The Neuroanatomy of Phenomenal Vision: A Psychological Perspective

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ABSTRACT: Somewhere in the visual system, phenomenal vision—the seeing of colors, brightness, depths, shades, and motion—is generated not only from the distribution of light on the retina, but also when the eyes are closed, in dreams, hallucinations, phosphenes, and (possibly) imagery. Whether these different forms of phenomenal vision share a common substrate although their origins are different (optical, mechanical, electrical, endogenous) is discussed in the light of evidence from neuropsychological and functional imaging studies. Whereas extrastriate visual cortical areas appear to be involved in all types of phenomenal vision that have been studied, the necessity of a contribution from primary visual cortex is demonstrated by the loss of conscious vision that follows its destruction. If both extrastriate and primary cortical activation are needed, the latter may not just provide an indispensable input, but may also need to receive the output of the extrastriate processing via reentrant connections.

KEYWORDS: Veridical vision; Blindsight; Nonveridical vision; Phosphenes; Dreams; Hallucinations; Imagery; Afterimages; V1; extrastriate cortex; Vision

INTRODUCTION

Where in the visual system does the neuronal processing of visual information become conscious? In the past ten years, several suggestions have been put forward: they focus on the extrastriate cortical areas, either in isolation[1,2] or conjointly with the primary visual cortex,[3,4] or on those extrastriate visual areas that form the ventral, occipitotemporal stream of visual cortical processing only,[5] or they include extra-visual areas in the frontal lobes,[6–8] or in the reticular formation.[9] In addition to this already wide range of structures, within the broader discussion of the neuronal basis of consciousness, thalamocorticothalamic loops linking the visual cortical areas with specific and unspecific thalamic nuclei are discussed.[10–12]

Focusing specifically on visual consciousness instead of on the general question of conscious representation restricts the problem to the best-studied of the sensory systems: Whatever mechanisms mediate the mysterious transformation of neural information into sensory awareness in the visual system would, presumably, play a similar role in other sensory systems. Unfortunately, however restricted conscious vision appears when compared to the entirety of conscious experience, it is still very complex. It includes awareness of brightness and darkness, of colors and motion, of

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depth and shapes and objects; it includes recognition of what one sees and what it may be used for; it includes veridical as well as non-veridical vision as in dreams and hallucinations, and ultimately it includes the conscious organism, a self, who sees, recognizes, and acts upon the visual information. Embedding conscious vision within the full range of the organism's experience requires reference to structures outside of the visual system and, because sensation serves action, requires the use of inclusion of the motor system as well. Nevertheless, while recognizing that conscious vision is something that only a conscious organism has, and that it is there to serve this organism by guiding its actions, I prefer, on heuristic grounds, to restrict the inquiry to the conscious representation of visual information, Moreover, I shall mainly address what I regard as conscious vision's most basic aspect, namely, that of seeing brightness and darkness, colors, and movements. This is phenomenal vision, whose elements are the visual qualia from which objects are constructed. It comes in veridical and nonveridical forms.

ORGANIZATION OF THE VISUAL SYSTEM

Let us first look at the visual process and the neural system which mediates it. Our current scientific paradigm and our immediate apprehension agree that there exists a real, physical world in which we live and whose properties we perceive. The physical reality we visually perceive consists of a small part of the electromagnetic spectrum, which, as light waves or particles emitted from light sources or reflected from surfaces, falls through the eye upon the retina. Here the spatial and spectral distribution of the light is transformed into a physiological code—nerve impulses that carry information about contrast, location, and chromaticity. The nerve impulses are transmitted from the retinal ganglion cells along the optic nerve and via the optic chiasm to the ten brain nuclei known to receive direct retinal input. The lion's share of the information is sent, first, to the dorsal lateral geniculate nucleus (dLGN), and from there on to the primary visual cortex (V1, or striate cortex) on the medial aspect of the occipital lobe. From here, the information is forwarded to the many functionally specialized extrastriate visual cortical areas that together comprise the visual cortex. These areas receive their visual input not only from V1, but also from the various retinorecipient nuclei which project to the visual cortical areas either directly or via other subcortical nuclei. In addition to their lateral connections, the visual areas are interconnected both in serial and in parallel, and in both the forward (caudorostral) and backward (rostrocaudal) directions[13] (FIGS.1 and 2).

The subcortical visual nuclei are functionally specialized. The nucleus suprachiasmaticus is involved in the entrainment of the circadian rhythms to the light–dark cycle, the pretectum in the pupillary light reflex, the nucleus of the optic tract in optokinetic nystagmus, and the superior colliculus in saccadic eye-movements and attention. Evidence for functional specialization of visual cortex is provided (1) by electrophysiological recording studies, which have shown that neurons in different cortical regions have different receptive field properties, (2) by functional imaging studies which have demonstrated activation of different cortical areas by different types of stimuli, and (3) by clinical (neuropsychological) studies correlating selective impairments of visual functions with lesions in different visual cortical areas. The specialized areas tentatively identified by such studies, may themselves be seg-

