Brain modules of hallucination: an analysis of multiple patients with brain lesions

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We systematically reviewed the localization of focal brain lesions that cause isolated hallucination in a single sensory modality. Case reports of post-lesion nonparoxysmal hallucination in 1 (and only 1) of 3 sensory modalities (i.e., visual, auditory, somatic) were reviewed, and the content of the qualitative descriptions was analyzed for each modality. The lesion is practically always located in the brain pathway of the sensory modality of the hallucination. There seem to exist localized sensory brain circuits that in healthy people diminish the intensity of internal sensory representation. After a lesion, hallucinosis seems to be caused also by compensatory overactivation of tissue in the nearby brain sensory pathway. This type of hallucination may indeed be termed a "release" form, whereby patients are aware of the hallucinatory nature of their experience, but not usually of "dream centres" as proposed by Lhermitte. Instead, we propose that it is dreaming that should be considered a special case of neural "release."

Nous avons passé en revue systématiquement l'emplacement des lésions cérébrales focales qui causent des hallucinations isolées dans un seul mode sensoriel. On a analysé des rapports de cas portant sur l'hallucination non paroxystique postlésionnelle dans un mode sensoriel (et un seulement) sur trois (c.-à-d. visuel, auditif, somatique), et on a analysé le contenu des descriptions qualitatives de chaque mode. La lésion est presque toujours située dans la voie cérébrale du mode sensoriel de l'hallucination. Il semble y avoir des circuits cérébraux sensoriels localisés qui, chez les gens en bonne santé, «atténuent» l'intensité de la représentation sensorielle interne. Après une lésion, l'hallucinose semble être causée aussi par une suractivation compensatoire de tissus de la voie sensorielle cérébrale voisine. On peut en fait qualifier ce type d'hallucination de forme de «libération», dans laquelle les patients sont conscients de la nature hallucinatoire de leur expérience, mais non habituellement de «centres oniriques» comme le propose Lhermitte. Nous proposons plutôt de considérer le rêve comme un cas spécial de «neurolibération».

Introduction

The purpose of this article is to systematically review the localization of brain lesions that cause isolated hallucination in a single sensory modality, specifically, vision, audition or somesthesia. Here we define hallucination widely as any false perception.

Everyone has experienced hallucinosis, that is, has

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recognized a false perception in oneself, such as the black points or "phosphenes" from eyeball rubbing, the tinnitus or "acouphenes" after a rock concert or with a fever, or "somatophenes" such as the illusion of weightlessness caused by the legs being crossed for too long. However, when the false perception is more spectacular, we immediately think of psychosis. The lesion approach, that is, the notion that psychosis can be caused by brain lesions, to the understanding of psychosis is full of pitfalls. Psychosis is a heterogenous phenomenon: patients present with various neurotic traits and affective symptoms, differing modalities of hallucination, paranoid or nonparanoid delusions, negative or positive symptom complexes, dementia, delirium, paramnesia, agitation and adynamia, among other symptoms. Finding patients who have developed such a mixture of disorders after a focal lesion probably would not provide a neat set of lesion loci. Cummings[1] and Pollack et al[2] have argued that the diverse lesion sites apt to produce psychosis tend to involve dopaminergic circuits. However, dopaminergic circuits are widespread, and this idea must be considered tentative.

We reviewed single case reports of post-lesion psychosis involving hallucination and delusion and found that the temporal lobe was the most common lesion site[3–12] followed by the frontal lobe[13–18] and, then, tissue around the third ventricle.[8,19–22] We hesitate to believe that this is indicative of a predilection for lesion loci to be located within dopaminergic pathways. Limiting oneself to a review of case reports of pure delusional states also does not yield a neat set of lesion loci. Cases have been described of patients with frontal lesions,[23,24] temporal lesions,[7,25] parietal lesions[18,26] and lesions of the basal ganglia.[27,28] It has been suggested that psychotic auditory hallucination is modulated by internal speech, and brain metabolic imaging of hallucinating psychotic patients and measurement of perioral musculature in psychotic patients both support that point of view.[29] This possible mechanism has not, as far as we know, been discussed in post-lesion cases, although there are several reports of patients who have reported hearing voices (more commonly singing). Indeed, the "internal speech" mechanism seems unlikely in post-lesion cases for a number of reasons. Namely, the lesion involves the auditory pathway and not the expressive speech areas, the content of the hallucinated speech does not correspond to any apparent emotional or moral obsession of the patient (as seems to occur in psychosis), the speech is often a conversation among several people known to the patient or singing by a person heard previously by the patient, and the speech is often of a person of the opposite sex. This leads us to wonder whether the perioral activity of patients with schizophrenia during hallucinated speech might not consist of a form of subliminal echolalia, a phenomenon that we would not expect in post-lesion hallucinations. Still, measurement of perioral activity during post-lesion hallucination of speech would be worth investigating.

With the ever-increasing number of single-case post-lesion reports, it has now become possible to review meaningfully large sets of patients with focal lesions who subsequently developed highly specific and limited false perceptions. The present literature review, which is based on multiple single case reports, will therefore focus on cases of pure hallucination, in specific sensory modalities, without neurotic, affective or delusional symptoms. Our method for reviewing this literature consisted of searches of the entire MEDLINE database and then detailed follow-up using the bibliographies of the articles thus obtained. This approach allows for a clear hypothesis that has been formulated on several occasions in single case reports and more limited multiple case reviews, namely, that pure isolated syndromes of hallucination tend to result from lesions located in the sensory brain pathways of the modality in question. Although it would be conceivable to review all the senses, post-lesion gustatory or olfactory hallucinations are too rare to support inference testing. Thus, we shall limit this review to the 3 most epicritic of the senses, namely vision, audition and somesthesia, restricting ourselves to nonparoxysmal cases manifesting hallucinations in only 1 of these modalities with the localizing hypothesis in mind. By primary visual pathway of the brain, we mean the main nuclei of that pathway and important cortical radiations: retina; optic nerve; chiasma; diagonal bands; lateral geniculate body; temporal, parietal and occipital lobes; and superior colliculus. By primary auditory pathway, we mean the pons, inferior colliculus, medial geniculate body and temporal lobe. By the somesthetic pathway, we mean the spinal cord, medulla, pons, thalamus and parietal lobe.

Epilepsy that accompanies a lesion may or may not override and even contradict lesion effects by irritating functional tissue and is sufficient cause of psychiatric symptoms including hallucination.[30] Depending upon whether a symptom complex is released or destroyed by a lesion, or is activated or inhibited by pathologic irritation of brain tissue, any symptom complex could

conceivably result from many brain disorders. Hallucination in epileptic patients is known to abate after resection.[31] To the extent that we can clearly distinguish cases with or without irritative conditions based on clinical electroencephalography (EEG), then some order may be brought to the whole issue. In fact, epilepsy is only 1 irritative condition of the brain. Migraine is another, as are toxic conditions, some tumours, and so on. Certain mitotic lesions have proximal electrical and metabolic irritative effects that can override the effect of tissue loss in the tumour area. This is well illustrated by Filley and Kleinschmidt,[5] who reported alleviation of psychiatric symptoms in all 5 of their operable neoplastic cases by surgical removal of the tumour. Consequently, in the multiple case review that we are proposing here, careful attention was paid to the origin of the lesion, as well as to clinical EEG parameters and any mention of seizures.

