

Visual command hallucinations in a patient with pure alexia

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Around 25% of patients with visual hallucinations secondary to eye disease report hallucinations of text. The hallucinated text conveys little if any meaning, typically consisting of individual letters, words, or nonsense letter strings (orthographic hallucinations). A patient is described with textual visual hallucinations of a very different linguistic content following bilateral occipito-temporal infarcts. The hallucinations consisted of grammatically correct, meaningful written sentences or phrases, often in the second person and with a threatening and command-like nature (syntacto-semantic visual hallucinations). A detailed phenomenological interview and visual psychophysical testing were undertaken. The patient showed a classical ventral occipito-temporal syndrome with achromatopsia, prosopagnosia, and associative visual agnosia. Of particular significance was the presence of pure alexia. Illusions of colour induced by monochromatic gratings and a novel motion-direction illusion were also observed, both consistent with the residual capacities of the patient's spared visual cortex. The content of orthographic visual hallucinations matches the known specialisations of an area in the left posterior fusiform gyrus—the visual word form area (VWFA)—suggesting the two are related. The VWFA is unlikely to be responsible for the syntacto-semantic hallucinations described here as the patient had a pure alexic syndrome, a known consequence of VWFA lesions. Syntacto-semantic visual hallucinations may represent a separate category of textual hallucinations related to the cortical network implicated in the auditory hallucinations of schizophrenia.

The acquisition of visual language—the visual representation of words and the ability to read them—is likely to be of recent evolutionary origin. Yet, surprisingly, several lines of evidence indicate that the human brain has developed distinct cortical modules devoted to visual linguistic representations. Nobre *et al* recorded directly from the inferior surface of the temporal lobe while presenting text stimuli (both words and word-like letter strings) and identified two regions of the fusiform gyrus with a relative specialisation for text.¹ The first, a posterior region, responded both to real words and non-word letter strings, while the second, an anterior region, only responded to real words and was sensitive to the linguistic context in which they were presented. The authors concluded that the posterior region represented the prelexical formation of a word percept, while the anterior region represented high level semantic attributes. Functional imaging studies have also identified a region in the left posterior fusiform gyrus which responds selectively to text (both words and non-word letter strings) compared to non-textual control stimuli.^{2–4} The imaging and neurophysiological findings help explain a classical neuropsychological syndrome—pure alexia (alexia without agraphia)—in which patients are unable to read complete words or even individual letters but have preserved spoken, heard, and written language functions.

The specialisation of the cortex for visual language also sheds light on a curious perceptual phenomenon encountered in a range of ophthalmological and neurological disorders—visual hallucinations of text. Visual hallucinations are common perceptual pathologies with estimates of between 11% and 57% of patients with visual pathway lesions, anywhere from the retina to the primary visual cortex being affected.^{5–7} The hallucinations fall into a restricted range of categories with about a quarter of patients with hallucinations related to eye disease experiencing text, isolated words, individual letters, numbers, or musical note hallucinations.⁸ The nature of such hallucinations is best conveyed by the reports of patients themselves. Typical descriptions include:

“...these hallucinations manifested themselves as very distinct lines of typed letters, in a random sequence, in my central vision...The phenomenon persisted for quite a period, during which time none of the letters ever made sense...I still get them from time to time but they appear extremely jumbled and at all angles...” (67 year old man, wet type macular degeneration).

“...thin black letters in neat rows, sensible words often surrounded by masses of nonsense words....” (85 year old man, dry type macular degeneration).

“...writing on the ceiling which changes every time I look at it. Lists of words that do not make sense, an oval with inverted commas around it and inside the oval the letters AT. Labels with prices on them, eg ‘Super Something’ with one shilling or a pound sign and the size below....” (76 year old man: glaucoma, bilateral retinal haemorrhages).