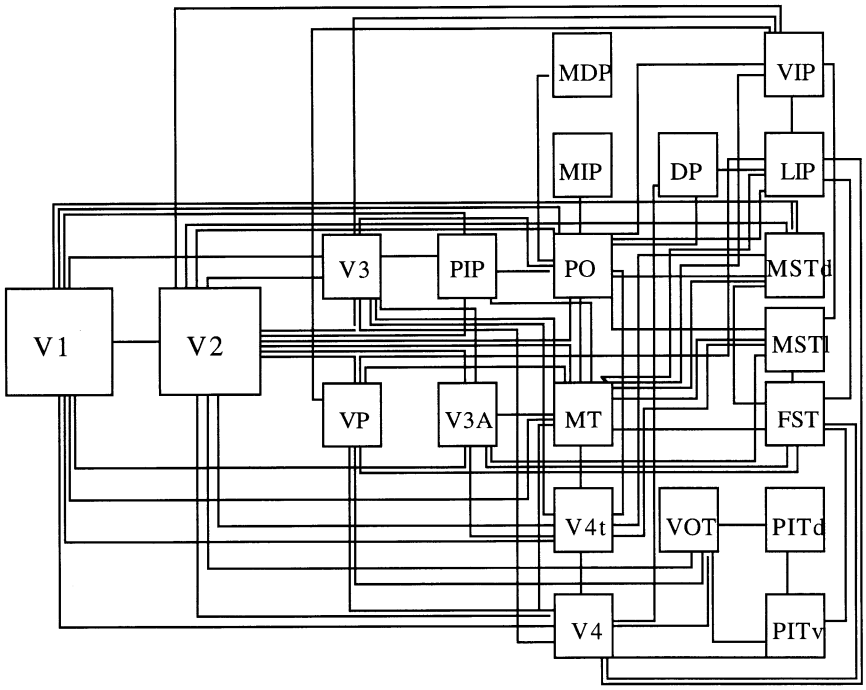

FIGURE 1. A schematic representation of functionally specialized visual cortical areas. Almost all known connections are bidirectional. (Data from Felleman and Van Essen.[13])

regated into still smaller functional compartments, increasing the difficulty of determining how and where vision becomes conscious. Were the visual system organized in a quasi-Cartesian fashion so that all retinal input eventually converged onto a single structure whose destruction abolished all conscious vision, we should happily accept that structure as "the mind's eye." Instead, we find a network of heavily interconnected, functionally specialized structures at both cortical and subcortical levels. Visual signals originating at the same retinal locus will be conveyed along different routes, via differing numbers of relay nuclei, by axons with different conduction velocities, arriving at their destination at different times.

How can perceptual unity arise from such a distributed network? Where in this dense mesh of interconnected visual neurons does the visual neural code get transformed into the phenomenal visual world? After all, qualia do not exist in the physical world, nor are they properties of neuronal processes. Instead, they represent a mental level of reality; their perception by an animal defines its vision as conscious. The construction of qualia may depend uniquely upon a single neuronal processing feature, or a combination of such features, from microtubules, synapses, and neurotransmitters, to neuronal morphology and connectivity, velocity of signal transmission, and the synchronous activity of cell assemblies distributed in cortical and subcortical structures. Several of these possibilities are dealt with in other chapters

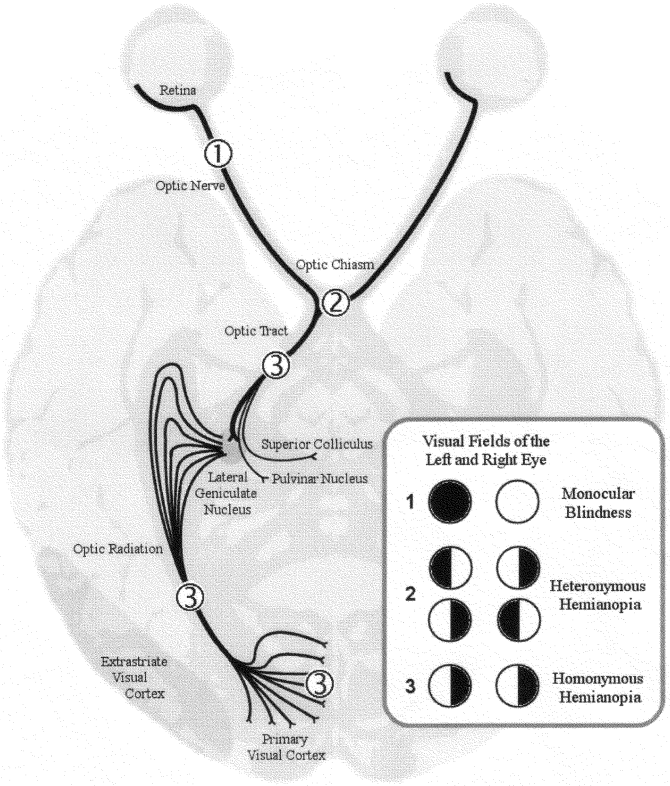

FIGURE 2. The primary visual pathways and (*inset*) the field defects resulting from lesions within this system. While destruction of the eye (1) or the optic nerve causes blindness in one eye, lesions involving the optic chiasm (2) will, depending on their precise location, cause a bitemporal or binasal heteronymous hemianopia. Post-chiasmatic lesions (3), due to the crossing of fibers from the nasal retinae, affect homonymous parts of the visual fields of the two eyes, regardless of whether they involve the optic tract, the radiation, or the primary visual cortex.

of this volume. In this chapter I shall focus on the macroscopic level, that is, the level of structures and networks of structures, and ask: where are visual qualia made?

