# Visual hallucinations

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## ABSTRACT

Visual hallucinations have intrigued neurologists and physicians for generations due to patients' vivid and fascinating descriptions. They are most commonly associated with Parkinson's disease and dementia with Lewy bodies, but also occur in people with visual loss, where they are known as Charles Bonnet syndrome. More rarely, they can develop in other neurological conditions, such as thalamic or midbrain lesions, when they are known as peduncular hallucinosis. This review considers the mechanisms underlying visual hallucinations across diagnoses, including visual loss, network dysfunction across the brain and changes in neurotransmitters. We propose a framework to explain why visual hallucinations occur most commonly in Parkinson's disease and dementia with Lewy bodies, and discuss treatment approaches to visual hallucinations in these conditions.

## INTRODUCTION

A visual hallucination is the experience of seeing something that is not actually there. Those involving the perception of people or animals are often referred to as being complex, whereas those involving simple geometrical patterns, for example, in migraine, are called simple visual hallucinations. The experience of mistaking an object for an animal (eg, seeing a pile of clothes as a dog) is known as an illusion or misperception. Another form of minor visual hallucination is a passage hallucination, where an object or animal appears briefly to pass in the peripheral field.[1] Pareidolia is closely related to visual hallucinations and is defined as a tendency to perceive a specific meaningful image in an ambiguous visual pattern (box 1 provides a glossary of terms).

Perhaps 60% of people with Parkinson's disease experience complex visual hallucinations at some point during their illness,[2] and in the related dementia with Lewy bodies their presence forms part of the diagnostic criteria.[3] The Charles Bonnet syndrome is another common cause of complex visual hallucinations in neurological practice that occurs in up to 20% of people with eye disease causing low vision.[4] We propose that several interconnected processes are responsible for visual hallucinations and that these may explain their high frequency in Parkinson's disease and dementia with Lewy bodies.

### Case 1

A 76-year-old man with Parkinson's disease for 2 years began seeing several people dressed in dark clothes in the garden, particularly in the evenings. He described them as strangers, but they were not at all frightening (figure 1). He would often bring out cups of tea for them but they never thanked him or engaged in conversation. At first, he thought they were real people, but later came to accept that his mind was playing tricks with him and was no longer concerned about their presence.

Visual hallucinations associated with Parkinson's disease and dementia with Lewy bodies frequently involve the perception of people or animals. They are usually non-threatening; and may occasionally be welcomed in people who are socially isolated. They are most likely to occur in the early evening or in dim light and often are seen in the same place.[1] They are now considered to be a harbinger of dementia in Parkinson's disease.[5]

### Case 2

A 94-year-old woman described seeing thousands of small animals and children. They did not make sounds and they disappeared when she tried to touch them. She was aware that they were not real. She had a history of age-related macular degeneration but no other significant medical history. She took no medications and lived independently. Neurological examination was unremarkable apart from significantly reduced visual acuities, at 20/400 in the right eye and 20/800 in the left eye. Cognitive examination was normal. Funduscopy identified advanced geographic atrophy of both maculae.[6]

**Box 1 Glossary of terms linked to visual hallucinations**

- Complex visual hallucination: visual hallucination of a formed object, usually a person or an animal.
- Extracampine hallucination: the sense of a person being present, without seeing them.
- Minor hallucination: a term that includes visual illusions, passage hallucinations and extracampine hallucinations.
- Pareidolia: seeing meaningful objects (usually faces) in patterned surfaces or scenes.
- Passage hallucination: seeing an object fleetingly in the periphery, where that object is not there.
- Visual hallucination: the experience of seeing something in the absence of an external stimulus.
- Visual illusion: mistaking one object for another, for example, seeing a towel on the floor as a dog.

In 1769 Charles Bonnet described his blind grandfather's experience of visual hallucinations in the context of visual loss (figure 2). Although a syndrome of visual loss with visual hallucinations in the absence of dementia is now considered suggestive of Charles Bonnet syndrome, formal diagnostic criteria need to be devised. The level of impairment of visual acuity required to result in visual hallucinations is still unclear, and the possibility of mild cognitive impairment in many cases reported in the literature is unresolved. The usual cause in the developed world is age-related macular degeneration[7] but any reason for visual loss along the visual pathway can cause it; retinal, optic nerve and occipital lesions with visual hallucinations have all been described.[8] Visual hallucinations due to eye disease are usually simple and unformed, such as with posterior vitreous detachments; the term Charles Bonnet syndrome implies more complex images including people, faces and objects.[9]

**Figure 1** A painting by a man in his 80s with dementia with Lewy bodies. He had visual impairment due to cataract as well as hearing disability. His symptoms were of visual hallucinations, episodic confusion and great tiredness. Courtesy of Dr Sibylle Mayer, Skane University Hospital and Dr Elisabet Londos.