Functional magnetic resonance imaging (fMRI) studies of hallucinating patients have shown a direct relation between the location of transient pathological increases in brain activity and the visual attribute hallucinated.⁹ Thus increased activity within cortex specialised for colour is associated with hallucinations of colour, increased activity within cortex specialised for objects, with hallucinations of objects, and so forth. The presence of a cortical region which responds more to text (either complete words or nonsense letter strings) than non-text stimuli provides a plausible explanation for the text hallucinations found clinically. In particular, it accounts for an almost invariable characteristic of the hallucinations that, while patients recognise individual letters, numbers, or

Abbreviations: VWFA, visual word form area

words, there is little or no meaning to the text as a whole. The text hallucinations are primarily orthographic without prominent syntactical or semantic content.

Here we report a patient with text hallucinations of an entirely different character which followed bilateral occipito-temporal infarcts. Instead of hallucinating individual letters or words the patient hallucinated entire sentences whose meaning was clear and, on occasion, acted upon. Taken together, the meaningful grammatical content of the hallucinations, the location of the lesions, and, most significantly, the patient's alexic syndrome point to an origin of the experiences outside the orthographically specialised cortex.

CASE REPORT

GW, a 78 year old married white woman, was admitted to hospital in May 2001. She had been an enthusiastic crossword puzzler, completing several puzzles every day and, on one occasion, noted that the letters of her crossword became "all jumbled" for a few minutes. The experience was not reported to her doctor and, two weeks later, she suffered a stroke. Computed tomography at the time showed bilateral ventral occipito-temporal lesions with extension of the right hemispheric lesion to the parietal lobe. Later magnetic resonance imaging showed the lesions in more detail (fig 1). The patient's right hemispheric stroke affected the upper and lower banks of her right calcarine cortex, resulting in a dense left hemianopia, while her bilateral occipito-temporal lesions resulted in profound visual deficits within the "spared" right hemifield (detailed below).

Following discharge from hospital she developed a mild depressive illness, treated with sertraline (50 mg daily). In October 2001 she began to experience visual hallucinations of written messages. Although the experiences began several months after the initiation of antidepressant treatment, the sertraline was withdrawn as a precautionary measure. The withdrawal of this drug had no effect on the hallucinations, and she was started on olanzapine (5 mg daily). This did not lead to a significant improvement, although after a dose increase to 7.5 mg daily it modified her interpretation of the experiences and her response to them (see below).

At the time of testing the patient was hallucinating written messages several times a week. Apart from a brief admission in November 2001 following a single generalised convulsion, she had lived at home since discharge and was attending a

day hospital. Other than the hallucinations she had no evidence of a major psychiatric disorder. In particular she did not meet diagnostic criteria for major depressive illness, late onset schizophrenia, or dementia (her mini-mental test score was 27 of a possible score of 28, after taking her visual impairment into account). She had no parkinsonian features and was neurologically intact apart from the visual dysfunction described below and a mild left sided distal weakness. An EEG showed marked slow wave activity consistent with her stroke but no evidence of epileptiform activity.

Visual hallucinations

The patient was able to differentiate her visual hallucinations from visual imagery experiences. Her hallucinations had the vivid perceptual quality of real objects, were localised in external space, and were entirely outside her control. They would last several seconds and appeared on walls in front of her or to her left (her blind hemifield). When they first appeared, they could occur at any time but, by the time of her examination, they occurred predominantly in the evening. They consisted of warning messages or instructions in a scrawling hand written red script or black font print. Examples include:

"Don't eat the fish."

"Don't take your tablets."

"They're after your money, we have got some of them but we are trying to find the others."

"Throw the water at them to see if they are one of them."

There was a conversational aspect to the messages, with questions, comments, and replies. She was able to read the hallucinated messages without having to scan each word. On one occasion she experienced hallucinations of figures consisting of ladies in bowling costume climbing over the television. She had not experienced other commonly reported hallucination categories such as phosphenes, grid patterns, or disembodied faces. Whatever their content, the hallucinations were always silent with no associated tactile or olfactory experiences. She did not develop a complex delusional system

