The Functional Anatomy of Visual Awareness

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What is the relationship between our subjective world of perceptions, thoughts, and memories and the activity of nerve cells that must somehow be responsible for this experience? The past 5 years have witnessed a transformation in the way the neuroscience community is approaching this complex of problems. As reported below, psychophysical and electrophysiological experiments are being carried out to elucidate the neuronal correlate of sensory awareness. If this aim can be realized and the anatomical structures and neuronal groups representing the contents of sensory awareness can be identified, we will be one step closer to understanding the most mysterious and central aspect of our existence.

In the visual modality, there is a preponderance of evidence, not yet conclusive but certainly persuasive, that visual awareness does not depend equally on all parts of visual cortex. In particular, it seems that visual awareness does not arise from neural activity in striate cortex (area V1, primary visual cortex) but in certain extrastriate cortical areas. The matter is complicated by the fact that an intact and functioning striate cortex is required for most forms of visual awareness (as are the eyes). Evidence pointing to some extrastriate visual areas, but not others, as being necessary for visual awareness is less compelling but certainly deserves a closer look. Here our point of view seems likely to evolve rapidly in the near future.

Once the link between visual awareness and certain visual cortical areas is confirmed, this will open subjective experience to the kind of systematic attack by neuroscience to which other mental functions—perception or language, for example—are already being subjected. How do areas that contribute to subjective experience differ from those that do not? Do these areas contain different cell types that express different proteins? Do they exhibit particular types of connectivity? Are they characterized by particular temporal patterns of neuronal firing?

The possibility that extrastriate, but not striate, cortex is necessary for visual awareness is consistent with a recent prediction to this effect, advanced mostly on theoretical and anatomical grounds (Crick and Koch 1995). The emphasis of this paper is not on these theoretical arguments, however, but on the gratifying amount of supporting evidence that has recently come to light.

We review evidence from patients suffering lesions in striate and extrastriate cortex, from single-cell recordings in nonhuman primates which have attempted to correlate the subjective experience of the animal with neuronal firing, and from psychophysical studies with displays that selectively stimulate early levels of processing and that have succeeded in manipulating visual awareness in normal human subjects.

FUNCTIONAL ANATOMY OF VISUAL AWARENESS

As neuroscientists, our point of departure must be that awareness is a property of the brain and thus, in principle, no more mysterious than other mental functions (for a different point of view, see Chalmers 1996). If this is so, awareness can be expected to depend to different degrees on different parts of the brain; in other words, it can be expected to possess a functional anatomy.

Recently, Crick and Koch (1995) have attempted to deduce some features of this functional anatomy by postulating that visual awareness imposes a dual requirement: (1) an "explicit" neuronal representation of visual information and (2) a representation that is directly available to the "planning stages" of the brain. The distinction between explicit and implicit neuronal representations is best understood from an example: Illusory contours are known to elicit neuronal firing in visual area V2 (but not in striate cortex) (Peterhans and von der Heydt 1991). This activity explicitly represents the presence of illusory contours, whereas firing in the retina, lateral geniculate nucleus, and striate cortex represents such features merely in an implicit manner.

This dual requirement of an explicit representation that is available to the planning stages narrows the range of cortical areas that can contribute to visual awareness. The planning stages that initiate voluntary motor action are generally identified with frontal areas, and visual areas that project directly to frontal areas include all visual cortical areas with the notable exception of striate cortex (Felleman and Van Essen 1990). At least, this is the case in the macaque monkey. Ex-
trapolated to humans, this implies that visual information that is explicitly represented only in striate cortex (but not in extrastriate cortex) will not be available to visual awareness (Crick and Koch 1995). This does not in any way question, of course, that striate cortex is crucial for vision and visual awareness (just like the retina). It simply means that an explicit representation in striate cortex alone is not sufficient for visual awareness.

Drawing on a series of provocative findings, Milner and Goodale have proposed that different extrastriate visual areas contribute differentially to visual awareness (Milner and Goodale 1995). Specifically, they hypothesize that the ventral extrastriate areas which form the occipito-temporal pathway (areas V2, V3, V4, TEO, and inferotemporal cortex; “what” or “object” pathway) are far more consequential for subjective visual experience than the dorsal extrastriate areas of the occipito-parietal pathway (areas V3a, MT, MST, FST, STP, and posterior parietal cortex; “where,” “spatial,” or “action” pathway) (Ungerleider and Haxby 1994).

