

# The perceptual consequences of visual loss: ‘positive’ pathologies of vision

D. H. ffytche and R. J. Howard

*Institute of Psychiatry, London, UK*

*Correspondence to: Dr D. H. ffytche, Institute of Psychiatry, De Crespigny Park, Denmark Hill, London SE5 8AF, UK  
E-mail: d.ffytche@iop.kcl.ac.uk*

## Summary

Fifty patients with visual hallucinations and illusions secondary to degenerative eye disease reported remarkably stereotyped experiences. Questionnaire responses revealed five previously recognized categories of pathological vision (perseveration, illusory visual spread, polyopia, prosopometamorphopsia and micro/macropsia) and three novel categories (tesselopsia, hyperchromatopsia and

dendropsia). Identical pathologies of vision occur in a range of clinical and experimental settings, suggesting that they reflect fundamental visual processes. The known neurophysiology of the visual cortex helps explain the phenomenology of the experiences and provides the basis for a neurobiologically based classification of positive and negative visual perceptual disorders.

**Keywords:** visual hallucinations; palinopsia; metamorphopsia; tessellopsia; hyperchromatopsia; dendropsia

**Abbreviation:** fMRI = functional magnetic resonance imaging

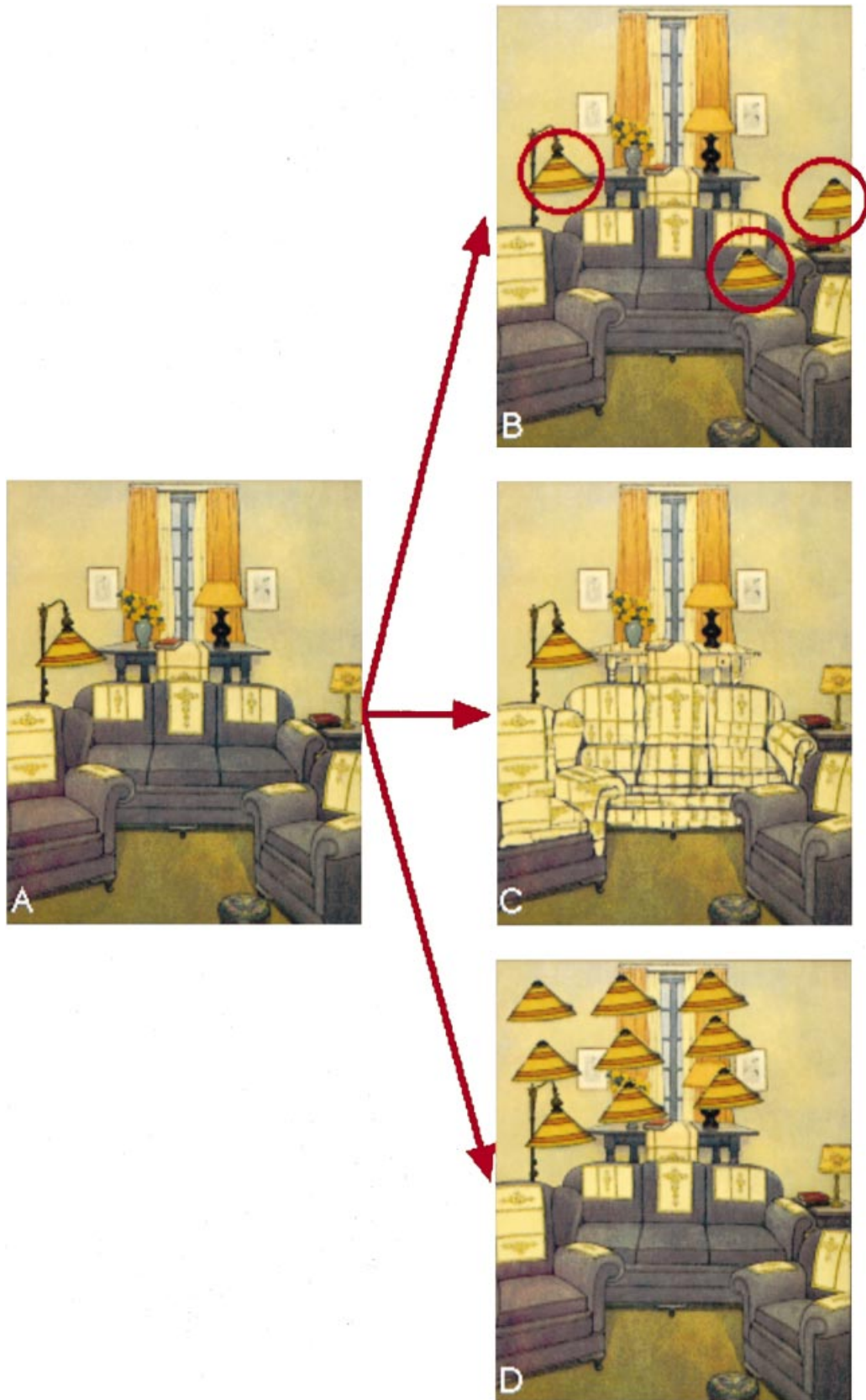
## Introduction

Neuroanatomical and neurophysiological studies of the macaque visual cortex anticipated that the human visual system would be found to consist of a series of maps, each specialized for a different visual attribute. For example, an area in the posterior fusiform gyrus is specialized for colour (human area V4: Zeki *et al.*, 1991; McKeefry and Zeki, 1997), a ventrolateral area is specialized for motion (human area V5: Watson *et al.*, 1993) and an area anterior to V4 is specialized for faces (Puce *et al.*, 1996). The specialized areas identified by PET and functional MRI (fMRI) studies and the location of cerebral lesions in patients with specific perceptual deficits are mutually consistent. Thus, unilateral lesions in the posterior fusiform gyrus (V4) lead to hemiachromatopsia (Kölmel, 1988), bilateral ventrolateral occipital lesions (V5) lead to akinetopsia (Zeki, 1991) and bilateral ventral occipitotemporal lesions lead to prosopagnosia (Meadows, 1974).

