

MAJOR REVIEW

Complex Visual Hallucinations in the Visually Impaired: The Charles Bonnet Syndrome

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Abstract. Visually impaired patients may experience complex visual hallucinations, a condition known as the Charles Bonnet Syndrome. Patients usually possess insight into the unreality of their visual experiences, which are commonly pleasant but may sometimes cause distress. The hallucinations consist of well-defined, organized, and clear images over which the subject has little control. It is believed that they represent release phenomena due to de-afferentation of the visual association areas of the cerebral cortex, leading to a form of phantom vision. Cognitive defects, social isolation, and sensory deprivation have also been implicated in the etiology of this condition. This condition, which is most common in the elderly, frequently goes unrecognized in clinical practice, due to both lack of awareness among doctors and patients' reluctance to admit to hallucinatory experiences, for fear of being labeled mentally unstable. Furthermore, patients who comprehend the unreality of their hallucinations may be distressed by the real fear of imminent insanity. Sensitive and sympathetic history taking is essential to ascertain the existence of hallucinations. Reassurance and explanation that the visions are benign and do not signify mental illness have a powerful therapeutic effect. Hallucinatory activity may terminate spontaneously, on improving visual function or on addressing social isolation. There is no universally effective drug treatment but anticonvulsants may play a limited role in aborting the hallucinations. Physician awareness and empathy are the cornerstones of management. (*Surv Ophthalmol* 48:58–72, 2003. © 2003 by Elsevier Science Inc. All rights reserved.)

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Visual hallucinations occur in a variety of settings: psychiatric disease, drug ingestion, sleep–wake transitional states, metabolic and endocrine disorders, epilepsy, cerebral ischemia, and other forms of neurological disease.^{8,94,107,128}

Deterioration of vision secondary to a wide spectrum of ophthalmic and neurological disease is a common accompaniment of advancing age. It is however, perhaps not widely appreciated that a considerable proportion of visually impaired individuals experience formed visual hallucinations, which are

sometimes distressing. This condition is known as the Charles Bonnet Syndrome (CBS).

I. History

The eponym was coined by de Morsier,^{33,34} recognizing the renowned Genoese naturalist, philosopher, and biologist Charles Bonnet (1720–1793), who in 1769 described the hallucinatory experiences of his grandfather Charles Lullin,¹⁸ in probably the first scientific documentation of a hallucinatory experience.³² Lullin, an intelligent and articulate 89-

year-old magistrate, described subjective perception of silent visions of men, women, birds, carriages, and buildings, varying in size, shape, and place, occurring in association with visual deterioration after bilateral cataract surgery,⁴⁶ while maintaining intact cognition and fully realizing that his visions were “fictions” of his brain.^{31,42} Ironically Bonnet, deaf from the age of 7, himself suffered deterioration of vision early in life, and he manifested symptoms typical of the syndrome which now bears his name.^{32,90}

II. Definitions

Perception is the intuitive recognition of stimuli presented through the sense organs, while imagery is an experience within the mind, usually without the sense of reality that characterizes perception. Imagery so intense as to accord a photographic quality is referred to as *eidetic*. Eidetic imagery may persist, especially when looking at poorly structured backgrounds. This phenomenon, characterized by the simultaneous existence of real and unreal images (the subject being aware of the unreality of the latter), is called *pareidolia*. This is distinct from *pallinopsia*, in which visual perseveration results in persistence of a prior image when a new direction of gaze is assumed. An *illusion* is a misrepresentation of an external stimulus, exemplified by the mistaking of a rope for a snake.^{19,38,51} A *hallucination* is a sensory experience, possessing the compelling sense of reality of a true perception, but occurring without external stimulation of the relevant sensory organ.^{5,19,80} A similar phenomenon, where the subject is aware of the unreality of the sensory experience may be termed a *pseudohallucination*,^{80,83,131,134,137} though the utility of this term has been challenged.^{51,119}

Visual hallucinations may be elementary or complex. *Elementary* hallucinations (also called phosphenes, photopsiae, or unformed visual hallucinations) consist of colored or colorless bright lights, such as points, flashes, stars, and sparks. In contrast, *complex* visual hallucinations consist of formed images of objects or persons, sometimes related to past experiences of the subject.^{38,83,94}

De Morsier³² defined CBS as the occurrence of visual hallucinations in elderly people with intact cerebral function. Noting that ocular pathology was often present in these elderly subjects, he nevertheless considered visual impairment not to be causative and therefore not obligatory for diagnosis.^{32,33}

CBS has subsequently been described as the occurrence of recurrent or persistent complex visual hallucinations often of a pleasant nature,³¹ which may be considered pseudohallucinations,^{11,24} in individuals with preserved insight⁵³ and intellectual function³¹ without altered consciousness, cognitive or psychiatric disturbances, sleep disorders, or focal

neurological lesions^{10,12,24,30,122,128} and often associated with ophthalmic pathology.^{31,64,132}

III. Epidemiology

CBS occurs predominantly in elderly, visually impaired people.^{116,144}

A. PREVALENCE

Historically, hallucinatory experiences have been deemed to signify mental instability;⁸ patients are therefore often reluctant to admit to their hallucinatory experiences. The prevalence of CBS is thus likely to be underestimated.^{12,83,116,132}

The prevalence of complex visual hallucinations in patients with visual impairment has been estimated at between 11% and 15%.^{21,44,70,82,86,109,145,147} A similar incidence of complex visual hallucinations has been reported in patients subjected to sensory deprivation by means of ocular masking prior to cataract surgery.⁹² In psychogeriatric patients over the age of 65 the prevalence of CBS has been estimated at between 1.84% and 3.5%.^{107,108}

Elementary visual phenomena however, occur more frequently; their incidence has been estimated at between 41% and 59% in visually impaired patients.^{21,52,89,124,151}

B. AGE DISTRIBUTION

CBS is more common in the elderly.^{24,32,71,116,121,128} Major case series reveal mean ages of incidence of 83.8,¹⁰⁷ 78.4,¹⁴⁵ 75.7,¹¹⁶ and 74.9⁷⁰ years. Other investigators report similar figures.^{53,77,146,148,150} However, the condition has also been reported in children sustaining rapid visual loss.^{130,155} It has been suggested that the predilection for the elderly merely reflects the increased incidence of sudden profound visual loss in that age group.¹³⁰

C. SEX DISTRIBUTION

Different case series yield varying figures on the relative prevalence of CBS in men and women. De Morsier³² originally reported a male preponderance. Other studies suggest a clear preponderance of women,^{53,71,107,108,128,145,148} yet others recognize no sex bias.^{42,49,116,146}

D. UNDER-RECOGNITION

CBS frequently goes unrecognized in clinical practice.^{21,89,147} Patients who admit to experiencing hallucinations are often labeled demented or psychotic,⁶⁰ while potentially treatable causative factors are ignored.¹²²

Many authors draw attention to the unfamiliarity of medical personnel with CBS and the tendency to misdiagnose mental illness.^{21,59,89,107} In one series of 60 CBS patients, only 16 had consulted a doctor about

their hallucinations; of these 16, one was diagnosed correctly.¹⁴⁷ There is also the account of an elderly woman with CBS who would have been confined to a mental institution, had it not been for the intervention of a knowledgeable social worker.⁶⁰

Arguably one of the most illustrative examples is a study of 50 patients with probable Alzheimer's disease, in which hallucinatory activity was exclusively confined to those who were visually impaired.²² It has been subsequently pointed out that, despite recognizing the association between hallucinations and impaired visual acuity, and that optimizing visual acuity by optical means often reduced the occurrence of hallucinatory experiences, the possibility of CBS as part of the differential diagnosis tends not to be considered.¹⁴⁰

Awareness of CBS among medical personnel is therefore essential to avoid the risk of inappropriate therapy for non-existent psychiatric disease.^{104,107}

IV. Clinical Features

One of the earliest descriptions of the clinical features of CBS is that of Ernest Naville,¹⁰³ who described his own visual hallucinations as intriguing, non-deceptive, non-distressing, exclusively visual experiences (without auditory accompaniment), occurring during clear consciousness and normal perception. He was unable to consciously control the appearance and disappearance of his visions; they vanished on closing his eyes.

A. CONTENT

Hallucinatory content has been variously described. The most common image is that of a person.¹¹⁶ Disembodied distorted faces; small costumed figures and branching structures;¹²⁵ vivid images of animals and figures;⁹⁴ subtle geometric forms; well-defined complex figures; faces; Lilliputian (miniaturized),^{21,31,32,83} normal-sized,^{32,104} and "larger than life" images,^{32,71} in black and white—or more commonly—in color,^{31,49,83,104,116,128,147,156} of varying degrees of complexity¹²⁸ have also been described.