Two types of neurogenic hallucination were distinguished early on (1973) by Cogan:[32] the irritative and release forms. The *irritative* forms are associated with migraine, tumour and, of course, paroxysmal EEG. They are reputed to be stereotyped and simple, as in migraine auras, and to have some localizing value. They may or may not be associated with brain lesions. When they are, the association is with diffuse radiologically unidentifiable lesions. The patient is not typically aware of the hallucinatory nature of his or her perception (the false perception is then termed *hallucinosis*). The *release* forms are associated with silent localized lesions, have little or no localizing value, often represent remarkably complex scenes and vary in the same patient. The patient is typically perfectly lucid concerning the "hallucinatory" nature of the perception. The image is often recognized from memory, suggesting that it is in fact more an overly vivid recollection, recognized as such or not, than a false perception.

Lhermitte[33] proposed in 1922 a special midbrain syndrome, which he termed "*peduncular hallucinosis*," possibly involving release, presumably via destruction of an inhibitory influence, of dream-like activity. Sleep disorders have on a few occasions been associated with this type of hallucination. Lhermitte associated the sleep disorder with the hallucination and likened the hallucinosis to a mechanism similar to narcoleptic hallucinosis. Several cases have since been reported of post-lesion hallucinosis without sleep disorder. In addition, we will demonstrate that the lesion loci for these types of hallucinations can be anywhere from the pons to the thalamus to the cerebral cortex. Thus, Lhermitte's idea that such highly lucid and articulated hallucinations might be a form of midbrain release of dream processes seems doubtful.

S*ensory deprivation* due to peripheral damage, as in Charles Bonnet syndrome, is believed to remove competition with endogenous representation (imagination) and thus to lower the threshold for hallucination.[34] Indeed, most reports of post-lesion hallucinosis in the visual modality, for example, consist of representations located in the hemianopic field,[35] and they commonly occur at twilight or at night.[36] Hearing loss is common in post-lesion auditory hallucinosis,[37] and somesthetic deficits are common in hallucinations of the body image.[38] It is interesting to note that Lhermitte's 1922 patient also presented ophthalmologic problems, retrospectively making that case a reasonable candidate for Charles Bonnet syndrome.

At present, it is not known whether children may also manifest pure, namely, isolated hallucinations subsequent to lesions, as is the case for adults. Consequently, we will pay special attention to that issue as well.

Visual hallucinosis

Where are visual hallucinations generated in the brain?

Evidence from stimulation and lesion studies

Simple visual hallucinations from electrocortical stimulation are most typically evoked by occipital probing, whereas complex ones involving people, animals and scenes are more readily evoked by temporo-occipital or parieto-occipital stimulation, according to Penfield and Perot.[31] In addition, the same authors found that occipital stimulation tended to produce hallucinations in the contralateral field, whereas temporal stimulation produced hallucinations in both fields. Although the stimulation site that is likely to produce visual hallucinations is typically in the visual pathway, a few sites outside the visual pathway have also been reported. For example, stimulation of the subthalamic nucleus produced visual hallucinosis in a parkinsonian patient that was promptly relieved by clozapine.[39]

Visual hallucinations in the adult have been stated to result from lesions anywhere in the visual pathway, including the retina, brainstem pathways and occipital lobe.[40] Lesions in the suprasellar area diencephalon have been reported to produce visual hallucinosis in 2

patients,[41] presumably because of the lesions' proximity to the diagonal bands or chiasma. Smith et al[42] described a patient with visual hallucinations with several lesions, but only 1 of which involved the visual pathway, namely, the optic chiasm. In his study of 120 patients with hemianopia or quadrantanopia, Kölmel[35] found that for the 16 with hallucinations, the associated lesions located by computed tomography were especially in the occipital lobe but extended sometimes toward the parietal lobe or the temporal lobe, or both. Parkinson et al[43] studied patients with occipital lobe lesions and found that this lesion locus was associated with hallucinations in 12 cases. In a vast study of patients with visual loss and spontaneous visual phenomena, Lepore[44] showed that visual hallucinations were associated with lesions of the visual pathway, no matter the exact location, including the brain stem. Galasko et al[45] also found, in a review of the literature (MEDLINE, 1966–1987) on the psychiatric disorders associated with mass lesions, an association between visual hallucinations and lesions to various parts of the visual pathway.

Evidence from metabolic imaging studies

Ffytche et al[46] carried out functional magnetic resonance imaging (fMRI) of visual hallucination in 5 cases of Charles Bonnet syndrome. They found selective activation in the parts of the visual cortex that are known to process the types of images that appeared in each subject's hallucinations. In addition, the patients as a group had tonic activation in the visual cortex even when not hallucinating. Howard et al[47] published similar findings and interpretation. Adachi et al[48] reported single photon emission computed tomography (SPECT) using iodine 123 iodoamphetamin (IMP) and MRI studies of 5 patients with Charles Bonnet syndrome while they were having visual hallucinations. All patients developed complex visual hallucinations after suffering from eye disease. SPECT disclosed hyperperfusion areas with some asymmetrical appearances in the lateral temporal cortex, striatum and thalamus. This suggested to the authors that when elderly people suffer from eye disease, subsequent excessive cortical compensation in the lateral temporal cortex, striatum and thalamus may precipitate the development of visual hallucinations.

Case reports of pure visual hallucination subsequent to radiologically or surgically objectified focal lesions of the brain are listed in Table 1,[49–65] Table 2[66–73] and Table 3.[74]

What is the content of nonparoxysmal post-lesion visual hallucinations?

Charles Bonnet syndrome is usually characterized by the presence of vivid and complex visual hallucinations, which are recognized as unreal (i.e., pseudohallucinations, parahallucinations or hallucinosis) and occur in the absence of any other psychiatric symptoms or cerebral lesion. Some researchers suggest that isolated visual hallucinations in older adults, often with visual impairment, and frequently sedentary and sensorially deprived, may be an indication of early stages of dementia. Contrary to what was considered for a long time, the syndrome seems to occur frequently. Santhouse et al[75] reported a detailed content analysis of the hallucinations of 39 patients with Charles Bonnet syndrome. They found that the most common hallucination involved landscapes with small figures in costumes with hats. The next most common hallucination comprised grotesque faces with prominent eyes and teeth. Finally, the third content category consisted of visual perseveration or palinopsia, or both.

We were intrigued by a statement by Kölmel[35] to the effect that whenever a directional movement of the hallucinated representation was well identified (6 patients), it was from the periphery to the macular area. We found 12 reports that specified the direction of the movement of a hallucinated object: indeed, the movement was from the periphery to the macular portion of the field in 8 cases.[51,57,58,76,77] However, 4 patients have been described[40,57,62] whose hallucinated object always moved from the meridian to the periphery. In short, the evidence refutes any suggestion of a systematic direction of moving hallucinated objects.