VERIDICAL PHENOMENAL VISION

Absolute blindness is an absence of all visual qualia. It may result from destruction of one or more of the following visual structures: the eye, the retina, the optic nerve, the optic tract, the dorsal lateral geniculate nucleus (dLGN), the optic radiation, and the primary (V1) and secondary visual cortex (V2). A unilateral lesion will affect the visual field of one eye if it is prechiasmatic, and the visual field of both eyes if it is behind the chiasma (FIG. 2). The more distant it is from the retina, the more fibers projecting to extrageniculate visual nuclei will be spared, and the more

visual functions will remain intact. While a retrochiasmatic lesion may spare only the projection to the nucleus suprachiasmaticus, a retrogeniculate lesion will leave intact all retinofugal fibers projecting to extrageniculate nuclei. Visual reflexes such as the pupillary light reflex can then be elicited from the blind visual field, and so can nonreflexive visual functions, provided procedures such as forced-choice guessing are used to circumvent the blindness that the patients experience. The visual functions that have thus far been demonstrated include localization, detection, and discrimination of visual flux, and of the size, orientation, motion, motion direction, wavelength, and shape of stimuli presented in the blind field. These types of visual function, which are demonstrable in a subject's blind field, have been termed *blindsight* (see Weiskrantz *et al.*,[14] Weiskrantz,[15] and Stoerig and Cowey[16] for reviews). This phenomenon demonstrates that nonreflexive visual processing is possible in the absence of visual *awareness* of the processed information. Since the extrageniculostriate cortical visual system is undamaged in these patients, we must infer that the extrageniculo-striate system is, by itself, insufficient to mediate visual awareness.

Like destruction of the dLGN, destruction of the primary visual cortex causes a complete loss of phenomenal vision, but only the cortical lesion will permit the various nonreflexive visual functions of blindsight. Destruction of the secondary visual cortical area which surrounds V1 on all sides appears to have similar effects to those of a lesion in V1. Destruction of lower V2 causes a quadrantanopia in the upper contralateral hemifield, and destruction of upper V2 is followed by an anopia of the lower quadrant.[17] Disconnection of the primary visual cortex both from its geniculate input and from the higher visual cortical areas thus causes a loss of visual qualia. However, if the lesion selectively destroys extrastriate visual cortical areas *without* disconnecting V1 from the remaining cortical regions, visual qualia remain. Thus, when the occipitotemporal areas V4/V8 (the so-called "color complex") is selectively affected, color vision is lost, but movement and brightness remain. Conversely, when area MT, which is part of the human motion complex (hMT+), is affected, conscious motion vision is compromised, but color and brightness vision remain. It follows that phenomenal vision depends on the functional integrity of the early visual cortical areas surrounding and including V1/V2, with different areas supporting different qualia.

THE ROLE OF V1

The role that V1 plays in the concerted action mediating visual awareness is unclear. One hypothesis suggests that V1 functions like a cortical retina,[18] providing an input into the array of extrastriate areas without which they are incapacitated and cannot mediate phenomenal vision.[6] Alternatively, V1 could participate in visual awareness directly, supporting a quale of its own; indeed, the mediation of brightness has been suggested as its primary contribution to conscious vision.[19,20] Finally, its role could be to receive the results of extrastriate cortical visual processing via its extensive recurrent fibers, this feedback providing the crucial information necessary to generate qualia.[4,21] The first of these hypotheses affords V1 only a comparatively trivial role, but, like the third, is consistent with the loss of phenomenal vision produced by destruction of V1. The second is based largely upon a body of data suggesting the gradual development of some forms of conscious vision in a hemianopic

patient, GY, who suffered unilateral damage to his occipital lobe at age 8. This patient, now in his forties, has participated in a large number of studies of his residual visual functions, which have suggested that he is now aware of visual stimuli provided they have some salient feature, such as movement.[22–25] This visual awareness is phenomenal, or at least that is the conclusion drawn by the authors from GY's ability to find a perceptually satisfactory match in the intact field for a stimulus presented in the impaired field.[26] We have observed a similar slow change from absolute to relative blindness in another patient (FS) whose lesion occurred later in life (age 42), but who, like GY, has extensively used his hemianopic field in numerous blindsight studies.[27–30] In neither patient was any evidence for ipsilesional V1 activation found in functional imaging studies.[1,8,30–32] Within the limits of current technology, this demonstrates that some conscious vision may become possible without ipsilesional V1.

VERIDICAL VISION WITHOUT V1

What evidence suggests that it is specifically brightness that is missing in this kind of V1-less, low-level vision? Morland *et al.,*[19] using a forced-choice procedure, asked GY to try and match colors, motion velocity, and brightness between his normal and impaired hemifield. This involved his manually adjusting the visual properties of the matching stimuli so as to make a stimulus presented in one hemifield resemble another presented in the other field. [As in all forced-choice procedures, guessing was an available option]. While the patient's color and velocity matches were quite successful, his brightness matches bore little resemblance to those of a normal observer, leading the authors to suggest that brightness, rather than color and motion, depended on V1. These results, taken alone, are insufficient to support the hypothesis. Not only may a forced-choice match in a blindsight subject be based on phenomenal properties quite different from those that mediate a match in unimpaired subjects, but also it may reflect isomorphic processes underlying quite different perceptual representations such as conscious and unconscious ones. More importantly, GY strongly denies "seeing" colors, and even his extensively studied motion processing performance[1,22,23] may reflect his inferences about positional information rather than motion perception *per se.*[23,27,32] It is therefore premature to conclude, even in GY's case, that brightness is the only quale dependent upon V1's integrity.

