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Visual hallucinations in sensory deprived patients: Charles Bonnet Syndrome?

María Yoldi Negrete,[1] Jesús Ramírez-Bermúdez,[1] Angel Ruiz-Chow,[1] Luis Carlos Aguilar-Venegas,[1] Daniel Crail-Melendez,[1] Rodrigo Pérez-Esparza,[1] Luis Daniel Alviso de la Serna[2]

Article Review

ABSTRACT

Introduction

Some patients, after brain or peripheral injuries, lose a sensory function such as sight or hearing, but paradoxically experience complex hallucinations related to the function they have lost. It is known that this phenomenon may appear with injuries at any level in the visual pathway, especially in the retina.

Objective

To review the existing bibliography on Charles Bonnet syndrome to establish the latest advances with regard to this phenomenon.

Method

The databases PubMed and PsychInfo were searched for articles containing the following keywords: Charles Bonnet syndrome; visual hallucinations; Peduncular Hallucinosis; Charles Bonnet; sensory deprivation. We included those related to the subject. We also included the classic texts referring to this phenomenon and the articles mentioned in the literature.

Results

In the present study, we describe the history of Charles Bonnet syndrome, clinical presentation, risk factors, diagnostic criteria, treatment employed, similar conditions, and the theories seeking to explain it.

Discussion and conclusion

To date, the diagnostic criteria for Charles Bonnet syndrome remain controversial, especially those concerning the absolute preservation of insight as a *sine qua non* factor to establish the diagnosis. Conclusion: Described since the 18th century, Charles Bonnet syndrome corresponds to the prototype of visual hallucinations in patients with visual deprivation, although according to the present review, its phenomenology is vast, remaining unclear whether it corresponds to the prototype of hallucinations with retained insight.

Key words: Charles Bonnet syndrome, formed visual hallucinations, insight, neuropsychiatry.

RESUMEN

Introducción

Existen pacientes que, después de lesiones cerebrales o periféricas, pierden alguna función sensorial, como la vista o el oído. Paradójicamente, después de perder esta función, presentan alucinaciones complejas relacionadas con la función perdida. Se sabe que este fenómeno puede presentarse ante lesiones en cualquier nivel de la vía visual, especialmente en el nivel de la retina.

Objetivo

Revisar la bibliografía existente acerca del síndrome de Charles Bonnet para conocer los últimos avances con respecto a este fenómeno.

Método

Se revisaron las bases de datos de PubMed y PsychInfo con las siguientes palabras clave: síndrome de Charles Bonnet; alucinaciones visuales; alucinosis peduncular; Charles Bonnet; privación sensorial. Se incluyeron aquellos artículos que efectivamente trataran del tema. Asimismo, se revisaron los textos clásicos referentes a este síndrome y los artículos mencionados en la bibliografía encontrada.

Resultados

En el presente artículo se describe la historia del síndrome, el fenómeno clínico, los factores de riesgo, los criterios diagnósticos, los tratamientos empleados, otros fenómenos similares y las teorías propuestas para explicarlo.

Discusión y conclusión

A la fecha continúan siendo controvertidos los criterios diagnósticos del síndrome de Charles Bonnet, en especial en lo referente a la preservación absoluta del *insight* como condición *sine qua non*. Descrito desde el siglo XVIII, el síndrome de Charles Bonnet corresponde al prototipo de alucinaciones visuales en pacientes con privación visual, si bien, de acuerdo con la presente revisión, la fenomenología de éste es bastante variada, siendo cuestionable si resulta también el prototipo de las alucinaciones con *insight* preservado.

Palabras clave: Síndrome de Charles Bonnet, alucinaciones visuales formadas, *insight*, neuropsiquiatría.

[1] Neuropsychiatry Unit, Manuel Velasco Suárez National Institute of Neurology and Neurosurgery (INNN).
[2] Department of Psychiatry, Institute of Neurosciences and Emotional Development.