We were also intrigued by Kölmel's statement to the effect that "identical replication," namely, synchronous systematic repetition of a visual image (variably termed polyopia, tesselopsia and palinopsia) may be interpreted as an expression of the functional architecture of the primary visual cortex.[35] Various such recurring forms are frequent in the cases reported by other authors (see Table 4 for details of our content analysis) and are obtained by electrical stimulation of the occipital cortex.[78]

Finally, content analysis of hallucinosis in eye disease led Ffytche and Howard[79] to report an intriguing hallucination consisting of "tree" shapes, which they termed "dendropsia" (14% frequency). The authors entertained the fascinating idea that this perception could result from intrusion of retinal vasculature but opted, rather,

for long-range release of inhibition as a potential cause. Dendropsia was not observed in our extra-ocular cases (Table 4), suggesting that there might be more grounds for the former than the latter speculation.

Auditory hallucinosis

Where are auditory hallucinations generated in the brain?

Evidence from stimulation and lesion studies

Electrical brain stimulation that is apt to produce reports of auditory experiences is typically located in the temporal cortex, in either hemisphere. Spontaneous ictal auditory hallucinations are most common in temporal lobe epilepsy but also occur, less frequently, in cases with foci well localized in other lobes. Complex hallucinations such as voices or music are nearly universally associated with temporal cortex stimulation or irritation, whereas simple ones such as hissing, buzzing, and so on, can result from subcortical or insular stimulation or irritation.[31]

Post-lesion auditory hallucination, though less frequent than visual, follows the same logic as the visual forms: the typical lesion is situated anywhere along the auditory pathway, including the brain stem.[71] It is quite common in post-lesion auditory hallucination for the patient to manifest deficits of audition such as distortion, poor localization, hypoacusis[80] or even deafness,[37] suggesting that deafferentation can mediate the effect.

Evidence from metabolic imaging studies

Dierks et al[81] used an fMRI protocol to study brain activation during auditory hallucinations in 3 patients with schizophrenia. They found maximal activation in the transverse temporal gyrus of the dominant hemisphere and other activation in the posterior superior temporal gyrus, middle temporal gyrus, frontoparietal operculum, hippocampus, amygdala and sensorimotor cortex. The patients were asked to press a button when their auditory hallucinations started, and to release it when they stopped. Unfortunately, the authors did not use a button-pressing task as a control condition. Activation reported therefore contains mixed signal produced by the auditory hallucination and the motor task. A recent fMRI study[82] of 6 patients with schizophrenia found maximal activation in the right inferior colliculus, right and left insula, left parahippocampal gyrus, right temporal gyrus, right thalamus, middle frontal and anterior cingulate gyri, and right inferior and superior temporal lobe during auditory hallucinations. The authors conclude that, because the areas involved in auditory verbal hallucinations are also involved in inner speech, auditory hallucinations result from defective monitoring of inner thoughts. In another study, continuous whole-brain fMRI with a 3-T magnet was used to map the cerebral activation associated with auditory hallucinations in 4 subjects with schizophrenia.[83] The subjects experienced episodes of hallucination while in the scanner, so periods of hallucination could be compared with periods of rest in the same individuals. Group analysis demonstrated shared areas of activation in the right and left superior temporal gyri, left inferior parietal cortex and left middle frontal gyrus. When the data were examined on an individual basis, the temporal cortex and prefrontal cortex areas were activated during episodes of hallucination in all 4 subjects. The authors concluded that these findings support the theory that auditory hallucination reflects abnormal activation of normal auditory pathways. As is to be expected from the functional imaging method, activated areas tend to be numerous and several of these are probably epiphenomenal. Positron emission tomography (PET) was used to study the brain state associated with the occurrence of hallucinations in 5 patients with schizophrenia with classic auditory verbal hallucinations despite medication.[84] During the hallucinations, there was activation in the subcortical nuclei thalamic, striatal, limbic structures (especially hippocampus and paralimbic regions, parahippocampal and cingulate gyri, as well as the orbitofrontal cortex). The authors propose that activity in deep brain structures, identified with group analysis, may generate or modulate the hallucinations, and the particular neocortical regions involved in individual patients may affect the specific content of their hallucinations. Our reading of this literature suggests to us that in psychotic auditory hallucination, the most likely mechanism is release of inhibition of the auditory cortex by other cortical auditory neural assemblies, including those in the contralateral hemisphere.[85] This appears to be the same mechanism as in post-lesion musical hallucinosis: activation in one such case was in the areas (posterior temporal lobes) that would have been expected in normal hearing of such sounds in healthy individuals.[86] Case reports of post-lesion auditory hallucinosis are presented in Table 5[87–91] and Table 6.[92–94]

Table 1: Nonparoxysmal visual hallucinations resulting from radiologically or surgically objectified telencephalic lesions (adult cases)

