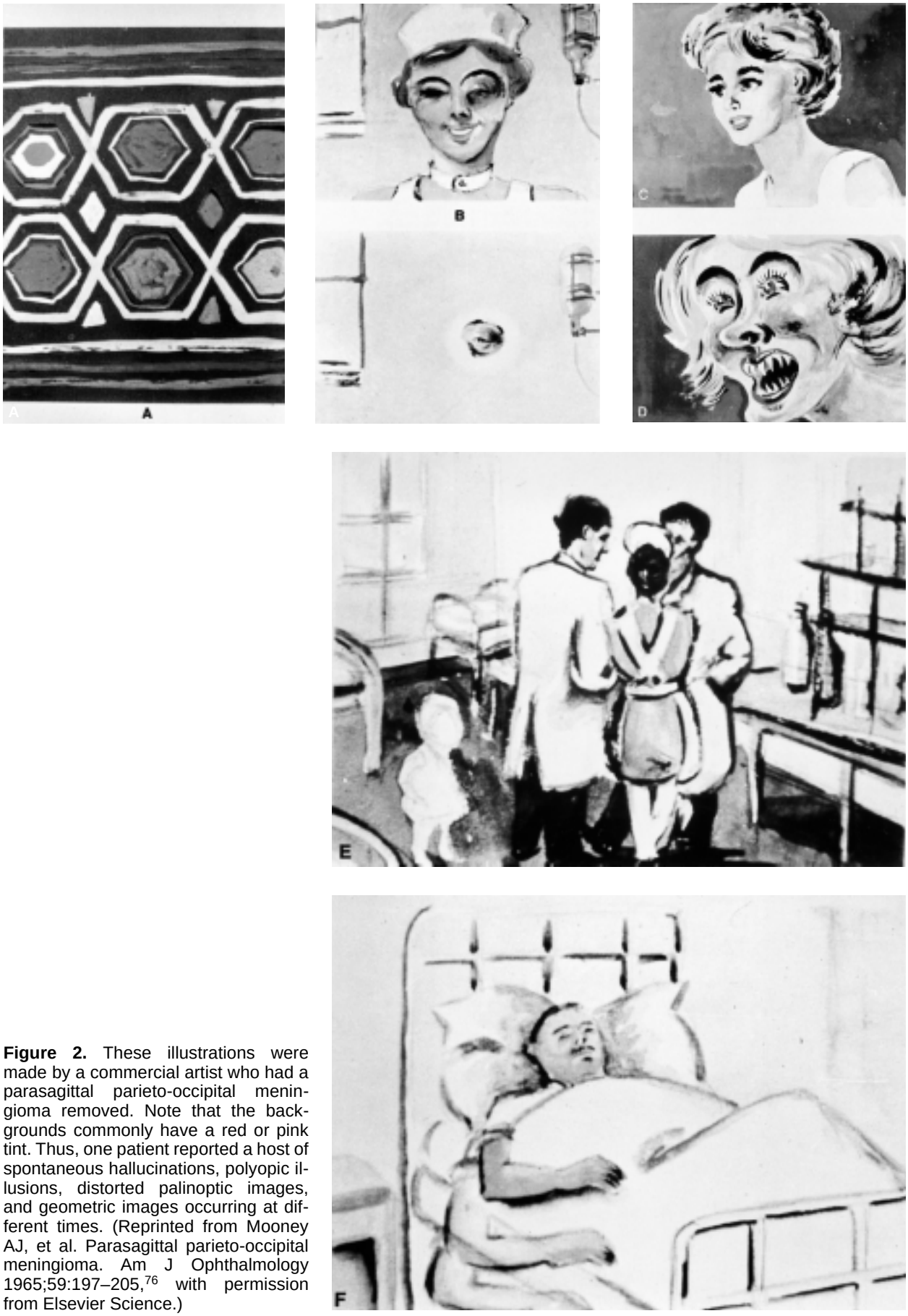

Figure 2. These illustrations were made by a commercial artist who had a parasagittal parieto-occipital meningioma removed. Note that the backgrounds commonly have a red or pink tint. Thus, one patient reported a host of spontaneous hallucinations, polyopic illusions, distorted palinoptic images, and geometric images occurring at different times. (Reprinted from Mooney AJ, et al. Parasagittal parieto-occipital meningioma. Am J Ophthalmology 1965;59:197–205,[76] with permission from Elsevier Science.)

episodes of seconds-long moving colored lights that were red, yellow, and blue with red as the predominant color. These occurred in the right visual field. Postoperatively he experienced complex visual hallucinations. Visual images could be (A) colorful geometric figures, or a (B) series of unpleasant transformations of images that remained behind after a person had been seen and left (palinoptic images). In (B) the nurse's face and left eye, is discolored and the eye is distorted, remaining behind after the nurse had left the room. In (C) spontaneous hallucinations of "girls one might see on magazine covers; one like all the rest, (D) would be laughing horribly; she had bright yellow hair but her lips (screaming vermilion red color) and very white teeth . . . [became] long and pointed." When people were in groups and talking, a small pale childlike image would appear with the group—like "lilliputian hallucinations" (E). Polyopia—multiples of objects (F) especially involving hands and arms were common.[76]