In 1951, Critchley attempted to classify a set of visual disorders he had observed in his patients and which he had noted in earlier clinical reports. He named the disorders palipsia (from Greek *palin*, again), but the term has changed to palinopsia in the intervening 50 years. Critchley divided palinopsias into spatial and temporal varieties and further subdivided spatial palinopsia into two subcategories: illusory visual spread and polyopia. To illustrate these experiences, panel A of Fig. 1 shows a room as correctly observed while

panels B–D show the same room from the perspective of the palinoptic patient. In Fig. 1B the patient fixes on the lampshade in the left-hand side of the room (shown as the red circle on the left) and a percept of the lampshade persists as he moves his gaze to successive fixation points (the circles on the right)—a temporal palinopsia. Critchley described cases in which the palinoptic percept remained present while the gaze moved (as in Fig. 1B) and cases in which the palinoptic percept reappeared after a few seconds or minutes. Kölmel (1982) further subdivided temporal palinopsias into immediate perseverations, short-latency palinopsias and long-latency palinopsias (in which the percept reoccurred months or even years later). Figure 1C illustrates Critchley’s first subcategory of spatial palinopsia: illusory visual spread. Here the patient perceives a pattern extending beyond its true boundaries to cover neighbouring objects. In the example shown, the pattern on the antimacassar has spread to fill the table and settees. In his second category of spatial palinopsias, polyopia, Critchley cited a group of patients described by Bender (1945). The typical polyopic image is shown in Fig. 1D. The lampshade on the left of the room becomes multiplied and the repeated copies form geometric rows or columns.

Critchley believed that palinopsia was only one component of a wider spectrum of disorders—the metamorphopsias (Critchley, 1953). These included a variety of perceptual



**Fig. 1** The palinopsias. (A) A room as correctly observed. (B) Perseveration: each red circle marks a successive fixation point. (C) Illusory visual spread: the pattern of the antimacassar spreads to other objects. (D) Polyopia: the lampshade is repeated in rows and columns.

distortions including those involving the size of objects (macropsia and micropsia), the fragmentation of lines, the waviness of contours, the apparent movement of stationary objects and a distortion specific to faces (prosopometamorphopsia). In a translation of Bodamer's original report of a patient with an occipital gunshot wound (Bodamer, 1948), Critchley described how 'All faces were strangely contorted and the features displaced; e.g. the ward sister's nose was deviated to the side by several degrees; one eyebrow was higher than the other; the mouth lay at a diagonal; the hair was dishevelled like a wig askew. Objects, places, colours contours, in fact anything other than a face, were seen correctly just as before his wound'.

How might these perceptual pathologies be understood within the current model of a functionally specialized and modular visual cortex? Each of the cases described by Critchley had a posterior cerebral lesion, but it would seem unlikely that the defects described were the perceptual consequences of damage to a particular specialized module, as found in akinetopsia, achromatopsia and prosopagnosia. What sort of module would be damaged in a patient with polyopia? It seems unlikely that part of the brain is specialized to edit out multiple copies of a percept so that, without the module, percepts are duplicated.

While patients with the same range of perceptual disorders continue to be described (for example, see Müller *et al.*, 1995; Vaphiades *et al.*, 1996), the sum of the palinopsia literature amounts to fewer than 50 case reports, while that of prosopometamorphopsia amounts to fewer than 10. Few clinicians have an opportunity to collect together more than a handful of such cases, and the fact that many of the disorders are transient means that systematic psychophysical studies are difficult to pursue. With such small numbers of cases it is tempting to ignore these phenomena.

The occurrence of visual hallucinations in association with visual impairment was first described by the Swiss philosopher Charles Bonnet, who reported his grandfather's visual experiences. Charles Bonnet later went on to develop the disorder himself, and in 1936 de Morsier named the syndrome after Bonnet (de Morsier, 1936, 1967). We have been investigating patients with the Charles Bonnet syndrome and have noted descriptions of perseveration, polyopia, illusory visual spread, prosopometamorphopsia and micropsia/macropsia that seemed identical to Critchley's case reports. We present below a qualitative picture of these phenomena together with descriptions of three new pathologies, and we attempt to relate them to the known neurobiology of the visual system.

## Methods

Patients assessed by the Kent and Buckinghamshire Associations for the Blind between March 1997 and March 1998 were asked about the occurrence of visual hallucinations and, if they had experienced them, to fill in a questionnaire with the aim of identifying a subset of patients for further

fMRI investigations. The questionnaire covered demographic and clinical details as well as the temporal characteristics of the hallucinations, phenomenology and factors influencing their onset and offset. Apart from specific questions as to the perceived size of the hallucinations, whether the hallucinations were in colour and whether the patients had ever hallucinated a face or a pattern, there were no questions related to any of the categories of perceptual deficit described below. Fifty-three per cent of the patients were further interviewed by telephone or at the Institute of Psychiatry. All patients gave informed consent and the study was approved by the Maudsley Hospital Ethical Committee.

## Exclusion criteria

Patients were considered to have visual hallucinations as a result of eye disease alone if, in addition to their blind-registration diagnosis, there was no history of cerebrovascular disease, migraine, Parkinson's disease, epilepsy or symptoms suggestive of complex partial seizures, or a fixed relationship between the hallucinations and sleep.

## Analysis

A preliminary analysis of the questionnaire responses identified three new classes of unprompted stereotyped descriptions (tesselopsia, hyperchromatopsia and dendropsia; see Results). Each questionnaire was subsequently scored for the presence of (i) visual perseveration, defined as a true (non-hallucinated) percept that persisted after the patient looked away, (ii) illusory visual spread, defined as the spread of a non-hallucinated pattern, (iii) polyopia, defined as multiple copies of a percept, (iv) micropsia/macropsia, defined as an abnormality in perceived size, (v) prosopometamorphopsia, defined as the distortion of a face, (vi) tessellopsia, (vii) hyperchromatopsia and (viii) dendropsia. We did not include categories of short- and long-latency palinopsias because of the difficulty in differentiating such phenomena from spontaneous hallucinations (see Discussion). Patients were classified into two groups: those with residual vision (able to read with visual aids) and those without (completely blind, perception of light or perception of vague outlines). As our definitions of perseverations and illusory visual spread required patients to be able to see, the percentages of these phenomena were based on the number of patients with residual visual abilities. The percentage frequency of micropsia/macropsia was based on the number of patients in whom the content of the hallucination allowed a size judgement to be made (e.g. hallucinations of a face or a figure but not an abstract pattern).