These hallucinations are always localized in external space,^{31,42,49,132} and the contents are well organized, defined and brilliantly clear.^{104,109,125,128,156} In the context of coexistent visual impairment, the clarity of the visions contrasts sharply with the blurred perception of real objects.^{11,31,49,132,156}

Patients may perceive visions of themselves, sometimes at earlier stages in their lives, a phenomenon referred to as *heautoscopia*^{46,82} or *autoscopia*.^{80,83} Déjà vu experiences are rare; subjects rarely recognize the figures as living or deceased acquaintances.¹¹⁶ It is unclear whether the images represent playback of previous visual memories.¹²⁸

The typical CBS hallucination has been variously described as a sudden sharply focused, immobile image, most often the face or figure of a person, which occurs when the patient is alert, with eyes open and vanishes spontaneously after a period of seconds.¹²⁹ Alternatively it has been described as a solitary constant solid object in the central visual field, most commonly a flash, but often a complex grid, a disembodied distorted face, a small costumed figure with a hat or a branching structure.¹²⁵

Atypical CBS hallucinations, classified into atypical sensory-perceptual (ASP) and atypical psychodynamic (APD) varieties, have also been described. ASP hallucinations differ from typical complex visual hallucinations in one or more characteristics such as duration, movement, volition, or participation of another sense (the latter widely considered an exclusion criterion for Charles Bonnet Syndrome), whereas APD hallucinations, similar to dreams, are more complex, mirroring the subject's psychological state, changing in content and frequency with alterations in the person's inner life or daily activities and may be repetitive.¹⁰⁵

B. MOVEMENT

There is no uniformity of opinion regarding the presence or absence of movement of hallucinatory images. Images have been described as static,¹²⁹ moving *en bloc* (without internal change)^{42,49,116} or dynamic (with internal movement).^{31,63,128}

C. STEREOTYPY

The criteria of Gold and Rabins⁵³ specify that the visual hallucinations in CBS be stereotyped. Recurrent themes^{21,82,100} and minimal variability in individual subjects¹⁰⁹ have been noted in many subjects; however, many investigators have reported hallucinatory content varying between patients and even in the same patient at different times.^{21,83,104,147,148} It has therefore been recommended that the criterion mandating stereotypy of hallucinatory content should be abandoned.¹⁴⁸ However, when images are dynamic (animated), the pattern of internal movement tends to be stereotyped.¹²⁸

D. TRIGGERING AND RELIEVING FACTORS

These hallucinations typically occur independently of any triggering factors or exercise of volition in the genesis of the image.^{104,129} In some individuals however, they may be triggered by a wide variety of stimuli,⁴² such as conditions of general sensory reduction,⁹¹ fatigue,¹⁰⁹ stress,⁸² low levels of illumination^{122,128} or even by bright light.¹⁵⁶

Once manifest, images last for periods varying from seconds through minutes to hours^{12,21,49,83,94,96,104,109,116,122,128,152} and subsequently disappear, either spontane-

ously, or in response to actions such as closing the eyes^{27,32,42,94,128,147,148} and executing ocular saccades.^{27,54,82,83,128} Measures such as looking directly at the images,⁸² attempting to approach them,¹²² and conversing with them⁸⁷ have also been reported to terminate hallucinations. A clinical approach, which investigates the utility of any such strategies, may be warranted.

Eye closure may both trigger and terminate hallucinations.^{31,53,148} Hallucinations have been reported to occur when the eyes are closed.^{141,147}

E. COURSE

After impairment or loss of vision, hallucinations usually appear after a latent period lasting from hours to days.⁸² They may commence gradually, or more commonly exhibit a sudden onset, often occurring several times a day.^{21,49,83,94,104,116,128} Hallucinations may then continue to occur over varying periods from days through months to years.^{31,82,116,122,128} They tend to fade as sight is finally lost,^{42,156} usually disappearing several months after the onset of the disorder, but are prone to reappear under conditions of physical or emotional stress.⁸³

Three patterns of disease—episodic, periodic, and continuous—have been described.^{32,116} The episodic variety, in which hallucinations occur over a period of days to months and then permanently resolve, is the least common.¹³ The periodic pattern of disease is characterized by phases of hallucinatory activity alternating with phases of remission, whereas patients manifesting the continuous pattern experience no hallucination-free intervals.

Elementary visual hallucinations may progressively evolve into complex visual hallucinations;^{31,87,116,152} less frequently complex visual hallucinations regress to simple visual phenomena as they gradually cease to occur.⁸⁷

F. HALLUCINATIONS OF OTHER MODALITIES

Most authorities maintain that the hallucinatory experiences of CBS are exclusively visual, unaccompanied by noise^{18,31,32,87} or hallucinations of other senses.^{11,109,132} It has been recommended that such silent visual hallucinations should alert clinicians to the possibility of dysfunction of the visual pathway.¹⁵¹ Auditory hallucinations in association with complex visual hallucinations have, however, been described;^{74,108} it has therefore been suggested that the coexistence of hallucinations of other modalities does not preclude a diagnosis of CBS, provided subjects retain insight into the unreality of their experiences.⁷⁴

G. PATIENTS' REACTION TO THEIR HALLUCINATIONS

Reports on patients' reaction to the images are variable. Some investigators emphasize that patients find their hallucinations pleasant,^{3,12,31,53,116,128} pur-

portedly due to preserved central nervous function and adaptability.⁹⁴ Others however, counter that CBS hallucinations are not invariably pleasant experiences.^{16,21,48,62,71,96,108,129,147,148} Emotional response may be related to the nature of hallucinatory content.³¹ A quarter of CBS patients manifest anger, anxiety, or mild paranoia in response to the imagery.^{49,125} Reactions to these visual experiences vary from indifference, through curiosity and irritation to terror.^{31,38,48,62,104,128}

Based on numerous reports describing patient reluctance to share their experiences^{16,108,109,148,156} and personal observation, it has been suggested that the positive response to hallucinations documented in the literature may be over-rated.¹⁴⁸ Other investigators however, counter that patients with CBS discuss their hallucinations freely and objectively.¹⁵²

Unlike the images seen in psychiatric states such as schizophrenia and depression, CBS hallucinations infrequently consist of images capable of generating emotional distress.^{94,148,152} Some authors draw a distinction between a predominantly positive reaction to hallucinatory content on the one hand, and anxiety and distress at the existence of hallucinatory activity on the other.^{104,116} Though most patients experience no practical problems, continuous visual hallucinations occurring while the subject is in motion can interfere with navigation.¹⁰⁴

H. INSIGHT

Most investigators agree that patients with CBS retain full insight into the unreality of their hallucinations,^{31,82,89,94,109,132,143,147} which may therefore may be considered pseudohallucinations.¹¹

However, insight may not be immediate. On initial appearance of hallucinations, patients may not recognize the unreality of their visions.^{31,83,116} Accurate insight is often achieved after initial deception,^{31,49,143} especially if the perceived images are appropriate to and fit realistically into the surroundings.¹⁴³ Accordingly, patients may react to their hallucinations, trying to hit them away or grab them.⁷¹ The utility of the terms hallucinations and pseudohallucinations in CBS has been challenged on this basis.³²

Furthermore, insight is not an all-or-none phenomenon; it may come and go, and even when present, may vary in degree.¹³⁷ Accordingly, studies suggest that a considerable proportion of CBS patients possess partial or fluctuating insight into their symptomatology.^{53,108}

I. THE MENTAL STATE

Lack of agreement exists regarding the mental state in CBS patients. Many authors emphasize the absence of depression or other demonstrable psychopathology in CBS.^{31,70,71,109,127,128,146,148} It has been

suggested that subtle confusional states in the elderly may be relevant in the development of CBS.¹⁶ Depression has also been observed in CBS patients with considerable frequency.¹²⁷

Cognitive impairment has been documented in CBS patients;³⁰ cases of CBS have developed dementia on follow-up.⁵³ The prevalence of CBS may be higher in individuals with lower cognitive scores and impaired cognitive status may be a predisposing factor for CBS.^{70,71} Subjects lacking insight into the unreality of their hallucinations typically demonstrate significant cognitive impairment compared with those with normal insight. On this basis, it has been suggested that patients with CBS manifest neuropsychological changes characteristic of early dementia, which are however, often overlooked due to lack of sensitivity of screening test measures such as the Mini-Mental State Examination (MMSE), to subtle cognitive decline in the early stages of dementia.¹¹⁴ It is therefore proposed that visual hallucinations may represent an early marker for dementia in the elderly and that visual impairment occurring in the early stages of cognitive decline unmasks these hallucinations.^{113,114} This has, however, been contested on the grounds that patients who lack insight do not meet criteria⁵³ for the diagnosis of CBS,¹⁴³ and on the basis of documented stable cognitive scores on follow up of CBS patients with macular degeneration.⁶⁹

It has also been argued that the failure of patients to experience distress on account of their hallucinations reflects compromised awareness of the significance of the symptom and provides further evidence that CBS and dementia lie on the same continuum of cognitive decline.¹¹⁵

The term *Charles Bonnet Plus* has been proposed in the presence of cognitive impairment.¹⁰ This has however, been deemed unnecessary, on the admittedly controversial basis that some degree of cognitive impairment is universally present in CBS patients.¹¹³

It is interesting that hypoperfusion of the mid-parietal and occipital areas of the brain, a well-recognized pattern in Alzheimer's disease and other dementias, has been documented on single photon emission computed tomography (SPECT) in a patient with CBS.⁵⁸

It has been argued that cognitive or visual deficits alone are unlikely to cause hallucinations, but together they may contribute to a state of sensory deprivation.³⁰ CBS patients with significant cognitive impairment may well represent a different sub-group to those who present to ophthalmologists primarily on account of poor vision, which may account for the diversity of views in this area.