Correspondence: María Yoldi Negrete. Affective Disorders Clinic, Ramón de la Fuente Muñiz National Institute of Psychiatry, Calz. México-Xochimilco 101, San Lorenzo Huipulco, Tlalpan, 14370 Mexico City. Phone: 4160 - 5323, E-mail: myoldine@conacyt.mx

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INTRODUCTION

After specific cerebral or peripheral injuries, some patients lose a sensory function such as vision or hearing. Paradoxically, after losing this function, they can have complex hallucinations related to the function they have lost. For example, a patient who loses visual function can start to have complex visual hallucinations. It is likely that this may happen for all senses, such as sight, hearing, touch, taste, or smell, as there is already evidence of musical hallucinations in patients with hypoacusia[1] and this could also be the case with phantom limb syndrome, even if not all known mechanisms behind this phenomenon are shared with the hallucinatory phenomenon of the visual path[2] that is our concern in the present work.

A characteristic of these patients is that most of the time, they retain their judgment of reality at least partially (i.e., they recognize the unreal nature of the hallucinations).

In spite of various case reports, case series, and epidemiological studies having been published on Charles Bonnet syndrome, Peduncular Hallucinosis, and other hallucinatory phenomena, and even when criteria have been proposed for the diagnosis of Charles Bonnet syndrome, it seems that important controversies still persist: for some authors, the term "Charles Bonnet syndrome" should be exclusively used for patients with ocular pathology. For others, it should be a syndrome utilized only in elderly patients. Still others think that if the injury is to the midbrain it should be called Peduncular Hallucinosis, regardless of whether or not there exists an accompanying visual deficit; there are those who consider that the appearance of hallucinatory phenomena in epileptic patients corresponds to a different phenomenon even if they are accompanied by a visual deficit. Finally, use of the term is also restricted only to patients who have integral capacities of *insight* in terms of the hallucinatory phenomena. However, authors have set out controversies in all of these matters.

As such, it is still unclear whether it is a single phenomenon with different generating mechanisms but the same clinical presentation (suggesting a common final path) or whether it is a multiplicity of different phenomena (with different causes, presentation, development, and response to treatment).

OBJECTIVE

The present article seeks to carry out a revision of the history, current knowledge, and controversies around Charles Bonnet syndrome.

CHARLES BONNET SYNDROME

History of the syndrome

Charles Bonnet (1720-1793), an 18th century naturalist and philosopher, described this type of phenomenon in his grandfather, Charles Lullin, and it was later known as Charles Bonnet syndrome.

At the age of 89, Lullin, whom Charles Bonnet described as a respectable man, full of good health, innocence, judgment, and memory, had an episode of visual hallucinations three months long, during which he saw the images of men, women, birds, carriages, buildings, etc., during waking hours and without external stimuli. He saw that these forms had movement, moved close up and further away, and ran off; they reduced and increased in size; they appeared, disappeared, and reappeared. He saw buildings rise up in front of him, along with all the elements that formed part of their external construction. All of these images were perfectly clear and seemed to him as real as if the objects themselves were truly there; but they were nothing more than pictures. The men and women did not speak, and no noises affected the old man's hearing. Bonnet stresses that his grandfather did not interpret the visions as reality and emphatically writes, "he knows to healthily judge all of these apparitions, and to trust his first instincts". He had undergone cataract surgery in both eyes, and although it is not specified whether this was carried out before or after the hallucinations, Bonnet states that the early success of the surgery was later lost, leaving the grandfather without the use of his left eye and with a marked reduction of visual acuity in the right eye, which only allowed him to see objects close up. Later, one of Lullin's manuscripts was found, in which the old man himself specified certain characteristics of the hallucinations: they were only present when he was lying down or seated, they were clearer in the left field of vision, and they disappeared when he moved his eyes to the right.[3,4]

Years later, Charles Bonnet suffered a marked reduction of visual acuity and had a similar experience of observing "fantastical objects" which he recognized as illusions. It was De Morsier who coined the term Charles Bonnet syndrome.[4]

In the 19th century the description of Charles Bonnet became the paradigm of visual hallucinations existing in mentally healthy subjects.

Currently, in spite of it being a little-studied syndrome, various critical aspects are now known, which we will attempt to clarify in the following glossary.