Study	Age at onset, yr	Sex	Lesion location	Symptoms	Etiologic considerations, complications
Kölmel[35]	66	F	Right occipital	Complex visual hallucinations	Infarct and atrophy, right temporal EEG slowing
	74	F	Right occipitoparietal	Complex visual hallucinations	Infarct, no EEG or seizures reported
	75	M	Left occipital	Complex visual hallucinations	Infarct, no EEG or seizures reported
	68	M	Left occipitoparietal	Complex visual hallucinations	Oligodendroglioma, no EEG or seizures reported
Manford and Andermann[49]	55	F	Left occipital cortex to the left posterior thalamus	Complex visual hallucinations	Infarct, no EEG or seizures reported
	54	M	Left occipital cortex to the left posterior thalamus	Complex visual hallucinations	Infarct, no EEG or seizures reported
Michel and Troost[50]	68	M	Left occipital	Complex visual hallucinations	Infarct, no EEG or seizures reported
	71	F	Right occipital	Complex visual hallucinations	Infarct, no EEG or seizures reported
	66	M	Right occipital	Complex visual hallucinations	Infarct, no EEG or seizures reported
Critchley[51]	32	F	Right parieto-occiptal	Complex visual hallucinations	Infarct, no EEG or seizures reported
Bender[52]	20	M	Left occipital	Complex visual hallucinations	Penetrating wound, no EEG or seizures during hallucination reported
	22	M	Right occipital	Visual hallucinations	Penetrating wound, no EEG or seizures during hallucination reported
Meadows and Munro[53]	73	F	Right occipitotemporal	Complex visual hallucinations	Infarct, no EEG or seizures reported
	56	F	Right occipitotemporal	Complex visual hallucinations	Meningioma, EEG slowing on the right
La Mancusa and Cole[54]	66	F	Right occipital	Visual hallucinations	Infarct, no EEG or seizures reported
Medina et al[55]	58	M	Right occipital	Visual hallucinations and agitation	Cerebrovascular accident, slowing of EEG on left
	37	M	Left occipitotemporal	Visual hallucinations, agitation and aggressiveness	Infarct, slowing of EEG on left
Lance[56]	62	F	Right occipital	Complex visual hallucinations	Infarct (case 9), no EEG or seizures reported
	65	F	Right occipital	Complex visual hallucinations	Infarct (case 10), no EEG or seizures reported
	53	M	Right occipital	Complex visual hallucinations	Infarct (case 7), no EEG or seizures reported
	73	F	Right occipital	Complex visual hallucinations	Infarct (case 12), no EEG or seizures reported
	72	M	Right occipitoparietal	Complex visual hallucinations	Infarct (case 11), no EEG or seizures reported
	60	F	Right parietal	Complex visual hallucinations	Infarct (case 8), no EEG or seizures reported
	48	M	Left occipital	Complex visual hallucinations	Infarct (case 5), no EEG or seizures reported
	72	F	Left occipital	Complex visual hallucinations	Infarct (case 13), no EEG or seizures reported
	70	F	Left occipital	Complex visual hallucinations	Infarct (case 6), no EEG or seizures reported
Vaphiades et al[57]	76	F	Right temporal	Complex visual hallucinations	Infarct, right slow focus in EEG
	46	M	Right occipital	Complex visual hallucinations	Infarct, normal EEG
	62	M	Right occipital	Complex visual hallucinations	Infarct, no EEG or seizures reported
	56	M	Right occipital	Complex visual hallucinations	Infarct, normal EEG
	46	F	Right occipital	Complex visual hallucinations	Infarct, right slow focus in EEG
	67	F	Right occipital	Complex visual hallucinations	Infarct, normal EEG
	72	M	Right occipitotemporal	Complex visual hallucinations	Infarct, EEG slowing on left side
	71	F	Left occipital	Complex visual hallucinations	Infarct, normal EEG
Hoksbergen et al[58]	64	M	Right occipitotemporal	Visual hallucinations, metamorphopsia, left hyporeflexia, mild prosopagnosia	Ischemia, slowing of EEG at posterior right scalp
Lindner et al[59]	41	M	Right occipitotemporal	Visual hallucinations, left homonymous hemianopia	Ischemia, history of migraine with aura, EEG slowing over right parieto-occipital area
	64	F	Right temporal	Complex visual hallucinations	Infarct, no EEG or seizures reported
	67	M	Right temporal	Complex visual hallucinations	Infarct, no EEG or seizures reported
Benson and Rennie[60]	64	M	Left occipital	Complex visual hallucinations	Infarct, no EEG or seizures reported
Kasten et al[61]	56	M	Left occipital	Complex persistent visual hallucinations	Infarct, no EEG or seizures reported
Anderson and Rizzo[62]	79	M	Left occipital	Visual hallucinations	Infarct, normal EEG including during hallucinations
Cole[63]	63	M	Left occipital	Visual hallucinations, right hemianopia	Infarct, no mention of EEG or seizures
Werring and Marsden[64]	35	M	Left occipital	Visual hallucinations, palinopsia	Tuberculoma, no mention of EEG or seizures
Tapiador et al[65]	70	M	Right parietal	Complex visual hallucinations	Infarct (case 2), normal EEG

Note: EEG = electroencephalography.

What is the content of nonparoxysmal post-lesion auditory hallucinations?

Parallel to the visual modality, a repetitive simple sound is probably the most common form of post-lesion hallucination, and it will usually be diagnosed as an acouphene (tinnitus). The cause is often obscure, but it can be the result of a vascular abnormality of the cervical region, skull base or cranium, or a tumour. Interestingly, more complex auditory hallucinations, such as music and human voices, are not so unusual subsequent to brainstem lesions, evoking the surprisingly complex representations described by patients with visual post-lesion hallucinosis (Table 1, Table 2 and Table 3). In Table 7, we classify the contents of auditory hallucinations of cases reviewed in Table 5 and Table 6.

Somatic hallucinosis

Where are somatic hallucinations generated in the brain?

Evidence from stimulation and lesion studies

Somatic hallucination is crudely classifiable into 3 basic forms: pain, paresthesia and complex somatoparaphre-

Table 2: Nonparoxysmal visual hallucinations resulting from radiologically or surgically objectified nontelencephalic lesions (adult cases)

Study	Age at onset, yr	Sex	Lesion location	Symptoms	Etiologic considerations, complications
Feinberg and Shapiro[34]	83	M	Right thalamus	Visual hallucinations, vertigo, insomnia	Infarct, no mention of EEG or seizures
Shiga et al[40]	63	M	Left putamen, left optic radiation	Metamorphopsia, visual hallucinations, right hemiparesis, right homonymous hemianopia	Hemorrhage, EEG was normal
Vaphiades et al[57]	72	M	Right thalamus, capsule, basal ganglia	Complex visual hallucinations	Infarct, diffuse slowing of EEG
	69	M	Right thalamus and temporal lobe	Complex visual hallucinations	Infarct, EEG slowing on the right side
Tapiador et al[65]	35	M	Dorsolateral medulla	Complex visual hallucinations	Infarct (case 1), no mention of EEG or seizures
Howlett et al[66]	63	M	Left cerebral peduncle	Complex visual hallucinations	Infarct, EEG abnormalities unspecified
Hattori et al[67]	73	M	Right midbrain pons, right putamen	Complex visual hallucinations, proximal myoclonus	Unspecified infection, mild diffuse slowing of the EEG
Geller and Bellur[68]	61	M	Right midbrain tegmentum	Complex visual hallucinations	Infarct, no mention of EEG or seizures
McKee et al[69]	83	M	Bilateral pars reticulata of substantia nigra	Complex visual hallucinations	Infarct, diffuse slowing of EEG
Catafau et al[70]	68	M	Right thalamocapsular region	Left paresthesia, left hemiparesis, visual hallucinations	Infarct, right EEG slowing
Danziger et al[71]	56	M	Right pons, thalamus and cerebellum	Complex visual hallucinations	Infarct, no mention of EEG or seizures
De La Fuente Fernandez et al[72]	70	F	Left cerebral peduncle	Complex visual hallucinations	Infarct, normal EEG
Van Bogaert[73]	59	F	Left midbrain	Complex visual hallucinations	Infarct, no mention of EEG or seizures

Table 3: Nonparoxysmal visual hallucinations resulting from radiologically or surgically objectified nontelencephalic lesions (juvenile cases)

Study	Age at onset, yr	Sex	Lesion location	Symptoms	Etiologic considerations, complications
Kumar and Kaur[74]	4	M	Cerebral peduncle	Visual hallucination after surgery for peduncular medulloblastoma	Medulloblastoma, no mention of EEG or seizures
Kumar et al[41]	10	F	Sellar and suprasellar	Complex visual hallucinations	Resected astrocytoma, no mention of EEG or seizures

Table 4: Types of contents of nonparoxysmal visual hallucinations resulting from brain lesions (listed in Tables 1, 2 and 3)

