

## REVIEW

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# Hallucinations Experienced by Visually Impaired: Charles Bonnet Syndrome

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### ABSTRACT

Charles Bonnet Syndrome is a condition where visual hallucinations occur as a result of damage along the visual pathway. Patients with Charles Bonnet Syndrome maintain partial or full insight that the hallucinations are not real, absence of psychological conditions, and absence of hallucinations affecting other sensory modalities, while maintaining intact intellectual functioning. Charles Bonnet Syndrome has been well documented in neurologic, geriatric medicine, and psychiatric literature, but there is lack of information in optometric and ophthalmologic literature. Therefore, increased awareness of signs and symptoms associated with Charles Bonnet Syndrome is required among practicing clinicians. This review of the literature will also identify other etiologies of visual hallucinations, pathophysiology of Charles Bonnet Syndrome, and effective management strategies.

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Key Words: visual hallucinations, Charles Bonnet Syndrome, impaired vision, visual pathway, neurobiology, low vision, phantom vision

Charles Bonnet Syndrome, also known as “phantom vision,” is a condition where visual hallucinations occur as a result of damage along the visual pathway. People experiencing Charles Bonnet Syndrome have intact cognition, insight that the visual hallucinations are not real, and absence of other psychological conditions.<sup>1</sup> Visual hallucinations may also occur in neurological diseases, metabolic disorders, psychiatric conditions, illicit drug use, and use of certain medications. Patients do not willingly disclose that they experience visual hallucinations for fear of not being taken seriously or fear of being labeled as mentally unstable by their loved ones and/or health care providers. Charles Bonnet Syndrome has been well documented in the neurologic, geriatric medicine, and psychiatric literature. There is lack of information in the optometric and ophthalmologic literature; therefore, it is still poorly understood by eye care professionals. This review of the literature aims to increase

awareness about Charles Bonnet Syndrome, improve recognition of the symptoms, identify the condition, and offer management strategies among practicing clinicians.

### HISTORY

In 1760, a Swiss scientist named Charles Bonnet, who was well known for his contributions to botany and philosophy, first described visual hallucinations associated with Charles Bonnet Syndrome. Bonnet documented the complex visual hallucinations experienced by his 90-year-old grandfather, Charles Lullin, and published his findings in an essay.<sup>1</sup> His grandfather underwent cataract surgery in both eyes. The bilateral cataract surgery initially improved his vision, but his vision deteriorated over time. The visual hallucinations occurred shortly after his vision loss. Charles Lullin’s hallucinations included detailed figures of men, women, birds, animals, buildings, tapestries, and carriages with variations in size, shape, and place.<sup>1,2</sup> He knew that these visual hallucinations were not real, but he was uncertain why he was experiencing them. He was in good overall health without any psychiatric disorders.<sup>1</sup>

Ironically, Charles Bonnet suffered from an unknown cause of painful vision loss at 20 years of age. The vision loss progressed to severe visual impairment by the age of 40. He also experienced visual hallucinations. However, it was not until 1967 that another Swiss scientist, George De Morsier, coined the term, “Charles

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Bonnet Syndrome.” De Morsier was known well for his contributions in neurodevelopmental disorders.<sup>1</sup>

## PREVALENCE

The prevalence of Charles Bonnet Syndrome varies and ranges from 0.4% up to 30%, depending on the study.<sup>1,3–6</sup> Khan et al.<sup>4</sup> noted that the incidence of Charles Bonnet Syndrome was statistically higher in subjects with worse visual acuity. This finding demonstrated worse vision is associated to some degree with increased risk of experiencing visual hallucinations.<sup>4</sup>

There are several reasons why variability in the prevalence rates of Charles Bonnet Syndrome exist. First, there is no universal definition of Charles Bonnet Syndrome agreed upon by all healthcare professionals. Second, the diagnosis of Charles Bonnet Syndrome is made across different disciplines including optometry, ophthalmology, geriatric medicine, psychiatry, and neurology. Third, many people are hesitant to report that they experience visual hallucinations for fear of being labeled as mentally unstable.<sup>1,7–9</sup> As the geriatric population increases, the amount of acquired vision loss will also increase. As a result, there will be an increase in those affected by Charles Bonnet Syndrome. As health care providers, we need to understand and recognize this condition so these individuals can be properly managed. In a study by Gilmour et al., only 9% of people experiencing Charles Bonnet Syndrome sought medical advice. Disappointingly, only half of them received an explanation about Charles Bonnet Syndrome.<sup>48</sup>

In another study, 15.2% of the participants informed their eye health professional about the visual hallucinations.<sup>54</sup> Cox and Ffytche investigated 492 people with Charles Bonnet Syndrome. There was a trend association between the quality of information provided by their medical professional and whether a negative outcome was experienced. Forty-seven percent of the people with Charles Bonnet Syndrome were not given a clear explanation and experienced negative outcomes whereas 36% of people with Charles Bonnet Syndrome were given a clear explanation. They also found one third of the medical professionals were either uncertain or unaware about Charles Bonnet Syndrome.<sup>56</sup>

## DEFINITIONS

There are many definitions that have been used to classify Charles Bonnet Syndrome, but there has been no consensus on which definition is most accurate. In 1935, According to De Morsier’s definition,<sup>10</sup> Charles Bonnet Syndrome implied a localized neurodegeneration, such as is evident in Parkinson’s disease, Alzheimer’s disease, and peduncular hallucinosis. He strongly believed Charles Bonnet Syndrome occurred in the elderly with normal cognition and without correlation to ocular pathology. The fact that Charles Bonnet’s grandfather had impaired vision was merely a coincidence that ocular pathology is common in the elderly.<sup>10</sup> De Morsier was not able to provide evidence to confirm his hypothesis.

In 1936, L’Hermitte and de Ajuriaguerra’s definition implied visual hallucinations involve thalamic lesions in addition to ocular pathology.<sup>10</sup> They did not agree with De Morsier’s definition that Charles Bonnet Syndrome occurred in the absence of ocular pathology. L’Hermitte and de Ajuriaguerra’s definition coincided

with Bonnet’s original description of visual hallucinations experienced by his grandfather.

Gold and Rabins’ definition<sup>10</sup> (1989) is most commonly used in the psychiatric literature. They describe the visual hallucinations in Charles Bonnet Syndrome to be stereotyped, formed, complex, persistent, or repetitive in nature.<sup>10</sup> Further, they suggest that those with Charles Bonnet Syndrome are aware that the hallucinations are not real. In their definition, there are no delusions or hallucinations affecting other sensory modalities. Ocular pathology is not part of their diagnostic criteria.<sup>10</sup>

Ophthalmologists and neurologists use Charles Bonnet Syndrome to describe visual hallucinations that occur as a result of ocular pathology or visual pathway disease. The type of hallucinations and age of the patient are of secondary importance.<sup>12</sup> The definition used by ophthalmologists and neurologists supports Bonnet’s original description of the condition. An important consideration about the ophthalmologically defined Charles Bonnet Syndrome is that it carries an inherent ambiguity: Was ocular pathology or loss of acuity the contributing factor for the visual hallucinations? Diagnostic criteria for Charles Bonnet Syndrome still remains controversial.