The clinical phenomenon

Content

The content of the hallucinations has been widely described. The most commonly described image is that of a person,[5] although in a recent study which included simple halluci-

nations as a part of Charles Bonnet Syndrome, it was mentioned that the most commonly seen image was that of animals, followed by bright elements of light, strange people, and flowers. Where more than one object was seen, that order of presentation was the same, except that strangers were more common than bright lights.[6] Hallucinations can be very varied, from simple lights6 to complete scenes.[7] There have been descriptions of faces without a body, vivid images of animals and plants, subtle geometric shapes, and miniature, normal-sized, or gigantic images.[8] There have also been descriptions of familiar people, whether alive or dead.[5]

Hallucinations are described as being located in the external environment,[8] and the majority are described as very clear,[8] however, there are also authors whose patients have described hallucinations as being more blurry.[7] Movement has been described as static, blocky, and with internal movement.[8,9]

Gold and Rabins' criteria require that images are stereotyped, although there are various authors who differ from this, finding patients who describe different images among themselves and even in the same patient at different times.[6,8–10]

Triggers and mitigating factors

Images are typically described as appearing spontaneously outside of the subject's control, although various different patients identify factors that encourage their appearance, such as general situations of reduced alertness, tiredness, stress, low lighting levels, and bright light.[8] Actions have also been described which tend to make the hallucinations disappear: closing or rubbing the eyes, looking directly at the images, or trying to approach and talk to them.[8] Closing the eyes has been described as a factor which can both trigger and eliminate the images.[8]

Hallucinations of the other senses

Most authors have observed that the images are not accompanied by sound or any other type of hallucination.[8] However, auditory hallucinations have been described in relation to complex visual hallucinations.[11,12]

Patients' emotional reaction

Hallucinations may trigger neutral, positive, or negative emotional reactions,[6] and the description of these emotions is very varied (fear, anxiety, mild paranoia, desperation, anger).[8]

Insight

It is universally known that the main characteristic of this syndrome is that people who have it are aware that the images they perceive are not real.[4,6,8,9] However, various authors agree that this characteristic is variable from one patient to another,[8] and questionable in some moments. It takes some patients more than one event to realize that the images they perceive are not real[6] and this can even vary from one moment to the next in the same patient.[8]

Clinical course

The hallucinations can present themselves after loss of vision with a very variable latency period, from hours or days[8] to years later.[6] In fact, a study by Gilmour et al. found that the likelihood of having the syndrome was 24% higher if the diagnosis of ocular pathology was more than five years old.[6]

The course of the condition has been described as episodic, periodic, and continuous,[8] and the hallucinations tend to disappear when visual acuity improves or worsens significantly.[8]

The disappearance of the hallucinations is variable from patient to patient. One follow-up study in an ophthalmological center observed 152 first-time patients, and then again one year later. They found that 15 patients (28%) of the sample who had reported having hallucinations at the start of the study no longer had them; however, 23 of the 98 remaining patients (24%) who had not had them initially did present them at the end of the study.[13] Another study of 33 patients monitored for a year found that 10 (30%) no longer had hallucinations, seven (21%) found reduced frequency, eight (24%) found increased frequency, and eight (24%) stayed the same.[14]

Visual acuity

In their description of the syndrome, De Morsier thought that poor visual acuity was a characteristic only present in some patients, and not a necessary characteristic for the syndrome to be present.[15] Later study of the pathology led De Morsier to think that they were mistaken in this aspect and that in fact, the images appeared due to loss of vision,[4] although various later studies found patients who did not present reduction in visual acuity[6] and that it was a phenomenon that was not present in patients who had been blind from birth.[8] What does seem to be clear is that loss of vision is a factor that significantly raises the risk of having the syndrome[6,8] and that this risk raises after visual acuity of 20/40 through 20/1,600.[6] The greatest risk is present between 20/60 and 20/66[8] and it is more common in those with visual acuity between 20/100 and 20/800, and twice as likely if acuity is between 20/301 and 20/800.[8]

Furthermore, it is more frequent when the pathology is bilateral,[8] and some authors describe hallucinations having a predilection for blind spots in the field of vision;[16,17] although there are others who did not find a relationship between scotoma and hallucinated images.[18]

