Visual hallucinations

Hallucinations may be defined as perceptions that occur in the absence of a corresponding stimulus. They can involve any of the five senses, hence visual, auditory, tactile, gustatory and olfactory hallucinations. By contrast, illusions depend upon a misinterpretation of an external stimulus. In this review we outline the causes, and describe the characteristics of various hallucinatory states. This should help in the assessment of a patient presenting with visual hallucinations.

Throughout history, hallucinations have been associated with insanity and psychiatric disease. Patients who experience them may not reveal their symptoms for fear of being labelled as ‘mad’. These symptoms may therefore go unrecognised by doctors.

Visual hallucinations form a spectrum from photop-sias, which are simple visualisations of colours and shapes, to complex formed hallucinations of people, animals and events. If the unreal character of the hallucination is readily recognised by the individual, this is referred to as a ‘pseudohallucination’.

Common causes of visual hallucinations (Table 1) include acute confusional states, drugs, bereavement reactions, neurological, ophthalmological and psychiatric conditions. Though the form and the content of the visual hallucination may not be specific to the underlying disorder, some characteristics may be diagnostically useful [1,2].

Visual hallucinations associated with non-morbid conditions

Dreaming and hallucinations can be thought of as being part of the same spectrum, the visual hallucinations occurring during sleep in the former and while awake in the latter. Dreaming occurs during REM (rapid eye movement) sleep and appears to be the release of fantasy thought in a sequence of images.

Hypnagogic images are hallucinations perceived in the period between waking and sleeping, whereas hypnopompic hallucinations occur during the transition from sleep to wakefulness. They are most likely to be experienced when a subject is drowsy, yet aroused in situations of stress. Such images can include flashes of light and colour, geometric patterns and complex memory scenes. They are often accompanied by tactile kinaesthetic sensations of moving, falling or floating, as well as distortions of body imagery. They are common in the rare condition of narcolepsy in which REM sleep intrudes into the waking state without passing through the usual intervening sleep stages. Both these types of hallucination usually occur in physically healthy people and in the absence of major mental illness. Hallucinations in all these non-morbid conditions are usually recognised as being unreal perceptions or pseudohallucinations.

Hallucinations may also occur after sensory deprivation and isolation. Examples include those experiences reported in sensory deprivation studies, where subjects were confined to dark rooms or tanks for prolonged periods [3]. They have also been reported by hostages who had been subjected to conditions of isolation, visual deprivation, physical restraint and the threat of death [4]. They experienced a progression of visual hallucinations from simple geometric forms to complex memory images.

Bereavement hallucinations

These are one of the commonest types of hallucination, occurring in 50–60% of widowed people [5,6]. Experiences of contact with a dead person include an awareness of their continued presence, hearing them speak, being touched by them as well as ‘seeing’ the deceased relative. The visual hallucinations are commoner at night though they can be experienced throughout the day. A common experience is ‘seeing’ the loved one sitting in a chair. These visions and other hallucinations are usually a source of comfort but can at times be unpleasant. They are equally common in men and women, especially in those who have been married for many years, and occur irrespective of race or creed. They gradually decline with time but can persist for decades. There is no association between bereavement hallucinations and suddenness of death, or whether the family was present, an autopsy was done, or the bereaved person was clinically depressed. Most widowed people do not mention these experiences to doctors, clergymen or family: perhaps they fear being ridiculed or thought of as being mad. People who experience these hallucinations are less likely to re-marry, feeling that the dead spouse would be opposed to this.

Hinton [7] suggests that these hallucinations may make the loss of a loved one more bearable and that they might be a response to an incomplete acceptance of death. Parkes [8] considers them as part of the normal reaction to bereavement but points out that sometimes they are so vivid that people need reassurance that they are not unusual phenomena.