Content of hallucination	No. of reports
Animals, frequently moving	
Dogs	11
Birds	7
Cats	4
Lions	3
Horses	3
Turtles, bulls, wolves, foxes, snakes, insects, goats, butterflies, hedgehogs, rodents	39
Moving recurring forms, i.e., tessellopsia	
Drapes	4
Fortifications	2
Circles	2
Steps, pyramids, US flag, piano keys, tiles, wallpaper, checkerboard, geometric figures, lines, zigzags, billiard balls, coloured drawers, crescents, ring of feathers, beige fans moving, black spots, rippling, twirling, whirling, undulation, pylon with a spiral around it, Christmas candles, artificial eyelashes	37
People, frequently moving, frequently of reduced size*	
Rows of uniformed soldiers	4
Child	3
Floating people	2
Costumed miniature figures	2
Crawling men, genuflecting men, a hundred visitors, baseball players, toreadors, marching peasant women, row of top-hatted Lilliputians, nurse, oneself younger, oneself smaller, friends, armed assailants, teacher, pedestrian crossing a road, Santa Claus	33
Body parts	
People's faces	9
Hands	6
Heads	3
Claws, tubes coming out of people's heads, coloured octopus legs	22
Coloured light arrays	
Fireworks	2
Explosion of colour, coloured bright flashes, aircraft lights descending, purple blobs, a fountain of light	15
Objects, frequently moving	
Trains	3
Airplane, cylinders, telescope, motors, motorcycle, cars, televisions, spaceship, toys, rock, coloured stationery, tiny caravan, arrows, a painting on the wall, object fragments, floating objects	15
Phantasmagoric creatures	
Dainty fairy, toy monsters, angel, Garfield the cat	4
Buildings	4
Natural outdoor landscape	3
Visual perseveration, i.e., palinopsia	
Multiple objects	3
Bananas	1
$20 bills	1
Dress	1
Starburst	1
Moving shadows	
Macropsia, (e.g., self is 40 000 ft tall)	3
Micropsia (e.g., self is Lilliputian), zoom, scintillating scotoma, smoke, seaweed, flowers	11

Note: Scenes frequently have an artistic appearance. Goya, Bosch and Dali were mentioned by 3 patients.
*The hallucinated people are more often costumed and wearing hats than not and the faces hidden from view.

nia. In a review of 16 cases of post-lesion pain, Gonzales et al[95] concluded that "central pain" results from lesions anywhere in the somesthetic projection system. Bowsher et al[96] came to the same conclusion on the basis of their investigation of 73 cases of post-lesion pain. These cases include focal unilateral parietal lesions,[97] but thalamic lesions are most likely to produce pain resembling a psychiatric syndrome: "pain in the thalamic syndrome"[98] is of the burning sort, is generalized and may be provoked by benign stimuli (allodynia). Even spinal cord injuries

Table 5: Nonparoxysmal auditory hallucinations resulting from radiologically or surgically objectified telencephalic lesions (adult cases)

Study	Age at onset, yr	Sex	Lesion location	Symptoms	Etiologic considerations, complications
Filley and Kleinschmidt[5]	22	M	Left medial temporal	Auditory hallucinations	Oligoastrocytoma, no mention of EEG or seizures
Fujii and Ahmed[23]	20	M	Right frontoparietal	Auditory hallucinations	Cranial trauma, diffuse EEG slowing
Griffiths[86]	82	F	Right occipitotemporal	Complex auditory hallucinations	Cyst, normal EEG
Paquier et al[87]	NR	NR	Right temporoparietal	Complex auditory hallucinations	Hemorrhage, normal EEG
Hécaen and Ropert[88]	44	M	Left temporal	Auditory verbal hallucinations in contralateral ear	Glioma, no mention of EEG or seizures
Tanabe et al[89]	64	F	Left superior temporal gyrus	Auditory hallucinations, transient Wernicke's aphasia	Hemorrhagic infarct, left EEG slowing
Lesser et al[90]	63	F	Left temporoparietal	Auditory hallucinations	Subarachnoid cyst, no EEG or seizures reported
Bremer[91]	61	M	Right temporal	Auditory hallucinations pre-lesion post-traumatic stress disorder and panic attacks	Hemorrhage and lobectomy, no seizures, right EEG slowing

Note: We were unable to find any published description of pure post-lesion auditory hallucination in a child. NR = not reported.

Table 6: Nonparoxysmal auditory hallucinations resulting from radiologically or surgically objectified nontelencephalic lesions (adult cases)

Study	Age at onset, yr	Sex	Lesion location	Symptoms	Etiologic considerations, complications
Murata et al[37]	55	M	Right pontine tegmentum	Auditory hallucinations	Hematoma, EEG was normal
Cambier and Decroix[80]	54	M	Inferior colliculus	Complex auditory hallucinations	Infarct (case 5), no mention of EEG or seizures
	56	F	Right paramedian pons	Complex auditory hallucinations	Infarct (case 4), no mention of EEG or seizures
	55	F	Right paramedian pons	Complex auditory hallucinations	Infarct (case 3), no mention of EEG or seizures
	74	F	Right paramedian pons	Complex auditory hallucinations	Infarct (case 2), no mention of EEG or seizures
Tanabe et al[89]	61	M	Left paramedian pons	Complex auditory hallucinations	Hemorrhage (case 1), EEG slowing with no epileptiform signs
Inzelberg et al[92]	75	F	Left thalamic	Complex auditory hallucinations	Infarct, no mention of EEG or seizures
Lanska et al[93]	55	M	Left dorsal pons	Complex auditory hallucinations	Hemorrhage, mild EEG slowing without epileptiform signs
Cascino and Adams[94]	42	F	Right pontine tegmentum	Auditory hallucinations, right ear hearing loss, left hemianesthesia, right arm and leg ataxia	Hemorrhage (case 2), normal EEG
	33	F	Left pontine tegmentum	Auditory hallucinations, right ear hearing loss	Hemorrhage (case 1), no mention of EEG or seizures
	53	F	Bilateral midbrain at the level of the inferior colliculi, right cerebellum	Auditory hallucinations, deafness, bilateral ptosis, nystagmus retractorius, limited upgaze, spastic left hemiparesis, left hemisensory loss	Metastatic tumour (case 3), right EEG slowing

Note: We were unable to find any published description of pure post-lesion auditory hallucination in a child.

can induce pain below the lesion. Finnerup et al[99] studied 436 spinal cord injury cases and found that 77% of individuals reported pain or unpleasant sensations ("dysesthesias" in the authors' terminology). Complex somatic hallucinations involve feelings of supernumerary or abnormally sized or deformed body parts. This condition was termed somatoparaphrenia by Gerstmann in 1942. The lesion that produces this syndrome is nearly always left parietal, although a few cases with lesions localized elsewhere have been reported.