## CHARACTERISTICS OF CHARLES BONNET SYNDROME

Visual hallucinations experienced by Charles Bonnet Syndrome patients are typically simple or complex in nature, although a full spectrum of hallucinations can occur. Simple visual hallucinations, which are sometimes described as elementary or formed, are composed of photopsias, simple shapes, grid-like patterns, and branching patterns. Complex visual hallucinations are made up of vivid and complicated images of people, faces, vehicles, animals, flowers, trees, plants, and miniature images of people and objects.<sup>7</sup>

The visual hallucination episodes last seconds to a few hours, with recurrent episodes of visual hallucinations occurring for days to years. A majority of the visual hallucinations are strange or bizarre to the patients, but they are seldom disturbing. Generally, people experiencing visual hallucinations remain neutral about the hallucinations.<sup>1</sup>

In a study done in the United Kingdom, 38% of the 492 subjects described the visual hallucinations as frightening, terrifying, and startling during the initial onset of them. Over time, their emotions towards the hallucinations decreased to 8%.<sup>56</sup> They also found 60% of subjects reporting the visual hallucinations did not affect their lives, 33% of subjects felt it had a negative effect on their lives, and only 7% felt it had a pleasant effect on their lives.<sup>56</sup>

A Canadian study mirrors Cox et al.’s results citing 63% individuals experienced a neutral reaction to the hallucinations, 34% had a negative reaction, and 6% had a positive response to them.<sup>48</sup> These recent findings demonstrate the prevalence of negative reactions to visual hallucinations are higher than previously reported by Holroyd and Rabins.

In 2008, Vukicevic and Fitzmaurice found 51.5% of their participants report mild stress compared to 30% of participants who report severe stress as a result of visual hallucinations. The stress was not a result of the images seen, rather it was primarily due to concern about the underlying cause of the hallucinations.<sup>54</sup>

In a case report, Vukicevic demonstrated how a stressful life event can change the nature of hallucinations experienced in Charles Bonnet Syndrome from non-distressing to distressing. An 80-year-old woman with diagnosed age-related macular degeneration and closed-angle glaucoma had been experiencing Charles Bonnet Syndrome for 4 years. Her hallucinations became atypical on February 7, 2009. This date was marked by intense heat and bushfires in southern Australia known as “Black Saturday.” She lived in an area affected by the fires and evacuated from her house on that day. The patient likely suffered from acute stress disorder because she had a history of fire-related trauma. The change in nature of her hallucinations may represent a relationship between Charles Bonnet Syndrome and acute or posttraumatic stress disorder.<sup>53</sup>

Complex visual hallucinations are emphasized in the Charles Bonnet Syndrome definitions. However, Charles Bonnet Syndrome patients experience simple visual hallucinations most frequently. Even though these patients have some level of impaired vision, the visual hallucinations are in more detail and with better clarity than their residual vision. These visual hallucinations are vivid images such as that seen in normal viewing conditions and are not under voluntary control.<sup>13,45</sup>

Charles Bonnet Syndrome hallucinations are purely visual in nature and do not involve hallucinations in other sensory modalities.<sup>14,49</sup> Further, Charles Bonnet Syndrome visual hallucinations occur in a clear state of consciousness when the patient is alert and awake. These patients show no signs of dementia and maintain preserved intellectual functioning including mental awareness, perception, reasoning, judgment, processing information, and ability to apply knowledge.<sup>14</sup> People experiencing the hallucinations maintain insight that the hallucinations are not real.

In a Canadian study, 80% of participants knew that the visual hallucinations were not real after the first episode, followed by 8% knew by the second episode, and only 6% knew by 10 or more episodes. The researchers also found that a majority of the participants (57%) saw the same objects with every hallucinatory event. They also found 51% participants hallucinated in color whereas 49% hallucinated in black and white.<sup>48</sup>

### Charles Bonnet Syndrome and Visual Acuity

Madill and colleagues studied the occurrence of Charles Bonnet Syndrome in four patients with advanced glaucoma.<sup>12</sup> They challenged previous authors who stated that acuity loss is required for ophthalmologically defined Charles Bonnet Syndrome. In these advanced glaucoma patients, visual acuity ranged from 20/20 to 20/40 monocularly with extensive visual field defects. No hallucinations occurred in other sensory modalities, and all maintained insight that the hallucinations were not real. The results of Madill et al.’s case study coincide with the ophthalmological definition of Charles Bonnet Syndrome emphasizing ocular pathology or visual pathway disease as a cause of the visual hallucinations. It is important to understand this case series emphasizes visual acuity loss is not a requirement for Charles Bonnet Syndrome visual hallucinations to occur.<sup>12</sup>

With that said, an association between age-related macular degeneration (AMD) and colored visual hallucinations has been

proposed. Signals related to color vision pass through the parvocellular layers of the lateral genicular nucleus (LGN) and are transmitted along the ventral color pathway. In patients with macular degeneration, there is degeneration throughout the ventral color pathway.<sup>15</sup> Damage to cone photoreceptors in the macula creates a lack of visual input to the color areas of the visual association cortex. Because of reduced visual input to the visual association cortex, endogenous activation within the color area results in colored hallucinations. Functional magnetic resonance imaging (fMRI) provides support for this theory, as the color area of the visual association cortex remained hyperactive in Charles Bonnet Syndrome patients with macular degeneration.<sup>15</sup>

Because of the physiological deafferentation with ganglion cell loss in glaucoma, they can experience Charles Bonnet Syndrome without an acuity loss, whereas in AMD, there is loss of central retinal ganglion cells, which causes Charles Bonnet Syndrome to occur with an acuity loss.<sup>12</sup> Therefore, patients with significant ocular disease and preserved visual acuity may still be at risk for developing Charles Bonnet Syndrome.

In a study evaluating patients only with AMD, their extent of vision loss did not predict which patients would experience visual hallucinations. The investigators also found that the extent of vision loss did not determine the complexity of visual hallucinations experienced.<sup>57</sup> Gilmour and colleagues found no statistical significance in patients with dry or wet AMD and likelihood of having visual hallucinations. Patients with both types of AMD experienced Charles Bonnet Syndrome.<sup>48</sup>

### No Age Limit for Charles Bonnet Syndrome

According to Khan et al.<sup>4</sup> and Menon’s studies,<sup>16</sup> there were no increased risks of Charles Bonnet Syndrome with increased age. Although Charles Bonnet Syndrome affects geriatric populations because of increased incidence of eye disease in the elderly, it can affect all ages after vision loss.<sup>7</sup> In the original definitions of Charles Bonnet Syndrome, there have been no references to age being required as part of the inclusion criteria. Although very few studies of Charles Bonnet Syndrome in children are found in the literature, Schwartz and Vahgei reported two cases of Charles Bonnet Syndrome that occurred in children after profound vision loss.<sup>17</sup> Both children had a diagnosis of cone-rod dystrophy accompanied by vivid and complicated visual hallucinations.

The children’s visual hallucinations were very similar to hallucinations described by adults with the condition.<sup>17</sup> The complicated visual hallucinations included flashing lights, people, faces, wolves, snowballs, ballerina, houses, and colored balls. No other sensory modalities were affected. This study highlighted the important concept that age is not a criterion for the diagnosis of Charles Bonnet Syndrome.<sup>17</sup> Visual hallucinations have not been reported by those with congenital blindness. In fact, Charles Bonnet Syndrome has only been reported as being partially related to visual impairment.<sup>18</sup>

### ETIOLOGIES OF VISUAL HALLUCINATIONS

Determining the etiology responsible for the visual hallucinations is essential to implement the appropriate management.