J. FEAR OF INSANITY

Hallucinations have historically been deemed to signify psychiatric disease. Numerous reports describe

patients' reluctance to admit to their hallucinatory experiences for fear of being considered psychiatrically unstable.^{1,11,15,21,42,52,54,60,70,83,89,93,109,132,139,146,147,148,149,156}

Many investigators draw attention to the fear of imminent insanity in the mind of a patient who knows their visions are not real.^{49,60,68,83,101,116,129,149} Moreover, care providers, relatives, and friends may think the patient with CBS is mad.⁵⁴ It is well recognized that many patients experience relief when told that their hallucinatory experiences are normal and do not signify psychiatric disease.^{70,146,147}

K. VISUAL FUNCTION

There is no consensus on whether visual pathology is necessary for the development of CBS. Many investigators report a strong association between CBS and impaired vision;^{2,15,31,42,52,53,65,70,82,83,89,94,107,109,116,128,146,148,156} the most commonly associated ocular pathology is age-related macular degeneration,⁹⁴ although CBS has been documented in the context of visual impairment secondary to pathology anywhere along the visual pathway, from eye to occipital cortex.^{37,41,82,85,96,117} Such visual hallucinations do not occur in those born blind, but only in the context of acquired visual impairment.¹¹⁰

It has been suggested that CBS is almost invariably associated with impaired vision;^{122,128} indeed some authors mandate diminution of visual acuity,⁶⁴ obligatorily secondary to ocular pathology⁸³ to diagnose CBS. Others^{116,148} concur with de Morsier's original opinion³² that visual dysfunction, though common, is not mandatory for diagnosis.

Hallucinations may occur more commonly in the context of sudden and unexpected reduction in visual function.^{70,83} The degree of diminution of vision may be more relevant to the occurrence of visual hallucinations than the specific underlying ocular pathology.¹⁴⁶ CBS occurs more frequently in higher degrees of visual impairment^{52,70,89,146} and with bilateral as opposed to unilateral ocular pathology.^{21,70,146} In two separate studies, visual acuity of worse than 20/60 and 0.3 (20/66 approximate) in the better eye has been found to be associated with significant risk of developing visual hallucinations.^{70,146}

Numerous reports^{82,91,96,116} suggest that improvement of visual function, spontaneously or by interventional means (such as cataract surgery or neurosurgical procedures) results in cessation or improvement of hallucinatory phenomena.

Some reports suggest that complex visual hallucinations have a predilection for blind areas of the visual field,^{82,87} whereas other investigators recognize no relationship between the location of the hallucinatory images and objectively blind/scotomatous areas of the visual field.¹⁵²

It has however, been countered that hallucinations are unrelated to the degree of visual impairment.^{71,104,114} This is supported by the observation that hallucinations may paradoxically cease in response to further deterioration of vision.¹⁵⁰

L. QUALITY OF LIFE IN VISUALLY IMPAIRED PATIENTS

Loss of vision, with its profound effects on employment, independence, mobility, and self-esteem, can be an overwhelming personal tragedy. The reactions of adults who have recently suffered loss of vision, include psychic stress, depression, suicidal ideation, anxiety, tearfulness, withdrawal, hallucinations, and insomnia,⁴⁴ persisting as late as 5 years after the onset of visual loss.⁴⁵ Patients with macular degeneration manifest poor quality of life and emotional distress ratings, comparable with those of individuals with chronic illnesses such as obstructive airways disease and AIDS.¹⁵⁷

M. NEUROLOGICAL FEATURES

On the basis of normal visually evoked potentials (VEP) in a patient with CBS due to bilateral cataracts, it has been suggested that the diagnosis can be confirmed by a normal VEP, the geniculostriate pathways and occipital lobe being normal in this condition.¹²⁰ It is however, evident in the light of current knowledge that this is not so, and that CBS can occur due to pathology anywhere along the visual pathway.^{37,59,63,82,85,96,117,135,152}

N. OTHER RISK FACTORS

Multiple factors have been implicated as causative for the complex visual hallucinations seen in CBS. Postulated predisposing factors include social isolation,^{70,71,144} shyness,¹⁴⁴ and cerebrovascular disease.⁷⁰ Fatigue and disturbances of vigilance have been implicated as relevant,^{94,109,116,145,147} since hallucinations are more likely to occur during states of drowsiness. Other factors, including stress^{4,139} and suggestibility,¹⁴ have also been proposed as relevant to the genesis of visual hallucinations.

The pre-morbid personality structure and the ability to mobilize visual memories in the absence of normal stimuli has also been highlighted as relevant to the occurrence of complex visual hallucinations.⁸²

V. Diagnostic Criteria

There is lack of consensus regarding the diagnostic significance of ocular pathology, neurological disease, and the cognitive state in the context of CBS.

Damas Mora et al described CBS as a condition in which "persistent or recurrent visual pseudo-hallucinatory phenomena of a pleasant or neutral nature occur in a clear state of consciousness. Despite vividness,

clarity and impelling character, they are recognized as unreal. The condition tends to occur in the elderly with clinically preserved intellectual functions and is often associated with ocular pathology."³¹

Subsequently, various investigators have proposed diagnostic criteria for CBS. The criteria of Podoll et al¹¹⁶ are as follows:

1. The predominant symptom is the occurrence of visual hallucinations in elderly individuals in normal mental health.
2. There is no evidence of delirium, dementia, negative impact on intellectual capacity, deterioration as in the affective syndromes, paranoid developments, psychosis, intoxication, or neurological disease.
3. Loss of vision as a consequence of ocular disease is found in most cases as a specifying factor but is not obligatory for diagnosis.

Gold and Rabins,⁵³ noting that most investigators did not include patients with other psychopathology under the term *Charles Bonnet Syndrome*, suggested the diagnostic criteria, which focus on describing clinical phenomenology rather than etiology and course. Notably these criteria neither require nor exclude ocular or cerebral pathology:

1. Visual hallucinations which are
 - a. Formed
 - b. Complex
 - c. Persistent/repetitive
 - d. Stereotyped
2. Insight is fully or partially retained
3. Primary/secondary delusions are absent
4. Hallucinations in other modalities are absent

The criterion requiring stereotypy of visual hallucinations has been contested on the basis of observed variability of hallucinatory experiences in CBS patients.¹⁴⁸ It has also been suggested that these criteria be modified to include the presence of both ophthalmic pathology and cognitive defects on formal neuropsychological testing.¹¹⁴

VI. Associated Conditions

Charles Bonnet hallucinations have been documented in association with a wide spectrum of pathology of the eyes and visual pathway, including age-related macular degeneration,^{50,55,70,99,101,132,156} cataract,^{12,91} choroideremia,¹⁵⁶ corneal opacities,³¹ glaucoma,³¹ retinal detachment,^{31,132} enucleation,^{38,88} multiple sclerosis with optic neuritis,²⁵ retinitis pigmentosa,⁴⁸ occipital infarction with both homonymous hemianopia^{37,82} and bilateral loss of vision,¹³⁵ venous congestion of occipital cortex due to arteriovenous malformation (which improved after trans-arterial embolisation),⁸⁵ vertebro-basilar insufficiency,^{63,117} estrogen intake,⁴¹ in

association with macular translocation surgery⁹ and grief reaction.^{3,4}

CBS has also been reported in association with AIDS with CMV retinitis,⁶² suprasellar meningioma with visual loss (neurosurgical excision resulted in cure),⁹⁶ cranial arteritis with presumed cerebral involvement,⁵⁹ and pituitary tumors compressing the optic nerves and chiasm.¹⁵²

Charles Bonnet–type hallucinations have also been documented in the absence of ocular or neurological disease,^{7,30,145} in type II diabetes mellitus with normal vision,⁵⁶ in leprosy,¹ in association with HIV infection, where a possible undetectable effect of HIV on the brain was proposed,⁹⁵ and in the elderly, where they can occur in the absence of apparent cause.¹⁶

VII. The Neuroanatomic Basis of Complex Visual Hallucinations

Foerster,⁴⁷ studying the effects of faradic stimulation of various areas of the cerebral cortex, noted that stimulation of area 17 (the area striata) and area 18 resulted in subjective perception of elementary visual sensations/flashing lights, whereas that of area 19 (the visual association area) resulted in the subjective perception of complex formed visual sensations including figures, people, and animals. It has subsequently been suggested that complex hallucinations originate from activity in both areas 18 and 19, while elementary sensations signify activity in area 17, the primary visual cortex.¹¹⁷

It has been proposed that CBS hallucinations arise from entoptic sources,⁷⁶ but this is unlikely since visual hallucinations also occur in retrobulbar pathology. It is, however, interesting that acute termination of visual hallucinations has been documented in patients with macular degeneration treated with laser photocoagulation, implying that laser therapy may possibly silence spontaneously discharging retinal ganglion cells, in some way responsible for visual hallucinations.⁶⁹

On the basis of the occurrence of visual hallucinations in the context of calcarine infarction, it has been inferred that, since there cannot be activity in infarcted cortex, the origin must lie in the visual association areas.⁸⁷ Similarly, the ability to recall visual images from memory in individuals with cortical blindness indicates that primary visual (occipital) cortex does not subserve visual memory or imagination.²³

Smaller lesions of the occipital cortices are associated with hallucinations, while more extensive lesions, especially if extending anteriorly, result in loss of visual imagery, since the visual association area, located anterior to the striate cortex, is “released” by small posterior lesions and destroyed by larger, more anterior pathology.^{94,151}