Evidence from metabolic imaging studies

Using fMRI, Lotze et al[100] investigated 14 upper-limb amputees and 7 healthy controls during the execution of hand and lip movements and imagined movements of the phantom limb or left hand. Only patients with phantom limb pain showed a shift of the lip activation into the deafferented primary motor and somatosensory hand areas during lip movements. Displacement of the lip representation in the primary motor and somesthetic cortex was positively correlated with the amount of phantom limb pain. Thalamic activation was only present during executed movements in the healthy controls. The cerebellum showed no evidence of reorganizational changes. In amputees, movement of the intact hand showed a level of activation similar to movement of the right dominant hand in the healthy controls. During imagination of moving the phantom hand, all patients showed significantly higher activation in the contralateral primary motor and somatosensory cortices compared with imagination of hand movements in the controls. In the patients with phantom limb pain but not the pain-free amputees, imagined movement of the phantom hand activated the neighbouring facial area. These data suggest selective coactivation of the cortical hand and mouth areas in patients with phantom limb pain. This reorganizational change may be the neural correlate of phantom limb pain. With PET imaging, phantom pain has been found to be significantly related to activity in the thalamus, anterior cingulate and lateral prefrontal cortex.[101] Shergill et al[102] examined a patient with schizophrenia with fMRI. They compared the distribution of brain activity during somatic and auditory (verbal) hallucinations, occurring at different times. Somatic hallucinations were associated with activation in the primary somatosensory and posterior parietal cortex, areas that normally mediate tactile perception. Auditory hallucinations were associated with activation in the middle and superior temporal cortex, areas involved in processing external speech. They proposed that hallucinations in a given modality seem to involve areas that normally process sensory information in that modality. Case reports of somatic hallucination after brain lesions are reviewed in Table 8[103–108] and Table 9.[109–114]

What is the content of nonparoxysmal post-lesion somatic hallucinations?

There is a large neurologic and neuropsychologic literature on negative forms of complex disorders of body image, for example, neglect, anosognosia. These forms

Table 7: Types of contents of nonparoxysmal auditory hallucinations resulting from brain lesions (listed in Tables 5 and 6)

Content of hallucination	No. of reports
People's voices	
Talking	5
Singing	5
Voice telling patient where to go, haunting voices, radio announcer, the patient's own voice, children crying, murmuring crowd	15
Elaborate music	
Mozart, foreign music, songs accompanied by instruments, recurrent hymns	8
Bells ringing	4
Buzzing	2
Rain falling on a roof	1
Water out of a faucet	1
Drumming	1
Loud whistling	1
Barking	1
Paper crumpling	1
Combustion engine	1
Freight train	1
Tone	1
Organ note	1
Static white noise	1
Grinding as in heavy machinery	1
Banging	1
Humming	1
Whirring	1
Clicking	1
Chirping	1
Sirens	1
Airplanes	1
Dogs barking	1

Note: Much of the content of these hallucinations is overtly recognized as being drawn from memory.

are not the object of the present review. It is well recognized that somatic hallucinations can derive from organic conditions, such as the paresthesias of delirium tremens (e.g., insects creeping on the skin or vermin under it). The most common forms of post-lesion somatic hallucination are simple, namely, paresthesias such as burning, picking, tingling. These can readily result from thalamic lesions (Table 9). As for positive expressions of complex disorders of body image, these can include supernumerary limbs,[115] hatred of the affected limb (misoplegia),[116] overestimation of the strength of the affected limb,[117] over- or underestimation of the size of the affected limb or mislocation of the affected limb.[118] The content of hallucinations in cases reviewed in Table 8 and Table 9 is classified in Table 10.

Discussion

The localization of lesions that produce an isolated nonparoxysmal syndrome of hallucination in 1 sensory modality is strikingly coherent, more so than indicated by functional imaging studies. It is nearly always in the primary sensory pathway of that modality in the brain. Take the visual versus auditory modalities as a natural test of this idea. The primary visual pathway does not extend below the midbrain, and the primary auditory pathway contains a major part of its circuitry in the pons itself. If low-level lesions in the pathway can easily produce hallucinations in the relevant modality, then visual lesions should result from peduncular lesions, and auditory hallucinations should result from pontine lesions. This is exactly what occurs. Somesthesia offers an even clearer test of this basic principle: Table 9 illustrates that spinal lesions can cause somatic hallucinations, but such lesions are never reported to cause visual or auditory hallucinations.

Post-lesion hallucination in children?

Why are there so few cases of children manifesting isolated syndromes of unimodal hallucination after a focal lesion? One reason could simply be that few children have focal lesions and that case reports will accrue with time. However, there are numerous cases of children with brain lesions, though admittedly fewer than in adults. There is no penury of juvenile cases manifesting florid psychosis after a focal lesion with hallucinations and delusions.[3,4,8,9,11,17,21,22] We also suspect that there are few pediatric cases of pure nonparoxysmal post-lesion hallucinosis because the various comorbidities that facilitate hallucination are usually not present in children (incipient dementia, modal peripheral sensory loss, confusional state, and so on). At any rate, we were not able to find any case report of post-lesion unimodal hallucination in the auditory or somesthetic modalities. We found only 4 cases, limited to the visual modality, and 2 of them (not presented here) had paroxysms on EEG and seizures.

Toward a neurophysiologic model

We speculate that a hallucination, especially a complex

Table 8: Nonparoxysmal somatic hallucinations resulting from radiologically or surgically objectified telencephalic lesions (adult cases)

Study	Age at onset, yr	Sex	Lesion location	Symptoms	Etiologic considerations, complications
Halligan et al[103]	41	M	Right parietotemporal	Somatoparaphrenia	Hematoma, no mention of EEG or seizures
Hécaen and Ajuriaguerra[104]	48	M	Right parietotemporal	Foreign limb, limb neglect	Hemorrhage (case 16), no mention of EEG or seizures
	27	M	Right parietal	Foreign limb	Penetrating injury (case 67), no mention of EEG or seizures
Rode et al[105]	69	F	Right parietotemporo-occipital	Somatoparaphrenia, anosagnosia, limb neglect, logorrhea	Infarct, no mention of EEG or seizures
Assal[106]	86	F	Right parietofrontotemporal	Somatoparaphrenia, anosagnosia, limb neglect	Embolus, no mention of EEG or seizures
Miura et al[107]	77	F	Left lateral thalamus, internal capsule, lateral geniculate body, hippocampus, caudate nucleus and medial occipitotemporal gyrus	Somatoparaphrenia, anosognosia, disorientation, right unilateral spatial neglect and mild amnesic aphasia	Infarct, no mention of EEG or seizures

Note: We were unable to find any published description of pure post-lesion somatic hallucination in a child, except the case reported by Fujii et al[108] of paresthesia in a 4-year-old child with a left parietal lipoma.