Risk factors

Various risk factors have been proposed to be associated with Charles Bonnet Syndrome. These include social isola-

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tion,[19–21] shyness, a general lack of quality in social contact,[19] cerebro-vascular disease,[21] tiredness and disorders in the state of alertness, stress, the power of suggestion,[8] the level of vision, light levels, and time of day.[6] It should be noted that the study by Gilmour et al.[6] did not find age, living alone, or being widowed to be risk factors. Neither in this study did they find a correlation with the *Mini-Mental Status Examination* (MMSE), use of ophthalmological drugs, alcohol or tobacco consumption, or the number of laser or photodynamic treatments. Other frequently-found comorbidities in this syndrome are cognitive deterioration and depression.

There are authors who think that cognitive deterioration should be ruled out to be able to carry out this diagnosis and in their case series they perform an MMSE to exclude patients who do not pass that test.[6] However, there are various descriptions of patients with cognitive deterioration who have this phenomenon, and there are even authors who think that it may be about a pre-dementia state,[22,23] although there has already been a study comparing the MMSE of patients with and without hallucinations and this association has been rejected. Furthermore, after a year of follow-up, no greater deterioration was seen in MMSE scores with respect to the control group.[24] The term "Charles Bonnet plus" has even been proposed for cases which present cognitive deterioration, although this term is not currently considered useful because the age at which this syndrome is most prevalent is also that when cognitive deterioration is frequently present, and as such both diagnoses coincide.[8]

Depression has been an important finding in various articles on this subject.[7,8,10,14,21,25,26] Jackson et al.[14] found a prevalence of 33%.

One notable point that has even led to thinking that this syndrome is exclusive to elderly people[4] and has found that it is independent of age[6,8] is its high prevalence in the over-60s,[6,8,14,27] which is thought to be associated with an increase in the incidence of sudden, profound loss of vision in this age group.[28] Other authors consider that this syndrome is not exclusive to elderly patients, given that similar phenomena have even been found in children.[28] Another frequently reported finding is the syndrome's greater frequency in women,[8,11,27,29–31] although there are those who do not find differences in gender[8] and originally, De Morsier reported a greater frequency in men.[15]

Diagnostic criteria

In 1873, Naville proposed six criteria to recognize compatible hallucinations with the following reasoning:[4]

1. occurs in a clear sense and does not trick the subject,
2. occurs in combination with normal perceptions,
3. is exclusively visual,
4. is not accompanied by strange sensations,
5. appears and disappears with no apparent cause.
6. is more entertaining than concerning for the subject.

In 1989, Gold and Rabins[29] proposed the following diagnostic criteria specifically for Charles Bonnet syndrome:

- Presence of formed, complex, persistent or repetitive, and stereotyped visual hallucinations;
- *Insight* maintained totally or partially towards the hallucinations;
- Absence of hallucinations in the other senses;
- Absence of delusional ideation.

Later in 1995, Teunisse proposed other criteria on which their investigations on the subject have been based:[27]

1. Presence of formed and complex hallucinations;
2. Hallucinations which are persistent or repetitive:
   a. at least one complex visual hallucination in the past four weeks,
   b. a period of less than four weeks between the first and last hallucination;
3. *Insight* that is partial or complete of the unreal nature of the hallucinations;
4. Absence of hallucinations in the other senses;
5. Absence of delusion.

There are still various controversies on this subject: whether the name itself should be reserved for ocular pathology,[15] and for elderly people,[4] whether the presence of other types of hallucinations excludes diagnosis in all cases,[12] whether *insight* must be retained,[8] whether hallucinations are persistent or not;[13] there are even authors who mention that it can be present with normal vision.[6,15] As such, all criteria have been placed in doubt. As Germán Berrios mentions in his article,[4] all these controversies mean that there is little interest in using an eponym to refer to all of the hallucinatory states, and it is much more informative to describe the phenomenon itself, especially if there are no universally accepted diagnostic criteria. Because of this, it seems suitable to describe other hallucinatory states with retained *insight* as shall shortly be explained.