## Results

Of 116 patients who reported hallucinations, 49 (42%) had hallucinations secondary to eye disease. Fifty-eight per cent of these patients had senile macular degeneration as the

**Table 1** Phenomenology: eye disease

Condition	Frequency	Case	Description
Tesselopsia	37%	42	Wallpaper with lines; very fine golden wire netting
		57	Oval shape full of brickwork patterns; tiles; a fence made up of diamonds (squares on their sides)
		105	Nets in sharp geometric shapes
Hyperchromatopsia	16%	8	Shapes in vivid colours that wiggle
		113	Fireworks exploding in vivid colours
		105	Angular pattern in brilliant colour
Prosopometamorphopsia	16%	6	Missshapen and mutilated heads, two half heads are joined like Janus. Some faces have bulging eyes, some have blank eye-sockets; the expressions are all evil and malevolent
		50	Faces with distorted features like Spitting Image puppets
		62	Ugly faces with horrible teeth—like witches
Dendropsia	14%	11	Line of trees (conifers), or hedges
		67	Road map in black and white
		85	A bough full of green leaves
Perseveration	5%	41	I looked at a tree in the garden and, on looking away, the tree's leaves remained in the centre of my vision
Illusory visual spread	5%	105	My friend was working in front of a 10-foot privet hedge. I was suddenly aware he had disappeared. There was an orange peaked cap bobbing around in front of the hedge and floating in space by its own devices. The vanished figure had been replaced with a continuation of the hedge background complete with all the details of shadows and leaves, etc
Polyopia	6%	6	The screen became filled with numerous heads
		46	Rows of mugs fixed on a wall (three rows of four) for up to two minutes. Large mugs in the top row and cups at the bottom
		48	The lawn was completely covered by birds
Micro/macropsia	42%		Smaller 58%
			Larger 16%
			Variable 26%

primary blind-registration diagnosis, 18% had glaucoma and the remaining 24% had a variety of acquired ocular pathologies. Forty-six per cent of the patients had residual visual abilities as defined above.

Table 1 presents examples of patient descriptions, classified into different perceptual categories, together with their percentage frequency. Most reports are transcribed directly from the questionnaires. Thirty-seven per cent of the patients volunteered descriptions of regular, repeating patterns described as brickwork, lattices, netting, mosaics, chequerboards, wallpaper, grids, fences, roof-tiles, crazy paving and cobwebs, for which we propose the term *tesselopsia* to reflect the repeated geometry of the descriptions (from the Greek-derived Latin word *tessera*, a small tile used in mosaics). Sixteen per cent of the patients volunteered descriptions of hyperintense, vivid, brilliant colours, for which we propose the term *hyperchromatopsia*. Sixteen per cent of the patients volunteered descriptions of distortions in hallucinated faces. Fourteen per cent of the

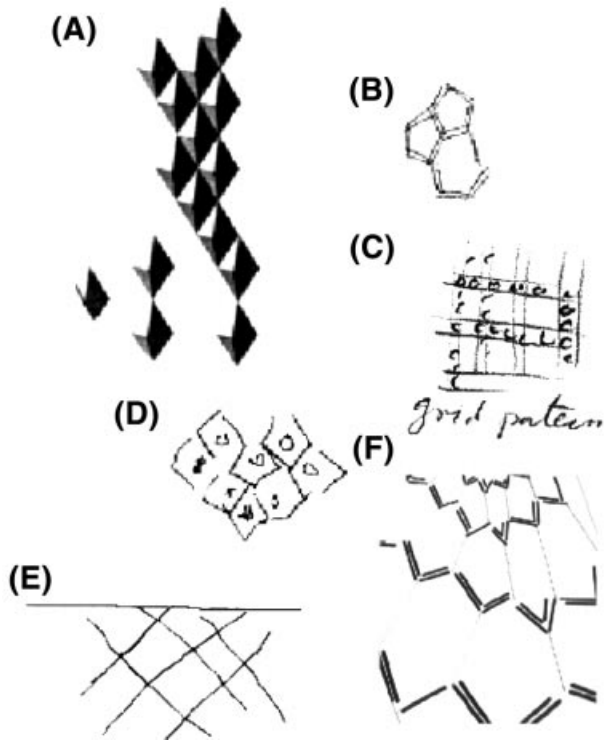
patients volunteered descriptions of irregular branching forms described as trees, branches or maps, for which we propose the term *dendropsia* (from the Greek *dendros*, tree). Three patients described a hallucinated polyopia while one patient described perseveration and one illusory visual spread. Abnormalities of size were reported by 42% of the patients, and of these 58% reported micropsia.

## Discussion

The unexpected consistency of the hallucinated percepts described by different patients and their similarity to Critchley's descriptions of patients with occipital pathology suggested to us that these experiences reflected fundamental visual processes. In what follows we show that the same experiences are reported in other clinical and experimental contexts, and we offer tentative neurobiological explanations for each perceptual experience.