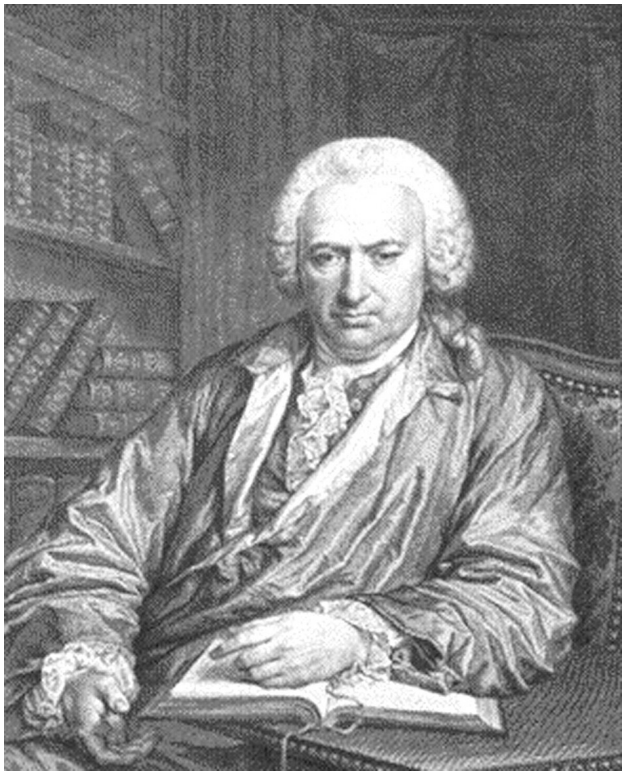

**Figure 2** A lithograph of Charles Bonnet. Reproduced from Jacob *et al*[51] with permission.

### Case 3

In 1922 Jean Lhermitte (figure 3) in his clinic at La Salpêtrière hospital in Paris, described a 72-year-old woman with double vision, headache and vomiting. She had a left abducens nerve paresis, pathologically brisk right-sided tendon reflexes and a right-sided intention tremor. Cerebrospinal fluid examination was normal. Two months later, she developed a left ptosis and left oculomotor and trochlear nerve palsies, with right tongue deviation and right-sided incoordination and weakness. It was at that juncture that she started seeing cats and chickens walking across the floor, but when trying to touch them they would disappear. She also saw people dressed in bizarre costumes and children playing with dolls.[10]

Although there was no postmortem examination on this case, Van Bogaert in 1927 described a similar patient with peduncular hallucinosis, who was confirmed at postmortem to have had a stroke involving the thalamus, cerebral peduncle, third nerve nucleus, superior colliculus, red nucleus, periaqueductal grey, decussation of the superior cerebellar peduncle and substantia nigra.[11] There have been several more recent reports of structural lesions associated with peduncular hallucinations all involving the thalamus, pons or midbrain.[12]

**Figure 3** Photograph of Jacques Jean Lhermitte. The words under the photograph are: À mon collegue et ami le Docteur Subirana. Bien cordial souvenir de la visite a Paris. (To my colleague and friend Dr Subirana. Cordial memories of the visit to Paris.) Reproduced with permission from the Spanish Society of Neurology's museum and historical archive (Sociedad Española de Neurología Museo Archivo Histórico).

These three vignettes illustrate that complex visual hallucinations with similar phenomenology involving living people and animals may occur in conditions with widely differing pathologies.

In sharp contrast, visual hallucinations in schizophrenia are relatively uncommon and invariably associated with auditory hallucinations, although in affected individuals the visions may occur sometimes unaccompanied by voices.[13] Although they can also involve life-size images of faces, people or events, they are often more bizarre and frightening,[14] and patients have poor insight.[15] Hallucinations induced by psychoactive substances (especially those active at serotonin receptors) are almost always visual,[16] and can include both geometric forms and complex hallucinations.[14] See table 1 for a summary of conditions associated with visual hallucinations.