Functional magnetic resonance imaging (fMRI) has demonstrated that while visual hallucinations are actually occurring, the occipital cortex manifests a reduced response to exogenous visual stimulation, suggesting that this reduced responsiveness to peripheral sensory input, may disinhibit endogenous visual memories, which then emerge into consciousness as hallucinations.⁷⁹ Neuroimaging studies have also shown that visual hallucinations correlate with phasic activity in specialized visual cortex, the location of which is reflected in hallucinatory content, implying functional specialization of the region, and that increased ventral extra-striate activity persists between hallucinations.^{43,125} Activation of different cortical areas may therefore produce different images.^{6,69,123}

An association between age-related macular degeneration and color hallucinations has been noted. In this context, it is thought that selective damage to cone photoreceptors at the macula (which subserve color vision) creates selective deafferentation of “color” areas of the visual cortex, with resultant localized hyper-excitability and the manifestation of colored hallucinations.¹²⁵

Studies of cerebral perfusion during actual hallucinatory experiences suggest that the lateral temporal cortex, corpus striatum, and thalamus are the regions most likely to be responsible for the genesis of complex visual hallucinations.² It is interesting that positron emission tomography (PET) in subjects experiencing musical hallucinations, in the context of acquired deafness, has demonstrated increased activity in auditory association areas rather than primary auditory cortex.⁵⁷

The tendency of CBS hallucinations to occur during states of drowsiness has led to the suggestion of a role for the ascending reticular activating system in the prevention of hallucinatory activity. This is supported by the observation that hallucinations occur in brain stem lesions, such as peduncular hallucinosis, affecting this system.⁹⁴

Further investigations, perhaps utilizing imaging techniques such as PET and SPECT, may prove useful in facilitating a deeper understanding of the neuroanatomical and neurobiological correlates of the Charles Bonnet Syndrome.

VIII. Theories of Pathogenesis

Bonnet himself suggested that these hallucinations have their origin in the part of the brain subserving visual function.¹⁸ In 1932 Jackson, conceptualizing the nervous system as a hierarchy of three levels, higher (cortical), middle (sub-cortical), and lower (brain-stem), postulated that higher centers exert a controlling influence on sub-cortical centers, loss of which allows the release of activity in disinhibited sub-cortical centers, resulting in hallucinations.¹³⁸

A. SENSORY DEPRIVATION—PHANTOM VISION

Visual hallucinations occurring in the context of visual loss have been conceptualized as phantom visions²⁸ due to de-afferentation¹⁶ and compared with the “phantom-limb” syndrome.^{12,16,104,128} Visual sensory cortex, when deprived of normal afferent input, may exhibit spontaneous independent activity with resultant conscious imagery,^{12,21,109,127} a hypothesis supported by the observation that such hallucinations can be abolished by normal or excessive visual stimulation.¹²

Reduction in sensory input to specific areas of the brain may allow memories of previous perceptions to enter consciousness as hallucinations.⁸ Visual hallucinations in destructive lesions of the central visual system may relate to pathological activation of other neural regions.^{6,69,123} On the basis that some hallucinatory states are associated with impaired cortical inhibition it has been suggested that images that are “screened out” or censored under normal circumstances, could, in the absence of normal inhibitory activity, reach higher centers and impinge onto consciousness.⁷⁶ It has also been suggested that visual impairment alone may be insufficient to cause complex visual hallucinations, but together with impaired cognition, may contribute to a state of sensory deprivation with resultant visual phenomena.³⁰

Musical pseudohallucinations with insight have been documented in the context of acquired deafness and have been compared with the visual pseudohallucinations of CBS.^{40,57}

B. SENSORY DEPRIVATION EXPERIMENTS

Hallucinations similar to those in CBS have been described by individuals subjected to sensory deprivation. The development of hallucinatory phenomena has been demonstrated under conditions such as ocular masking^{92,158} and exposure to monotonous sensory environments.⁶⁶

Heron et al⁶⁶ subjected themselves to sensory (including visual) deprivation, and they noted that after a single day all investigators invariably experienced visual hallucinatory experiences, initially elementary, which later evolved in complexity and exhibited movement, both *en bloc* and internal. The typical progression of visual hallucinations from simple to complex, observed during experiments on perceptual isolation, has been interpreted to signify progression from lower to higher centers in the nervous system.¹⁵⁹

Hallucinatory activity in patients undergoing bilateral ocular patching exhibits a strong relationship with duration of visual deprivation and manifests mainly during periods of reduced alertness, indicating that concurrence of sensory deprivation and re-

duced alertness is of causal significance in the development of hallucinatory experiences.¹⁵⁸

C. DREAMS AND HALLUCINATIONS

The neurophysiological mechanisms underlying dreams and hallucinations may be similar, the former occurring during sleep and the latter during wakefulness.³⁹ Dreams and hallucinations have been conceptualized as lying on a continuum, manifestation in the waking state being prevented by activity in ascending corticopetal nervous pathways. Disruption of such inhibitory mechanisms may “unmask” dreams—and the emergence of hallucinations.⁶¹

Reduced sensory input usually occurs during sleep; however, if specific sensory and non-specific ascending reticular input to sensory cortex drops below a threshold level even during the waking state, super-sensitivity to background activity in cerebral cortex may develop, with resultant hallucinatory experiences.^{94,126} In this context it is interesting that fatigue and disturbances of vigilance have been implicated as relevant to the emergence of hallucinations,^{94,109,116,145,147} as they are more likely to occur during states of drowsiness.

D. THEORY OF PERCEPTUAL RELEASE

The brain may actively exclude irrelevant sensory impulses from conscious perception, by means of an active censorship mechanism, in turn dependent upon normal sensory input. When afferent input is reduced below a threshold level (such as in disease of the eyes or visual pathway), the brain may then allow previously registered subconscious perceptions or engrams to emerge into consciousness, resulting in a hallucinatory experience.^{153,154}

E. THE NEUROMATRIX THEORY

Contending that the occurrence of visual hallucinations is independent of the degree of visual impairment, some authors¹⁰⁴ challenge the theory of perceptual release and refer to the neuromatrix theory,^{97,98} which proposes the existence of a network of neurons, the neuromatrix, extending throughout the brain, capable of generating sensory phantoms. This neuromatrix is said to impart a pattern, the *neurosignature*, on all afferent inputs from the body, so that sensory experiences may have a quality of self and possess affective tone and cognitive meaning. These authors argue that changes to sensory input may trigger or modulate the output of this neuromatrix, but do not dictate the qualities of experience.

F. IRRITATIVE AND RELEASE HALLUCINATIONS

Cogan²⁷ classified hallucinations into irritative and release variants and suggested that while the former are repetitive, stereotyped, and momentary, occur-

ring secondary to electrical discharge at any point along the visual pathway, the latter are more likely to cause formed, continuous, variable complex visual hallucinations. He believed that complex visual hallucinations in patients with multi-factorial visual impairment represented release phenomena secondary to attenuation of visual sensory input due to pathology anywhere along the visual system, as a result of which they have much less localizing value than those of the irritative type.

Many authors believe that CBS hallucinations represent release phenomena.^{27,42,82,89,94,109,111,128,132,150} It has also been suggested that coexisting pathology of the central nervous system may have a further permissive effect on the release of hallucinatory activity.¹³²

Ictal activity from a focal irritative center, possibly in the occipital or temporal cortices has also been implicated as causative in the genesis of visual hallucinations.^{16,87,122}

G. SOCIAL ISOLATION

Social isolation has been implicated as etiological in the genesis of CBS hallucinations,^{70,71,77,144,148} supported by the observation of temporary cessation of hallucinatory experiences during hospitalization. Social isolation may predispose to a state of sensory deprivation, a well recognized predisposing factor for visual hallucinations.³⁰

H. SENESCENCE

The possible role of senescence has been highlighted,²⁷ in that it may bring about the disintegration of higher centers and facilitate the release of sub-cortical phenomena, resulting in hallucinations. Complex visual hallucinations may be more likely to manifest in the context of macular disease if superimposed on age related central nervous system changes.⁹⁴ The requirement for disinhibition of higher cortical centers may therefore explain the almost exclusive incidence of CBS in the elderly.⁴⁹

I. PSYCHOLOGICAL FACTORS

It has been suggested that the psychological desire to see under conditions of partial visual loss may produce visual hallucinations. However, the disappearance of hallucinatory experiences when vision is totally lost negates this possibility.¹⁵⁰ Hallucinations have been postulated to involve the integrative activity of the mind¹⁵² and may be due to facilitation of a general capacity of the brain for imagery;²⁰ indeed, CBS is reported to occur predominantly in individuals with high levels of education and in the creative professions.^{65,121}

J. ANOMALIES OF CEREBRAL PERFUSION

Abnormalities of perfusion of the cerebral cortex have been implicated in the etiology of complex vi-

sual hallucinations. Vertebro-basilar insufficiency with ischemia of the visual pathway;^{107,117} asymmetrical hyperperfusion in the lateral temporal cortex, corpus striatum, and thalamus during hallucinatory experiences;² reduced occipital perfusion;¹³³ and mid-parietal and occipital hypoperfusion resembling a commonly described pattern in Alzheimer's disease⁵⁸ have been demonstrated in studies with SPECT and magnetic resonance imaging.