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Visual hallucinations

Table 1. Causes of visual hallucinations

| Normal or non-morbid conditions: | Drug-induced: |
|----------------------------------|---------------|
| *dreaming* | side-effects of therapeutic drugs |
| *hypnagogic/hypnopompic* | effects of addictive drugs |
| *grief reactions* | alcohol withdrawal/intoxication |
| *following prolonged isolation* | |

| Neurological disorders: | Toxic confusional states: |
|------------------------|---------------------------|
| *occipital lobe lesions +/- temporo-parietal lobe involvement* | *infection/infarction/organ failure* |
| *Parkinson’s disease* | *states of delirium/metabolic disturbances* |
| *epilepsy* | |
| *migraine* | |
| *peduncular hallucinosis* | |
| *head trauma, central nervous system (CNS) infections* | |
| *pituitary tumours* | |
| *Sydenham’s/Huntington’s chorea* | |

| Eye disease: | Psychiatric disorders: |
|-------------|------------------------|
| *cataracts* | *dementia - all types* |
| *macular degeneration* | |
| *glaucoma* | *schizophrenia, bipolar disorder, major depression* |
| *choroidal neovascularisation* | |

Neurological disorders

*Occipital lobe lesions*

Visual hallucinations may occur with lesions of the occipital lobe, with or without concurrent involvement of the temporo-parietal regions. The most common lesion here is an infarct, though haemorrhage or any space-occupying lesion may be responsible.

Kolmel [9] studied 120 patients with a homonymous hemianopia, of whom 16 (13%) experienced complex visual hallucinations in the hemianopic field. There was a latent period of a few hours to one week after the onset of the hemianopia before the hallucinations appeared. They often started as simple photopias, and later developed into complex visual forms. They were typically weak in colour and of the same form (stereotypical) in appearance. If persons appeared, their faces were often difficult to recognise or turned away from the observer. The hallucinations were easily recognised as unreal, and all patients reported that saccadic eye movements (rapid, jerky eye movements on changing fixation from one point to another) caused the hallucinations to disappear. This allows differentiation from hallucinations occurring in epileptic aura and seizures, which are not affected by eye movement. The hallucinations usually disappeared as the hemianopia resolved. In cases of persistent visual field defect, the hallucinations gradually became less frequent and eventually disappeared. It therefore seems that the best treatment strategy after the diagnosis has been made is reassurance that the hallucinations often spontaneously subside.

Parkinson’s disease

Visual hallucinations are common in patients with parkinsonism; auditory and olfactory hallucinations also occasionally occur in this disorder. The visual hallucinations are sometimes experienced before the patient has taken any drugs [10], but usually the symptom is related to treatment, and has been described with all anti-parkinsonian medication. Amantadine causes hallucinations, especially in higher doses [11], as do lysuride and pergolide [12]. Bromocriptine causes hallucinations [13] which may persist for weeks after the drug has been withdrawn. The hallucinations of parkinsonism are most common in patients on higher doses of levodopa, those who have been taking this drug for a long time, and those on a combination of levodopa and anticholinergic therapy.

Patients on levodopa may first develop vivid dreams, then visual hallucinations and may go on to experience paranoid delusions and confusion. The visual hallucinations appear and disappear suddenly. They are recurrent and tend to be non-threatening, but are sometimes frightening [14]. The hallucinations may be unformed (stripes, spots, patterns, shadows) or formed visions of people, animals or inanimate objects [15]. The people seen may be littleputian (reduced in size). The cause of parkinsonian hallucinations is uncertain but overstimulation of serotonin receptors and dopamine receptor hypersensitivity are probably important factors. Reducing the dose of levodopa or stopping the drug will lessen or abolish the hallucinations, but may cause intolerable worsening of the parkinsonian signs. Drugs which block serotonin
(5-HT) or dopamine receptors may be helpful. Ondansetron, a 5-HT\textsubscript{3} receptor antagonist, abolished or attenuated hallucinations in one small study [16], without any worsening of the patients' physical condition. Risperidone antagonises 5-HT\textsubscript{3} and dopamine D\textsubscript{2} receptors. It too may affect an improvement in the hallucinations [17], but recent case reports have suggested that a deterioration in motor symptoms may occur [18]. Larger scale trials are needed to confirm the findings of these pilot studies.

**Epilepsy**

The observation that psychic phenomena such as visual hallucinations may be a manifestation of an epileptic discharge was first noted by Hughlings Jackson in 1931 [19].