Table 1 outlines a sampling of the common etiologies of visual hallucinations.

## Ocular Pathologies

Crane et al. evaluated the prevalence of Charles Bonnet Syndrome. In their study of 284 individuals, 57.7% had macular disease, 19.7% had retinal disease, 15.4% had neuropathic disease, and 7% had ocular disease of other etiologies (amblyopia, cataract, central retinal artery occlusion, central retinal vein occlusion, cerebrovascular accident, corneal scar, nystagmus, pseudophakic bullous keratopathy, and uveitis).<sup>52</sup> O'Hare et al. found 37% individuals with advanced retinitis pigmentosa experienced Charles Bonnet Syndrome.<sup>55</sup>

## Neurologic Disorders

### Parkinson's Disease

The pathology of Parkinson's disease extends beyond the substantia nigra to include widespread brainstem changes and

various neurotransmitter systems. There is significant loss of neurons in the brainstem of Parkinson's disease patients. In these patients, there are defects in the visual pathway associated with dopamine deficiency in the retina and central pathways. Levodopa increases the dopamine levels, which should also improve the visual system's function.<sup>19</sup> In those with Parkinson's disease, visual hallucinations are attributed to decreased vision, cognitive impairment, dopaminergic medications, or anticholinergic medications.<sup>20</sup>

According to Manford and Andermann in 1998, visual hallucinations were not reported until after Parkinson's disease treatment was initiated with anticholinergics, Levodopa, and dopaminergic agonists.<sup>19</sup> More specifically, in patients who took Levodopa, their hallucinations began after 10 years of using the medication. It was thought that hallucinations were directly related to the dosage of Levodopa. However, a direct toxic effect of Levodopa on vision seems unlikely to cause hallucinations.<sup>19</sup> Visual hallucinations usually occur towards the end of day, associated with vivid dreams, disturbances in sleep, and changes in arousal. Parkinson's disease patients maintain insight that the hallucinations are not real.

**TABLE 1.**  
Etiologies of visual hallucinations

Neurologic disorders	Psychiatric disorders	Illicit drugs	Metabolic disorders	Miscellaneous conditions	Charles Bonnet Syndrome	Prescription medicines
Intracranial tumors	Schizophrenia	LSD	Drug withdrawal	Hypnagogic hallucinations (transitional state between waking and sleeping)	Macular diseases	See Appendix A
Peduncular hallucinosis	Delirium	Psilocin	Alcohol withdrawal	Hypnopompic hallucinations (transitional state between sleeping and waking)	Retinal diseases	Medication side effects
Alzheimer disease	Acute psychoses	Psilocybin	Cardiopulmonary insufficiency	Narcolepsy-cataplexy syndrome	Optic nerve disease	
Aneurysms	Bipolar disorder	PCP	Uremia	Sleep deprivation	Central retinal artery occlusion	
Seizure disorders	Depression	Angel dust	Hepatic disease	Food deprivation	Central retinal vein occlusion	
Lewy body dementia		Mescaline	Endocrine disturbances	Water deprivation	Field deficits without visual acuity loss	
Encephalitis		Cannabis	Vitamin deficiencies	Prolonged social isolation		
Meningitis		Amphetamines	Inflammatory disease	Excessive fatigue		
Head trauma		Methylphenidate	Infectious disease	Excessive stress		
Infarcts		Cocaine				
Migraines						
Epilepsy						

According to Biousse and colleagues (2004), one fourth of 30 Parkinson’s disease patients experienced complex visual hallucinations.<sup>20</sup> Some authors believe visual hallucinations in Parkinson’s disease indicate the development of dementia.<sup>14,19,21</sup> However, the Biousse study found no dementia after 2 years of follow-up.<sup>20</sup>

**Peduncular Hallucinosis**

Peduncular hallucinosis shows the closest clinical overlap with Charles Bonnet Syndrome.<sup>22</sup> The underlying etiology of peduncular hallucinosis is vascular in nature. Complex visual hallucinations occur as a result of lesions in the midbrain and/or thalamus. The term “peduncular” does not only imply cerebral peduncles but includes the midbrain and surrounding area. Peduncular hallucinosis has been associated with other central nervous system pathologies, including vascular and infectious pontine, midbrain, and thalamic lesions, local subarachnoid hemorrhage, compression by local and distal tumors, basilar vascular hypoplasia, basilar migraine, and after surgical and angiographic interventions.<sup>22</sup> Common hallucinations experienced by these individuals include people, animals, landscapes, grotesque and deformed faces, repeated patterns, and Lilliputian hallucinations. Lilliputian hallucinations are visual phenomena in which the hallucinations are miniature in size.

Peduncular hallucinosis typically occurs a few days after the infarct. Each hallucination episode will last for a few minutes to several hours and can be accompanied by tactile or auditory hallucinations differentiating it from Charles Bonnet Syndrome. Patients with peduncular hallucinosis may experience hallucinations for several weeks to years. These patients do maintain insight that the hallucinations are not real.<sup>19</sup> Treating the underlying central nervous system pathologies will resolve the hallucinations. Therefore, it is important to screen radiologically for central nervous system disease to rule out peduncular hallucinosis.

**Multiple Sclerosis**

With the ocular and cerebral manifestations of multiple sclerosis, there are reports of Charles Bonnet Syndrome visual hallucinations that can occur. These patients will usually have some cognitive impairment due to the disease. According to Chen and colleagues, a woman with multiple sclerosis lost vision from optic neuritis and began experiencing complex and vivid visual hallucinations.<sup>9</sup> In another case, a woman with multiple sclerosis noticed that her visual hallucinations disappeared once she regained vision.<sup>23</sup> These case studies highlight that dynamic changes in visual acuity may play a role in eliciting Charles Bonnet Syndrome visual hallucinations, although it has not been proven.<sup>24</sup>