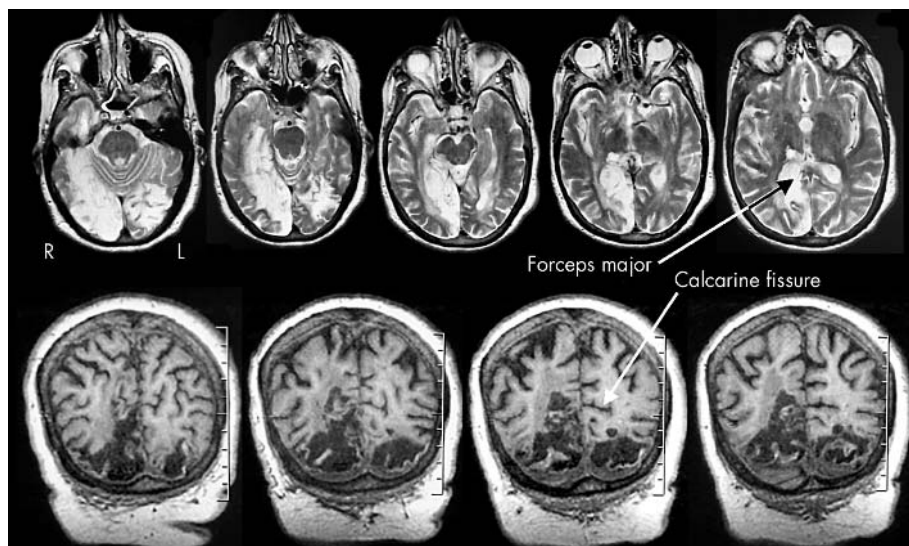


Figure 1 Magnetic resonance images of the patient's lesion. Axial T2 weighted images (top row) and coronal SPGR slices are shown (bottom row).

to explain them but did feel threatened on occasion and was convinced that the messages were real, floating in the air, even asking her family to read them to her. On occasion she obeyed the written commands, at worst throwing a cup of hot tea over her daughter. Her insight into the experiences improved after the olanzapine dose was increased (to 7.5 mg daily). By the time of the testing reported below she did not feel compelled to act on the hallucinations and accounted for them as “seeing her own thoughts written down.” She was observed while hallucinating on several occasions. The experiences occurred in clear consciousness without inaccessibility, drowsiness, or motor automatisms. During a hallucination, she was able to continue whatever she was doing and report the experiences as they occurred.

Visual function

The patient had a dense left hemianopia and was unaware of her field defect (a hemianopic anosognosia¹⁰). Formal perimetric plotting of the defect proved difficult owing to her inconsistent reporting of test stimuli because of her continuing illusions and hallucinations. The visual function in her “spared” right visual field was entirely consistent with the location of her lesions, consisting of intact low level and parietal abilities and profound occipito-temporal and right lateral occipital deficits. A series of customised tests was designed to examine her higher level visual function, based on standard assessment tools but using large, high contrast stimuli.

Low level vision

The patient’s visual acuity was relatively spared. Given her difficulty with letters (see below) we presented single or double line stimuli, of varying separation, and asked her to report whether she saw one or two lines. She was able to perform the task to a Snellen equivalent of 6:24. This level of acuity allowed her to recognise simple shape stimuli without difficulty.

Ventral occipito-temporal function

The patient had various ventral occipito-temporal deficits.

Dyschromatopsia

She was unable to identify correctly specific colours from a set of colour swatches and, when asked to name the swatches, did so incorrectly with consistent errors (for example, blue for green; green for blue).

Prosopagnosia

When presented images of famous faces (for example, the Queen, Princess Diana) she was able to identify the images as faces but not their identity.

Associative agnosia

She was unable to recognise images of common objects in canonical views (for example, a tea pot was called a bird), while able to copy the outline of the images.

Dorsal occipito-parietal function

In contrast to her profound occipito-temporal deficits, the patient’s visual parietal function was largely intact.

Motion

She was able to detect slow (2°/s) and fast (20°/s) moving targets and correctly identify their direction (although note the directional illusion reported below).

Optic praxis

She was presented with moving targets on a computer monitor and, 0.5 to 1 second after the disappearance of the

stimulus, was asked to trace its trajectory. With the exception of rightward horizontal motion (see below), her performance was entirely normal.