Evidence for this hypothesis comes from patients with lesions in striate cortex (discussed below) and especially from a patient with ventral occipital damage resulting from carbon monoxide poisoning (Goodale et al. 1991). Although this patient exhibits no sign of being aware of visual shapes and orientations, she nevertheless performs visually guided movements such as placing her flat hand into a narrow slot of varying orientation. Thus, information about visual orientation seems to be almost normally represented in the occipito-parietal pathway without contributing subjective visual experience. Another observation supporting the hypothesis of Milner and Goodale is that the familiar effects of size constancy (e.g., Titchner illusion) do not bias visually guided motor behavior in normal subjects, suggesting that size is processed independently in the two pathways and that only the size represented in the occipito-temporal pathway reaches subjective awareness (Milner and Goodale 1995).

SINGLE-CELL RECORDING AND VISUAL AWARENESS

Much of our knowledge about the way visual information is represented in the brain derives from single-cell recordings in cat and monkey (Fe1lerman and Van Essen 1990; Van Essen and Gallant 1994). Unfortunately, whether the information encoded by a particular cell or cell population is actually used by the organism—i.e., whether the cell or population exercises some discernible influence on behavior—is rarely obvious (Britten et al. 1992; Salzman and Newsome 1994). Determining whether a given cell or population contributes not only to behavior, but also to subjective visual experience poses an even greater challenge.

An ideal paradigm to probe the neuronal correlate of visual awareness with single-cell recording is binocular rivalry (Myerson et al. 1981; Sengpiel et al. 1995). Here, constant visual stimulation (e.g., a horizontal grating viewed by the left eye and a vertical grating viewed by the right eye) results in a discontinuously changing visual percept (horizontal and vertical gratings alternating on a time scale of a few seconds). Assuming that one can train the animal to promptly and truthfully report its subjective experience, one may thus ask where in the visual system there are single cells whose activity correlates with the alternating percept rather than the constant visual stimulation.

Earlier experiments with awake macaques viewing rivalrous stimulus motion indicated that up to 40% of the neurons in area MT show either a positive or negative correlation with the direction of motion reported by the animal (Logothetis and Schall 1989). Logothetis and colleagues extended these observations to rivalrous stimulus orientation and found 38% of neurons in area V4 and 18% of neurons in area V1/V2 were significantly modulated by the changing percept, with some neurons being strongly correlated and others strongly anti-correlated with the reported orientation (Leopold and Logothetis 1996). Their results leave open the possibility that the percentage of neurons modulated in this way is even smaller in striate cortex (area V1) alone. The vast majority of the cells whose activity changes with the percept are driven binocularly, rather than monocularly, arguing against the widespread notion (see, e.g., Sengpiel et al. 1995) that binocular rivalry results from the mutual inhibition of left- and right-eye-dominated cells (Logothetis et al. 1996). The most exciting aspect of these results in the present context is that subjective experience appears to correlate far better with activity in extrastriate cortex (area MT, area V4) than with activity in striate cortex (area V1).

Another compelling, if slightly less “clean,” situation in which subjective experience can be correlated with neuronal activity has been discovered by Andersen and colleagues (Qian and Andersen 1994; Qian et al. 1994a,b). Working with visual textures composed of two sets of dots moving in opposite directions, these researchers showed that the percept of two transparent, moving surfaces known as “motion transparency” (Adelson and Movshon 1982) is replaced by a percept of incoherent flicker when each dot moving in one direction is “paired” with another dot moving in the other direction. This perceptual breakdown of transparency occurs only when paired dots are in close spatial proximity (less than 0.1° of visual angle). Recording from single-direction-selective cells in areas V1 and MT, they further showed that the pairing of dots moving in opposite directions reduces neuronal responses in area MT far more than responses in area V1, suggesting that responses in area MT, not area V1, are responsible for the percept of motion transparency.
Thus, it again appears that subjective experience correlates better with extrastriate than with striate activity. Yet another instance may be provided by perceptual color constancy, which also seems to correlate better with neuronal responses in area V4 than in area V1 (Zeki 1983; Schein and Desimone 1990; Heywood et al. 1995).