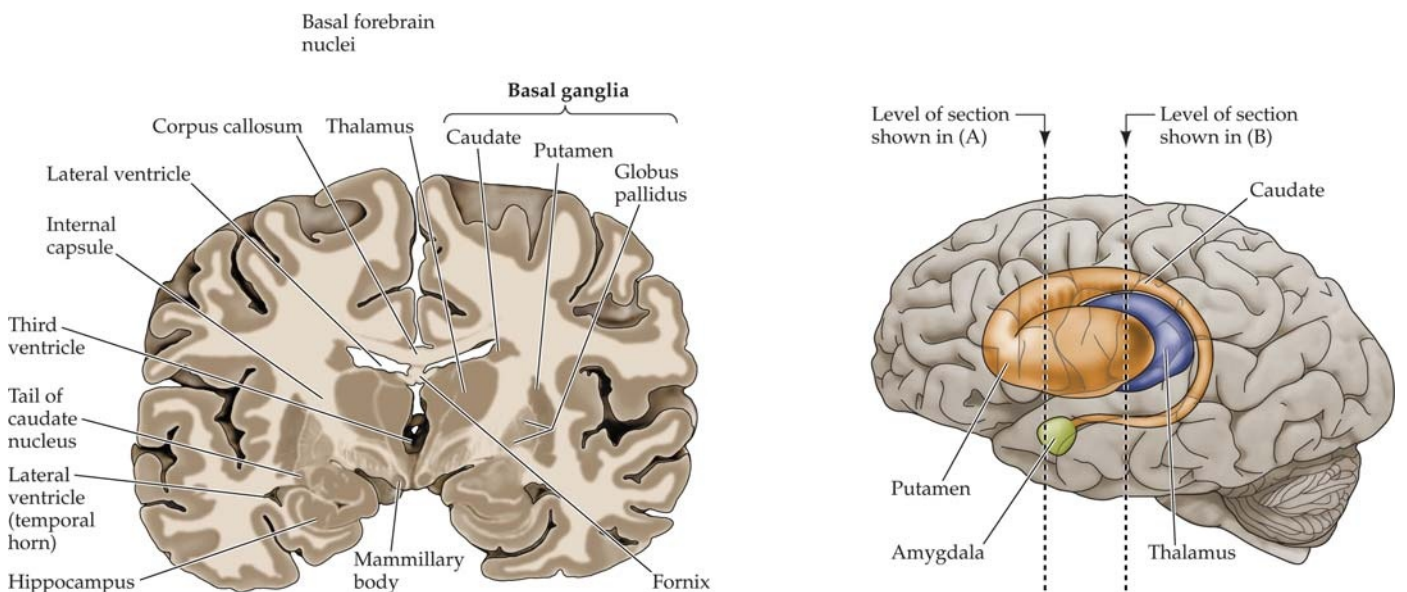
**Epilepsy**

Visual hallucinations associated with epilepsy are different from other hallucinatory events. These hallucinations are brief, simple, and tend to be associated with seizure manifestations. Intracranial electroencephalograph and direct cortical stimulation experiments demonstrate excitation of visual cortical areas. A hallmark sign of epileptic hallucinations is excitation in the visual association cortex.<sup>19</sup> Complicated visual hallucinations that occur in epilepsy involve the posterior parietal or temporal association cortex.<sup>19</sup>

**Conditions Affecting Blood Supply**

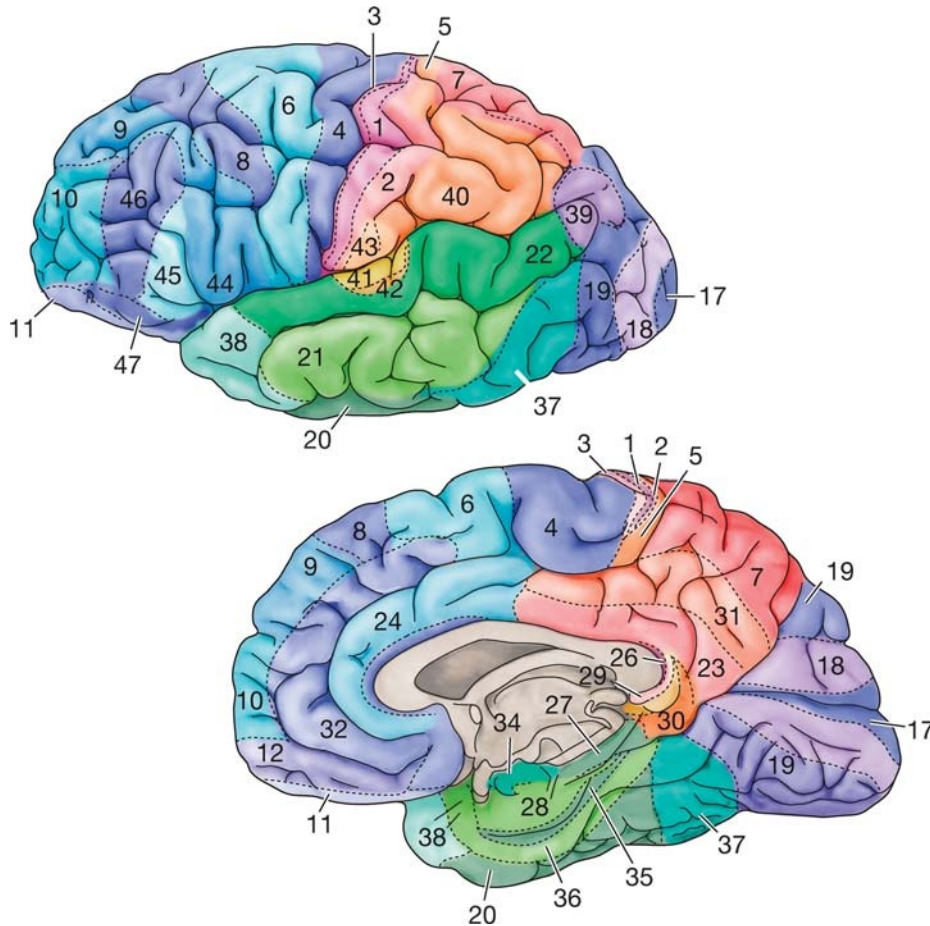
**Migraines**

Auras before migraine headaches have been well recognized as a cause of simple visual hallucinations. It is rare to experience complex visual hallucinations with migraines. Investigators suggest that depression of cortical activity, in conjunction with changes in blood flow, are responsible for generation of migraine auras.<sup>19</sup> The most common symptoms reported by patients with



**FIGURE 1.**

Diagram identifying the thalamus and striatum. Reproduced with permission from Purves DL, et al., eds. Neuroscience, 4th ed. Sunderland, MA: Sinauer; 2008.



**FIGURE 2.**

Visual cortical areas, including Brodmann Area 17 (primary visual cortex), Brodmann Area 18 (secondary visual cortex), and Brodmann Areas 19 and 37 (visual association cortices). Reproduced with permission from Purves DL, et al., eds. *Neuroscience*, 4th ed. Sunderland, MA: Sinauer; 2008.

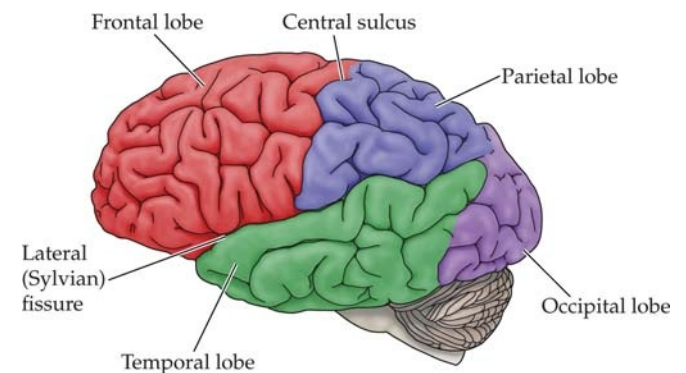
visual auras preceding migraines include photopsias and simple shapes and patterns, along with very bright and colorful images.<sup>7</sup> Ocular migraines without headaches are encountered frequently in eye care practices. Therefore, practitioners must distinguish between visual hallucinations related to ocular migraines, typical migraines, and Charles Bonnet Syndrome to appropriately manage the visual hallucinations.