Spatial vision

She was able to describe the relative positions of target objects placed in the fronto-parallel plane.

Neglect

She ignored the left part of visual space with a rightward line bisection bias, consistent with her hemianopic anosognosia.

Lateral occipital function

The lateral occipital cortex processes illusory contours such as those found in Kanizsa figures.^{11 12} The specialisation is related more to the right hemisphere, although the left hemisphere is also involved. Consistent with the extension of her right hemispheric lesion to the lateral occipital surface, the patient was unable to detect illusory contours despite being able to describe, and trace by finger, the component Kanizsa figure parts.

Illusions and hallucinations

The patient experienced hallucinations and illusions during visual testing. Some were transient, such as seeing individual words and objects on blank regions of the testing sheets (for example, additional copies of testing stimuli or a rose garden). Others were more consistent, and the factors influencing their induction could be investigated further.

Induced colours

When presented with black and white stripes, the patient saw the stripes in colour. The effect was first noted when she was shown an illusory contour stimulus consisting of parallel black horizontal lines on a white background, broken at their mid-point, the left and right halves being offset in the vertical axis. She did not perceive an illusory vertical line, as is the case for normal sighted subjects. Instead, she reported a series of blue and red lines. To explore this colour induction illusion further, she was presented with computer generated gratings and plaids (two superimposed orthogonal gratings) with varying spatial frequencies, luminance profiles (a smooth or abrupt change from black to white), and duty cycles (the relative thickness of black and white stripes). The results are summarised in fig 2. We found that it was not the break and vertical line offset in the illusory contour stimulus that induced colours in her, as standard horizontal or vertical line gratings produced the same effect. The important factor seemed to be the spatial frequency of the stimulus. If the grating contained a few thick stripes (low spatial frequencies) or many thin stripes (high spatial frequencies) she saw only black and white stripes or a homogeneous dark grey. In contrast, at intermediate spatial frequencies she saw colours (the 0.58 and 1.25 cycles per degree stimuli in fig 2). Changing the luminance profile of the stimulus did not seem to affect colour induction, nor did changing the duty cycle or the presentation of plaids.

Illusory direction of motion

As noted above, the patient correctly detected motion, identified its direction, and had normal optic praxis. However, during the motion experiments we noticed she made systematic errors for a specific stimulus direction. When the target dot appeared in her left (hemianopic field) and moved to the right, she traced the direction of motion as initially leftward, then doubling back on itself and ending in the correct, rightward direction. An example of the error is shown in fig 3. The error was specific for horizontal motion, as other directions which appeared in the left hemifield were

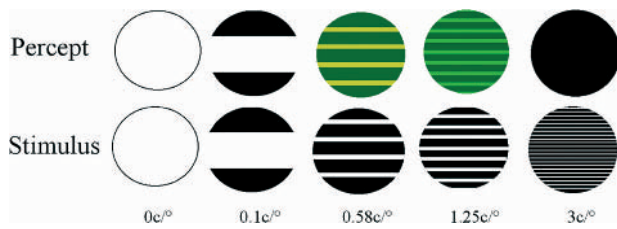


Figure 2 Colour illusions. The top row shows the grating stimuli as perceived by the patient, the bottom row the gratings as perceived by a normal observer. The spatial frequency of each stimulus is shown below each column. The patient sees colour in black and white gratings of intermediate spatial frequencies.

traced correctly (for example, targets passing from 8 to 2 o'clock). Furthermore, the error was specific for rightward and not leftward motion, as shown by the normal leftward performance in fig 3.

Language

The patient's heard and spoken language functions were entirely normal. She could understand simple and complex commands and name objects placed in her hand with a preserved knowledge about their functions and attributes. Her spontaneous speech was normal, although she made occasional phonemic paraphrastic errors (for example, strike for stroke), and she was able to repeat phrases and write short sentences (see fig 4 for an example). It was only in the visual domain that her language problems became apparent.