DAMAGE TO STRIATE CORTEX: BLINDSIGHT

Patients who have suffered damage to striate cortex generally believe themselves to be “blind” in all parts of the field of view affected by the lesion. Despite their extremely impoverished subjective experience, visual stimulation of the blind field may nevertheless have a strong influence on the response of such patients in forced-choice situations. This ability to discriminate stimuli without being aware of them has been termed “blindsight” (Pöppel et al. 1973; Weiskrantz et al. 1974). Although it has been suggested that some of these patients may retain islands of intact striate cortex (Fendrich et al. 1994), other patients clearly exhibit blindsight in parts of the field of view no longer represented by striate cortex (Weiskrantz 1986; Cowey and Stoerig 1991). Indeed, there is a strong case for blindsight even in macaque monkeys with complete (hemilateral) ablation of striate cortex (Cowey and Stoerig 1995).

The nature of subjective experience during blindsight may be somewhat variable. In some situations, there appears to be a complete lack of any subjective awareness of a stimulus (“I have no sense of anything. There is nothing at all. I am just guessing”; patient DB [Weiskrantz 1986]). In other situations, there seems to be a reduced form of awareness of a nonvisual character (“I did not see, but I was sure”; patient DB [Weiskrantz 1986]; a “content-less kind of awareness, a feeling of something happening, albeit not normal seeing”; patient GY [Weiskrantz et al. 1995]). The latter state tends to be elicited by more intense stimulation (higher luminance contrast or higher speed of motion), but it does not seem to be comparable to the normal visual awareness that characterizes healthy parts of the visual field (Weiskrantz et al. 1995). This is an important point, because it suggests that blindsight is mediated by visual pathways other than conscious vision.

The retention of visual function after ablation of striate cortex is not as paradoxical as it might appear (Fig. 1a). Although a striate lesion triggers massive retrograde degeneration in the lateral geniculate nucleus and in the retina, the retinal degeneration is incomplete, and approximately 20% of retinal ganglion cells remain functional (Cowey and Stoerig 1993; Stoerig and Cowey 1993). In addition, a striate lesion does not completely deafferentate extrastriate visual areas: in non-human primates, a striate lesion abolishes visual responsiveness in areas V2, V3, V4, and inferior temporal cortex, but not in areas V3a, MT, and the superior temporal polysensory area (STP) (for review, see Bullier et al. 1994). For example, lesion or inactivation of striate cortex considerably weakens responses in area MT, but leaves selectivity for direction of motion and receptive field size unchanged (Rodman et al. 1989, 1990; Girard et al. 1992). A strong indication that the same is true for humans comes from blindsight patient GY, whose striate cortex and optic radiation were almost completely destroyed hemilaterally, but in whom visual responsiveness of area MT can be discerned with positron emission tomography (PET) (Barbur et al. 1993).

Visual information can bypass striate cortex along a number of known anatomical pathways (Stoerig and Cowey 1993; Bullier et al. 1994). The largest alternative route leads from the retina to the superior colliculus and onward to the lateral and inferior pulvinar nucleus of the thalamus (Fig. 1a). From the pulvinar, information is widely distributed to different striate and extrastriate areas. Another, rather minor, pathway projects directly from the interlaminar and S layers of the lateral geniculate to extrastriate cortex, including inferior temporal cortex (Hernandez-Gonzalez et al. 1994). The existence of tecto-extrastriate pathways bypassing striate cortex finds further support in the fact that a combined lesion of striate cortex and superior colliculus abolishes all visual responsiveness in area MT (Rodman et al. 1990). When only superior colliculus is lesioned, response properties in area MT remain largely unchanged. Behavioral studies with nonhuman primates confirm the superadditive effects of individual and combined lesions of striate cortex and superior colliculus (Mohler and Wurtz 1976).

The importance of blindsight lies in the fact that an anatomically defined lesion impairs visual awareness while sparing a significant degree of visual function. Unfortunately, more specific inferences are complicated by the fact that a striate lesion adversely affects large parts of the visual system outside striate cortex (either by deafferentation or by triggering retrograde degeneration). Thus, we may put down the disruption of visual awareness either to the striate lesion itself or to the effective deafferentation of ventral extrastriate areas. The fact that some visual function is retained can be attributed to the continued visual responsiveness of subcortical nuclei and/or of dorsal extrastriate areas. Thus, as argued most prominently by Milner and Goodale (1995), the functional anatomy of blindsight suggests that dorsal and ventral extrastriate areas may contribute differentially to visual awareness. In any case, the question that remains is not why patients without striate cortex retain some faculty of “sight,” but why they experience themselves as “blind.”