The hallucinations caused by seizure discharges are typically brief, lasting less than three minutes (usually less than 30 seconds), and can be associated with hallucinations in other senses, eg olfactory and gustatory hallucinations with temporal lobe lesions [20] and uncinate gyrus fits. The visual hallucinations vary from simple photopsias to complex forms, and may be recognised as unreal, though some are true hallucinations. Dewhurst and Pearson [21] reported a man with seizures secondary to a shrapnel wound of the temporal lobe; initially he saw multicoloured lights but later reported approaching and receding 'crowds of tiny figures, all the colours of the rainbow—all myself'.

There is a marked variation in hallucinatory experiences among patients, but in any one patient these experiences tend to be of the same form, ie stereotypical. The electroencephalogram (EEG) is abnormal during the hallucination, but can be normal afterwards. Treatment with anticonvulsants is usually successful [22]. The hallucinations are not affected by eye movements.

Epileptic seizures in post-traumatic epilepsy almost always begin with photopsias or with complex visual hallucinations in the contralateral field. This is followed by movement of the head away from the focus and ultimately by a generalised tonic–clonic seizure in the most severe cases. The hallucinations may involve the actual events leading to the brain injury, a fascinating phenomenon which has been termed 'Erregungsfang' (flashback) by Pateisky [23].

**Migraine**

Visual hallucinations associated with migraine usually consist of simple flashes of light or colour, scintillating scotomas or zigzag lines (fortification spectra). Complex hallucinations accompanying migraine are a rare phenomenon. Autoscopic phenomena are visual reproductions of the self or parts of the body in external space, and can occur as a symptom of migraine or epilepsy. They can also rarely be associated with schizophrenia and states of extreme anxiety or fatigue. **Peduncular hallucinosis**

This is a rare syndrome of visual hallucinations, sleep disturbance and agitation, secondary to a lesion of the midbrain or thalamus. It is usually due to infarction in the posterior cerebral artery territory, damaging the reticular activating system and leading to sleep disturbance.

Lhermitte [24] described the first case in 1922, a 72 year-old woman with vivid, formed hallucinations and clinical features of a lesion of the upper midbrain (headache, nausea, vertigo, left-sided ptosis, ophthalmoplegia and ataxia). The hallucinations were of brightly coloured, kaledoscopic, illiputian people. Van Bogaert described another case in 1924 [25]. Autopsy revealed infarction of the midbrain structures, and he coined the term 'peduncular hallucinosis', 'pedunculaire' referring to the midbrain. Feinberg and Rapcsak [26] reported a similar case with a lesion of the thalamus, and Dunn et al [27] reported one following brain stem compression by a cranio-pharyngioma, this being reversible with surgery.

The hallucinations usually appear as isolated, vivid, visual images, often in bright colours. They tend not to invoke fear in the patient, who is usually aware that the image is not real, though in some cases the patient is unable to determine this.

Any overt or subtle signs of a brain stem disorder should therefore be carefully sought in patients with acute formed hallucinations. Radiological confirmation can be achieved by magnetic resonance imaging (MRI).

**Eye disease**

Visual hallucinations may arise in patients who have impaired vision secondary to eye disease [28] such as cataracts [29], macular degeneration [30], retinal ischaemia [31] and glaucoma.

Simple photopsias such as flashes of light and colour are most commonly seen. However, the hallucinations may be complex, formed, brightly coloured and brilliantly clear, in sharp contrast to the blurred and faded vision that these patients are used to. The hallucinations usually elicit a normal or pleasant emotional response, the patient frequently being aware that the image is not real (pseudohallucinations). They typically occur at night, when the level of illumination is low; however, with eyes closed or in total darkness the images may disappear. They are never accompanied by hallucinations of other senses. As blindness progresses, the clarity, frequency and duration of the hallucinations fade. Rosenbaum et al [32] described the case of an 82 year-old woman who saw people engaged in various activities, especially at night. She described it as watching the television with the volume turned off. She was noted to have bilateral cataracts leading to impaired visual acuity. There was no psychiatric history and her mental state examination was normal. Following
cataract extraction her visual acuity improved and the hallucinations disappeared. It is postulated that when visual sensory input is reduced, for example secondary to cataracts, the sensory deprivation leads to the release of previously recorded images which are perceived as hallucinations.