**Table 2** *Phenomenology: clinical and experimental*

	Cerebral pathology	Sensory deprivation	LSD/mescaline	Migraine
Perseveration	One day I was watching my wife gardening through the window in the bright sunlight. Returning to my chair I saw her in the corner of the room, set in a window-frame. I looked away but she appeared wherever I looked (Kinsbourne and Warrington, 1963)	When objects were moved across the visual field, part of the moving figure appeared to trail behind the rest. Thus, when a thin black line was rotated slowly against a dimly illuminated milk glass screen in a darkened room, the line seemed S-shaped because the ends 'lagged' behind the centre part (Heron <i>et al.</i> , 1956)	As a result of persistent afterimages, he can see different stages of this movement simultaneously. The overall effect is similar to that of time-lapse or strobe light photography (Grof, 1976)	Objects on which she has fixed can 'follow' when she changes the direction of gaze (Klee and Willanger, 1966)
Illusory visual spread	If she looked at anyone wearing a striped or chequered garment, the pattern would seem to extend over the person's face. The pattern of cretonne curtains would often seem to extend along the adjacent wall. When she came to hospital by taxi, the iron railings enclosing the garden of Queen Square appeared to extend across the road (Critchley, 1951)	Not described	Not described	Talking with a friend, I glanced just to the right of his face, whereupon his head disappeared. His shoulders and necktie were still visible, but the vertical stripes in the wallpaper behind him seemed to extend right down to his necktie (Lashley, 1941)
Polyopia	Everything around him seemed to be quadruple, no matter in which direction he looked. The four images were arranged in two parallel pairs, one above the other (Bender, 1945)	The visual field was often filled with a large number of identical small patterns or objects (such as geometrical forms, plants or animals); these were usually arranged in symmetrical rows (Heron <i>et al.</i> , 1956)	A small wooden face appears; it has the form of a small apple; then there comes a small yellow face; suddenly there are three, four faces in one row; above it new rows appear (Klüver, 1966)	She can also experience one person as four people (Klee and Willanger, 1966)
Tesselopsia	The patient sometimes had the impression that an entire net of colours appeared (Kölmel, 1984)	This tended to be 'simple' in form (rows of dots, geometrical patterns, mosaics etc. (Heron <i>et al.</i> , 1956)	Lattices, large transparent blobs that floated indefinitely in one's visual field and complex geometrical patterns (Abraham, 1983)	Latticed, faceted and tessellated motifs predominate: images reminiscent of mosaics, honeycombs, Turkish carpets etc., or moiré patterns (Sachs, 1995)
Dendropsia	Sometimes when she was watching television, a piece of timber or the branch of a tree emerged from the set and moved towards her, always stopping three feet away from her (Lance, 1976)	Not described	See Fig. 3A and B	Maps (Sachs, 1995)
Metamorphopsia (Prosopo-)	Faces may look torn, warped distorted or contorted. One of our patients said to her physician: you have stretched lips, a thick nose, and you are grinning. You don't look nice at all. Your eyes are stretched and have big circles under them (Hecaen and Angelergues, 1962)	Faces might expand and contract, bulge or writhe, if looked at for any length of time (Heron <i>et al.</i> , 1956)	Then a lady-doctor came to sit at our table. At first I thought she was very nice. Soon, however, her teeth, which protruded slightly, began to grow into long fangs; her glasses became perched precariously on the end of a crooked nose; her eyes became very protruding (Guttman and Maclay, 1936c)	During these episodes he has observed that portions of the visual image, in particular faces, may appear 'cut up', distorted and disjointed, being composed of sharp edge fragments. He compares this appearance to that of an early Picasso (Sachs, 1995)
Hyperchromatopsia	The colours were so intense that the patients initially closed their eyes in an attempt to shut out the light. One patient described his phosphenes as though a light as powerful as the sun had projected the colour, while another patient spoke of shocking colours and a third likened the visual effect to fire (Kölmel, 1984)	Colours appeared bright, highly saturated or luminescent. Colour and brightness contrast was exaggerated, giving objects a glittering appearance (Heron <i>et al.</i> , 1956)	The prevailing tint is blue, but the multitude of shades, each of such wonderful individuality, make me feel that hitherto I have been totally ignorant of what the word colour really means. The colour is intensely beautiful, rich, deep, deep, deep (sic), wonderfully deep blue (Knauer and Maloney, 1913)	These figments and elementary images tend to be brilliantly luminous, coloured, highly unstable, and prone to sudden kaleidoscopic transformation (Sachs, 1995)



**Fig. 2** Patients' drawings of tesselloptic hallucinations. (A) Cerebral lesion (Kölmel, 1984). (B) LSD (Stoll, 1947). (C) Mescaline (Guttman and Maclay, 1936a). (D and E) Patients with eye disease. (F) Teichoptic patterns integrated over time (Richards, 1971).

**Commonalities of visual experience**

Table 2 shows that identical phenomenological descriptions have been reported in a variety of clinical and experimental settings, including cerebral lesions, sensory deprivation, the administration of psychedelics (e.g. LSD and mescaline) and migraine. To illustrate this, Fig. 2 shows the consistency of tesselloptic patterns in a patient with a right-sided occipital infarct (Fig. 2A) (Kölmel, 1984), following the ingestion of LSD (Fig. 2B) (Stoll, 1947), following ingestion of mescaline (Fig. 2C) (Guttman and Maclay, 1936a), two of our patients with eye disease (Fig. 2D and E) and a composite diagram of migraine fortification spectra (Fig. 2F) [teichopsia (Plant, 1986)], which shows how teichoptic patterns, integrated over time, produce a tessellated appearance (Richards, 1971). Figure 3A is an artist's impression of his own mescaline hallucinations demonstrating tessellopsia (the brick wall), dendropsia (the stems and roots of the branching flowers growing from the wall) and polyopia [the three aspidistras in a column (Guttman and Maclay, 1936b)]. Figure 3B is another example of dendropsia from the same study (Maclay and Guttman, 1941).

The list of conditions in Table 2 is not complete. Visual hallucinations are found in Parkinson's disease, epilepsy, peduncular lesions, Alzheimer's disease, dementia with Lewy bodies, schizophrenia, late paraphrenia (Howard and Levy, 1994) and acute brain syndromes (for reviews, see Barodawala and Mulley, 1997; Manford and Andermann,



**Fig. 3** Artist's drawings of mescaline hallucinations. (A) Tessellopsia, dendropsia and polyopia (Guttman and Maclay, 1936b). (B) Dendropsia (Maclay and Guttman, 1941).