IX. Differential Diagnosis

The visually impaired are not immune to hallucinations secondary to other neuropsychiatric conditions or emotional disturbances;¹⁰⁴ it is therefore important to exclude other possible causes of complex visual hallucinations, such as peduncular hallucinosis, Alzheimer's disease, delirium, Parkinsonism and levodopa-induced hallucinations, Lewy Body dementia, (recovery from) migraine coma, schizophrenia, medication, epilepsy, and hallucinations experienced during sleep-wake transitions.^{94,136}

Up to one-third of normal individuals at some point experience hypnagogic hallucinations,⁹⁴ consisting of dramatic images in vivid colors, which, however, unlike those seen in CBS, invariably occur immediately before sleep, with the eyes closed and are frequently associated with auditory sensations.¹²

It has been suggested that the most common etiologies for visual hallucinations are dementia and delirium,¹³⁴ especially in psychogeriatric patients.⁶⁷ The possibility of delirium certainly merits careful consideration. However, in contrast with CBS, delirious subjects manifest impaired attention, disorganized thought processes, and abnormalities of sleep and orientation.¹⁵⁰

A wide spectrum of pathology of the visual system has been described in Alzheimer's disease. It is suggested that visual hallucinations occurring in such patients may be related to visual dysfunction (compromised visual acuity and visual agnosia) frequently seen in this most common of dementias.^{22,72,73}

Visual hallucinations in elderly subjects with Parkinson's disease may be similar to those in CBS. These patients often manifest impaired visual function, clear sensorium, and absence of hallucinations of other modalities. The pathogenesis may be a release phenomenon, similar to that of CBS.³⁵ Visual hallucinations in Parkinson's disease are significantly associated with poor visual acuity, impaired cognition, depression, and severity of disease, but not with psychiatric disease, dose or duration of anti-Parkinsonian medication, or duration of illness.⁶⁸

Brain stem lesions associated with hallucinations include peduncular hallucinosis, in which striking visual images, similar to those seen in CBS and the

hypnagogic state, occur with preserved insight and consciousness.⁹⁴

Visual hallucinations characteristic of CBS may occur in the early stages of Dementia with Lewy Body (DLB); it has therefore been suggested that patients with complex visual hallucinations be carefully followed up, in order to exclude DLB.¹⁴²

X. Management

Physician awareness and compassion are the mainstays of management for CBS. Though there is no universally effective therapy, treatment may not always be necessary, especially in cognitively intact patients,¹⁴⁹ since visual hallucinations often cease spontaneously, in response to either improvement or further deterioration of visual function.¹⁵⁰ Moreover, many patients with CBS are not distressed by the content of their hallucinations, as much as by anxiety about the significance of their occurrence.

A. HISTORY TAKING

Because most patients do not mention their symptoms unless specifically asked, sensitive, sympathetic, specific, and comprehensive history taking is essential to ascertain the existence of complex visual hallucinations in elderly visually impaired patients.^{15,19,21,42,107,139,147} When questioned by sympathetic, understanding, and accepting interviewers, more visually impaired subjects may admit to hallucinatory experiences.¹⁰⁴

B. EXAMINATION

Once hallucinations have been documented, thorough ophthalmic and neurological examination,^{113,149} including formal evaluation of the neuropsychological status,¹¹⁴ is recommended to determine any potentially treatable causal pathology. It is also important to exclude other causes of visual hallucinations, as discussed previously. Effective communication with psychiatrists and rehabilitation therapists involved in the care of the patient is valuable in this context. However, in the alert, well-oriented patient, a simple test of cognitive function such as the Mini Mental State Examination may be adequate, with further psychiatric evaluation perhaps indicated only in the context of cognitive impairment.

C. REASSURANCE AND COUNSELING

Patients with CBS may experience considerable anxiety, compounded by lack of awareness of the condition.¹⁰⁴ Many patients express disappointment and amazement at unsympathetic reactions received from healthcare professionals when seeking advice regarding their hallucinations.⁶⁰

Most patients experience relief when reassured that their condition is not a psychiatric disorder, but

a recognized phenomenon with a name.¹⁴⁷ Explanation and reassurance that the visions are benign and harmless, and do not signify mental illness therefore have a powerful therapeutic effect,^{15,36,104} so much so that it has been recommended that all blind individuals, even those who do not acknowledge the existence of hallucinations, should be informed of the possibility of their occurrence and advised that if they do occur, the hallucinations need not be a cause for distress.¹⁰⁴ Cognitively intact patients may need no further measures, though patients in the early stages of dementia may prove more difficult to reassure, especially if lacking insight and experiencing visions of a distressing nature.¹⁴⁹

It has been claimed that patients derive comfort from being told that their visual hallucinations will cease as their vision deteriorates further,^{122,150} though it is difficult to understand how the knowledge of impending blindness can be of comfort. Reassurance and counseling are therefore the cornerstones of treatment.

D. MAXIMIZING VISUAL FUNCTION

Optimizing visual function often has a beneficial effect on hallucinatory activity^{42,82,91,116,132} and may be the most effective treatment, though correction of the visual defect may not always be possible, especially in elderly patients.¹⁵⁰ Optical means such as prescription of spectacles or visual aids and surgical procedures such as cataract surgery may be employed for this purpose.

E. PSYCHOLOGICAL THERAPY

Techniques such as hypnosis, distraction, cognitive restructuring, and relaxation training, utilized in psychological therapy for phantom limb pain, have been advocated to minimize the unpleasant effects of persistent, intrusive, and troublesome visual hallucinations.¹⁰⁴

F. PHARMACOTHERAPY

There is currently no universally effective pharmacotherapy for CBS. The evaluation of any potential remedy is further complicated by the fact that even without treatment, hallucinations sometimes fade away over a period of weeks to months. Moreover, because patients are often not distressed by their visions, and may even enjoy them, a therapeutic approach may not always be necessary. However, if hallucinations are frequent, non-resolving, distressing, or impairing quality of life, effective therapy is required.¹¹²

Drug therapy is rarely useful in CBS.^{83,116} Anticonvulsants such as carbamazepine,^{17,24,25,55,77,141} clonazepam,¹⁴¹ and valproate⁷⁵ have been suggested as effective. A combination of carbamazepine and

clonazepam has proved useful in some patients.¹⁴¹ It is suggested that the efficacy of carbamazepine may be due to inhibition of inter-hallucinatory persistent increased ventral extra-striate neuronal activity.²⁴

Low-dose gabapentin (anti-convulsant) is reported to have produced permanent remission.¹¹² On the basis that serotonergic pathways may be relevant to the pathogenesis of visual hallucinations, cisapride, a potent 5HT-3 antagonist, has also been used with reported success.^{106,118}

Anti-psychotic drugs (neuroleptics) have been used in treatment of CBS, but are generally only partially effective at eradicating hallucinatory activity.¹⁴⁹ Varying degrees of success have been reported with thioridazine,⁶² haloperidol,²⁶ and the atypical neuroleptics risperidone⁷⁸ and melperone.¹³ Different neuroleptics may have varying effects on CBS (risperidone has been documented to worsen hallucinations and render them menacing); treatment should therefore be tailored to the individual patient.⁸⁴

G. SOCIAL AND ENVIRONMENTAL FACTORS

CBS is known to occur in the context of social isolation and sensory deprivation, improvement of which may have a beneficial effect.¹⁴⁹ Symptoms may abate when alternative interests are pursued, such as television, music, and the company of friends.

Environmental changes such as improved illumination may reduce hallucinatory activity,^{99,149} perhaps by increasing the overall level of sensory stimulation. Treatment of depression, modification of the environment, and establishment of support groups, in which patients can share their experiences, may also be useful.

XI. Discussion

There has been much controversy and disagreement about inclusion and exclusion criteria for CBS. Such criteria are arguably of limited relevance since they do not alter management in any way. In the pursuit of strict, well-defined criteria we perhaps overlook the clinical significance of this phenomenon—*visual hallucinations can and do occur in patients with visual impairment*. Such patients are often elderly and may be considerably distressed by their visions, the nature of which they do not understand. This is relevant and requires recognition and attention.

CBS can be misdiagnosed and inappropriately treated. It is significant that the majority of information on CBS is contained in psychiatric rather than ophthalmic literature. This may indicate that patients who do admit to their hallucinatory experiences are judged mentally unstable and referred to psychiatrists, who then make the diagnosis of CBS. Visually impaired patients often present first in oph-

thalmic clinics, where CBS should ideally be diagnosed, or at least suspected.

The majority of individuals with CBS suffer from impaired vision. Indeed both Lullin, the first described case, and later Bonnet himself, who lent his name to the syndrome, definitely manifested ocular pathology. In any case, ophthalmologists rarely encounter patients with formed visual hallucinations who do not have visual impairment. We therefore suggest that this eponym is reserved exclusively for patients experiencing visual hallucinations in the context of visual impairment secondary to pathology of the eyes or visual pathways. Charles Bonnet–type visual hallucinations in the absence of visual loss should be considered a separate entity.

Hallucinatory symptoms are well recognized to abate upon both further decline and improvement of visual function. In this light, although cognitive dysfunction may be a contributory factor, it is unlikely to be of primary etiological significance. The most widely accepted explanation for the phenomenology of CBS is that of sensory deprivation—deafferentation—of the visual association areas of the cerebral cortex, with resultant release of hallucinatory activity.