### Infarcts

In cases of infarcts causing visual hallucinations, ischemia is the underlying etiology of cortical lesions. The onset of visual hallucinations occurs days to weeks after the ischemic event. These hallucinations have a typical duration of days to weeks, but the frequency may persist over time. The majority of complicated visual hallucinations, which are reported after stroke, are due to occipital infarcts, rather than middle cerebral artery infarcts.<sup>19</sup> This is consistent with the hypothesis that epilepsy activates the visual association cortex directly. However, strokes stimulate the visual association cortex from a distance. Possible explanations for how strokes affect the visual association cortex include (1) loss of direct cortico-cortical inputs and (2) loss of striate cortex control of thalamic inputs to visual association cortices.<sup>19</sup>

### Regional Cerebral Blood Flow

A study evaluated regional cerebral blood flow using brain single-photon emission computed tomography (SPECT) in patients experiencing Charles Bonnet Syndrome visual hallucinations.<sup>25</sup> There was an increased perfusion in regional cerebral blood flow in the regions of the lateral temporal cortex, striatum,



**FIGURE 3.**

Diagram outlining the ventral occipitotemporal cortex (also known as the ventral occipital lobe). Reproduced with permission from Purves DL, et al., eds. *Neuroscience*, 4th ed. Sunderland, MA: Sinauer; 2008.

and thalamus in patients who experienced visual hallucinations (Fig. 1–3). It is suggested that people with vision loss have increased cortical activation in the areas of the lateral temporal cortex, striatum, and thalamus contributing to development of visual hallucinations.<sup>25,45,49</sup>

## PRESCRIPTION MEDICATIONS

Prescription medications can have a side effect of visual hallucinations by mechanisms that are still unknown.<sup>19,26,27</sup> Appendix A includes a comprehensive list of these medications (available at <http://links.lww.com/OPX/A252>).<sup>26,27</sup> Alpha-2 agonists such as brimonidine tartrate have been shown to cause systemic and neuropsychiatric phenomena, which may induce Charles Bonnet Syndrome.<sup>28</sup> Because it is a well-known lipophilic agent, it has the ability to penetrate the blood–brain barrier and cause systemic side effects such as visual and auditory hallucinations, depression, confusion, and anxiety in adults.<sup>29,44</sup> In children, it can cause adverse effects such as coma, hypotension, bradycardia, hypotony, and hypothermia.<sup>44</sup> The study by Tomsak et al.<sup>28</sup> reported four primary open-angle glaucoma subjects experienced visual hallucinations within 5 days to 2.5 months after starting brimonidine tartrate therapy.<sup>28</sup> The authors found the visual hallucinations resolved after discontinuation of brimonidine tartrate.<sup>28</sup> A case report by Garcia-Catalan et al. describes an 81-year-old woman with pseudoexfoliation glaucoma and age-related macular degeneration developed visual hallucinations after 1 month of using brimonidine with complete resolution of the hallucinations after suspending its use.<sup>44</sup> However, Rahman et al. concluded visual hallucinations are a rare side effect of the medication.<sup>29</sup>

## Illicit Drugs

Mescaline and lysergic acid diethylamine tartrate (LSD) are hallucinogens that act on specific serotonin (5-hydroxytryptamine: 5HT<sub>2</sub>) receptors. LSD is active on serotonergic neurons. With these hallucinogens, insight is maintained and there is no evidence of psychosis or delusions. The visual hallucinations may begin as visual distortions, colored patterns, or shapes. They may progress to figures of people and animals, have distortions in size, and involve feelings of fantasy.<sup>19</sup>

Stimulants, such as amphetamine and cocaine, more commonly cause auditory or tactile hallucinations. Cocaine acts on specific serotonin (5HT<sub>3</sub>) receptors, dopamine receptors, and catecholamine receptors, which are all concentrated in the limbic system. They may begin as simple hallucinations and later develop into vividly colored, complex hallucinations. They are more easily seen with eyes closed or in the dark.<sup>19</sup>

## Psychiatric Disorders

Visual hallucinations may be prominent in people with psychoses. However, those with psychosis will also have hallucinations affecting other sensory modalities which differentiates it from Charles Bonnet Syndrome.<sup>26</sup> There are many studies that evaluate whether isolated visual hallucinations may be a precursor for developing dementia.<sup>14,30,31</sup> In Guerra-Garcia's study (1997), Charles Bonnet Syndrome patients underwent a brain SPECT imaging test.<sup>31</sup> SPECT revealed reduced perfusion in the mid-parietal and occipital areas.

The hypoperfusion in the parietal area closely corresponds to SPECT patterns that exist in Alzheimer's disease and other forms of dementia. In older adults with Charles Bonnet Syndrome, the presence of other brain disorders should be investigated as a possible cause for early cognitive decline in addition to dementia.

Dementia with Lewy bodies is a common type of progressive dementia. Clinical features of dementia with Lewy bodies include progressive cognitive decline, fluctuations in attention and alertness, recurrent visual hallucinations, and motor features of Parkinson's disease. Symptoms of dementia with Lewy bodies are caused by buildup of Lewy bodies inside brain neurons responsible for memory and motor control.<sup>32</sup> Kazui et al. (2009) evaluated glucose metabolism of those patients having dementia with Lewy bodies.<sup>25</sup> Results showed glucose metabolism was decreased in the primary visual cortices; however, glucose metabolism was higher in visual association cortices of dementia with Lewy bodies patients who experienced visual hallucinations. The occurrence of visual hallucinations is a result of dysfunction in primary and secondary visual cortices but requires preserved function in visual association cortices.<sup>25</sup>

In a study of patients with AMD, Holroyd and Rabins (1996) found slightly lower cognitive scores for subjects who experienced visual hallucinations compared to cognitive scores of patients without hallucinations were not statistically significant.<sup>11</sup> Isolated visual hallucinations were not considered an early symptom of progressive dementia in this study. The relationship between visual hallucinations and development of dementia is still uncertain.

In schizophrenia, visual hallucinations occur more commonly than previously thought. It occurs typically with a delusional component that is not normally seen in organic causes. As schizophrenia progresses, the clarity of hallucinations may deteriorate, which is distinct from Charles Bonnet Syndrome.<sup>19</sup> Table 2 outlines the clinical characteristics which differentiate schizophrenia-related visual hallucinations compared to those originating from organic disease.

## PATHOPHYSIOLOGY OF CHARLES BONNET SYNDROME

The true underlying mechanism of visual hallucinations associated with Charles Bonnet Syndrome is not exactly known.<sup>1,3,4,17,23,26,33</sup> There are a few well-recognized theories. Hallucinations can result

**TABLE 2.**  
Differentiating schizophrenia-related visual hallucinations versus organic disease-related visual hallucinations

Schizophrenia	Organic disease
Waking hours, associated with increased arousal	Nocturnal, associated with drowsiness
More paranoia and thought disorder	Less paranoia and thought disorder
More personal experiences	Insight
Visual and auditory hallucinations	Visual hallucinations
Deterioration in clarity of visual hallucinations	Seen in better clarity than remaining vision
Delusions	No delusions

from release by the visual association cortex, acting by loss of corticocortical inputs, and alteration of serotonergic processes.<sup>19</sup>

## Irritative Theory

According to the irritative theory, diffuse irritative lesions in seizures and migraines<sup>26</sup> send abnormal input to the visual association cortices. From there, visual association cortices will generate excitatory discharges transmitted to occipital and temporal lobes,<sup>33</sup> which become interpreted as visual hallucinations.

## Release Phenomenon

The brain is constantly being bombarded with visual stimuli. When people see external visual information, stimuli from visual information will stop activation from within the visual association cortices.<sup>17</sup> As a result, no visual hallucinations are released.