1998). Where phenomenological descriptions of the hallucinations are given, examples of the same perceptual pathologies as those described above are noted. For example, Platz and colleagues (Platz *et al.*, 1995) reported fences, rods, house walls and scaffolding (tesselopsia) and ‘10 bottles of vodka’ (polyopia) in the visual hallucinations of patients with delirium tremens; Persaud and Cutting (Persaud and Cutting, 1991) reported visual distortions in schizophrenia (specific to faces in one subject but more generalized in three others), and Howard and Levy (Howard and Levy, 1994) reported polyopia and prosopometamorphopsia in late paraphrenia (six life-sized people dressed as gypsies; faces of persecutors at window with frightening expressions). Figure 4 is an example of prosopometamorphopsia in a schizophrenic patient’s drawing of her visual hallucinations with the characteristic facial distortions reported in our patients (Guttman and Maclay, 1937). The classical descriptions of hypnagogic hallucinations also contain elements of the same perceptual pathologies. For example, McKellar’s subjects (McKellar, 1957) reported ‘a large bloated yellow head, pouting red lips, wild blue eyes rolling, hair dishevelled’; ‘witches black and brown with hooked noses and bulging eyes’, and, with regard to colour, that objects are ‘frequently reported as coloured in unnaturally vivid hues. Some of our subjects likened these hues to those of Technicolor rather than of nature’.

### The Charles Bonnet syndrome

De Morsier, in his original paper (de Morsier, 1936), defined the Charles Bonnet syndrome as follows: ‘*Dans les syndromes séniles avec lésions oculaires—le syndrome de Charles Bonnet—(les hallucinations visuelles) peuvent être isolées avec intégrité complète des autres fonctions cérébrales*’. He recognized that visual hallucinations occurred in a range of clinical conditions and his intention was to differentiate the Charles Bonnet cases from visual hallucinations associated with parietal lesions, peduncular lesions and chronic hallucinatory psychoses. However, in the same paper he

noted that eye disease was not the cause of the hallucinations: ‘*Contrairement à la théorie soutenue par les oculistes les lésions oculaires qu’on trouve le plus souvent chez ces vieillards hallucinés ne sont pas la cause de ces hallucinations*’. In his 1967 review (de Morsier, 1967) he was adamant that eye disease was not the most important aetiological factor and removed it from the definition, choosing to emphasize old age and the absence of a neuropsychiatric disorder: ‘*En 1938, j’ai proposé de désigner sous le nom de <syndrome de Charles Bonnet> les hallucinations visuelles apparaissant chez les vieillards sans déficience mentale. Pour éviter toute confusion, il convient de conserver cette définition. Cette par erreur que quelques auteurs ont donné récemment <syndrome Charles Bonnet> comme synonyme d’<hallucinations chez des ophtalmopathes>. Il n’existe pas de corrélation entre les hallucinations visuelles et les lésions des globes oculaires. Les hallucinations visuelles ne peuvent pas être expliquées par une <privation> d’afférences visuelles. Elles sont toujours causées par une altération du cerveau*’. By de-emphasizing eye disease, de Morsier introduced an ambiguity that continues to confuse the literature: the index cases—Charles Lullin and Bonnet himself—both had eye disease, yet the Charles Bonnet syndrome was not intended to describe this association. As a result, some authors follow de Morsier and reserve the Charles Bonnet eponym as a purely phenomenological description (complex hallucinations in the psychologically normal) without specifying the aetiology (Damas-Mora *et al.*, 1982; Gold and Rabins, 1989; Teunisse *et al.*, 1996). These authors describe eye disease and old age as common clinical associations rather than diagnostic prerequisites. Other authors use the eponym to refer to those patients with complex visual hallucinations associated with eye disease (Burgermeister *et al.*, 1965; Kölmel, 1993; Manford and Andermann, 1998). Thus at one extreme the term is used to describe all patients with complex visual hallucinations with preserved insight regardless of whether the experiences are the result of cerebral lesions, metabolic disturbance or eye disease, while at the other extreme

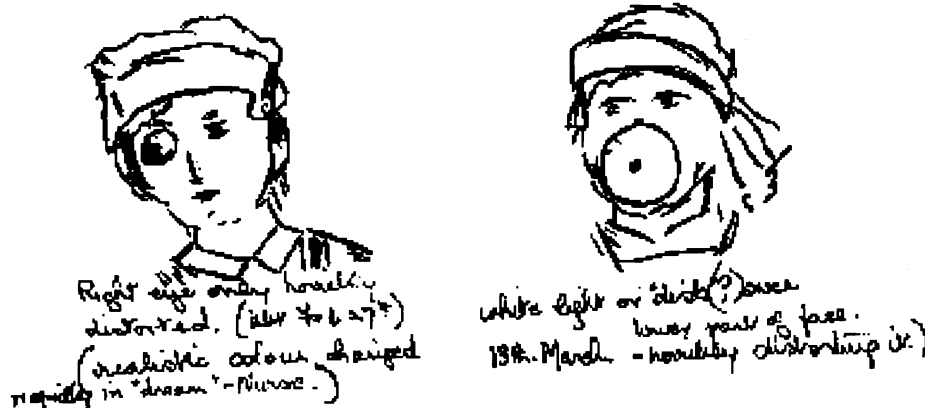


Fig. 4 Prosopometamorphopsia in a schizophrenic patient’s drawing of her visual hallucinations (Guttman and Maclay, 1937).

the term is used to describe patients with complex visual hallucinations and eye disease. While both uses of the term have their respective advantages and disadvantages, we favour the latter, which reminds us of Bonnet's and de Morsier's original observations. Like de Morsier, we recognize that not all patients with eye disease have visual hallucinations, in the same way that not all patients with posterior cerebral artery infarcts have such experiences; however, unlike de Morsier, we do not feel that this is evidence that eye disease is coincidental—it suggests that another factor plays a part (see below).