Treatments used in Charles Bonnet syndrome

The primary intervention described for this syndrome consists of providing the patient with a psycho-educational process, explaining to them that the hallucinatory phenomenon they have is not part of a mental illness. Nor should they be told that they are "losing their reason or sanity" (a fear frequently reported by patients); rather, it is a phenomenon with a neurological cause.[6,8,13,14,24,27,32,33]

There are various reports that improving visual capacity improves the hallucinations,[8] although it this is obviously only possible in some cases.

Psychotherapeutic techniques have been reported, such as those utilized in phantom limb syndrome with some improvement.[7]

Table 1. Drugs used in Charles Bonnet syndrome and response obtained

Drug	Response obtained	Level of evidence	Reference
Anticonvulsants			
Gabapentin	Remission of hallucinations two days after starting treatment with 300mg/d and free of hallucinations at three-month follow-up.	Case report	(Paulig, Mentrup 2001)
Valproate	Remission of hallucinations through starting carbamazepine (time not specified). Carbamazepine was withdrawn due to adverse effects and hallucinations returned. They disappeared once again on treatment with valproate. Treatment with valproate at 300mg/d "significantly reduced hallucinations" and an increase to 1,500mg/d "almost made them disappear". This was also accompanied by a reduction in hypermetabolism found in the left thalamus and inferior temporal area before treatment.	Case reports	(Hori et al. 2000; Jang et al. 2011; Segers 2009)
Levetiracetam	Did not reduce hallucinations, and even increased them, with a 500mg/day dose.	Case report	(Segers 2009)
Clonazepam	No response with a combination of 0.25mg/d of triazolam, 50mg/d of tiapride, and 100mg/d of pentoxifylline. Later started on carbamazepine up to 200mg/d with partial response, and total improvement with a combination of carbamazepine 100mg/d and clonazepam 0.5mg/d. Monotherapy was attempted first with carbamazepine and then with clonazepam, but only the combination was effective.	Case report	(Terao 1998)
Carbamazepine	Disappearance of hallucinations through starting carbamazepine 100mg every 12h (time not specified). Carbamazepine was withdrawn due to adverse effects and hallucinations returned.	Case report	(Segers 2009)
5HT3 antagonists			
Cisapride	Improved during the first week of treatment with cisapride 40mg/d (nights only, short duration and low clarity) and disappearance after three months of treatment in the first case. Improved in the first month (marked reduction in frequency and intensity of hallucinations) of treatment with cisapride 30mg/d and disappearance after two months of treatment.	Two case reports	(Ranen, Pasternak, Rovner 1999)
Antipsychotics			
Haloperidol	Reduction in frequency of hallucinations after two weeks of treatment with 0.5mg every 12h.	Case report	(Chen et al. 1996)
Olanzapine	Total disappearance of hallucinations after seven days of treatment with 10mg/d. Later reduced down to 2.5mg/d with continued response at one-year follow-up. Reduction in intensity of hallucinations through treatment with 5mg/d. Disappearance of hallucinations after 48 hours of treatment with 2.5mg/d*	Case report	(Coletti Moja et al. 2005), (Cumurcu, Elbozan 2005), (Mocellin 2006)
Risperidone	Reduction in frequency of hallucinations with 1mg every 12h. Reduction in frequency of hallucinations after one week of treatment with 1mg/d of risperidone and disappearance after two weeks of treatment. Partial improvement with 4mg/d but with poor adherence by the patient, which led to a change to treatment with cisapride. No response in one case.	Case reports	(Howard, Meehan, Powell 1994), (Maeda et al. 2003), (Ranen, Pasternak, Rovner 1999), (Cammaroto et al. 2009)
Thioridazine	No improvement after seven days of treatment with 25mg every 12h.	Case report	(Howard, Meehan, Powell 1994)
Melperone	Three case reports. Case 1 was lofepramine at low doses plus melperone at 67.5mg/d. Hallucinations diminished "rapidly" altogether and did not return at a later date. Time not specified. Case 2, gradual-to-total reduction in a week with melperone 25mg/d. Case 3 started with promazine with no response, changed to 100mg/d of melperone with a "clear and persistent" improvement.	Three case reports	(Batra, Bartels, Wormstall 1997)
Antidepressants			
Venlafaxine	Remission of hallucinations after four days of treatment with 75mg/d.	Case report	(Lang et al. 2007)
Citalopram	The remission obtained with venlafaxine was maintained despite changing to citalopram 20mg/d (due to increase in blood pressure with venlafaxine).	Case report	(Lang et al. 2007)
Mirtazapine	Remission of hallucinations after three days of treatment with 7.5mg/d.	Case report	(Siddiqui, Ramaswmay, Petty 2004)
Acetylcholinesterase inhibitors			
Donepezil	Reduction in hallucinations in one eye and in both upon increasing the dose to 5mg/d.	Case report	(Ukai et al. 2004)