one involving full-fledged articulated scenes or events, resulting from a focal lesion, is indeed a release phenomenon — though not necessarily dream-like. More specifically, we propose that the lesioned tissue (supposing that there is no paroxysmal activity and no irritation of brain tissue) must have previously contained a predominance of inhibitory over excitatory neurons for the sensory modality in question. These inhibitory neurons, operating normally, would be capable of "*gauging down*" brain circuits containing complex sensory representations, which would be presumably cortical (see Fuster[119]), even though they are sometimes situated in the brain stem. Perhaps such modules are less developed in children simply because juveniles have a less built-up store of such complex representations, or because of lack of experience or lack of re-experiencing (rehearsal) of such complex events, or both. Considering that there are frequent comorbidities of sensory impairment or deprivation in post-lesion hallucinosis and Charles Bonnet syndrome, it seems likely that compensatory overactivation of remaining nearby tissue after a lesion contributes greatly to the risk of hallucinosis. Again, children would be less at risk for these comorbidities (e.g., cataracts, hearing loss, prolonged immobilization, fatigue, incipient dementia) and thus for post-lesion hallucinosis. Indeed, sensory deprivation alone, in healthy adults, leads to hallucinosis.[120] Immediately after a retinal lesion, field-adjacent cortical cells increase their receptive fields.[121]

This interpretation suggests that Lhermitte's fascinat-

Table 9: Nonparoxysmal somatic hallucinations resulting from radiologically or surgically objectified nontelencephalic lesions (adult cases)

Study	Age at onset, yr	Sex	Lesion location	Symptoms	Etiologic considerations, complications
Hécaen and Ajuriaguerra[104]	Adult	M	Right spinal	Phantom arm	Compression of spine (case 8), no EEG or seizures reported
	34	M	Right spinal	Phantom leg	Compression of spine (case 9), no EEG or seizures reported
	32	M	Bilateral spinal	Hallucinated leg flexion	Trauma and laminectomy of T12 (case 10), no EEG or seizures reported
	Adult	M	Bilateral spinal	Hallucinated leg flexion and torsion	Trauma, (case 11), no EEG or seizures reported
	82	F	Bilateral spinal	Phantom feet and hands	Trauma C6, no EEG or seizures reported
Lorenz et al[109]	47	M	Right medulla	Painful "dysesthesias," allodynic pain	Infarct, no EEG or seizures reported
Van Bogaert[110]	60	M	Right thalamus	Hallucination of limb detachment and torsion	Infarct, no mention of EEG or seizures
Halligan et al[111]	65	M	Right thalamus, pulvinar	Somatoparaphrenia (phantom limb), left hemiplegia	Hematoma, no mention of EEG or seizures
Paciaroni and	69	M	Right thalamus	Paresthesia	Ischemia, no EEG or seizures reported
Bogousslavsky[112]	80	M	Right thalamus	Paresthesia	Ischemia, no EEG or seizures reported
	42	M	Left thalamus	Paresthesia, pain	Ischemia, no EEG or seizures reported
	69	M	Left thalamus	Paresthesia, pain	Ischemia, no EEG or seizures reported
	48	M	Right thalamus	Paresthesia, pain	Ischemia, no EEG or seizures reported
	47	M	Right thalamus	Paresthesia	Ischemia, no EEG or seizures reported
	63	M	Right thalamus	Paresthesia	Ischemia, no EEG or seizures reported
	84	F	Left thalamus	Paresthesia	Ischemia, no EEG or seizures reported
	65	M	Right thalamus	Paresthesia	Ischemia, no EEG or seizures reported
	45	M	Left thalamus	Paresthesia	Ischemia, no EEG or seizures reported
	46	F	Right thalamus	Paresthesia	Ischemia, no EEG or seizures reported
	39	F	Left thalamus	Paresthesia	Ischemia, no EEG or seizures reported
	51	F	Right thalamus	Paresthesia	Ischemia, no EEG or seizures reported
	61	M	Left thalamus	Paresthesia	Ischemia, no EEG or seizures reported
	57	M	Right thalamus	Paresthesia, pain	Ischemia, no EEG or seizures reported
	44	F	Left thalamus	Paresthesia	Ischemia, no EEG or seizures reported
	43	M	Left thalamus	Paresthesia	Ischemia, no EEG or seizures reported
Franzini et al[113]	64	M	Left thalamus	Paresthesia, pain	Ischemia, no mention of EEG or seizures
Tatu et al[114]	30	M	Right thalamus	"Dysesthesias," impression of complex movement of limb	Infarct, no paroxysms observed and EEG was normal

ing proposal concerning visual hallucinosis may have put things upside down: visual representation might not be generated during dreaming by a brainstem "dream centre," as Lhermitte speculated,[33] but by the visual pathway itself. In other words, we propose that in the visual pathway, neurons and neuronal assemblies specialized for waking vision and for voluntary visual imagination are responsible for the hallucinosis of people with narcolepsy and epilepsy, and of patients with migraine and patients with tumours, as well as the visual evocations of patients receiving exploratory electrical stimulation and, of course, hallucinosis of patients with Charles Bonnet syndrome, hallucination of patients with psychosis and of post-lesion hallucinosis, as well as visual aspects of normal dreams. After all, it is well established that cortical cell assemblies (occipital, inferotemporal and parietal) are repositories of fully formed dynamic images and are extraordinarily specialized for this function. There is also no evidence whatsoever to the effect that such fully formed conscious images could be generated in any subcortical tissue. Within this interpretation, all hallucinations need not derive from conscious memory: they may derive from unconscious memory (forgotten material), or they may even be constructions resulting from the architectonic structure of the neural assemblies stimulated and the specific local effect of any pathology of the neural assemblies. Each pathologic condition creates a particular context for activation of these assemblies, providing a specific coloration.

In their attempt to articulate a pathophysiologic model of hallucinosis, Manford and Andermann[49] proposed a neurotransmitter-based explanation. We do not dispute the plausibility of serotonin dysfunction as an important modulating factor of hallucination in narcolepsy, dementing processes, drug consumption and, importantly, a few cases of peduncular hallucinosis. However, the cases presented here are best explained by a neurotransmitter-independent, modality-specific neuronal loss, resulting in connection-based release of inhibition in the sensory cortex. The disconnection is often corticocortical (see Dierks et al[81] for fMRI evidence), but it can also be remote. Manford and Andermann[49] propose that in the latter case, a thalamocortical gate is usually unbridled. We find this quite plausible, because the thalamus is known to gauge sensory arousal and because thalamic lesions commonly suffice to produce hallucinosis. Not mentioned by Manford and Andermann is the strong piece of evidence to the effect that thalamic bursting is well known to occur during post-lesion pain.[122] We remain somewhat perplexed, however, by the paucity of published cases of auditory hallucinosis after a thalamic lesion. Noda et al[123] reported 2 patients with cases of thalamic infarction, 1 right and 1 left, who both had visual and auditory hallucinations consisting of clear recollections of events dating as far back as 50 years.