**Table 1** Conditions associated with visual hallucinations

| | |
|---|---|
| | Parkinson's disease |
| | Lewy body dementia (including dementia with Lewy bodies and Parkinson's disease dementia) |
| | Fronto-temporal dementia |
| | Strokes: brainstem or thalamic lesions: peduncular hallucinosis |
| | Strokes: due to occipital lobe infarcts |
| | Occipital epilepsy |
| | Migraine coma |
| | Posterior reversible encephalopathy syndrome |
| Neurological causes | Creutzfeldt-Jakob disease |
| Psychiatric causes | Schizophrenia (much less common than auditory hallucinations) |
| Ophthalmic causes | Charles Bonnet syndrome |
| Drugs | |
| Medications | Levodopa |
| | Dopamine agonists |
| | Anticholinergics |
| | Opiates |
| | ACE inhibitors |
| | Baclofen withdrawal |
| | Alcohol intoxication or withdrawal |
| | Barbiturate and benzodiazepine withdrawal |
| Psychoactive drugs | Lysergic acid diethylamine (LSD) |
| | Psilocybin |
| | Mescaline |
| | Cannabis |
| | Dimethyltryptamine |
| | Phencyclidine (Angel dust) |
| Miscellaneous | Narcolepsy-cataplexy syndrome |
| | Bereavement |
| | Sensory deprivation: shipwreck survivors |

### Potential mechanisms involved in visual hallucinations

#### Impaired visual processing

In Charles Bonnet syndrome, it is the absence of visual input, or de-afferentation that probably causes hallucinations through some form of cortical release.[17] This is based on the notion that bottom-up visual processing is usually inhibitory and when this is lost, for example, as a consequence of blindness, spontaneous cortical activity is released. This notion, however, fails to explain why most blind people do not experience complex visual hallucinations, and does not explain the particular animate nature of the visions.

Patients with Parkinson's disease and those with dementia with Lewy bodies who have visual hallucinations are more likely to have visual deficits. Two large epidemiological studies showed that visual deficits due to any cause were linked with poorer outcomes in Parkinson's disease, including depression, dementia and even death.[18 19] We also recently showed that patients with Parkinson's disease and visual dysfunction are more likely to show cognitive deficits at follow-up, with more widespread changes in white matter structure.[20] Patients with Parkinson's disease and visual hallucinations have more deficits in higher order vision compared with those with no visual hallucinations.[21] A study from Canada showed that Parkinson's patients with colour vision deficits are also more likely to develop dementia[22] and changes are even seen earlier in the visual pathway, with retinal thinning found in Parkinson's disease patients with visual hallucinations.[23] In dementia with Lewy bodies, impaired colour vision is specifically related to the presence of and severity of visual hallucinations.[24] It seems probable, therefore, that there is a connection between

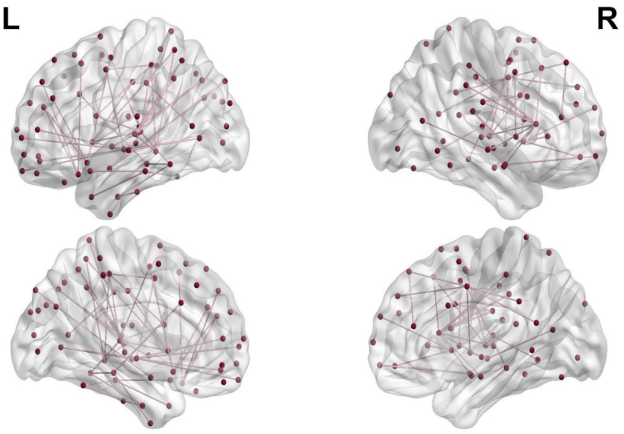


**Figure 4** Network-based statistics analysis comparing the structural brain networks of patients with Parkinson's disease and visual hallucinations compared with Parkinson's disease without hallucinations. A subnetwork of reduced connectivity is shown. Reproduced from Zarkali *et al*[28] open licence.

visual processing deficits in Parkinson's disease and dementia with Lewy bodies and visual hallucinations.

### Changes in brain networks

No consistent region of cerebral atrophy has been found in patients with visual hallucinations[25] and recent work has suggested that it is more likely that they arise from a shift in the relative weighting of different brain networks. We recently showed that patients with Parkinson's disease who had visual hallucinations were more dependent on prior knowledge compared with patients with no hallucinations, suggesting that information received from higher cognitive centres was more heavily weighted.[26]

The default mode network becomes activated during daydreaming and introspection. It has been suggested that it is over-activated in patients who develop visual hallucinations and that at the same time there is an associated failure in activating the dorsal attention network.[27] Reduced connectivity in a subnetwork involved in the integration of information across the brain has also been reported in Parkinson's disease patients with hallucinations[28] (figure 4).