According to release phenomenon, any deprivation to the visual system interferes with normal circuitry that inhibits activity in visual association cortex. This lack of inhibition will result in inappropriate excitation of visual association cortices<sup>18</sup> and subsequently cause visual hallucinations to be released.<sup>35,49</sup>

This causes visual images to be released from the subconscious to the conscious.<sup>45</sup>

In other words, lesions at any level of the visual pathway cause defective electrochemical impulses to be released in addition to the normal impulses. This abnormal visual processing results in visual hallucinations.<sup>54</sup>

Phantom sensations, such as auditory hallucinations, in the recently deaf or phantom tactile sensations in amputees represent similar phenomenon as Charles Bonnet Syndrome except in different sensory domains. Due to the absence of afferent sensory input to the brain, central nervous system increases activity. This increased activity is misinterpreted as hallucinations in the corresponding sensory system.<sup>26,54</sup> Some investigators describe Charles Bonnet Syndrome as phantom vision.

Researchers at Harvard-Thorndike General Clinical Research Center conducted a study to determine whether they could induce visual hallucinations by deprivation of the visual system.<sup>18</sup> The researchers utilized fMRI to study brain activation in response to stimulation. The results revealed rapid, complete, and prolonged deprivation to the visual system is enough to induce visual hallucinations in fully sighted individuals. It was also found that visual hallucinations occur more frequently with higher levels of visual impairment and bilateral blindness.<sup>18</sup> The Release Phenomenon also explains what occurs with sleep deprivation, psychoses, and use of hallucinogenic drugs.<sup>26</sup>

## Deafferentation Theory (Sensory Deprivation Theory)

Deafferentation theory is the hypothesis that is most widely accepted to explain Charles Bonnet Syndrome. Deafferentation means loss of visual input into the brain will lead to change in excitability of visual association cortex.<sup>12</sup> When sensory visual input into cortex is removed from ocular pathology or damage to visual pathways, spontaneous neuronal discharge in visual association cortex occurs, increasing excitability within visual association cortex. As a result, visual hallucinations are released.<sup>1,25,49</sup>

The increase in excitability in visual association cortex occurs for a variety of reasons: (1) increase in the number of neurotransmitters released in the presynaptic neuron caused by the presence of increased vesicles; (2) increase in the number of receptors in the postsynaptic membrane caused by prolonged inactivity, which also leads to an increase in the intensity of response; and (3) changes in the amount of gamma-aminobutyric acid and glutamatergic *N*-methyl-D-aspartic acid within the synapse that has been shown to contribute to hyperexcitability of neurons.<sup>45</sup>

Because the visual system has plasticity, it can lead to sprouting new axons in damaged areas and reorganization of receptive fields. Therefore, small amounts of remaining stimulus from the retina, optic tract, or other structures within the visual system can elicit visual hallucinations because the neurons are more sensitive.<sup>45</sup>

The sensory deprivation theory is similar to the release theory that abnormal electrochemical impulses are released in the brain. The main difference in sensory deprivation theory is due to the reduction in sensory input to the brain, which causes spontaneous discharge in visual association cortex. This is directly a result of a decrease in visual stimuli caused by ocular pathology.<sup>54</sup> In a study, 70% of those with visual hallucinations also suffered from hearing loss. It has been found that people with dual sensory loss in vision and hearing are more likely to suffer from visual hallucinations because of lack of sensory stimulation, thus reinforcing the sensory deprivation theory as cause of Charles Bonnet Syndrome.<sup>54</sup>

## VISUAL PATHWAYS

After visual information leaves the LGN, it passes along optic radiations and transmitted to the primary visual cortex (Brodmann Area 17). From the primary visual cortex, it will transmit information to the secondary visual cortex (Brodmann Area 18) and finally transfer visual information to higher processing areas of the visual association cortices (Brodmann Areas 19, 37).<sup>25</sup>

The primary visual cortex (Brodmann Area 17) is responsible for simple visual hallucinations. The secondary visual cortex (Brodmann Area 18) and visual association cortices (Brodmann Areas 19, 37) are accountable for complex visual hallucinations.<sup>2</sup>

## NEUROBIOLOGY OF VISUAL HALLUCINATIONS

Comprehending the underlying functional neurobiology will help to understand areas of the visual cortex, which are responsible for stimulating specific visual hallucinations. Neuroimaging studies are providing insight into the science of visual hallucinations. Functional MRI studies have been conducted on patients experiencing visual hallucinations. In these studies, fMRI is used to identify the cerebral location relating to visual hallucinations. Santhouse et al. (2000) found that visual hallucinations were related to increases in activity within specialized areas of the visual association cortex.<sup>15</sup>

Functional MRI studies revealed that when visual hallucinations occur, the occipital cortex has a decreased response to external visual stimuli. The decreased response to sensory input allows endogenous visual stimuli to be perceived as hallucinations.<sup>2</sup>

There is a relationship between the content of hallucinations and corresponding regions within the ventral occipital lobe. The ventral occipito-temporal cortex, also known as ventral occipital



**TABLE 3.**

Type of visual hallucinations related to specific locations within the ventral occipital lobe

Visual hallucination content	Location
Color	Posterior fusiform gyrus
Faces	Left middle fusiform gyrus
Objects	Right middle fusiform gyrus
Visual textures (bricks, fences, maps)	Collateral sulcus
Black and white hallucinations	Behind and above the posterior fusiform gyrus

lobe, is highly specialized for complex visual hallucinations, such as landscapes, figures, faces, people, vehicles, trees, shrubs, and Lilliputian hallucinations.<sup>10,13,45,49</sup> The types of visual hallucinations in relation to their specific cortical locations are outlined in Table 3.

The amount of cortical tissue available in the corresponding region has been suggested as being responsible for why certain simple or complex hallucinations occur more frequently than others.<sup>7</sup> The greater the amount of cortical tissue, the more frequently it will be endogenously activated.<sup>7</sup>

The ventral occipital lobe lies closely to the amygdala, which is responsible for emotional response to hallucinations.<sup>15</sup> In a study by Santhouse et al., they conducted a questionnaire regarding variables associated with visual hallucinations.<sup>15</sup> Half of 34 Charles Bonnet Syndrome patients did not notice an emotional connection to their hallucinations. Of the patients who did have an emotional connection to the hallucinations, 50% of them found the hallucinations to be unpleasant.<sup>15</sup>

## MANAGEMENT STRATEGIES

In management of these patients, it is important to remember Charles Bonnet Syndrome is a diagnosis of exclusion. Other etiologies that can cause visual hallucinations must be investigated and ruled out before concluding that it is Charles Bonnet Syndrome.<sup>1</sup> Charles Bonnet Syndrome may be difficult to discriminate among other comorbidities. Vukicevic and Fitzmaurice created a validated questionnaire to assess characteristics of visual hallucinations<sup>54</sup> (see Table 4).

Asking the necessary questions, understanding the predisposing factors, and providing anecdotal methods of alleviating visual hallucinations (see Table 5) aid in the differential diagnosis. Patients who meet the requisite criteria are managed using (1) low vision rehabilitation including optical intervention (spectacles, contact lenses, and optical aids), (2) surgical intervention, (3) multidisciplinary approach, (4) medical therapy, and (5) patient education.