Pure alexia (alexia without agraphia)

Consistent with her left occipito-temporal lesion, the patient was unable to read individual letters or complete words. She could see the outline of letters and trace them with her finger but was only able to name them after deducing their identity from the contour. Figure 4 shows an example. She correctly identified a "backwards C" shape in the letter P but was unable to recognise the P or to read APPLE. Like many patients with pure alexia, she was unable to read her own writing (or even recognise it as her own) when it was presented after a distracter task.

DISCUSSION

The patient's visual text hallucinations differed from the typical visual text hallucinations of eye disease. Instead of

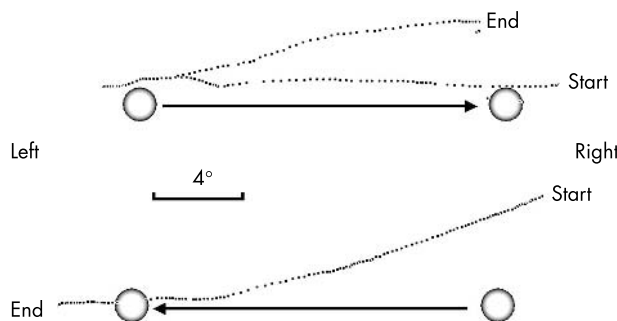


Figure 3 Direction of motion illusion. Two trials from the motion experiment are displayed. The circles show the start and end position of the target, with the patient's screen trajectory shown as a dotted line. The patient is asked to look at the centre of the screen at the beginning of each trial (midpoint of the trajectory). For rightward horizontal motion (top trial), the patient traces an initial leftward path and reverses direction to complete the trial in a rightward direction. For leftward motion the patient traces the correct target path.

Reading

Writing

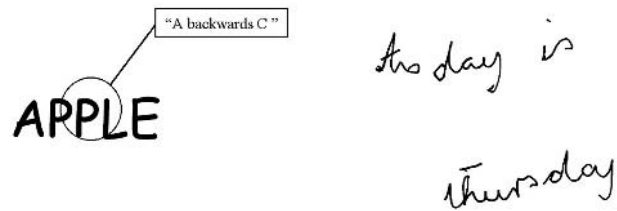


Figure 4 Pure alexia. The patient cannot read words despite being able to see the outlines of individual letters. Her spontaneous writing and writing to dictation is normal.

seeing single identifiable words or nonsense letter strings, she experienced entire sentences and phrases. Below we speculate on the clinical aetiology of her hallucinations and consider the implication of their content for the cortical organisation of visual language.

Clinical aetiology

Visual hallucinations occur in a range of clinical contexts, several of which are of relevance here.

Deafferentation—The patient's stroke infarcted the right primary visual cortex, involving both inferior and superior banks of the calcarine fissure, leaving her with a dense left homonymous hemianopia and deafferenting higher visual areas. Around 40% of patients with such visual pathway lesions suffer from visual hallucinations, typically within the hemianopic field.^{5 6} The patient's hallucinations sometimes occurred to her left, supporting a stroke related deafferentation explanation. However, deafferentation at the level of primary visual cortex is unlikely to be the most significant aetiological factor, as the onset of the experiences was several months after the infarct, not the hours, days, or weeks typically described.

Epilepsy—Isolated visual hallucinations also occur as auras in partial seizures of temporal or occipital lobe origin, with complex content, such as that reported here, associated with anterior temporal foci.¹³ In support of an epileptic aetiology, the patient had a generalised seizure on one occasion. However, the seizure was not preceded by visual hallucinations, nor was there evidence of inaccessibility or automatisms during or after the hallucinations to indicate a complex partial seizure origin. While the hallucinations may reflect localised cortical "irritative" activity, they do not seem to be "epileptic" in the clinical sense of the term.