## **Methodological issues**

### **Classification**

The eight categories of pathological vision are not intended to be a complete classification of perceptual dysfunction. We assume that there are other experiences (such as the waviness of contours) that our patients have chosen not to volunteer either because they are subtle and not easy to describe or because they are regarded as commonplace. We adopted Critchley's original description when defining illusory visual spread, requiring the presence of a true (non-hallucinated) percept of a pattern. Had we relaxed the definition to include hallucinated patterns, then many of our patients would have described this category. In contrast, our definitions of prosopometamorphopsia, polyopia and micro/macropsia allowed for distortions of hallucinated as well as true percepts. Had we insisted only on the latter then none of our patients would have reported these phenomena (we expand on this point below). We have not included separate categories of short- and long-latency temporal palinopsias (Kölmel, 1982) because of the difficulty in differentiating these experiences from *de novo* visual hallucinations. Classification of a particular percept as perseveration or short- or long-latency palinopsia rests entirely on whether the patient remembers having seen the image before. For example, Kölmel classifies the following description as an example of long-latency palinopsia (1982: Case 4, left occipital infarct): 'I took a bus to see my ophthalmologist. I looked out the window and couldn't believe my eyes. A huge wall made of blue tiles towered in front of me from the ground to the sky. I remembered that these were the same tiles that, with the sweat of my brow, I had tiled the bathroom walls some four months before. When I rubbed my eyes in order to look at the tiled wall in greater detail it disappeared'. All of our patients have seen a wall, tiles, fences, unfamiliar faces and animals at some time in the past and, by Kölmel's criteria, all our patients would be classified as examples of palinopsia. We would argue that many of the patients with short- or long-latency palinopsia have associated visual hallucinations so that the distinction between the experiences becomes less apparent [see for example Critchley, 1951 (Cases 1 and 3); Bender *et al.*, 1968 (Cases 1, 2 and 3); Lance, 1976 (Cases 7 and 9); Müller *et al.*, 1995 (Case 2); Vaphiades *et al.*, 1996 (Case 5)].

### **Comparison with previous phenomenological surveys**

Several studies have surveyed the phenomenology of visual hallucinations in patients with eye disease using questionnaire, semi-structured interview or literature review methods (Lepore, 1990; Schultz and Melzack, 1991; Holroyd *et al.*, 1992; Teunisse *et al.*, 1995, 1996; Schultz *et al.*, 1996). In general, visual hallucinations have been shown to be associated with increasing age, increasing severity of visual impairment and low arousal. Those studies which report the content of the hallucinations are in broad agreement with the results reported here, with geometric forms, repetitive patterns, faces, trees and size distortions all represented in the lists of experiences described. None of the studies has reported specific phenomenological details such as the distortions of faces, the vividness of colours or the nature of the repetitive geometry. We attribute this difference between our study and previous surveys not to a difference in the patient sample but to the fact that we have analysed our data with *post hoc* perceptual categories constructed from recurrent descriptive themes arising within our patient group and across a range of clinical conditions.

### **Prevalence confounds**

Our analysis is based on unprompted reports and thus has little epidemiological validity. In essence we are reporting answers given to questions that we have not asked. The frequency of each perceptual category is therefore related more to whether it was considered sufficiently interesting or unusual by the patients to mention, rather than to its true prevalence in the cohort. A disadvantage of our approach is that we cannot provide meaningful statistical associations between the *post hoc* perceptual categories and a range of factors we have asked about (e.g. visual acuity, duration of hallucinations, diagnosis), since the absence of a volunteered response need not imply that a given class of pathology has not been experienced. It is unlikely that the patient's reports were biased, as (i) with the exception of micropsia/macropsia, none of the perceptual categories was mentioned in the questionnaire, (ii) none of the disorders is traditionally associated with eye disease, and (iii) three of the categories were formulated *post hoc*.

### **Non-ophthalmological aetiologies**

We were reliant on questionnaire-based exclusion criteria, and some of our patients may have had abnormal visual experiences secondary to other causes. However, since our aim is to point out the similarities of experience with different aetiological factors, any inhomogeneity in the cohort strengthens rather than weakens our argument. We are currently undertaking a systematic survey of abnormal visual perception in ophthalmic, psychiatric and neurological populations.

### ***Visual neuroscience and the pathologies of visual perception***

Are the similarities between the visual experiences of unrelated clinical and experimental contexts meaningful in neurobiological terms? Traditional classification schemes would place much emphasis on a number of factors that we have chosen to ignore. For example, we have not differentiated between hallucinated (without afferent sensory signals) and illusory percepts (false percepts with afferent sensory signals) nor have we differentiated between the perceptual experiences that are recognized as real by the patient and those that are not. We would argue that by separating the phenomenology into different descriptive categories (hallucinations with insight, illusions without insight etc.) the underlying neurobiological message may be lost. For example, the perception of a distorted face might be a hallucination recognized as unreal in a patient with eye disease; a hallucination believed to be real in a patient with schizophrenia; or an illusion (a real face appears distorted) in a patient with an occipital lobe lesion. From the clinical point of view all three conditions are entirely different; however, it seems unlikely that the neural substrate of the distorted face percept differs between the three examples given. We believe that the presence or absence of insight or the fact that the face is an illusion or a hallucination reflects the neurobiological context in which the visual experience arises. The visual percept itself—the final common pathway—tells us something of the neurobiology of vision.

We are not the first to point out commonalities in abnormal visual experience or to realize their significance. Klüver (1966), based on a consideration of the mescaline literature, recognized three classes of perceptual pathology common to a range of clinical conditions: (i) the appearance of ‘form’ constants—grating, lattice, fretwork, filigree, honeycomb or chessboard patterns; (ii) alterations in the number, size and shape of objects; and (iii) changes in spatiotemporal relations (e.g. parts of objects are transferred to other objects or objects that have appeared reappear after relatively long periods of time). Klüver believed that such ‘constants’ represented fundamental mechanisms at work in the cortex: ‘... the occurrence of these symptoms in aetiologically different conditions suggest that we are dealing with some fundamental mechanisms involving various levels of the nervous system. To elucidate these mechanisms, we must rely on future research to provide the necessary anatomical, pathological, biochemical and clinical data’. The ideas we present below are an attempt to reformulate Klüver’s intuition in the light of what we now know of the neurophysiology of vision.