Independently of whether V1 plays a special role in brightness perception, one wonders what the source is of residual conscious vision in the absence of ipsilesional V1? Given the observation that destruction of V1 produces cortical blindness, how can its absence be compensated? One possibility is that qualia may be mediated by extrastriate cortex alone; another that qualia require the joint activation of both extrastriate and extravisual systems. The first alternative is contradicted by the fact that stimulation of the impaired hemifield, although it activates extastriate cortical areas in patients with absolute cortical blindness, does not produce even rudimentary awareness of the stimuli.[33] Extrastriate cortical activation alone is therefore insufficient, even if it involves not just dorsal but also those ventral extrastriate cortical areas[30] that have been implicated in the mediation of conscious vision.[5] But perhaps ipsilesional extrastriate cortical activation, though initially insufficient, could with practice recover to the point of supporting low-level phenomenal vision. Increasing

use of the hemianopic field might be the most likely process mediating such recovery, but there may be others.

There is the alternative possibility, that for visual awareness, extrastriate cortical activation needs to be coupled to activation in extravisual structures, that is, structures outside the classically defined visual system. This hypothesis was suggested by the results of two functional magnetic resonance imaging studies on GY. As GY's visual awareness depends on the velocity of a moving stimulus,[24] both studies used a dot stimulus moving at different speed through the patient's impaired field, and compared a condition evoking awareness (fast motion) with one that did not (slow motion). Both studies found activation in the motion complex (hMT+) *which appeared stronger in the aware condition.*[9] In addition, both found differential activation of extravisual structures, the laterodorsal frontal cortex in the first study (Sahraie *et al.*[8]; note numerous additional foci) and the brainstem reticular formation in the second study.[9] The different conclusions of the two studies illustrate some of the problems associated with imaging studies of visual awareness. First, high-resolution images of the entire brain are needed to avoid biasing the findings, and, second, very good quality images are needed to avoid false positives (if the quality is poor, many of the putative foci of activation are probably meaningless). Finally, differences in activation, although often attributed to differences in awareness, may arise from differences in properties of the stimuli themselves;for example, different velocities may themselves trigger differential activation patterns. [Note: Zeki and ffytche [9] attempted an analysis designed to control for stimulus speed].

In view of the divergent results and the problems inherent in this approach, we do not yet know how the low-level conscious vision of patient GY is mediated. It may involve extra-visual structures, as suggested by the studies cited above, or reorganization of the functional connectivity of visual structures. Either way, it appears to be independent of involvement of ipsilesional V1. However, these exceptional cases should not obscure the fact that the vast majority of patients with complete destruction of V1 are cortically blind. Indeed, it is this observation that provides the empirical foundation for the traditional neurologists' view that V1 is the substrate of conscious vision. Finding the neuronal correlate of phenomenal vision in the absence of ipsilesional V1 should tell us something interesting about long-term plasticity in the visual system. Both patients have regained their conscious vision in the course of many years of experiments that forced them to use their hemianopic field. This type of practice has resulted not only in better performance in blindsight tasks and more residual visual functions, but, at least in their case, to the return of some conscious vision. How the recovery of vision in these cases is mediated is an exciting and important question which may have therapeutic implications. However, the neuronal correlate of visual awareness in these individuals is very likely to be different from that of the normal observer, in whom phenomenal vision depends on the integrity of V1.

Early visual cortical areas that include V1, V2, V3, V4, V8, hMT+ and possibly others seem to partake in the mediation of phenomenal vision since their destruction causes a partial or complete loss of visual qualia in the affected part of the visual field. As noted earlier, V1 could function either as an indispensable provider of input to processing mechanisms in higher cortical areas, and/or as the recipient of feedback generated by processing in these areas. The latter hypothesis implicates the

very extensive feedback connections among the visual cortical areas whose inactivation, via inactivation of an up-stream visual area, markedly alters the functional tuning of V1 neurons (e.g., Hupé *et al.*[34]). If the results of processing by higher-order areas were fed back to V1, this could explain the unambiguous positioning of objects in the visual field on the basis of the high spatial resolution of V1, which is in contrast to the much lower resolution in increasingly higher areas. This hypothesis gains support from a number of recent neurophysiological studies that used very different approaches. They have shown that the late response components (80–100 ms) of V1 neurons differ from the early ones in orientation tuning,[35] in preserving figure-ground segregation,[36] and most importantly, in the perceptual interpretation of stimuli.[37] Independent confirmation of this result comes, first, from experiments in binocular rivalry showing that a small percentage of V1 neurons respond according to the monkey's present percept and independent of its visual input,[38] and, second from a study showing that the responses of V1 neurons in cats reflected brightness rather than physical contrast.[39] Additional support for the hypothesis comes from studies of the effects of experimental interventions, such as masks and transcranial magnetic pulses, upon the conscious perception of a stimulus. These psychophysical studies have identified two different time windows for effective disruption of visual awareness. In addition to an early time window (ca. 20–30 ms), which coincides with the arrival of the retinal information in V1, a much later one (ca. 100–120 ms) was particularly effective at suppressing the conscious perception of a visual stimulus. The earlier window coincides with the initial processing of visual inputs, and will prevent those inputs from being forwarded to higher areas. But at 100–120 ms, when the information has long since reached extrastriate cortex, presentation of a masking stimulus still interferes with the after-discharges of V1 neurons,[40] and a magnetic pulse over the occipital pole still suppresses stimulus perception.[41] If visual awareness were indeed dependent upon the reception by V1 of feedback from extrastriate visual processing, V1 would play a more interesting role than that of a visual relay. The "feedback" hypothesis is certainly consistent with the observation that the late-response components of V1 neurons reflect the perceptual rather than the physical properties of a visual stimulus, since these late components are most likely to be affected by the results of feedback from extrastriate areas. Interestingly, lesions in higher visual cortical areas do not abolish or diminish the patient's repertoire of visual qualia, but cause higher-order perceptual deficits.