Treatment includes surgery for the eye disease if possible (eg cataract extraction). In some cases, such as those with macular degeneration, the physician may only be able to reassure the patient that the hallucinations usually subside as visual failure proceeds. Some authors [33] have found improvement in certain cases with anticonvulsants. Neuroleptics are of little benefit.

The Charles Bonnet syndrome was initially described in 1769 as a combination of formed visual hallucinations in an individual with preserved insight, usually associated with decreased visual acuity. However, since then, a much wider view of the syndrome has developed, and includes any state of visual hallucinations in older people, irrespective of accompanying symptomatology. Unfortunately, this makes the syndrome of little clinical significance [34].

Holroyd et al [30] found that in patients with macular degeneration the likelihood of developing visual hallucinations increased with sensory deprivation (such as living alone), and with a history of previous brain injury (such as poor cognition and a prior history of strokes).

Drug-related visual hallucinations

Visual hallucinations can occur as a side-effect of certain therapeutic drugs or can be induced by various illicit drugs (Table 2).

They initially tend to consist of unformed images such as abstract shapes and flashes of light. There is often an illusory component with altered size, colours and shapes, eventually leading to more complex forms with people and scenes in vivid colours. The nature of the hallucinated material is greatly influenced by the individual’s psychological background. The images are more readily seen with the eyes closed or in darkened surroundings. They can be associated with hallucinations in other senses such as the tactile hallucinations of cocaine and amphetamine intoxication, in which insects are felt crawling on the skin (formication). Removing the suspected agent is a simple means of diagnosis and treatment.

Some illicit drugs may lead to synaesthetic hallucinations. Here, sensory stimuli are experienced in another quality of sensation, eg a colourful visual hallucination after hearing a loud noise or an auditory hallucination in response to a bright light. It is believed that this phenomenon occurs because of drug-induced cortical hypersensitivity which allows a strong stimulus in one area to trigger other areas of the cortex.

Alcohol withdrawal

Alcohol withdrawal can lead to visual hallucinations as part of the syndrome of delirium tremens. They usually occur on the second or third day after reduction or cessation of drinking, and typically involve small moving objects or animals such as mice. They usually provoke fear in the individual and treatment is important. Other sensory hallucinations such as auditory and tactile may also be involved.

Toxic confusional states (delirium)

This is one of the commonest causes of hallucination, in which the hallucinations themselves may be a small part of a transient psychotic syndrome. Vivid nightmares and illusions are often noticed first, followed by frank hallucinations. Images of snakes, mice and other small animals are characteristic. Patients experiencing such hallucinations are often noted to pick repeatedly at the air or bedclothes (carphologia). Treatment of the underlying disorder will result in resolution of these hallucinatory phenomena. Similar hallucinations can occur in patients after cataract extraction. Here, a combination of visual sensory deprivation and a relative overdose of the drugs used before, during and after operation are thought to be responsible. They often subside without treatment.

Visual hallucinations associated with psychiatric disease

Although auditory hallucinations are far more common in psychiatric disease, visual hallucinations can occur. In schizophrenia, the visual hallucinations are usually formed and may be described by the patient as images of people, animals or events taking place in front of them. They are thought of as real,
and can be in colour or black and white. Rarely, they may consist of simple photopsias alone. Visual hallucinations in psychiatric disease appear suddenly and without prodromata in a setting of intense affective need or delusional preoccupation. Unlike visual hallucinations induced by drugs, they do not change if the eyes are open or closed. Tactile, olfactory and gustatory hallucinations have also been reported. Hallucinations that occur during manic states, major depression, and other psychotic conditions may be no different from those described in schizophrenia. Therefore, the form and the content of the hallucination may not allow differentiation between the various psychotic conditions but may well be useful in differentiating psychiatric disorders from organic conditions.

Treatment of visual hallucinations secondary to psychiatric conditions usually involves the use of neuroleptic medication but may include techniques such as psychotherapy and increasing the external visual stimulus.