The importance of brain networks, rather than particular brain regions, was also recently supported by a meta-analysis of peduncular hallucinosis.[12] Although traditionally thought of as arising from the pons, midbrain or thalamus, the lesions causing peduncular hallucinosis were distributed heterogeneously with little overlap in location. The lesions however, did share functional localisation to extra-striate visual regions.[12]

### Thalamus as a driver of network change

The thalamus is a diencephalic hub critical for filtering sensory information. Although traditionally considered to be a relay station between different brain regions, its importance in controlling shifts between brain networks is now becoming clearer and it has recently been suggested to be a driver of the network changes seen in patients who hallucinate.[29] Hypometabolism and atrophy of the thalamus have been found in patients with Parkinson's hallucinations[30] and also in patients with fronto-temporal dementia reporting hallucinations.[31] Reduced connectivity between the thalamus and the prefrontal cortex has also been reported in psychosis.[32] We recently reported that specific thalamic subnuclei are affected in people with Parkinson's disease who have visual hallucinations, with most changes occurring in the right medio-dorsal medial nucleus and the tracts connected to it.[33]

The model of thalamo-cortical dysrhythmia links thalamic dysfunction with network changes in the default mode network.[34] This proposes that hallucinations arise through thalamic-driven decoupling of the default mode network from fronto-parietal and attention networks and is accompanied by thalamic-driven theta EEG rhythms. This idea is largely based on studies showing that Parkinson's disease dementia and dementia with Lewy bodies are associated with progressive theta rhythms,[35] which in turn predict cognitive decline and fluctuations in Parkinson's disease.

## Neurotransmitters affecting visual hallucinations

### Dopamine

For many years after the introduction of dopaminergic therapy to treat Parkinson's disease, visual hallucinations were considered a reversible drug-related adverse effect rather than related to the disease process itself. The observation of 'off period' visual hallucinations in a few patients[36] and the emergence of dementia with Lewy bodies as an entity distinct from Alzheimer's disease or vascular dementia (where visual hallucinations are a striking feature) called this into question. There are now clear reports of frequent visual hallucinations, including complex hallucinations in early-stage Parkinson's patients who have not yet started dopaminergic treatment.[37 38] Nevertheless there is little doubt that dopaminergic therapy can lower the threshold for the emergence of visual hallucinations in Lewy body disease although with no clear link to dosage.[39] Dopamine has a role in encoding the salience or importance of an object[40] and dopaminergic therapy can also make hallucinations more distressing by enhancing the perceived salience of a given visual stimulus.[41]

### Acetylcholine

Visual hallucinations are a common adverse effect of anticholinergic medications in the elderly and after poisoning with solanaceous plants such as deadly nightshade. Acetylcholine is linked with enhancing the precision of sensory signals, possibly thorough nicotinic and muscarinic receptors as part of the thalamo-reticular nucleus. This helps to modulate

sensory information by increasing relevant stimuli and suppressing signals thought to be noise.[42] Greater availability of acetylcholine might therefore enhance sensory precision and bottom-up information, making visual hallucinations less likely. This is likely to be the mechanism for particular benefit of rivastigmine, an acetylcholinesterase inhibitor, on Parkinson's patients with visual hallucinations.[43]

### Serotonin

The serotonergic system influences both early and late stages of sensory processing, as well as affecting behavioural responses to visual information.[44] Particular receptors involved in visual processing include 5HT2A receptors, which are strongly expressed in the visual cortex, and 5HT3 receptors, which modulate the release of other neurotransmitters, especially acetylcholine, dopamine and glutamate. Many psychedelic drugs including lysergic acid diethylamide (LSD-25) induce hallucinations through their effects on 5HT2A receptors. There has also been interest in agents that are active at serotonergic receptors as potential treatments for visual hallucinations. Clozapine has activity at serotonergic receptors[45] and pimavanserin is a 5HT2A inverse agonist, now licensed in the USA to treat psychosis and visual hallucinations in Parkinson's disease.[46] It has been claimed that it is more effective in patients with greater cognitive impairment.[47] Ondansetron is a 5HT3 antagonist that is licensed for use as an anti-emetic. Open label studies previously showed some potential benefits in Parkinson's hallucinations,[48] but the higher cost of ondansetron at the time of those studies meant that larger trials were not conducted. However, it now has a lower cost, and a randomised controlled trial is underway to formally test its effectiveness in Parkinson's hallucinations (TOP HAT | Psychiatry - UCL – University College London).