Dysmetropsia is also termed dysmegalopsia or metamorphopsia and is a disorder of size perception. Objects can either appear smaller (micropsia) or larger (macropsia) than their objective size. This has been described in such disparate conditions as retinal edema, retinal folds, epilepsy, migraine, hallucinogenic drugs, and conversion disorder. It may present as an episodic phenomenon in adolescence most likely associated with migraine (Alice-in-Wonderland effect). Permanent dysmetropsia following focal cerebral lesions is not common. A patient was described with a right ischemic prestriate lesion and she developed micropsia in her left homonymous visual field. The patient was aware of her visual difficulty and other aspects of visual processing were intact. The patient was neither alexic nor achromatopsic. In this case it seemed that size perception could be dissociated from other aspects of visual processing such as color of form. This case suggests that the prestriate cortex is likely involved in the perception of an object's shape and size.[66]

The Pulfrich Phenomenon is widely accepted to be due to an interocular latency difference in the two eyes of the observer, due to a variety of causes. Carl Pulfrich, who first described the phenomenon, was one-eyed and could not appreciate what he theorized would occur. Most often this effect is caused by a reduction in the amount of light reaching one optic nerve compared to the other. Patients have described oncoming traffic as crossing over lanes toward the observer, people in crowds appearing to walk into the observer, and misperceptions of how to pour liquids, do needlework, or play tennis. A spontaneous Pulfrich phenomenon can be produced in any condition in which nerve conduction is delayed in one optic nerve especially with tumor compression, demyelination, and ischemia. This can also occur in conditions in which ocular pathology causes interocular illumination differences such as a unilateral cataract. Treatment of this condition involves placing a neutral density filter in front of the good eye in a quantity sufficient to reduce illumination to match that of the affected eye.[67]

Visual allesthesia occurs when visual stimuli are transposed from one homonymous field to another. This can occur with other modes of stimuli as well. This may be a manifestation of parieto-occipital or occipital lobe disease caused by neoplasm, infection, trauma, or seizure activity. It has also been described in multiple sclerosis and migraine headache.[68]

Sensation of environmental tilt is usually associated with lateral medullary syndrome. Reports of 90- or 180-degree tilts terrify the patient and mystify the doctor, who commonly believes the patient to have a psychiatric problem. The large tilts are abrupt in onset and brief (minutes long) in duration, whereas smaller deviations of the perpendicular may persist for prolonged periods.[69,70] In addition to the lateral medullary syndrome, this illusion of environmental tilt may occur in hemispheric locations or as the result of other brain stem or cerebellar lesions.[71] The vestibular system takes a combination of linear and rotatory sensory information and produces an internally perceived "model" of head orientation and movement. Unilateral disruption of the input from the vestibular system may produce a sensation of rotation or the illusion of tilt of the environment.[72] One patient had a VP shunt placed in the right parieto-occipital cortex due to normal pressure hydrocephalus. On the first postoperative day the patient developed episodes where the environment would transiently rotate 90 degrees. These episodes occurred three to six times per day and resolved by the sixth day after surgery. This patient's head CT and EEG were within normal limits. On follow-up these events had not recurred.[68] Rotatory illusions are most common with lateral medullary stroke.

Visual synthesia occurs when auditory, tactile, or gustatory stimuli produce visual images. This phenomenon can occur with lesions in all parts of the visual pathway.[73] In visual dysesthesia, various unpleasant visual sensations are experienced when looking into an area of visual loss. This is often associated with a lesion localized to the optic radiations.[73] Simultanagnosia is neither a hallucination nor illusion but can be confused with either. This condition presents with an inability to experience the visual field as a whole, with only certain aspects being attended to. Patients tend to use only macular vision.[73] Balint's syndrome presents with simultanagnosia, optic ataxia, and ocular apraxia. This syndrome is often a sequela of a watershed posterior cerebral artery and middle cerebral artery infarct and localizes to the occipito-parietal region. Ocular apraxia involves an inability to gaze directly at a certain part of the visual field. Optic ataxia involves an inability to direct a limb in space using visual guidance.[74] (For more details, see the article in this journal by Rizzo.)

Sensory deprivation can cause simple or complex visual hallucinations. The patient who is sensorily isolated reports abstract patterns such as geometric designs or recognized people or animals. The latter may be in color and have atypical features such as wearing bright caps or walking around. When ophthalmology patients were bilaterally patched, kept in a dark room and sedated while receiving atropinic medications to keep pupils dilated, a common sensory deprivation scenario of visual hallucinations and agitation would arise. On occasion, there may also be auditory or tactile hallucinations. Dur-

ing sensory deprivation an increase in EEG slow wave activity occurs, which resolves when the deprivation ends.[75]

Also, prolonged sleep deprivation may be associated with visual hallucinations. One American Vietnam Prisoner of War was deprived of sleep for many days and experienced colorful rescue hallucinations that occurred as if staged in a small theater. A curtain would open and muscular, well-armed rescuing Marines would charge in only to have the curtains pulled and a repetition of the same event to occur (personal information).

Visual perception is an essential component for experiencing the environment. As is evident, a wide variety of perceptual abnormalities can occur, which can have a wide variety of neurological localizations and a multitude of causes. The conditions span the fields of medicine, neurology, and psychiatry. A systematic approach to these conditions will result in more accurate diagnosis and better treatment options for patients who develop perceptual abnormalities.

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