Visual hallucinations and dementia

Visual hallucinations can occur in a variety of dementing processes, including Alzheimer's disease [49], multi-infarct dementia [50], and senile dementia of the Lewy body type (SDLT) [51]. The visual hallucinations are usually complex and formed, often of people, and can arouse fear in the patient if they are thought of as real. They are often worse at night when the level of illumination is low. Haddad et al [52] described a case of dementia in an 84-year old woman. Here the visual hallucinations were the first symptom to occur. She saw strangers in her house, mainly at night, and would leave the light and fire on for them. Mental state examination at that time was normal, though signs of generalised cognitive impairment developed four months later. Computed tomographic (CT) scan showed generalised atrophy, most marked in the parietal region. Her condition continued to deteriorate and she died six months later. Neuroleptics were ineffective in controlling her hallucinations. This accords with the observation of Berrios and Brook [53] that neuroleptics are of little benefit in treating persistent visual hallucinations in older subjects. By lowering seizure threshold, they may actually increase the tendency to hallucinate if the aetiology is epileptiform. However, they may be useful in certain cases as they are known to be potent dopamine receptor antagonists.

Lerner et al [54] have found that visual hallucinations in Alzheimer's disease patients are common, often occur in the presence of specific behavioural disturbances, and may therefore have management implications. Burns et al [49] have shown that their presence leads to a more rapid decline in cognitive function.

Table 3. A scheme for classifying visual hallucinations

| Visual hallucinations that are usually perceived as real by the patient |
|--------------------------------------------------------------------------|
| These are mainly complex formed hallucinations, which may be associated  |
| with other features of psychosis, and with hallucinations in other       |
| sensory modalities. This group includes visual hallucinations associated |
| with:                                                                    |
| (a) psychiatric disorders such as schizophrenia                         |
| (b) all types of dementia                                                |
| (c) Parkinson's disease                                                  |
| (d) acute confusional states                                             |
| (e) delirium tremens                                                    |
| (f) some drug-induced states                                             |

| Visual hallucinations that are usually recognised as unreal by the patient (pseudohallucinations) |
|-----------------------------------------------------------------------------------------------|
| These can be divided into hallucinations that are purely visual, and those that involve other |
| sensory modalities:                                                                         |
| 1. Exclusively visual hallucinations                                                        |
| (a) Visual hallucinations secondary to eye disease—these tend to occur in the whole visual   |
| field, and usually disappear with eye closure                                               |
| (b) Hemianopic hallucinations—these occur in the hemianopic field (ie unilateral), and usually |
| disappear with saccadic eye movements                                                       |
| 2. May involve hallucinations in other sensory modalities                                  |
| (c) Peduncular hallucinations—very variable. Some patients have difficulty recognising these |
| as unreal                                                                                   |
| (d) Visual hallucinations in epileptic aura or seizures—these are short-lived, usually <30 |
| seconds, and not affected by eye movement. They can be perceived as true hallucinations in   |
| some cases.                                                                                 |
| (e) Some drug-induced visual hallucinations                                                 |
| (f) Visual hallucinations associated with migraine—mainly simple photopsias                 |
Perry [51] studied 12 patients with senile dementia of the Lewy body type (SDLT), with frozen tissue available for neurochemical analyses. Six of these patients had visual hallucinations. They found lower levels of choline acetyltransferase activity in all SDLT cases than in normal subjects, and much lower levels in the hallucinating group (50–55% reduction). If confirmed, this observation could be potentially important in selecting individual cases of dementia for cholinergic therapy.

Summary

In this review we have shown that visual hallucinations are associated with many drugs and medical conditions as well as psychiatric disease. Therefore, in order to try and ascertain the aetiology in any particular patient, and hence rule out a treatable cause, a complete evaluation is necessary.

History

A thorough history including a complete drug history is vital. The following features of the hallucination should be elicited, as they may indicate certain aetiologies:

- whether the hallucination is perceived as real by the patient, and its effect upon the patient
- the content of the hallucination, simple or complex
- whether hallucinations in other senses are involved
- whether the hallucination is in the whole visual field or just half a field
- whether the hallucination is affected by factors such as eye closure or saccadic eye movements
- the duration of the hallucination.

A scheme for classifying the different visual hallucinations is presented in Table 3.

Examination

A full examination should include:

- mental state examination
- visual acuity and fundoscopy
- full neurological assessment, eg visual fields, brain stem signs, parkinsonian signs.

Investigations

Appropriate investigations might include:

- full blood count, full metabolic screen, arterial blood gas estimation, infection screen
- CT scan or MRI of the brain
- EEG
- urinary/plasma drug screen.

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