Delirium—Visual hallucinations occur in delirium, of which stroke is an important precipitant. The patient's hallucinations occurred in clear consciousness and were remembered in detail, both factors arguing against a delirious aetiology. Furthermore delirious hallucinations are typically multi-sensory and not purely visual.¹⁴

Late onset schizophrenia—The patient's initial lack of insight, the persecutory threatening content of her hallucinations, and their "command" nature are all consistent with a diagnosis of schizophrenia; however, like delirium, the visual hallucinations of schizophrenia rarely occur without hallucinations in other modalities¹⁴ and, when occurring in old age, tend to be associated with complex systematised delusions.¹⁵ The absence of these features, together with the fact that non-threatening hallucinations continued after the patient's insight was restored, argues against a purely schizophrenic aetiology.

Dementia—The relative preservation of cognitive function excludes dementia as a cause and there were no parkinsonian features to suggest Lewy body disease.

Iatrogenic—Hallucinations may be precipitated by a range of drugs, particularly those associated with anticholinergic side effects.¹⁶ Selective serotonin reuptake inhibitors may (rarely) induce visual hallucinations¹⁷ but the temporal relation between the start and end of the patient's sertraline treatment and the onset and continuation of her hallucinations makes it unlikely that sertraline caused them.

As no single diagnosis provides a satisfactory explanation for the patient's hallucinations it is likely that several factors acted in combination to cause the experiences. Furthermore, their delayed onset may indicate that cortical reorganisation after the stroke played a part. While the cause of the hallucinations is unclear, it is not this aspect of the case that is of primary interest here. The significance of the hallucinations is the fact that they occurred at all. Hallucinations of meaningful, grammatical visual sentences in the presence of pure alexia have important implications for the cortical representation of visual language.

Hallucination content as a guide to cortical specialisation

Like its monkey counterpart, the human visual system consists of multiple richly interconnected, map-like areas, each with a degree of specialisation for different visual attributes.¹⁸ Such specialisations range from low level visual attributes such as colour, motion, and textures to high level visual attributes such as objects, faces, and extended landscapes. The functional specialisation of different cortical regions for different visual attributes accounts for an important characteristic of visual perceptual deficits—they each fall into distinct, dissociable categories, with a lesion of cortex specialised for one attribute leading to a perceptual deficit of that attribute. For example, lesions of V4 are associated with selective deficits of colour vision (achromatopsia¹⁹), of V5, motion vision (akinetopsia²⁰), and of the fusiform face area,²¹ of familiar faces (prosopagnosia²²). Visual hallucinations also fall into specific categories, each reflecting the specialisations of a different cortical region.²³ This segregation of hallucinations into categories is thought to reflect the fact that pathological activity within a given cortical area leads to a hallucination of its specialised attribute.⁹ If the pathological activity extends beyond an area to include its neighbours, the hallucination categories associated with each of the areas involved will link perceptually to form visual hallucinatory syndromes.⁸ What causes a given patient to experience some categories of hallucination but not others is not clear. On clue may lie in the observation that some categories of hallucination are associated with specific diseases. For example, colour hallucinations tend to occur in patients with age related macular disease, a condition known to cause degenerative changes within cortical colour pathways.⁸ The implication is that cortical colour areas in such patients are particularly susceptible to the pathological activity underlying hallucinations and that the patients are therefore more likely to hallucinate colours.

Visual perceptual deficits and visual hallucinations are thus two sides of the same coin—deficits resulting from an absence of activity and hallucinations from pathological activity within a specialised cortical region.²³ As the cortical specialisation of an area defines the content of its associated hallucination, the converse must also be true—each category of hallucination indicates a cortical specialisation. Visual hallucinations thus provide important clues as to the specialisations of the visual system, complementing insights

derived from the neuropsychological deficits of cortical lesions.