An Australian study found that 60.6% participants reported their visual hallucinations began when the surrounding room was bright, either with natural or artificial light. They also found those who hallucinated in bright light had less episodes than those who hallucinated in dim light. A total of 39.4% participants were unaware of methods to stop the hallucinations once they started; however, 30% indicated that blinking or closing their eyes made

**TABLE 4.**

Questions to ask regarding the differential diagnosis for visual hallucinations<sup>54</sup> (authors created a validated questionnaire to assess visual hallucinations because no previous validated questionnaire was available)

- We had a patient here the other day that had a similar problem with their eyes to yourself. The condition made it difficult to see things and they also noticed that they could see things that were really not there or that other people didn't see. Has this ever happened to you?
  - Yes
  - No
- What images were you seeing during a hallucinatory episode?
  - Complex patterns
  - Animals
  - Plants, trees, foliage
  - People
  - Inanimate objects
- How frequently do you experience the hallucinations?
  - Daily
  - Weekly
  - Monthly
  - Few episodes
- How long do the hallucinations last?
  - Seconds
  - Minutes
  - Hours
- How long have you been experiencing the hallucinations?
  - 1–3 months
  - 4–6 months
  - 6–12 months
  - >12 months
- What are the lighting conditions during a hallucinatory episode?
  - Bright light
  - Dim light
  - Darkness
  - Unsure
- What are the circumstances at the onset of a hallucinatory episode?
  - Upon waking
  - Relaxed state
  - Alone at home
  - Unsure
- What do you do to make the hallucination stop?
  - Continue with activity
  - Close eyes
  - Blink
  - Turn on lights
  - Unsure
- How stressful are the hallucinations to you?
  - Mild
  - Moderate
  - Severe
- Who have you told about the hallucinations?
  - Family member
  - No one
  - Ocular health professional

the images disappear. This suggested that interrupting visual images in some way may end the hallucination episode.<sup>54</sup>

**TABLE 5.**

Predisposing factors for Charles Bonnet Syndrome (CBS) and anecdotal techniques to minimize or eliminate visual hallucinations

Predisposing factors for CBS	Anecdotal techniques to eliminate visual hallucinations
Bright light conditions <sup>54</sup>	Blinking <sup>1,12,45,46,54</sup>
Low light levels <sup>1,2,49</sup>	Improved lighting conditions <sup>1,2,9,12,45</sup>
Evening hours <sup>1,14,49</sup>	Concentrating on something else <sup>1,12</sup>
Poor social interaction with others <sup>1,2,12,37,42,45,47,49</sup>	Encouraging interpersonal contact <sup>1,9</sup>
Fatigue <sup>2,45,47,48</sup>	Talking to the hallucinations <sup>45</sup>
Sudden onset of vision loss <sup>1,45,49</sup>	Walking toward the hallucinations <sup>45</sup>
Stress <sup>2,45,47,48</sup>	
Bilateral visual impairment <sup>37</sup>	
Cognitive deficits <sup>9,37</sup>	
Decreasing vision <sup>37</sup>	
Cerebral damage <sup>9,37,42</sup>	

It is necessary to refer patients to their primary care physician and psychologist or psychiatrist who meet the following criteria: (1) are experiencing other neurological symptoms, (2) have hallucinations affecting other sensory modalities, (3) believe their visual hallucinations are real, or (4) have visual hallucinations that did not occur after vision loss.

In a cohort of AMD patients, 71% of them had no previous knowledge of Charles Bonnet Syndrome and did not know visual hallucinations could be a feature of their low vision. This reaffirms that a greater awareness of Charles Bonnet Syndrome is critical among eye care practitioners for both provision of emotional relief and avoiding inappropriate psychiatric labeling and treatment.<sup>57</sup>

As can be imagined, there is a low reporting rate to medical professionals about visual hallucinations. A Canadian study found only 9% sought medical advice about them with half of subjects reported to an ophthalmologist and the other half reported to their general practitioner.<sup>48</sup>

An Australian study found 15.2% participants informed their eye health professionals about their visual hallucinations.<sup>54</sup> Another study found 47% individuals discussing their visual hallucinations with a medical professional, yet one third of the medical professionals did not know about Charles Bonnet Syndrome or were uncertain about the condition.<sup>56</sup>

## Optical Intervention

The first step in managing these patients with visual hallucinations is to maximize their remaining vision either with spectacles, contact lenses, optical aids, and/or low vision rehabilitation. By improving vision, it is possible to reduce the frequency of visual hallucinations.<sup>1,45–47</sup>

In 2004, Eperjesi and Akbarali reported three case studies where visual hallucinations decreased in frequency when patients used

optical devices. In these studies, patients were prescribed ground-in prism, monocular telescope, best-corrected spectacles, tinted sunglasses, and nightlights. All of these interventions were effective in reducing or eliminating visual hallucinations for these patients. A hypothesis for the success of these interventions in resolving visual hallucinations was because they provide the sharpest images possible to the retina by reducing blur, glare, and field loss.

## Surgical Intervention

If surgical intervention is appropriate, treating the underlying cause of visual impairment can halt visual hallucinations. Cataract extraction improved vision and eliminated visual hallucinations in a patient with bilateral cataracts.<sup>8</sup> Laser intervention for the treatment of proliferative diabetic retinopathy also reduced visual hallucinations in a patient.<sup>8</sup> Laser photocoagulation in a patient with subretinal hemorrhage reduced the occurrence of visual hallucinations.<sup>8</sup>

In a study with 220 patients with neovascular age-related macular degeneration who had treatment with intravitreal ranibizumab injections, 22 of them experienced Charles Bonnet Syndrome. Five of the patients (23%) with Charles Bonnet Syndrome reported an improvement in their symptoms (decrease in frequency, decrease in intensity, or complete resolution of visual hallucinations) if their visual acuity improved after the injection treatment. However, if the patients' visual acuity remained unchanged after the injections, then the visual hallucinations also remained unchanged.<sup>50,51</sup>

Reports of visual hallucinations that occurred after laser or surgical treatment have also been documented. According to Cohen et al. in 2000, none of the 60 patients with macular disease experienced visual hallucinations before macular photocoagulation was instituted.<sup>37</sup> After treatment with macular photocoagulation for choroidal neovascularization, 37 of the 60 patients reported visual hallucinations that occurred immediately after treatment and 23 of the 60 patients did not experience any visual hallucinations after treatment. In a different study in 2003, Cohen et al.<sup>38</sup> investigated the effects of photodynamic therapy on visual hallucinations, finding only 20 of the 100 patients with AMD experiencing visual hallucinations after the treatment. Surprisingly, only 1 of the 100 AMD patients reported visual hallucinations before the treatment, although there is a flaw in how the information was elicited. At 6 to 12 weeks after photodynamic therapy was performed, the investigators asked if the subjects experienced visual hallucinations. If they did experience visual hallucinations, the last question asked was “are you sure that these phenomena were not present before the treatment?” By asking all subjects whether they experienced visual hallucinations before any treatment, this will establish a baseline and better compare the treatment effect on visual hallucinations.

Cohen et al.<sup>37,38</sup> proposed that the underlying mechanism of visual hallucinations in these cases occurred from dynamic change in acuity after treatments, not the level of acuity itself. It was suggested that a decrease in macular edema after treatment resulted in anatomic changes in the fovea. These anatomical changes stimulated visual hallucinations to occur from temporary changes in the quality of vision or through an unknown mechanism that has not been well established.<sup>37–39</sup>

**TABLE 6.**  
Pharmacological therapy based on classification

Antipsychotics	Anticonvulsants	Anti-anxiety	SSRI
Melperone	Gabapentin	Diazepam	Ondansetron
Olanzapine	Phenytoin		Cisapride
Clozapine	Valproate		Venlafaxine
Haloperidol	Carbamazepine		
Risperidone			

## Multidisciplinary Approach

To rule out pathological causes of visual hallucinations and confirm intact cognition, a multidisciplinary approach must be utilized. As an eye care professional, it is important to rule out other ocular causes of visual hallucinations such as photopsias caused by vitreous degeneration, vitreoretinal tractions, or retinal detachments.