* This case is reported as Peduncular Hallucinosis by the authors, however, as discussed in this revision, it could also be categorized as Charles Bonnet syndrome.

To date, there is no universal pharmacological treatment for this condition. It should be noted that research into treatments is made even more complicated because of the fact that in some patients, these hallucinations disappear without any intervention.[13,14] Also, not all patients react with emotional discomfort towards their hallucinations, because of which intervention is not always recommended.[8] Some authors suggest that treating the factors around unpleasant hallucinations, such as anxiety, can improve the nature of them and make them more pleasant, if not eliminate them altogether.[34] However, if the hallucinations are frequent, do not disappear, cause anxiety, or affect the patient's quality of life, an effective therapy is indeed necessary.[8,35]

What follows is a synthesis of the pharmacological therapies that have, to our knowledge, been reported in medical literature[12,35–49] (Table 1).

OTHER PHENOMENA WITH VISUAL HALLUCINATIONS AND RETAINED *INSIGHT*: THE PHENOMENON OF PEDUNCULAR HALLUCINOSIS

In the 20th century, Jacques-Jean Lhermitte described the case of a woman who had suffered infarcts in the bridge and midbrain, and afterwards could see strange people, full of color, and groups of children in the twilight.[50] Van Bogaert later gave this the name of "Peduncular Hallucinosis".[50]

There are now numerous reports of this pathology, some associated with cerebral peduncles as first described, others associated with various diseases of the brain stem, thalamus, visual pathway, cerebellum, and even the visual pathway in various sections.[50] Although these patients may have visual defects, they are not necessary in order to have the phenomenon.[51] In this pathology, garish visual images can also be observed, similar to those of Charles Bonnet syndrome and to those of hypnagogic states with retained *insight* and alertness[8] (Table 2).

THEORIES PROPOSED AROUND PHYSIOPATHOLOGICAL MECHANISMS

Sensory deprivation: phantom vision

Visual hallucinations associated with loss of vision have been conceptualized as Phantom Vision due to deafferentation, and compared with phantom limb syndrome.[8] When the visual cortex is deprived of normal afferent *inputs*, it can exhibit independent spontaneous activity, resulting in conscious imagery, a hypothesis maintained by the fact that the hallucinations can be eliminated by normal or excessive visual stimulation.[8,52] Furthermore, this theory is also partially maintained in functional neuroimaging studies: in one study with functional magnetic resonance imaging, four patients with Charles Bonnet syndrome showed that active hallucinations were associated with independent spontaneous activity in the ventral occipital lobe.[53] The content of the hallucinations was related to activations in specific regions which correlated with the known specialized function of this area of the visual cortex.

The role of the serotonin/acetylcholine balance

In their article *Neuropsychiatry of Complex Visual Hallucinations*,[43] Mocellin et al. explained the relevance of cholinergic excitatory centers (parabrachial and parabigeminal) and serotonergic inhibitors which come from the dorsal raphe, explaining that lesions of the retina or other proximal lesions remove or alter the *inputs* from the external environment to the visual system, resulting in spontaneous complex visual hallucinations of portions distal to these systems. Lesions of the brain stem can therefore interrupt the serotonergic inhibitory *inputs* of the raphe nucleus, causing an excitation of the dorsal lateral geniculate nucleus, and deregulation of the *inputs* of the retina, causing complex visual hallucinations.