Orthographic hallucinations

In our survey of visual hallucinations secondary to eye disease, textual or musical note hallucinations occurred in around one quarter of the patients.⁸ This differs from other surveys in which such experiences have not been reported. However, many of these studies excluded simple hallucinations like notes or letter shapes, or pooled together rarer hallucinations under a single "miscellaneous" category. Ours is the only study to have asked specifically about the occurrence of text hallucinations, and we would argue that a similar prevalence would have been found in previous studies had patients been asked about them. The experiences consist of isolated words and nonsense letter strings, invariably without an overall meaning to the text. Similar visual text hallucinations have been described as a visual aura in a patient with epilepsy following a left parieto-temporal lesion.²⁴ This patient saw isolated words written in black letters (for example, "chameau," "ciseaux," "magnetophone"—the patient was French) and hallucinations of objects. In common with ophthalmological patients, the epileptic aura text hallucinations conveyed no meaning beyond that of the individual words and never occurred as extended sentences or phrases.

What cortical specialisation might be responsible for hallucinations of individual words and nonsense letter strings? Invasive neurophysiological recordings¹ and functional imaging studies²⁻⁴ identify a localised region of the left posterior fusiform gyrus which responds more to text—whether nonsense letter strings or words—than to non-textual control stimuli. The area has been termed the visual word form area (VWFA) and seems to be indifferent to the semantic attributes of words, responding to the same degree whether or not the words are contextually appropriate.¹ We propose that single word and nonsense letter string "orthographic" hallucinations are related to the VWFA. Although we do not have direct evidence to support the hypothesis (none of the hallucinating patients in our fMRI study had text hallucinations⁹), it is consistent with other findings. For example, our factor analysis of hallucination content identified three independent hallucinatory syndromes related to the anterior ventral temporal lobe, the parietal lobe, and the superior temporal sulcus.⁸ The factor loading for orthographic text hallucinations was below our threshold for inclusion into any of the syndromes; however, it was highest for the anterior ventral temporal syndrome (see figure 2 in Santhouse *et al*⁸). The implication is that orthographic text hallucinations are related to a region in the ventral temporal lobe but that this region is not located anteriorly—entirely consistent with the *posterior* ventral temporal location of the VWFA. Further evidence for a posterior temporal origin of orthographic text hallucinations is that the seizure auras of the patient described by Rousseaux *et al* consisted of objects on some occasions and words on others.²⁴ Cortex specialised for objects is localised in the ventral temporal lobe,²⁵ in close proximity to the VWFA. It would not be surprising for a seizure focus to affect one or other of the regions on different occasions.

Visual command hallucinations

The hallucinations of the patient described here were very different from those reported above. The patient experienced phrases and sentences with a conversational quality, endowed with meaning and often commanding action. She did not "read" the hallucinated sentences but registered their meaning without having to scan the text and, as her psychotic features resolved, reported that the experience

was like seeing her own thoughts written down. The sentence hallucinations might be considered atypical orthographic hallucinations were it not for the fact that the patient also suffered from a pure alexia. Pure alexia (alexia without agraphia) was traditionally considered a disconnection syndrome—either between hemispheres or within a single hemisphere.²⁶ However, the presence of a cortical specialisation for visual word forms suggests an alternative explanation, recently confirmed by Leff *et al*,²⁷ namely that pure alexia is caused by lesions affecting the VWFA. The patient's bilateral occipito-temporal infarcts include the VWFA, explaining her pure alexic syndrome.

If the patient does not have a functioning VWFA, the region cannot be the origin of her visual command hallucinations as, we would argue, loss of a specialised cortical module precludes hallucinations of its specialised attribute. What other cortical area or areas might be responsible? One clue lies in the linguistic content of the visual messages with their command nature, threats, and second person form of address. From a linguistic point of view, the patient's hallucinations were identical to the auditory hallucinations of schizophrenia, the only difference being that they were read and not heard. Imaging studies of schizophrenic auditory hallucinations have implicated a network of regions, including a lateral temporal region (a portion of the superior temporal gyrus, extending beyond primary auditory cortex), the inferior-lateral frontal lobe, and the temporo-parietal cortex.^{28–30} Interestingly, parts of the same network are activated in sentence reading and semantic processing tasks,³¹ implying that syntactical and semantic linguistic processes play a role in the generation of the auditory hallucinations. Of significance to the patient described here, some of the regions in the network respond to sentences whether presented visually or aurally (see, for example, Bottini *et al*³² and Mazoyer *et al*³³). While the exact details are not known, it is likely that visual and auditory syntacto-semantic processing takes place in different sub-compartments of these cortical areas, just as, within visually activated cortex, different subcompartments process textual and pictorial versions of the same semantic task.³⁴