NONVERIDICAL PHENOMENAL VISION

The previous section dealt with veridical vision—the situation in which light falling onto the retina is transduced into nerve impulses, and eventually transformed into visual qualia. But there are other means to evoke phenomenal vision. Afterimages are seen although the stimulus that induced them has disappeared. Phosphenes are phenomenal events, caused by mechanical, electrical, or magnetic stimulation of the retina or the visual cortex. Visual dreams are phenomenal, and result from involuntary intrinsic neuronal activation, as do hallucinations, while visual imagery may also be phenomenal, and is caused by voluntary intrinsic activation. Do all these kinds of phenomenal vision share a common mechanism? Do they all depend on V1

and its extrastriate partners? Or is the neuronal correlate of phenomenal vision so varied that quite different structures may mediate it under different conditions, so that it is dependent not on a particular structure or set of structures but upon such features as the strength or the temporal patterning of nerve impulses?

Let us begin with the phenomenon of **phosphenes**. Rubbing one's eyes causes visual experiences that can be complexly patterned and colorful, while a bump on the head causes one to "see" the "stars" with which we are familiar from cartoons. Electrical stimulation of the eyes or brain also causes phosphenes, as does the much newer and painless transcranial magnetic stimulation (TMS). Both kinds, electro- and magnetophosphenes, can be evoked in people who have lost their eyesight because of damage to the eye or optic nerve: A retinal input is not needed. But what about cortical areas? In the majority of studies using TMS, the technique has been used to suppress rather than evoke vision[42] (see Walsh and Cowey[43] for recent review). These studies have found that the timing of the magnetic pulse in relation to the presentation of the visual stimulus is critical (see above). However, several studies have also described magnetophosphenes evoked from stimulation over the occipital lobe,[44–46] suggesting that phosphenes were mediated by activity in early visual cortical areas or even the optic radiation. An obvious problem with attempts to infer the site of phosphene production from the location of visual structure most directly stimulated is that the strong TMS pulse may activate a relatively wide network of structures. Nevertheless, the kind of phosphenes—simple arrangements of dots or lines or stars—provide a cue as to the involvement of early visual cortex, because it is direct electrical stimulation of these structures that evokes this type of phosphene.[47] If V1 is necessary for the seeing of phosphenes, its destruction should prevent their appearance. As yet, there is no published study, but in our own tests of three patients with homonymous visual field defects we failed to elicit phosphenes in the blind field. Because we used stimulation parameters optimized for *normal* observers, these data are preliminary; however, they do suggest a critical role for the early visual cortex in the production of magnetophosphenes.

Afterimages are another instance of exogenously induced phenomenal vision. A recent functional magnetic resonance imaging study in normal observers has revealed that both the presentation of a saturated color stimulus, and the long-lasting colored after-image induced by prolonged viewing of the stimulus are accompanied by activation in V1 and in the color complex (V4/V8). In addition to these areas, the motion complex hMT+ was activated during the afterimage phase only, which may account for the observation that the subjective dynamic component was seen only during the after-image.[48] Thus activation in the cortical motion complex area produces a phenomenal motion effect in normal observers even though the stimulus itself is not moving (see Tootell *et al.*[49] for MT's role in the motion aftereffect). The role of V1 in perception of the afterimage was first explored by Bender and Kahn[50] in patients with V1 lesions using colored figures that they presented entirely or partly in their patient's field defect. Their results showed that afterimages were not reported when the stimulus fell entirely into the blind field, but that it was subject to some perceptual completion upon central fixation, that is, when only part of the figure was invisible to their patient (FIG. 3, top). These findings agree with our own data (FIG. 3, bottom) as well as those of Marcel,[51] demonstrating that information from the cortically blind field may complete or otherwise influence the percept, but that, by