### ***Perseveration***

Brindley and Lewin (1968) found that suprathreshold stimulation of the medial occipital lobe resulted in a phosphene that could persist for up to 2 min after the cessation of stimulation and that the position of the phosphene

followed eye movements. There are some similarities between the perceptual experiences provoked by stimulation and the visual perseveration described by our patients and in the literature, suggesting that the two are in some way related. In a typical palinoptic episode, objects or features are described as following the patient’s gaze for a period ranging from 30 s to a few minutes. Alternatively, the gaze may be held constant while viewing a moving object, resulting in a trail of images. It is easy to understand how, by looking at each person in a room, for example, a patient with perseveration will report: ‘After watching a character on the television set, faces were transposed to others in the room’ [Michel and Troost, 1980 (Case 2: right occipital lobe infarction)] or ‘She noticed that a replica of the white beard of the attendant Santa Claus was superimposed on the face of everyone she spoke to’ [Meadows and Munro, 1977 (Case 1: right lingual/fusiform infarct)].

### ***Polyopia***

Brindley and Lewin (1968) noted that the stimulation of a single point on the medial occipital cortex resulted in rows or irregular clusters of phosphenes. This unexpected finding is analogous to descriptions of polyopia which often note the arrangement of multiple copies of an object in rows or columns [Bender, 1945 (Cases 1 and 2); Kinsbourne and Warrington, 1963 (Case 1); Fisher, 1991 (Case 1); Kölmel, 1993]. While polyopia is traditionally explained as the consequence of defective eye movements and a failure of visual extinction (Bender, 1945; Kölmel, 1993), the eye-movement hypothesis does not explain the characteristic appearance of rows and columns, nor can it be the cause of the hallucinated polyopia found in our patient group. Why single points on the cortex should produce multiple arrays is not known.

### ***Tesselopsia***

Studies of migraine ‘fortifications’ often relate the geometry of the percept to cortical anatomy. Lashley (1941) wrote that ‘such repetitive patterns should be predicted from the free spread of excitation through a neural field having the structural arrangement of reverberatory circuits described by Lorento de Nó’. Some years later, Richards (Richards, 1971) interpreted the phenomena in terms of Hubel and Wiesel’s model of V1 (Hubel and Wiesel, 1977). However, Richards calculated that the cortical distance for each teichoptic line was 1.2 mm, five times larger than the known diameter of individual orientation columns in the monkey. Richards argued that the scale discrepancy suggested that orientation columns were not responsible for the phenomena, proposing hypothetical larger-scale units of organization. Kölmel (1984) also doubted the validity of a simple receptive field-based explanation as the tetrahedral geometries in his group of patients with cerebral lesions did not increase in size from the central to the peripheral visual field as would be predicted. In fact, a

matrix of periodic, lattice-like, long-range excitatory connections in different layers of V1, V2 and V4 (Rockland and Lund, 1983) are better anatomical candidates for the phenomena. These lattices connect neurons with similar orientation preference, although a third of the connections are targeted at neurons angled at  $45^\circ$  (Malach *et al.*, 1993). This angle bears a striking resemblance to the mean angle of the zigzag in migraine fortifications [ $\sim 45^\circ$  in the central  $30^\circ$  of visual field (Richards, 1971)] and the apices of the tesselloptic rhomboids (Fig. 2A). We would argue that increased activity within these lattices might be responsible for both teichoptic zigzags and tesselloptic patterns depending on the spatial configuration of the increase. If the activity formed an approximate straight line parallel to the surface of the cortex but only extended in one dimension (such as might be found at the edge of an ischaemic region or the edge of a wave of spreading depression) the former percept would be predicted. If, instead of being restricted to the edge, the activity were extended in two dimensions, a tesselloptic pattern would emerge. Activity in different extrastriate regions would be expected to produce lattices at different spatial scales and with different colour attributes. The fact that activity in some cortical neuronal populations correlates with consciousness while other cortical activities do not is well established (for review, see ffytche, 1999). We have no explanation as to why the matrix of long-range connections might have privileged access to consciousness.

### *Dendropsia*

We have, somewhat arbitrarily, chosen to differentiate geometrical tesselloptic patterns from irregular dendroptic patterns. Hallucinations of trees, maps and branches have traditionally been explained as silhouettes of retinal vasculature—one's own retinal vessels may be seen by shining a bright light on the sclera, which casts a shadow on parts of the retina that are not normally covered by vessels. We would argue that this explanation is unlikely to account for the appearance of rows of trees or the presence of additional features such as colours and leaves. It may be that dendroptic hallucinations are the result of activity in the same lattices of long-range connections as those described above, and we note that the angle of the branched intersections in Fig. 3B is  $\sim 45^\circ$ .

### *Prosopometamorphopsia*

Single-cell studies of face processing in the macaque monkey have identified neurons that respond to specific facial features or combinations of features, e.g. eyes, mouth and hair (Perrett *et al.*, 1982), while human studies have identified a negative visual evoked potential component at 170 ms specialized for eyes and insensitive to the spatial relations of the individual features (Bentin *et al.*, 1996). We would argue that increased activity within these neural populations would lead to an over-representation of the eyes, an indifference to spatial

relations between facial features, and hence the perception of distorted faces with characteristically prominent eyes.

### *Hyperchromatopsia*

A ventral extrastriate region in the human brain is specialized for colour (area V4) (Zeki *et al.*, 1991; McKeefry and Zeki, 1997). We hypothesize that hyperchromatopsia is the result of pathological increases of activity within this region. Our fMRI study of patients with the Charles Bonnet syndrome (ffytche *et al.*, 1998) lends partial support to this view in that phasic increases in V4 activity were associated with hallucinations of colour, often reported as vivid (see also Ramachandran and Blakeslee, 1998); however, the relationship between increased activity in V4, hyperchromatopsia and colour hallucinations is unclear.

### *Micropsia and macropsia*

The Gestalt psychologists demonstrated that retinal afterimages change their size depending on where the image is projected (Emmert's law; Emmert, 1881). The illusion is the result of size constancy, the process by which the perceived size of an object is made independent of the extent of its retinal projection; cerebral mechanisms take into account the distance of the object from the observer to make a judgement of size. These experiments are simple to replicate by looking at a bright light to produce an after-image and then looking successively at a near and a distant blank wall. The image appears larger when projected onto the distant surface and smaller when projected onto the near surface. Micropsia/macropsia in hallucinated percepts is likely to be the consequence of the same effect. A quarter of our patients noted that the apparent size of the hallucinations was variable, and some patients observed that the hallucination 'appeared as a projection, the nearer to the 'screen' the smaller was the size of the picture'. Le Beau and Wolinetz [Le Beau and Wolinetz, 1958 (Case 6)] and Kölmel [Kölmel, 1984 (Case 4)] noted the same phenomenon in patients with cerebral lesions. We argue that the perceived size of a hallucination depends on where it is projected, near projections resulting in micropsia and far projections resulting in macropsia. However, this projection-based explanation does not account for the micropsia/macropsia of true (non-hallucinated) percepts found in complex partial seizures and migraine, for example. It is likely that, in these examples, it is an abnormality of the constancy mechanism itself that is responsible.