In both Alzheimer's disease and Parkinson's disease hallucinations occur more commonly in the context of visual impairment. In the light of suggestions that cognition may be impaired in CBS, it is tempting to speculate that the underlying pathology is similar, that both Alzheimer's and Parkinson's lie on a continuum with CBS, indeed that the occurrence of visual hallucinations in these contexts constitutes a form of CBS.

XII. Conclusion

Eponymous labels are of limited value; however the term *Charles Bonnet Syndrome* may serve the essential function of reminding ophthalmologists that visual hallucinations can occur in the context of visual loss.²⁹

Visual impairment is increasingly common in the elderly. Age-related macular degeneration, the leading cause of new irreversible blindness in the elderly,¹⁰² has been estimated to occur in 1 in 5 people over the age of 65 years.⁸¹ It is important that we recognize the occurrence of visual hallucinations in this large segment of our elderly population.

The unfortunate patient with visual hallucinations, elderly, visually impaired, isolated, and afraid to seek help, has a genuine fear of impending insanity. The most powerful therapeutic approaches at our disposal remain empathy, sensitivity, communication, and reassurance.

XIII. Method of Literature Search

We undertook a Medline search using the following keywords: *Charles Bonnet, visual hallucinations,*

pseudohallucinations and phantom visions. Relevant citations from the reference lists of selected articles were also reviewed. Inclusion or exclusion of any article in the text was based on relevance and the necessity to avoid redundancy.

References

- Adachi N: Charles Bonnet syndrome in leprosy; prevalence and clinical characteristics. *Acta Psychiatr Scand* 93:279–81, 1996
- Adachi N, Watanabe T, Matsuda H, Onuma T: Hyperperfusion in the lateral temporal cortex, the striatum and the thalamus during complex visual hallucinations: single photon emission computed tomography findings in patients with Charles Bonnet syndrome. *Psychiatry Clin Neurosci* 54:157–62, 2000
- Adair DK, Keshavan MS: The Charles Bonnet syndrome and grief reaction. *Am J Psychiatry* 145:895–6, 1988
- Alroe CJ, McIntyre JN: Visual hallucinations. The Charles Bonnet syndrome and bereavement. *Med J Aust* 2:674–5, 1983
- American Psychiatry Association Committee on Nomenclature and Statistics: Diagnostic and statistical manual of mental disorders. Washington DC, American Psychiatry Association, 1994, ed 4, p 767
- Anderson SW, Rizzo M: Hallucinations following occipital lobe damage: the pathological activation of visual representations. *J Clin Exp Neuropsychol* 16:651–63, 1994
- Arya DK: Charles Bonnet syndrome. *Br J Psychiatry* 167:114–5, 1995
- Asaad G, Shapiro B: Hallucinations: theoretical and clinical overview. *Am J Psychiatry* 143:1088–97, 1986
- Au Eong KG, Fujii GY, Ng EW, et al: Transient formed visual hallucinations following macular translocation for subfoveal choroidal neovascularization secondary to age-related macular degeneration. *Am J Ophthalmol* 131:664–6, 2001
- Ball C: Charles Bonnet syndrome. *Br J Psychiatry* 166:677–8, 1995
- Barodawala S, Mulley GP: Visual hallucinations. *J R Coll Physicians Lond* 31:42–8, 1997
- Bartlett JEA: A case of organised visual hallucinations in an old man with cataract, and their relationship to the phenomena of the phantom limb. *Brain* 74:363–73, 1951
- Batra A, Bartels M, Wormstall H: Therapeutic options in Charles Bonnet syndrome. *Acta Psychiatr Scand* 96:129–33, 1997
- Bentall RP: The illusion of reality: a review and integration of psychological research on hallucinations. *Psychol Bull* 107:82–95, 1990
- Berrios GE, Brook P: Visual hallucinations and sensory delusions in the elderly. *Br J Psychiatry* 144:662–4, 1984
- Berrios GE, Brook P: The Charles Bonnet syndrome and the problem of visual perceptual disorders in the elderly. *Age Ageing* 11:17–23, 1982
- Bhatia MS, Khastgir U, Malik SC: Charles Bonnet syndrome. *Br J Psychiatry* 161:409–10, 1992
- Bonnet C: *Essai analytique sur les facultes de l'ame*. Copenhagen and Geneva, Philibert, 1769, ed 2, pp 176–8
- Borruat FX: [Visual hallucinations and illusions, symptoms frequently misdiagnosed by the practitioner]. *Klin Monatsbl Augenheilkd* 214:324–7, 1999
- Brasic JR: Hallucinations. *Percept Mot Skills* 86:851–77, 1998
- Brown GC, Murphy RP: Visual symptoms associated with choroidal neovascularization. Photopsias and the Charles Bonnet syndrome. *Arch Ophthalmol* 110:1251–6, 1992
- Chapman FM, Dickinson J, McKeith I, Ballard C: Association among visual hallucinations, visual acuity, and specific eye pathologies in Alzheimers disease: treatment implications. *Am J Psychiatry* 156:1983–5, 1999
- Chatterjee A, Southwood MH: Cortical blindness and visual imagery. *Neurology* 45:2189–95, 1995
- Chaudhuri A: Charles Bonnet syndrome: an example of cortical dissociation syndrome affecting vision. *J Neurol Neurosurg Psychiatry* 69:704–5, 2000
- Chen CS, Lin SF, Chong MY: Charles Bonnet syndrome and multiple sclerosis. *Am J Psychiatry* 158:1158–9, 2001
- Chen J, Gomez M, Veit S, ODowd MA: Visual hallucinations in a blind elderly woman: Charles Bonnet syndrome, an underrecognized clinical condition. *Gen Hosp Psychiatry* 18:453–5, 1996
- Cogan DG: Visual hallucinations as release phenomena. *Albrecht Von Graefes Arch Klin Exp Ophthalmol* 188:139–50, 1973
- Cohn R: Phantom vision. *Arch Neurol* 25:468–71, 1971
- Cole M: Charles Bonnet Syndrome an example of cortical dissociation syndrome affecting vision? *J Neurol Neurosurg Psychiatry* 71:134, 2001
- Cole MG: Charles Bonnet hallucinations: a case series. *Can J Psychiatry* 37:267–70, 1992
- Damas-Mora J, Skelton-Robinson M, Jenner FA: The Charles Bonnet syndrome in perspective. *Psychol Med* 12:251–61, 1982
- de Morsier G: Le Syndrome de Charles Bonnet: hallucinations visuelles des vieillards sans deficiance mentale. *Ann Med Psychol* 125:677–702, 1967
- de Morsier G: Les Hallucinations. *Rev Otoneuroophthalmol* 16:244–352, 1938
- de Morsier G: Les automatismes visuels. Hallucinations rétrochiasmatisques. *Schweiz Med Wochenschr* 66:700–8, 1936
- Diederich NJ, Pieri V, Goetz CG: [Visual hallucinations in Parkinson and Charles Bonnet Syndrome patients. A phenomenological and pathogenetic comparison]. *Fortschr Neurol Psychiatr* 68:129–36, 2000
- Dlugon U: [Charles Bonnet syndrome]. *Psychiatr Pol* 34:307–16, 2000
- Dodd J, Heffeman A, Blake J: Visual hallucinations associated with Charles Bonnet Syndrome—an ever increasing diagnosis. *Ir Med J* 92:344–5, 1999
- Duke-Elder S, Scott GI: Disorders of perception: visual hallucinations, in Duke-Elder S (ed): *System of Ophthalmology*, Vol. XII. London, Henry Kimpton, 1971, pp 562–9
- Evarts, EV: A neurophysiologic theory of hallucinations, in West LJ (ed): *Hallucinations*. New York, Grune and Stratton, 1962, pp 1–14
- Fenelon G, Marie S, Ferroir JP, Guillard A: Hallucinoses musicales: 7 cas. *Rev Neurol (Paris)* 149:462–7, 1993
- Fernandes LH, Scassellati-Sforzolini B, Spaide RF: Estrogen and visual hallucinations in a patient with Charles Bonnet syndrome. *Am J Ophthalmol* 129:407, 2000
- Fernandez A, Lichtsheim G, Vieweg WV: The Charles Bonnet syndrome: a review. *J Nerv Ment Dis* 185:195–200, 1997
- ffytche DH, Howard RJ, Brammer MJ, et al: The anatomy of conscious vision: an fMRI study of visual hallucinations. *Nat Neurosci* 1:738–42, 1998
- Fitzgerald RG: Visual phenomenology in recently blind adults. *Am J Psychiatry* 127:1533–9, 1971
- Fitzgerald RG, Ebert JN, Chambers M: Reactions to blindness: a four-year follow-up study. *Percept Mot Skills* 64:363–78, 1987
- Fluornoy T: Le case de Charles Bonnet. *Arch Psychol Suisse Romande* 1:1–23, 1902
- Foerster O: The cerebral cortex in man. *Lancet* 2:309–12, 1931
- Fong SY, Wing YK: Charles Bonnet syndrome with major depression in a Chinese middle-aged man. *Aust NZ J Psychiatry* 31:769–71, 1997
- Fuchs T, Lauter H: Charles Bonnet Syndrome and musical hallucinations in the elderly, in Katona C, Levy R (eds): *Delusions and hallucinations in old age*. Royal College of Psychiatrists, Gaskell, 1992, pp 187–98
- Galynker I, Kampf R, Rosenthal R: Dose related visual hallucinations in macular degeneration patients receiving phenelzine. *Am J Psychiatry* 151:450, 1994
- Gelder M, Gath D, Mayou R: *Oxford Textbook of Psychiatry*, Oxford, Oxford University Press 1991, ed 2, pp 5–8
- Girkin CA, Miller NR: Central disorders of vision in humans. *Surv Ophthalmol* 45:379–405, 2001