If a patient reports visual hallucinations, the primary care practitioner needs to rule out pharmacologic side effects, illicit drug use, miscellaneous conditions, and neurologic and metabolic etiologies of the visual hallucinations. A physical and neurological examination, blood workup, and medication review are indicated.<sup>1,49</sup>

Referral to a psychologist or psychiatrist is indicated for evaluating cognitive function with formal cognitive tests such as the mini-mental state examination. Neuroimaging tests such as MRI or computed tomography (CT) may be necessary in determining the cause of visual hallucinations.<sup>1,49</sup>

## Medical Therapy

When optical or surgical interventions are ineffective, referring patients to a psychiatrist for pharmacological therapy may be initiated if visual hallucinations are causing distress. A total of 51.5% of subjects report mild stress compared to 30% who report severe stress as a result of visual hallucinations. Investigators found that stress was not a result of the images seen, rather stress stemmed from concern about the underlying cause of the hallucinations.<sup>54</sup>

Certain pharmacologic agents have been identified to treat individuals who experience visual hallucinations (see Table 6 for a brief listing). Serotonin and acetylcholine are important neurotransmitters for the development of visual hallucinations. These neurotransmitters are concentrated in the visual thalamic nuclei and visual cortex, which are not found in other sensory modalities. This explains why Charles Bonnet Syndrome visual hallucinations do not affect hallucinations in other sensory modalities.<sup>19</sup>

Olanzapine has a broad spectrum of activity for dopaminergic, cholinergic, serotonergic, and adrenergic receptors.<sup>33,40</sup> Typically, dopaminergic, cholinergic, and serotonergic systems are involved in the generation of visual hallucinations.<sup>40</sup>

Venlafaxine, prescribed at low doses, acts as a selective serotonin reuptake inhibitor. Selective serotonin reuptake inhibitors change the excitability of neurons in occipital lobe responsible for visual hallucinations. Because symptoms of visual hallucinations are decreased with venlafaxine, it suggests that visual hallucinations have serotonin involvement.<sup>36</sup>

Research has shown sensory deprived cortex has significantly lower serotonin levels.<sup>36</sup> When there is interruption in relaying

visual information to the visual cortex due to ocular pathology or deprivation of the visual system, serotonergic processes are reduced. As a result, endogenous excitatory signals are released in the visual association cortex. These excitatory signals become misinterpreted as visual hallucinations. Medications, which restore serotonin to the visual cortex, have been effective in reducing symptoms of visual hallucinations.<sup>36</sup>

Antipsychotics, anticonvulsants, anti-anxiety, and selective serotonin reuptake inhibitors have been shown, albeit with inconsistent results, in the literature to reduce or eliminate visual hallucinations associated with Charles Bonnet Syndrome.<sup>8,17,33,34,41</sup> The studies that investigated efficacy of these medications utilized small sample sizes and provided short-term results.<sup>42</sup> Therefore, it is difficult to determine whether the same results will transfer to a larger population of patients and whether the effects can be sustained for a long period of time.

Currently, there are no well-established medications for treatment of Charles Bonnet Syndrome given the majority of reported cases documented in the literature are small case reports or case series; therefore, results are not generalizable for all patients with Charles Bonnet Syndrome.<sup>46</sup> It is imperative to conduct randomized clinical trials under age- and gender-matched conditions to evaluate side effects of medications and how they will affect Charles Bonnet Syndrome patients.<sup>41</sup> Pharmacological treatment needs to be reserved only for those Charles Bonnet Syndrome patients who are very distressed by the hallucinations because of the inconclusive results in effectivity.<sup>43,46</sup>

## Patient Education

One of the most important elements with managing Charles Bonnet Syndrome patients is properly educating patients and their families about the condition.<sup>8</sup> Many elderly patients affected by Charles Bonnet Syndrome fear that visual hallucinations are signs of dementia or mental instability. Educating and reassuring patients that the condition is benign and may be a common response after vision loss may relieve distress in those suffering from the condition.<sup>10,45–48</sup> One study suggests eye care practitioners should proactively warn low vision patients about the common possibility of visual hallucinations after diagnosing their eye condition.<sup>48</sup>

Learning that Charles Bonnet Syndrome is unrelated to mental illness relieves concern among patients and their families.<sup>8</sup> It is beneficial to inform patients that although visual hallucinations usually resolve spontaneously, they may persist for years in some cases.<sup>10,22</sup>

In London, researchers found that Charles Bonnet Syndrome continued for 2 years or more in 88% of participants with resolution of visual hallucinations at 9 years duration in 25% individuals. In 75% of participants, it persisted for 5 years or more.<sup>56</sup> They suggested that the frequency and duration of hallucinatory episodes decreased over time, so participants no longer considered them problematic. It is also suggested that people get used to Charles Bonnet Syndrome symptoms with time.<sup>56</sup> This finding was supported by another study where investigators found the frequency of visual hallucinations was related to how long the person had been experiencing hallucinations. If the onset of visual hallucinations was more than 6 months ago, then people are more

likely to hallucinate less frequently than people who had recently begun to experience visual hallucinations. They also found people adapting to their visual hallucinations and their frequency of hallucinatory episodes abate with time.<sup>54</sup>

These patients should be encouraged to join support groups to meet other individuals suffering from ocular pathologies who may be experiencing similar phenomena. They can discuss their experiences with others, provide reassurance to one another, and offer techniques for reducing the hallucinations.<sup>8</sup>

## CONCLUSIONS

Although awareness of Charles Bonnet Syndrome in the medical literature has increased over the past few decades, there remains a paucity of information about this condition in the optometric and ophthalmologic literature. Criteria for diagnosing Charles Bonnet Syndrome include visual hallucinations, absence of hallucinations in other sensory modalities, partial or full insight that hallucinations are not real, absence of psychological disorders, preserved intellectual functioning, no lower limit of visual acuity, and no specific age.

Knowledge of Charles Bonnet Syndrome allows for proper diagnosis and appropriate management of patients. Recognizing the symptoms of Charles Bonnet Syndrome permits the healthcare professional to share appropriate information about the condition with patients and their families. An increased awareness about Charles Bonnet Syndrome among eye care professionals will benefit and ensure effective management of patients with symptoms of visual hallucinations. If patients experience visual hallucinations, eye care professionals play a critical role in reassuring patients that it can be a normal consequence of their vision loss, informing them and family members about the condition, and coordinating care with other health care professionals to rule out other etiologies of visual hallucinations to effectively manage Charles Bonnet Syndrome. With our patients aging and living longer lives, there will be more individuals affected by vision loss due to age-related conditions. Therefore, we can expect more individuals experiencing Charles Bonnet Syndrome. By recognizing symptoms and identifying signs of Charles Bonnet Syndrome, eye care practitioners will avoid misdiagnosis and/or unnecessary psychiatric or medical treatment causing more distress or burden to patients.

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## APPENDIX

Appendix A, which includes a comprehensive list of these medications, is available at <http://links.lww.com/OPX/A252>.

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