## Combination of factors at play?

It is of considerable interest that visual hallucinations are common in Parkinson's disease and in dementia with Lewy bodies, but rare in other neurodegenerative conditions with different pathological signatures and regions of selective vulnerability. It may be that a combination of dysfunction across lower precision ascending sensory inputs combined with neurotransmitter imbalance in subcortical structures and loss of network integrity promotes visual hallucinations. The observation that visual hallucinations strongly predict poor outcomes, including dementia and death, in Parkinson's disease raises the possibility that more extensive network involvement including thalamic dysfunction has already occurred in these susceptible individuals.

## Practical management of visual hallucinations in Parkinson's disease and dementia with Lewy bodies

The large majority of patients with Parkinson's disease who develop visual hallucinations need no symptomatic treatment other than a recommendation to light their home well in the evenings. Some patients report that they can make the apparitions disappear if they stare at the people that they see, or talk to them.

On the other hand, frightening or threatening visual hallucinations, often associated with paranoia and delirium, are a medical emergency sometimes requiring urgent hospital admission. In this situation the first step always is to examine the patient carefully and to exclude and treat intercurrent urinary or respiratory tract infections, metabolic abnormalities, refractory constipation and to enquire about recent falls. Patients receiving anticholinergic medications need these to be slowly reduced before starting to reduce the dopaminergic medication. Adjuvant dopaminergic therapies including dopamine agonists and both monoamine oxidase and catechol-O-methyl transferase inhibitors should be tailed off in steps over 10 days. If severe immobility ensues, the levodopa dose may need to be increased. If a patient is receiving opioids or other medications known to provoke visual hallucinations, these should also be stopped.

Cholinesterase inhibitors reduce the frequency and intensity of visual hallucinations. The evidence for benefit is best for rivastigmine and donepezil but if these are ineffective or poorly tolerated, memantine and galantamine can also be tried.[49] In some cases, persistent and distressing visual hallucinations require antipsychotic medication. These should be used with caution as they are linked with higher rates of morbidity and mortality, with side effects including worsening of parkinsonism and cognitive function as well as falls and sedation. Current guidance from the National Institute of Health and Care Excellence recommends quetiapine as first-line treatment, despite weak evidence for its efficacy, due to its relatively good safety profile. Treatment is best started at a very small dose of 12.5 mg at night and built up slowly until the psychosis has settled, following which an attempt should be made to slowly withdraw it.

Clozapine is active at serotonergic receptors (5HT2A, 5HT2C, 5HT6 and 5HT7), as well as having affinity for dopaminergic receptors and has much better evidence for efficacy than quetiapine. However, due to concerns of agranulocytosis, it can be prescribed only in specialist mental health settings and requires daily heart rate and blood pressure checks initially and weekly blood tests for the first 18 weeks. It should be considered in anyone who fails to benefit from quetiapine, starting at a dose of 6.25 mg and increased up to a maximum of 50 mg/day.

Other treatment approaches for visual hallucinations are currently being evaluated, including antidepressants and also apomorphine. Interventions such as electroconvulsive therapy, transcranial magnetic stimulation and transcranial direct current stimulation are also being investigated (see Powell *et al*[50] for a more detailed review on these techniques).

## Key points

- The most common causes of complex visual hallucinations are Parkinson's disease and dementia with Lewy bodies; these usually manifest as silent people and animals.
- Similar phenomena occur in the Charles Bonnet syndrome associated with visual loss, and more rarely in peduncular hallucinations associated with thalamic and midbrain structural lesions.
- Certain drugs that affect dopaminergic, cholinergic or serotonergic transmission can provoke visual hallucinations.
- Visual impairment, central neurotransmitter derangement and changes in thalamic connectivity may all be causally involved

## Further reading

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## SUMMARY

The phenomenology of visual hallucinations in Parkinson's disease and in dementia with Lewy bodies closely resembles that in the Charles Bonnet syndrome and the peduncular hallucinosis of Lhermitte, but is distinct from the complex visual hallucinations reported in other neurological and psychiatric conditions. A combination of visual perceptual impairment, defective modulation of thalamocortical circuitry and involvement of ascending cholinergic and serotonergic transmitter pathways may be responsible.


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