53. Gold K, Rabins PV: Isolated visual hallucinations and the Charles Bonnet syndrome: a review of the literature and presentation of six cases. *Compr Psychiatry* 30:90–8, 1989
54. Goldberg KB, Goldberg RE: Is seeing believing? Visual hallucinations in age-related macular degeneration and Charles Bonnet. *J Ophthalmic Nurs Technol* 19:39–42, 2000
55. Gorgens K, Liedtke M: [Charles Bonnet syndrome]. *Psychiatr Prax* 25:85–6, 1998
56. Gray M, Jones IR: Type II diabetes mellitus presenting as the Charles Bonnet syndrome. *J R Soc Med* 90:503, 1997
57. Griffiths TD: Musical hallucinosis in acquired deafness. Phenomenology and brain substrate. *Brain* 123: 2065–76, 2000
58. Guerra-Garcia H: Charles Bonnet syndrome and early dementia. *J Am Geriatr Soc* 45:893–4, 1997
59. Hart CT: Formed visual hallucinations: a symptom of cranial arteritis. *Br Med J* 3:643–4, 1967
60. Hart J: Phantom visions: real enough to touch. *Elder Care* 9:30–2, 1997
61. Hartmann E: Dreams and other hallucinations: an approach to the underlying mechanism, in Siegal RK, West LJ (eds): *Hallucinations: Behaviour, Experience and Theory*. New York, John Wiley and Sons, 1975, pp 71–9
62. Hartmann PM, Kosko DA, Cohn JA: The Charles Bonnet syndrome (pseudohallucinations) in an AIDS patient with cytomegalovirus retinitis. *J Nerv Ment Dis* 183:549–50, 1995
63. Hauge T: Catheter vertebral angiography. *Acta Radiol* 109(Suppl):1–219, 1954
64. Hécaen H, Albert ML: Disorders of visual perception, in Hécaen H, Albert ML (eds): *Human Neuropsychology*. New York, John Wiley & Sons, 1978, pp 144–167
65. Hécaen H, Badarocco JG: Les hallucinations visuelles au cours des ophthalmopathies et des lésions des nerfs et du chiasma optiques. *Evol Psychiatr (Paris)* 21:157–79, 1956
66. Heron W, Doane BK, Scott TH: Visual disturbances after prolonged perceptual isolation. *Can J Psychol* 10:13–8, 1956
67. Holroyd S: Visual hallucinations in a geriatric psychiatry clinic: prevalence and associated diagnoses. *J Geriatr Psychiatry Neurol* 9:171–5, 1996
68. Holroyd S, Currie L, Wooten GF: Prospective study of hallucinations and delusions in Parkinsons disease. *J Neurol Neurosurg Psychiatry* 70:734–8, 2001
69. Holroyd S, Rabins PV: A three-year follow-up study of visual hallucinations in patients with macular degeneration. *J Nerv Ment Dis* 184:188–9, 1996
70. Holroyd S, Rabins PV, Finkelstein D, et al: Visual hallucinations in patients with macular degeneration. *Am J Psychiatry* 149:1701–6, 1992
71. Holroyd S, Rabins PV, Finkelstein D, Lavrisha M: Visual hallucinations in patients from an ophthalmology clinic and medical clinic population. *J Nerv Ment Dis* 182:273–6, 1994
72. Holroyd S, Sheldon-Keller A: A study of visual hallucinations in Alzheimers disease. *Am J Geriatr Psych* 3:198–205, 1995
73. Holroyd S, Shepherd ML: Alzheimers disease: a review for the ophthalmologist. *Surv Ophthalmol* 45:516–24, 2001
74. Hori H, Terao T, Nakamura J: Charles Bonnet syndrome with auditory hallucinations: a diagnostic dilemma. *Psychopathology* 34:164–6, 2001
75. Hori H, Terao T, Shiraiishi Y, Nakamura J: Treatment of Charles Bonnet syndrome with valproate. *Int Clin Psychopharmacol* 15:117–9, 2000
76. Horowitz MJ: The imagery of visual hallucinations. *J Nerv Ment Dis* 138:513–23, 1964
77. Hosty G: Charles Bonnet syndrome: a description of two cases. *Acta Psychiatr Scand* 82:316–7, 1990
78. Howard R, Meehan O, Powell R, Mellers J: Successful treatment of Charles Bonnet Syndrome type visual hallucinosis with low-dose Risperidone. *Int J Geriatr Psychiatry* 9:677–8, 1994
79. Howard R, Williams S, Bullmore E: Cortical response to exogenous visual stimulation during visual hallucinations. *Lancet* 345:70, 1995
80. Johnson FC, Smith LD: Cognition, in Thompson T, Mathias P (eds): *Lyttle's mental health and disorder*. London, Bailliere Tindall, 1994, ed 2, pp 175–240
81. Kahn HA, Leibowitz HM, Ganley JP, et al: The Framingham Eye Study. I. Outline and major prevalence findings. *Am J Epidemiol* 106:17–32, 1977
82. Kolmel HW: Complex visual hallucinations in the hemianopic field. *J Neurol Neurosurg Psychiatry* 48:29–38, 1985
83. Kolmel HW: Visual illusions and hallucinations, in Kennard C (ed): *Baillieres Clinical Neurology*, Vol.2, No. 2. London, Bailliere Tindall, 1993, pp 243–64
84. Kornreich C, Dan B, Verbanck P, Pelc I: Treating Charles Bonnet syndrome: understanding inconsistency. *J Clin Psychopharmacol* 20:396, 2000
85. Kurata A, Miyasaka Y, Yoshida T, et al: Venous ischemia caused by dural arteriovenous malformation. Case report. *J Neurosurg* 80:552–5, 1994
86. Lalla D, Primeau F: Complex visual hallucinations in macular degeneration. *Can J Psychiatry* 38:584–6, 1993
87. Lance JW: Simple formed hallucinations confined to the area of a specific visual field defect. *Brain* 99:719–34, 1976
88. Lauber HL, Lewin B: A clinical and psychological study of optic hallucinations in the elimination of vision. *Arch Psychiatr Nervenkr* 197:15–31, 1958
89. Lepore FE: Spontaneous visual phenomena with visual loss: 104 patients with lesions of retinal and neural afferent pathways. *Neurology* 40:444–7, 1990
90. Levêque de Pouilly JS: *Eloge de Charles Bonnet*, Lausanne, Henbach, 1794, pp 120–121
91. Levine AM: Visual hallucinations and cataracts. *Ophthalmic Surg* 11:95–8, 1980
92. Linn L, Kahn RL, Coles R: Patterns of behaviour disturbance following cataract extraction. *Am J Psychiatry* 110: 281–9, 1953
93. Loewenstein JI: Visual hallucinations in patients with choroidal neovascularization. *JAMA* 272:243, 1994
94. Manford M, Andermann F: Complex visual hallucinations. Clinical and neurobiological insights. *Brain* 121: 1819–40, 1998
95. Maricle RA, Turner LD, Lehman KD: The Charles Bonnet syndrome: a brief review and case report. *Psychiatr Serv* 46: 289–91, 1995
96. McNamara ME, Heros RC, Boller F: Visual hallucinations in blindness: the Charles Bonnet syndrome. *Int J Neurosci* 17:13–5, 1982
97. Melzack R: Phantom limbs and the concept of a neuromatrix. *Trends Neurosci* 13:88–92, 1990
98. Melzack R: Phantom limbs, the self and the brain. (the D.O. Hebb Memorial Lecture). *Can Psychol* 30:1–16, 1989
99. Mojica TR, Bailey PP: Hallucinations in the vision-impaired elderly: the Charles Bonnet syndrome. *Nurse Pract* 25:74–6, 2000
100. Murphy RP, Storer D, Bressler NM, Orr P: Visual hallucinations in patients with age related macular degeneration [abstract]. *Invest Ophthalmol Vis Sci* 31 (Suppl):48, 1990
101. Nadarajah J: Visual hallucinations and macular degeneration: an example of the Charles Bonnet syndrome. *Aust NZ J Ophthalmol* 26:63–5, 1998
102. National Advisory Eye Council: *Vision Research*, a National Plan, 1994–1998. Bethesda, MD: US Dept of Health and Human Services, NIH publication, 1993, pp 93–3186
103. Naville E: Hallucinations visuelles a letat normal. *Arch Psychologie* 8:1–8, 1908
104. Needham W, Taylor RE: Benign visual hallucinations, or phantom vision in visually impaired and blind persons. *J Vis Impair Blind* 86:245–8, 1992
105. Needham WE, Taylor RE: Atypical Charles Bonnet hallucinations: an elf in the woodshed, a spirit of evil, and the cowboy malefactors. *J Nerv Ment Dis* 188:108–15, 2000
106. Nevins M: Charles Bonnet syndrome. *J Am Geriatr Soc* 45: 894–5, 1997
107. Norton-Willson L, Munir M: Visual perceptual disorders resembling the Charles Bonnet syndrome. A study of 434 consecutive patients referred to a psychogeriatric unit. *Fam Pract* 4:27–35, 1987