The three regions implicated in auditory hallucinations were spared bilaterally in the patient, raising the possibility that her visual command hallucinations were generated within visual subcompartments of the syntacto-semantic network. Why such visual command hallucinations are not more common in patients with hallucinations related to eye disease is not clear. Their rarity may suggest that the VWFA lesion was an important aetiological factor, perhaps through the selective deafferentation of downstream visual subcompartments in the syntacto-semantic network.

Illusions of colour and motion direction

The patient saw illusory colours when presented with monochromatic gratings of certain spatial frequencies. A similar though less intense colour inducing effect is found in normal subjects (the Luckiesh illusion), although the spatial frequencies involved are higher than those reported here.³⁵ While the cause of the colour induction in normal subjects is unknown, in our patient it is unlikely to be related to activity in the human colour centre, area V4. As pointed out above, we would argue that the loss of a specialised cortical module precludes the possibility of a hallucination of its specialised attribute. As V4 was infarcted bilaterally in the patient, leading to a syndrome of cerebral dyschromatopsia, this would preclude her from having colour illusions or hallucinations. However, the situation is complicated by the fact that, while colour is coded in V4, cells in other cortical regions are wavelength selective (see Zeki¹⁹ for the distinction between colour and wavelength) and contribute to the

wavelength based visual capacities of patients with V4 lesions.³⁶ Cortical areas V1 and V2 respond to orientated lines and gratings of specific spatial frequencies,³⁷ and contain wavelength selective regions. One possibility is that the patient's "colour" illusions arose through abnormal neural crosstalk between these orientation and wavelength specialised cells.

The patient also saw brief leftward motion for horizontal rightward moving stimuli. Motion in one hemifield activates the motion specialised cortex (V5) in both hemispheres, the signals from contralateral V5 passing to ipsilateral V5 through the corpus callosum for motion towards the vertical meridian and through a non-callosal pathway for motion away from the vertical meridian.³⁸ Thus in normal subjects signals related to stimuli in the left hemifield moving rightward pass callosally from right V5 (which has a relative specialisation for leftward motion) to left V5 (which has a relative specialisation for rightward motion).³⁸ Although the patient had a lesion in her right primary visual cortex, right V5 could be activated through direct subcortical pathways.^{39–40} However, once activated, signals would be unable to pass to left V5 (as would occur in normal subjects), as the patient's infarct transected the bundle of fibres connecting left and right V5 (the forceps major; see arrow in the top row of fig 1). In the patient, the rightward moving target would initially activate right V5 alone—an area specialised for leftward motion. In consequence she might perceive an initial leftward trajectory. Once the target had reached the vertical meridian and crossed to her sighted hemifield she would be able to perceive the trajectory correctly as rightward. This change from leftward to rightward percept is recorded as a doubling back on itself of her traced trajectory. The same error would not be made for leftward motion starting in the right hemifield, as the left primary visual cortex could provide its own directional signal. Although we have no direct evidence to support our hypothesis, the explanation does account for the salient features of the patient's direction illusion, namely its axis specificity (horizontal only), its direction specificity (rightward only), and its correction after the target crosses the vertical meridian.

Conclusions

The linguistic content of the hallucinations described here differs from that found in association with eye disease. In contrast to the predominantly orthographic content described by ophthalmological patients, the hallucinations contained syntacto-semantic elements similar to those found in the auditory hallucinations of schizophrenia. The findings point to two independent, dissociable categories of text hallucinations. The first consists of word forms and nonsense letter strings and relates to pathological increases in activity within the VWFA. The second consists of meaningful and grammatically correct phrases and sentences. Although the cortical origin of this category of visual hallucinations is unknown, the similarity of its content with those of schizophrenic auditory hallucinations points to the network of areas involved in auditory or visual syntacto-semantic linguistic processing.

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