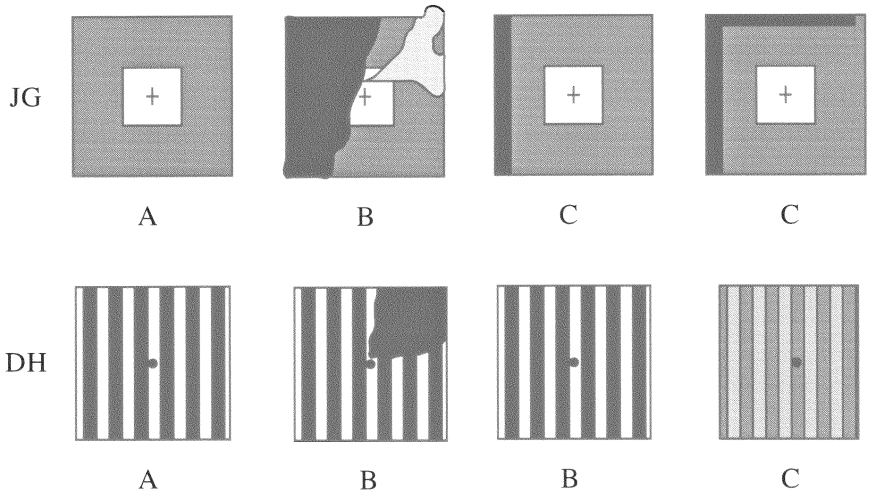

FIGURE 3. Perceptual completion and afterimages in patients with visual field defects from post-geniculate lesions. *Upper row*: Bender and Kahn's[52] patient JG; l*ower row*: patient DH (own unpublished data). **A:** The stimulus was a green 5 × 5 cm square superimposed on a red 10 × 10 cm square for JG, and a high-contrast yellow-blue grating for DH. **B:** The patient's rendering of the stimulus when fixation was in the center of the figure. The black region was not seen, and the light gray one appeared blurred to JG. Note that patient DH reported perceptual completion under direct fixation on some trials. **C:** The afterimages drawn by both patients show extensive completion. The opponent colors of the negative afterimage appeared somewhat paler, as indicated by the reduced contrast (*bottom right*).

itself, it is not sufficient to produce a phenomenal image; this also agrees with other experiments on perceptual completion.[26,52,53]

Little agreement exists, however, regarding V1's role in **dreaming**. Functional imaging studies agree in reporting extrastriate cortical activation during rapid eye movement (REM) sleep, with its higher incidence of dream reports. They disagree as to whether or not primary visual cortex is also activated, some studies reporting an absence[54] and others the presence[55] of significant activation. Unfortunately, this controversy cannot be resolved by neuropsychological observations. For normal (i.e., intact) individuals, our visual awareness during dreaming is "projected" upon a perceptual area defined by the borders of the normal visual field—and those borders are not something we perceive "positively." Similarly, patients with visual field defects from V1 lesions report that their blindness is "negative" under normal viewing conditions, that is, the *missing* part of the visual field does not appear black or stand out in any way, any more than do the borders of the *intact* visual field in normal people. Because no one has developed a paradigm for testing the visual field during dreaming, it remains unclear whether patients with visual field defects from V1 lesions have a shrunken "internal screen" for dreaming. In contrast, the involvement of extrastriate cortical areas in dreaming seems clear and is further supported by reports from patients with achromatopsia or prosopaganosia associated with lesions of

extrastriate visual cortex who experience an absence of color or recognizable faces not only in their waking life, but in their dreams as well.

Studies on the role of V1 in visual **imagery** are as controversial in their results as they are plentiful in number. Whether the system that mediates veridical vision also mediates visual imagery is the core question of the still unresolved "imagery debate." The available psychophysical, functional imaging, and neuropsychological data are in agreement with respect to the involvement of higher visual cortical areas. This was first shown convincingly by Roland *et al*.[56] in a positron emission tomography (PET) study on normal observers, and confirmed in numerous subsequent studies. What remains at issue is the involvement of early visual cortex. Several studies have demonstrated activation only in higher visual cortical areas,[56–58] while others have found it to extend all the way down to areas V1/V2[59–61] or even the lateral geniculate nucleus.[62] Kosslyn's psychological investigations of the properties of visual imagery led him to conclude that imagery must be supported by the "normal" visual system including early, topographically organized visual cortex (see Kosslyn[65] for review). He has argued that the failure to find activation in early visual cortex was caused by the lack of experimental control during the subject's "resting" condition, which serves as the control against which activation in the "imagery" condition is compared. If the subjects do not "wipe their screen clean" during the resting condition, but instead imagine something of their own accord, they will generate sufficient activation during the resting condition to minimize the contrast between the resting and imagery conditions. As a result, the study will fail to implicate V1/V2 as part of the visual network activated during visual imagery.

Another complicating factor in imagery studies is the variation in the imagining tasks used. These may range from imagining simple geometric pattern and letters to visualizing complex scenes, or even to imagining walking from home to a familiar place. It is generally accepted that simple visual patterns are processed largely in the early cortical areas, while complex spatial and object recognition tasks involve higher extrastriate cortical processing. In the complex tasks, less activation would be expected in the early cortical areas even if the results of the higher processing were to be fed back to earlier areas, because such feedback should require much less "capacity" than the processing task. Lower activation levels in the early areas would then be more likely to be missed. Finally, the task's demands on spatial resolution may influence the extent of early visual cortical involvement, with higher-resolution tasks requiring more processing by the early areas with their superior spatial maps of the visual field. This hypothesis is consistent with the report that imagining the same object in different sizes and at different positions within the visual field leads to early visual cortical activation.[60–62]

If the extent of top-down activation of the visual cortical areas is task-dependent then the deficits of patients with lesions in different visual cortical areas should reflect the presumed processing capacities of the specific areas. The results of several studies of patients with early visual cortical damage are consistent with this assumption. These patients did <u>not</u> show a deficit in tasks requiring answers to "high imagery content" questions such as "Does a bear have round or pointed ears?" or "Is a grapefruit larger or smaller than an orange?," which despite their referring to concrete objects can be answered on the basis of stored visual information.[64,65] Failure to correctly respond to such questions is more common in patients with visual agno-