108. O'Reilly R, Chamberlaine C: Charles Bonnet syndrome: incidence and demographic and clinical features. *Can J Psychiatry* 41:259-60, 1996
109. Olbrich HM, Engelmeier MP, Pauleikhoff D, Waubke T: Visual hallucinations in ophthalmology. *Graefes Arch Clin Exp Ophthalmol* 225:217-20, 1987
110. Ormond AW: Visual hallucinations in sane people. *BMJ*: 367-7, 1925
111. Özsancak C, Auzou P: Palinopsie au cours d'un syndrome de Charles Bonnet. *Presse Med* 27:359, 1998
112. Paulig M, Mentrup H: Charles Bonnets syndrome: complete remission of complex visual hallucinations treated by gabapentin. *J Neurol Neurosurg Psychiatry* 70:813-4, 2001
113. Pfeiffer RF, Bodis-Wollner I: Charles Bonnet syndrome. *J Am Geriatr Soc* 44:1128-9, 1996
114. Pliskin NH, Kiolbasa TA, Towle VL, et al: Charles Bonnet syndrome: an early marker for dementia? *J Am Geriatr Soc* 44:1055-61, 1996
115. Pliskin NH, Towle VL, Kiolbasa TA: In reply to Teunisse RJ: The Charles Bonnet Syndrome, insight and cognitive impairment. *J Am Geriatr Soc* 45: 892, 1997
116. Podoll K, Osterheider M, Noth J: [The Charles Bonnet syndrome]. *Fortschr Neurol Psychiatr* 57:43-60, 1989
117. Price J, Whitlock FA, Hall RT: The psychiatry of vertebro-basilar insufficiency with the report of a case. *Psychiatr Clin (Basel)* 16:26-44, 1983
118. Ranen NG, Pasternak RE, Rovner BW: Cisapride in the treatment of visual hallucinations caused by vision loss: the Charles Bonnet syndrome. *Am J Geriatr Psychiatry* 7:264-6, 1999
119. Rao V: Charles Bonnet syndrome, time to drop the name? *Int J Geriatr Psychiatry* 12:413, 1997
120. Raudino F: Visual evoked potentials (VEP) in the Charles Bonnet Syndrome: Report of a case. *Acta Neurol (Napoli)* 9:53-5, 1987
121. Reyes-Ortiz CA, Camacho ME, Mulligan T: Charles Bonnet syndrome in a centenarian. *JAMA* 276:451-2, 1996
122. Rosenbaum F, Harati Y, Rolak L, Freedman M: Visual hallucinations in sane people: Charles Bonnet syndrome. *J Am Geriatr Soc* 35:66-8, 1987
123. Rousseaux M, Debrock D, Cabaret M, Steinling M: Visual hallucinations with written words in a case of left parieto-temporal lesion. *J Neurol Neurosurg Psychiatry* 57:1268-71, 1994
124. Safran AB, Kline LB, Glaser JS: Positive visual phenomena in optic nerve and chiasm disease: photopsias and photophobia, in Glaser JS (ed): *Neuro-ophthalmology*, Vol. 10. St Louis, CV Mosby, 1980, pp 225-31
125. Santhouse AM, Howard RJ, ffytche DH: Visual hallucinatory syndromes and the anatomy of the visual brain. *Brain* 123:2055-64, 2000
126. Scheibel ME, Scheibel AB: Hallucinations and brain stem reticular core, in West LJ (ed): *Hallucinations*. New York, Grune and Stratton, 1962, pp 15-35
127. Schultz G, Melzack R: Visual hallucinations and mental state. A study of 14 Charles Bonnet syndrome hallucinators. *J Nerv Ment Dis* 181:639-43, 1993
128. Schultz G, Melzack R: The Charles Bonnet syndrome: phantom visual images. *Perception* 20:809-25, 1991
129. Schultz G, Needham W, Taylor R, et al: Properties of complex hallucinations associated with deficits in vision. *Perception* 25:715-26, 1996
130. Schwartz TL, Vahgei L: Charles Bonnet syndrome in children. *J AAPOS* 2:310-3, 1998
131. Sedman G: A comparative study of pseudohallucinations, imagery, and true hallucinations. *Br J Psychiatry* 112:9-17, 1966
132. Siatkowski RM, Zimmer B, Rosenberg PR: The Charles Bonnet syndrome. Visual perceptual dysfunction in sensory deprivation. *J Clin Neuroophthalmol* 10:215-8, 1990
133. Sichart U, Fuchs T: [Visual hallucinations in elderly patients with reduced vision: Charles Bonnet syndrome]. *Klin Monatsbl Augenheilkd* 200:224-7, 1992
134. Sims A, Mundt C, Berner P, Barocka A: Descriptive phenomenology, in Gelder MG, Lopez-Ibor JJ, Andreasen NC (eds): *New Oxford Textbook of Psychiatry*. Oxford, Oxford Medical Publications, 2000, pp 55-70
135. Symonds C, MacKenzie I: Bilateral loss of vision from cerebral infarction. *Brain* 80:415-55, 1957
136. Takata K, Inoue Y, Hazama H, Fukuma E: Night-time hypnopompic visual hallucinations related to REM sleep disorder. *Psychiatry Clin Neurosci* 52:207-9, 1998
137. Taylor FK: On pseudo-hallucinations. *Psychol Med* 11:265-71, 1981
138. Taylor J, Holmes G, Walshe FMR: Evolution and dissolution of the nervous system, in Taylor J, Holmes G, Walshe FMR (eds): *Selected writings of John Hughlings Jackson*, Vol. 2. London, Hodder and Stoughton, 1932, pp 3-120
139. Taylor RE, Mancil GL, Kramer SH: Visual hallucinations: meaning and management. *J Am Optom Assoc* 57:889-92, 1986
140. Terao T: Hallucinations in Alzheimers disease and Charles Bonnet syndrome. *Am J Psychiatry* 157:2062, 2000
141. Terao T: Effect of carbamazepine and clonazepam combination on Charles Bonnet syndrome: a case report. *Hum Psychopharmacol Clin Exp* 13:451-3, 1998
142. Terao T, Collinson S: Charles Bonnet Syndrome and dementia. *Lancet* 355:2168, 2000
143. Teunisse RJ: Charles Bonnet syndrome, insight and cognitive impairment. *J Am Geriatr Soc* 45:892-3, 1997
144. Teunisse RJ, Cruysberg JR, Hoefnagels WH, et al: Social and psychological characteristics of elderly visually handicapped patients with the Charles Bonnet Syndrome. *Compr Psychiatry* 40:315-9, 1999
145. Teunisse RJ, Cruysberg JR, Hoefnagels WH, et al: Risk indicators for the Charles Bonnet syndrome. *J Nerv Ment Dis* 186:190-2, 1998
146. Teunisse RJ, Cruysberg JR, Verbeek A, Zitman FG: The Charles Bonnet syndrome: a large prospective study in The Netherlands. A study of the prevalence of the Charles Bonnet syndrome and associated factors in 500 patients attending the University Department of Ophthalmology at Nijmegen. *Br J Psychiatry* 166:254-7, 1995
147. Teunisse RJ, Cruysberg JR, Hoefnagels WH, et al: Visual hallucinations in psychologically normal people: Charles Bonnets syndrome. *Lancet* 347:794-7, 1996
148. Teunisse RJ, Zitman FG, Raes DC: Clinical evaluation of 14 patients with the Charles Bonnet syndrome (isolated visual hallucinations). *Compr Psychiatry* 35:70-5, 1994
149. Thorpe L: Treatment of psychiatric disorders in Late life. *Can J Psychiatry* 42(Suppl 1):19S-27S, 1997
150. Tueth MJ, Cheong JA, Samander J: The Charles Bonnet syndrome: a type of organic visual hallucinosis. *J Geriatr Psychiatry Neurol* 8:1-3, 1995
151. Vaphiades MS, Celesia GG, Brigell MG: Positive spontaneous visual phenomena limited to the hemianopic field in lesions of central visual pathways. *Neurology* 47:408-17, 1996
152. Weinberger LM, Grant FC: Visual hallucinations and their neuro-optical correlates. *Arch Ophthalmol* 23:166-99, 1940
153. West LJ: A general theory of hallucinations and dreams, in West LJ (ed): *Hallucinations*. New York, Grune and Stratton, 1962, pp 275-91
154. West LJ: A clinical and theoretical overview of hallucinatory phenomena, in Segal RK, West LJ (eds): *Hallucinations: Behaviour, Experience and Theory*. New York, John Wiley and Sons, 1975, pp 287-311
155. White CP, Jan JE: Visual hallucinations after acute visual loss in a young child. *Dev Med Child Neurol* 34:259-61, 1992
156. White NJ: Complex visual hallucinations in partial blindness due to eye disease. *Br J Psychiatry* 136:284-6, 1980
157. Williams RA, Brody BL, Thomas RG, et al: The psychosocial impact of macular degeneration. *Arch Ophthalmol* 116:514-20, 1998
158. Ziskind E, Jones H, Filante W, Goldberg J: Observations on mental symptoms in eye patched patients: hypnagogic symptoms in sensory deprivation. *Am J Psychiatry* 116:893-900, 1960
159. Zuckerman M, Cohen N: Sources of reports of visual and auditory sensations in perceptual isolation experiments. *Psychol Bull* 62:1-20, 1964

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