Furthermore, they explain that dorsal raphe nuclei have been involved in the sleep regulation cycle and in the reg-

Table 2. Comparison between Peduncular Hallucinosis and Charles Bonnet syndrome, modified by Mocellin, Walterfang, and Velakoulis[45]

Clinical aspect	Peduncular hallucinosis	Charles Bonnet syndrome
Sensory predomination	Predominantly visual perceptions	Predominantly visual perceptions
Attributes of visual experience	Vivid, well-formed, and color perceptions	Vivid, well-formed, and color perceptions
Lilliputian hallucinations	May be present	May be present
Movement of hallucinated figures	Variable	Usually present
Stereotype of hallucinations	Variable	Usually non-stereotyped images
Predominant timing	Predominantly at night	Predominantly at night
Sensory	Generally clear	Generally clear
Ocular pathology	May be present	Generally present
Pathology of Central Nervous System	Generally present	May be present
Age range	May occur at any age	Generally occurs in elderly adults
Insight	Generally retained, although there may sometimes be hallucinatory behavior	Generally retained, although there may sometimes be hallucinatory behavior

ulation of rapid eye movement (REM) and non-REM sleep. It has been seen that complex visual hallucinations can be seen in normal sleep, sleep disorders, *delirium*, Lewy body disorders, psychosis associated with Parkinson's disease, and that they are seen to be accentuated in Charles Bonnet syndrome in situations of reduced alertness or light, which suggests an important role of the dorsal raphe nucleus system in generating said hallucinations.

In this context, some authors think that it may be related to a sleep "unmasking" mechanism and the emergence of hallucinations.[8]

Irritative theory

Ictal activity from an irritative center, possibly the occipital or temporal cortices, has also been proposed as a cause in the genesis of visual hallucinations.[4,8,16] Ossola et al.[54] presented a case with hallucinations typical of Charles Bonnet in a woman with hemorrhagic cerebrovascular events which presented periodic lateralized epileptiform discharges (PLEDs) and which improved through adjustment of antiepileptics.

Figure 1 summarizes these theories.

Sensory Deprivation: If the visual cortex is deprived of extrinsic afferent information due to lesions in the visual pathway, it can exhibit independent spontaneous activity, resulting in conscious imagery. *Serotonin/acetylcholine imbalance*: The passing of information from the lateral geniculate nucleus from the thalamus to the cortex is modulated by excitatory projections from the cholinergic system and inhibitors from the serotonergic system. Lesions of the brain stem can interrupt inhibitory serotonergic modulation, causing excitation of the dorsal lateral geniculate nucleus, deregulation of the information coming from the retina, and complex visual hallucinations. *Irritative theory*: Ictal activity in the occipital-temporal cortex can cause visual hallucinations.

DISCUSSION AND CONCLUSION

Complex visual hallucinations secondary to deprivation of the visual pathway remain a little-known, albeit frequent, issue. The wide variability in their clinical presentation and in the diseases that accompany them means that there is still no consensus around whether or not they constitute a syndrome in themselves, which limits their systematic study. However, we believe that continuing to analyze case-by-case with the variables already described in the bibliography may in future allow for better classification among nosological bodies.

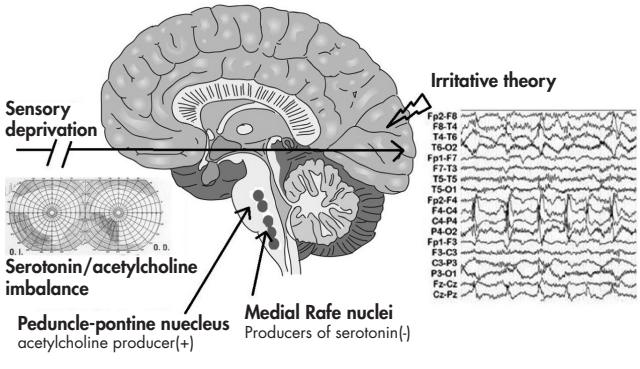

Figure 1. Possible physiopathological mechanisms

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Conflict of interest

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