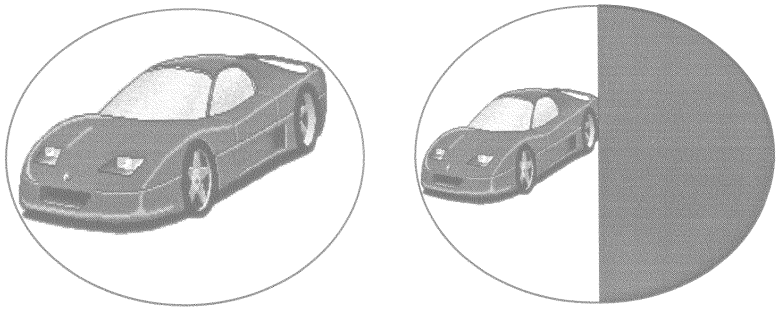

FIGURE 4. Farah *et al.* [68]reported that their patient required approximately twice the distance to imagine an object of a definite size after unilateral occipital lobectomy, indicating a shrinkage of the "field of visual imagery" that reflects the hemianopia induced by the surgery.

sia, suggesting that interference with this type of visual information retrieval reflects a disturbance of higher visual function rather than of cortical blindness. In contrast, in a patient available for testing both pre-and postoperatively, unilateral removal of the occipital lobe did reveal a striking change associated with the lesion and the subsequent hemianopia.[66] The patient was asked to imagine herself walking towards various objects of well-defined size, to stop when the object filled her internal screen to the point of overflow, and then to estimate the imagined distance between herself and the object. On the preoperative test, the patient said, for example, 33 cm for a kitten, and 188 cm for a car. During the postoperative test she more than doubled the distance, as if her internal screen had shrunk to the same extent as the visual field (FIG. 4). Furthermore, by repeating the tests with a ruler that had to be imagined in a horizontal or vertical orientation, this shrinkage was found to affect only the horizontal, but not the vertical extent of the image, as it should if the cortical blindness was responsible. Possibly, this task is more visuo-perceptual in nature, although it too draws on visual memory and could theoretically be solved without phenomenal imagery. An impairment of imagery requiring relatively high spatial resolution was also found in Butter *et al.*'s[67] study of hemianopic patients. When patients were asked to indicate whether an arrow was pointing at one of the dots in a pattern they had been shown in free vision but that was no longer visible, they were found to make more errors with arrows on the side of their hemianopia. Taken together, studies of imagery have usually reported involvement of higher extrastriate visual cortical areas, while the evidence for participation of early visual areas is much less consistent. The outcomes of these studies will be influenced both by task demands and individual problem-solving strategies, and, in the case of functional imaging studies, the design of the protocol and the analysis will be critical.

The last form of nonveridical vision to be addressed here are **hallucinations.** They may appear to patients with blindness due to (post-) retinal pathology—-Charles-Bonnet syndrome[68]—in whom the visual cortex remains excitable, as well as to patients with cortically blind visual fields caused by post-geniculate lesions.[69–72] They can be simple or highly complex, ranging from phosphenes to

complex geometric patterns to objects and people that move about. The comparatively simple forms—lines, dots, clouds, stars, triangles—are attributed to irritation within the primary visual pathway up to V1[69]; they closely resemble the magnetophosphenes elicited by TMS over the occipital pole. The complex ones, in contrast, are more likely to originate in temporal cortices, where images of scenes and people can also be evoked by electrical stimulation applied during neurosurgery.[73] Whether the precise content of the hallucinated images reflects the major focus of hyperexcitation, as argued by ffytche *et al.*[2] for patients with Charles-Bonnet syndrome who underwent fMRI during hallucination, is still uncertain.

The simpler hallucinations that are attributed to the early visual structures are regarded as indicative of some visual recovery,[68,74] while the complex ones may reflect a hyperexcitatory response to the lesion that caused the field defect, and usually disappear within a relatively short time.[71] That they are perceived in the cortically blind field demonstrates that strong endogenously generated activity may produce phenomenal images even in the absence of V1/V2. How this pathological activation differs from that caused by TMS is presently unclear. That it at least temporarily causes phenomenal visual images is uncontroversial (or almost so; see Pollen[20]) so that hallucinations are the only instance of fully conscious phenomenal vision without early visual cortex that is present immediately after the blindness-producing incident. The type of veridical phenomenal vision that may develop over long periods of training in blindsight subjects like GY is very crude and low-level when compared to such complex hallucinations as a series of identical gray–green men strolling through the cortically blind hemifield.[69]

SUMMARY AND OUTLOOK: COMPLEXITY AND UNITY

Phenomenal visual images can be caused by a variety of processes, ranging from exogenous (optical, mechanical, electrical, and magnetic events in the eyes or the visual cortex) to endogenous processes (which may be involuntary, as in visual dreams and hallucinations, or voluntary as in visual imagery). Studies using functional imaging techniques have consistently found that visual images, whether veridical or nonveridical, are associated with activation of extrastriate visual cortical areas. In contrast, activation of early visual cortical areas V1/V2 was reported in some, but not all such studies (see TABLE 1).