### *Illusory visual spread*

In the examples reported by Critchley (Critchley, 1951), Lashley (Lashley, 1941) and in our patient group, illusory visual spread is invariably associated with a visual field defect, described as becoming 'filled in' with a surrounding pattern or texture. The same phenomenon occurs in normal

subjects across the blind spot, where lines and contours may be seen as continuous even when no continuity is present (e.g. Sergent, 1988; Ramachandran and Blakeslee, 1998). A delayed rather than instant filling in occurs across artificial scotomas formed by stabilized retinal images (Gerrits *et al.*, 1966) and across circumscribed homogeneous areas within dynamic visual noise or within static visual textures (Ramachandran and Gregory, 1991). Different visual modalities such as colour, motion and texture are filled in by independent processes with different time courses, suggesting that several extrastriate areas contain their own fill-in mechanisms (Ramachandran and Gregory, 1991; Ramachandran and Blakeslee, 1998). It seems likely that filling in and illusory visual spread describe the same phenomenon. Recent neurophysiological studies have provided a mechanism by which the effect may be mediated. Compromising a small area of the retina, either by a lesion or an artificial scotoma, silences those cortical neurons that responded to visual stimuli prior to the lesion. Instead of remaining silent, these same neurons develop visual responses within a period of seconds to minutes (Gilbert and Wiesel, 1992; Pettet and Gilbert, 1992; De Weerd *et al.*, 1995). It has been argued that this change in responsiveness is responsible for perceptual filling in (Gilbert and Wiesel, 1992; De Weerd *et al.*, 1995), although the mechanism by which the responses are modulated is disputed. While scotoma-induced modulations have been found in areas V1, V2 and V3, De Weerd and colleagues (De Weerd *et al.*, 1995) have pointed out that the time scale of the V1 modulation is too long—minutes rather than seconds. They argue that it is ‘climbing activity’ in extrastriate neurons that is responsible.

### ***A classification of pathological visual perception***

We recognize that the hypotheses presented above are tentative and simplistic and do not reveal the neurobiological message of each perceptual symptom. Some symptoms are likely to be normal brain responses, e.g. illusory visual spread and macropsia/micropsia. Others go beyond normal visual experience and we must assume that they result from abnormal activity within as yet undescribed perceptual modules or mechanisms. Whether each symptom is linked to a specific functionally specialized area or is related to a process common to many areas is a question that will require further investigation.

One feature in common to each neurobiological explanation is the presence of increased neurophysiological activity. For tessellopsia and dendropsia the hypothesis is merely speculative; however, we know that direct stimulation of the visual cortex produces experiences which, although simpler, have features of the perseveration and polyopia described clinically. The evidence of our previous fMRI study of Charles Bonnet hallucinators is

that phasic increases in activity within specialized visual cortex underlies hallucinations of vivid colours and distorted faces (ffytche *et al.*, 1998). A unified account of the different visual experiences based on increases in visual cortical activity is attractive as it explains why the phenomena occur together and why they are associated with a variety of different causes (release due to loss of inhibitory inputs, epilepsy, drug effects, etc.). We do not know why eye disease leads to an increase in visual cortical activity, although the evidence of our fMRI experiments is that it does (ffytche *et al.*, 1998). A loss of excitatory visual inputs to the lateral geniculate nucleus/pulvinar has been shown to result in low-threshold calcium spike bursts (Llinás and Jahnsen, 1982) which, when propagated to the cortex, might lead to ‘positive’ perceptual phenomena (Jeanmonod *et al.*, 1996; Manfred and Andermann, 1998). However, the thalamic theory does not explain the penchant for ventral occipital dysfunction found in our fMRI study (ffytche *et al.*, 1998) or why some patients but not others experience the phenomena. Clarke (Clarke, 1994) has shown how, at post-mortem, a patient with senile macular degeneration had selective deficits of cytochrome oxidase staining in the parvocellular compartments of areas V1, V2 and V4. The parvocellular system tends to project ventrally into the occipitotemporal cortex rather than dorsally into the occipitoparietal cortex (Livingstone and Hubel, 1988), providing a possible explanation for the ventral bias. Recent molecular biological studies of 5-HT<sub>2A</sub> and 5-HT<sub>2C</sub> receptor polymorphisms have shown an association between specific receptor alleles and the presence of visual hallucinations in patients with Alzheimer’s disease (Holmes *et al.*, 1998). Such receptor studies may provide an answer as to why the same disease leads to pathological percepts in some patients but not in others.

A unifying aetiology of increased activity suggests a neurobiological classification of pathological visual percepts based on two broad classes—the ‘positive’ disorders of increased function and the ‘negative’ disorders of decreased or lost function. The former class includes the categories described above: visual hallucinations, the palinopsias, the metamorphopsias, hyperchromatopsia and tessellopsia. The latter group includes the agnosias, achromatopsia, akinetopsia—the deficits of destructive lesions. We do not yet know whether for each functionally specialized area there is a pair of such disorders, as might be the case for V4 (hyperchromatopsia versus achromatopsia).

### ***Conclusion***

We have shown that patients with eye disease experience the same pathologies of visual perception as patients with cerebral lesions and, under certain circumstances, normal subjects. Based on these constants of visual experience we have derived a neurobiologically based classification of

positive and negative pathological visual percepts. The classification is not intended to be complete and is descriptive rather than explanatory. We hope it provides a theoretical framework of use to both clinicians and neuroscientists and that the spectrum of disorders we have described will begin to be recognized as common clinical findings rather than rare neurological and neuropsychiatric curiosities.

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