Finally, it has been proposed that lesions of the right hemisphere are more likely than lesions of the left to produce visual hallucinations.[50,52] In the current analysis, visual hallucinations resulted from right hemisphere lesions in 35 cases and from left hemisphere lesions in 20 ($\chi^2 = 4.1$, $p < 0.05$), suggesting some truth to that assertion. The other modalities yielded nonsignificant prevalence differences by hemisphere, but there were not enough cases to support meaningful inference tests. The laterality of the expected lesion in visual hallucinosis suggests (but does not prove) a corticocortical mediation related to hemispheric specialization, again leading us to argue that neither a neurotransmitter-based nor a thalamic-gating mechanism can suffice to explain entirely the neural basis of post-lesion hallucination.

Table 10: Types of contents of nonparoxysmal somatic hallucinations resulting from brain lesions (listed in Tables 8 and 9)

Content of hallucination	No. of reports
Dysesthesias, paresthesia	20
Supernumerary limb	7
One's limb is detached	6
Burning pain	6
One's limb appears to belong to someone else	5
Limb behind 1 side	2
Limb larger on 1 side,	1
Limb heavier on 1 side	1
One body side is evil	1
Limb is moving	1

What remains to be done with regard to the neural bases of hallucination?

The study of hallucinosis and hallucination could contribute to the advancement of psychophysics and sensory neuroscience, as well as to better health care. We have presented a modest test of a statement by Kölmel[35] claiming peripheral-to-macular directionality of hallucinated movement in the damaged field. We

have provided another modest test of one of Ffytche and collaborator's models of dendropsia.[46] The same authors propose a relation between "near projection" and "far projection" and micropsia and macropsia, respectively. This remains to be investigated systematically. There are many other questions about the relation between sensation, perception, memory and the brain that only studies of hallucination can answer.

It would be interesting to determine, in detail, whether the contents of paroxysmal hallucinations in cases without identifiable lesions (migraine, drug induced, epileptic) are similar to those of lesioned cases without paroxysms. Post-lesion hallucinations seem akin to hallucinations evoked in epileptic patients by the neurosurgeon's stimulating probe exploring the cortex for epileptic foci: they are often complex, nonmorbid, lucidly identified as unreal, and they are evoked by lesions or stimulation in the same general areas of the cortex (see Stephane et al[124] for a comprehensive review), though the effects of subcortical lesions are another matter.

It would be even more informative to compare, in detail, quantitatively, large corpuses of contents of hallucinations as a function of each cause of hallucination, including focal lesions, psychosis, narcolepsy, idiopathic Parkinson's disease, migraine coma, Charles Bonnet syndrome, drugs, and, of course, epilepsy.[49] Let us consider one of these comparisons more closely. Similar to the hallucinations of patients with psychosis, the post-lesion hallucination is often of a complex scene (moving humans or animals, or both, or music including symphonies or voices, or body image distortion, supernumerary body parts, and so on). However, in contrast to the hallucinations of psychotic patients, the content of the post-lesion hallucinations seems benign rather than morbid (i.e., life-threatening visual scenes, molestation, ominous compulsive dangerous voices, feelings of body decay, worm infestation and so on). The patient with brain lesions seems to be typically more lucid about the hallucinatory nature of the experience than the patient with psychosis. Post-lesion hallucination often involves important components derived from memory. The proportion has been found to be 33% in patients with Charles Bonnet syndrome.[125] The contents of psychotic hallucination may appear stranger to the patient. Another difference between hallucinations of psychotic patients and post-lesion cases is that the former are more frequently auditory and the latter more frequently visual. We need to find out why such differences exist.

Let us consider another contrast, between patients with Charles Bonnet syndrome and patients with lesions. Post-lesion hallucination, in the visual modality, is very much akin to that of Charles Bonnet syndrome, except that palinopsia and dendropsia seem to be less frequent in the former. Otherwise, content analysis of the case reports reviewed in the present investigation (Table 1, Table 2 and Table 3) yields a classification of the same types and in the same proportions as the content analysis of 39 patients with Charles Bonnet syndrome recently published by Santhouse et al.[75] There are several important implications to this:

- Charles Bonnet syndrome is probably none other than a post-lesion syndrome in *forme fruste* (ischemia; mild neuronal or glial degeneration, or both; metabolic insufficiency).
- Expression of Charles Bonnet syndrome is simply facilitated by emerging dementia, advanced age, visual pathology, sleep disorders and surrounding darkness (e.g., deafferentation, sensory deprivation).
- Damage to the peripheral components of the neurosensory system may actually *add* characteristics of hallucinosis in Charles Bonnet syndrome (e.g., dendropsia).
- The logic of Charles Bonnet syndrome applies just as readily to the other sensory-perceptual modalities, audition and somesthesia, as to vision, and more attention should be paid to these other modalities, and the 3 syndromic entities should be recognized and labelled coherently.

Auditory post-lesion hallucinations are also very similar to those described in direct electrical stimulation of the temporal cortex (see Table 4 and Penfield and Perot[31]). Much less is known about somatic hallucinations, especially the complex ones.[126] Parietal epileptic foci are rare in the somesthetic cortex, such that this cortex has not been probed much electrically, and post-lesion positive somesthetic syndromes are rare (Table 9). Post-lesion case reports will thus remain a precious commodity in this modality for a long time to come.

What can be done to help patients with post-lesion hallucinosis?

The first implication of the literature on post-lesion hallucination is that post-lesion hallucination is underreported, probably because patients do not wish to appear "crazy."[127] Thus, patients with documented or sus-

pected fresh lesions should be questioned about hallucination. A second important implication is that an isolated report of hallucination should be taken as a potential symptom of a brain lesion and should be diagnostically explored as such. Medical treatment of a brain lesion in addition to the more obvious provision of "psychologic" support could be life saving.

According to Pelaez,[128] pattern completion in a neural network model of the thalamus and a biologically plausible model of synaptic plasticity are key concepts for analyzing cognitive disorders that involve hallucinations of several kinds: visual hallucinations in Charles Bonnet syndrome and psychedelic drug consumption, somatic hallucination in phantom limbs, and cognitive hallucinations in schizophrenia and even in multiple personality disorders. It has been suggested that all these types of hallucination are the result of pattern completion dynamics in thalamic deafferented areas. Effective treatments of some of these disorders would thus, suggests Pelaez,[128] involve peripheral stimulation jointly with central inhibition so that the neural circuits that generate the disorders are depressed according to the proposed model of synaptic plasticity.

Certain antipsychotic drugs such as olanzapine or risperidone[129] are known to exert a specific anti-hallucinatory effect. Could such agents be recommended for isolated unimodal hallucinations resulting from a lesion? If one is to take Charles Bonnet syndrome as a benign example of post-lesion hallucinosis, we must concur with Batra et al[130] that few successful methods of treatment have been described. Therapies with classic neuroleptics, antidepressants or benzodiazepines have generally been found to be unpromising. Batra et al[129] treated the condition successfully in 3 patients using the atypical neuroleptic melperone. Improvement resulting from anticonvulsant treatment has been observed in a few cases,[76] of course primarily cases with epileptiform signs on EEG. Burke et al[131] reported the successful treatment of a case of probable Charles Bonnet syndrome with the cholinesterase inhibitor donepezil.

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