Despite their remarkable contribution to the study of functional neuroanatomy, imaging techniques are of limited use in demonstrating causation (that is, in establishing whether or not a particular structure is necessary for a particular function). While imaging studies can identify brain regions activated during performance of a particular task, they do not differentiate between activation necessary for the task and that merely associated with its performance. Moreover, interpretation of the results of imaging studies is always linked to the design of the experimental protocol, which may not always do justice to the physiological reality, as well as to the nature of the data analysis. Methodological problems associated with the former include the definition of the resting state in protocols used in studies of visual imagery, the selection of the specific temporal offset used between stimulus and response, or the control of the subject's psychophysiological state during the measurement. Exam-

TABLE 1. Summary of evidence from functional imaging and neuropsychology regarding the participation of primary and higher visual cortical areas in the mediation of veridical and nonveridical forms of vision

Structures	Extrastriate Areas		Primary Area	
Evidence from	Imaging	Neuropsychology	Imaging	Neuropsychology
Function				
Veridical				
Normal vision	yes	yes	yes	yes
Residual vision	yes	yes	no	no
Blindsight	yes	yes	no	no
Nonveridical				
Phosphenes	?	?	?/yes	yes
Afterimages	yes	?	yes	yes
Dreams	yes	yes	yes/no	?
Imagery	yes	yes	yes/no	yes/no
Hallucinations	yes	?	no	no

NOTE: All results, including those from studies on Blindsight where conscious vision is absent, agree on extrastriate visual cortical activation.

Yes/no reflects controversial results; ? indicates the answer is unknown.

ples of the latter type include the use of data smoothing vs. averaging, or the choice of statistical criteria. Such factors may yield contradictory results from comparable data sets (see Sibersweig *et al.*[75]and Dierks *et al.*[76] for an example). While functional imaging studies are undoubtedly exciting, such interpretive considerations make them less than conclusive when considered in isolation. Their results need to be complemented by neuropsychological studies of patients with lesions of circumscribed brain regions, which can provide evidence of the functional significance of the damaged regions.

VISUAL CONSCIOUSNESS AND THE ROLE OF V1/V2

The available data (summarized in TABLE 1) show that striate and extrastriate visual cortical areas need to be activated in all normal, nonpathological forms of veridical or nonveridical phenomenal vision. Patients who have suffered destruction of primary visual cortex are blind. They do not see stimuli presented in the blind field, they do not see afterimages of stimuli that were presented to the blind field, they have not been reported to see phosphenes from magnetic stimulation of the lesioned hemisphere, their imagery appears unaffected when their visual memory is tapped but not when a visuo-perceptual task requiring good spatial resolution is given. Whether these patients have visual dreams involving the cortically blind field is unknown. Only in hallucinations and in the rare cases of low-level vision re-established in former blindsight subjects such as GY and FS is phenomenal vision without ipsilesional V1 known to occur.

Neuropsychology thus strengthens the case for V1, but does not prove its necessity without exception—the hallmark of a good rule. The exceptions, both patholog-

ical, may indicate that V1's absence can be compensated for under certain conditions. In the case of hallucinations, the spontaneous extrastriate cortical activation is quite strong, and may therefore spread to other structures, subcortical and cortical, in the ipsi- and contralesional hemisphere. The complexity of this activation pattern is in contrast to the very focal activation observed from visual stimulation of fields of absolute and relative cortical blindness. Stimulation of the normal hemifield results in activation of the normal primary and extrastriate visual cortex, and in ipsilesional extrastriate activation as well, just as in normal observers. Stimulation of the blind field activates ipsilesional extrastriate cortex, but the activation appears quite isolated and focal, involving little if any activation of surrounding ipsilesional or contralesional cortex.[30] These findings make it tempting to speculate that the blind field is blind because the neuronal activation it elicits lacks the capacity to intiate a sufficiently complex pattern of visual cortical activation.[77] Destruction of V1 may prevent the development of such complex patterns, not only by destroying a large part of the input to the extrastriate cortex, but also by interfering with the extrastriate feedback to V1.[78] Unusually strong extrastriate activation, such as present in hallucinations, may allow a phenomenal representation by causing a complex widespread pattern of activation not normally evoked without V1, and extensive training of blindsight may induce processes that to some extent can compensate for the loss of this structure.

If conscious vision is always mediated by widespread striate–extrastriate cortical activation, could the presence of such activation in an organism's brain prove that it is consciously seeing something? Although this is likely to be the case in organisms who are in a conscious state, and not comatose or anesthetized, it is not true in unconscious organisms. Provided the animals were effectively anesthetized, the fact is clearly demonstrated by recent fMRI experiments in monkeys who showed ample cortical activation in response to visual stimulation despite being under general anesthesia.[79] As only conscious organisms can have conscious perception, evidence for strong activation of striate and extrastriate visual cortical areas cannot prove conscious vision and thus cannot prove consciousness in animals whose possible consciousness we cannot yet assess unequivocally. This *caveat* demonstrates the need to eventually account for the substrate of conscious vision, isolated here for simplicity's sake, within the larger context of the neural substrates of conscious (as opposed to unconscious) states in general. Such an account would attempt to explain how the presence of a general state of consciousness—presumably mediated by unspecific systems and temporarily abolished by anesthesia (see Lamme *et al.*[80] for effects of anesthesia on neuronal activity in V1)—transforms the visuo-cortical activation into phenomenal awareness.

Finally, let us consider that while visual processing is modular, its result is a unified percept. Whether the primary visual cortex provides a neural substrate for that perceptual unity remains an open question. The fact that it receives perceptually relevant cortico-cortical feedback, and that its destruction causes blindness despite the availability of extrageniculo-striate input, makes V1 a prime contender for that role. Having a structure at the bottom end of a presumed cortical processing hierarchy turns out to be responsible for the visuo-perceptual unity produced by that processing would add a nice twist, which the great Cajal, who cherished whodunits and who wrote one himself ("A secreto agravio, secreta venganza"), would have appreciated.

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