DAMAGE TO EXTRASTRIATE CORTEX

A number of recent cases of “cortical blindness” bear upon the role of extrastriate cortex in visual awareness. One of these concerns two patients with
profundely impaired vision (due to astrocytoma) in the lower quadrant of the visual field (Horton and Hoyt 1991). The remarkable feature of this "quadrant-anopia" is that the defective region ends precisely at the horizontal midline. This, in combination with magnetic-resonance imaging of the occipital lobe, led the authors to argue that the lesion is restricted to areas V2 and V3 (in which the respective representations of upper and lower quadrants are anatomically separate), rather than in striate cortex (where upper and lower quadrant representations are anatomically contiguous). Unless damage to white matter complicates the issue (Pollen 1995), this implies that the inexactness of areas V2/V3, rather than that of striate cortex, is critical for normal visual awareness.

Another case of interest is patient PB who, having suffered anoxia, combines profoundly impaired form perception, to the point of being unable to distinguish vertical from horizontal, with relatively preserved color perception (Humphrey et al. 1991, 1995). Remarkably, after adaptation to a grating of alternating red and green stripes, this patient exhibits the orientation-specific McCollough effect, demonstrating that orientation information continues to be processed, presumably in the intact striate cortex. If the presumed sparing of striate cortex can be confirmed, this patient will constitute another instance where visual awareness is impaired by damage to extrastriate rather than to striate cortex.

**Figure 1.** Expected activity distribution for different visual cortical areas during two types of "blindsight" (highly schematic and based on maps of macaque visual cortex of Van Essen and Gallant [1994]). (a) When striate cortex (area V1) is ablated or cooled in a monkey, responses are absent in V2, V3v, V3d, V4, and IT (white), while (reduced) responsiveness remains in V3a, MT, and STP (black). In patients with blindsight, a lesion in striate cortex triggers neuronal degeneration in the lateral geniculate nucleus (dashed) and retina, but leaves the superior colliculus, the pulvinar, and several extrastriate cortical areas visually responsive. The responsive areas of cortex form part of the occipito-parietal pathway (areas V3a, MT, STP, and PP) (Bullier et al. 1994; Milner and Goodale 1995). (b) Hypothetical activity distribution for conflicting visual displays that induce blindsight in normal observers (Kolb and Braun 1995). Due to opponency, the response to conflicting displays may be significantly reduced in many extrastriate areas (Qian et al. 1994a). Subcortical nuclei and parts of striate cortex are unaffected, possibly triggering extrastriate activity in the occipito-parietal pathway. (dLGN) Dorsal lateral geniculate nucleus; (SC) superior colliculus; (PUL) pulvinar nucleus of the thalamus; (V1) striate cortex; (V2, V3a, V3d, V3v, V4, MT) extrastriate visual cortical areas; (PP) posterior parietal cortex; (IT) inferior temporal cortex; (STP) superior temporal polysensory area. (Reprinted, with permission, from Koch and Braun 1996.)

**PSYCHOPHYSICAL MANIPULATIONS OF VISUAL AWARENESS**

In normal observers, deficits of visual awareness are necessarily fleeting and are thus difficult to document from an objective, third-person point of view (Merikle 1992; Shacter 1992). To overcome this problem, we need a behavioral measure of visual awareness that is at least as sensitive as the traditional behavioral measures of visual discrimination: Verbal debriefing of observers is not enough. Fortunately, there has been steady progress toward this goal in recent years, largely due to a technique involving a "comment key" (Weiskrantz et al. 1995). In this technique, the ob-
server is asked not only to make a forced choice between stimulus alternatives, but also to comment on his or her subjective experience in every trial. In its most elementary form, the only two comments allowed are whether the stimulus was "seen" or "not seen" (Graves and Jones 1992; Weiskrantz et al. 1995). This technique has even been used to demonstrate blindsight in nonhuman primates, challenging the notion that the inner life of other creatures must remain forever closed to us (Cowey and Stoerig 1995). A refinement of the comment-key technique involves giving observers a greater choice of comments and, more importantly, correlating comments with the success or failure of individual trials (Kolb and Braun 1995). This last measure establishes whether subjective experience reflects objective performance in a forced-choice task. Of course, this is almost always the case. In a hyperacuity discrimination, for example, subjective experience closely follows objective performance even in the threshold regime, where performance is barely above chance (Fahle and Koch 1996). However, in certain exceptional situations, subjective confidence becomes decoupled from objective performance, and the positive correlation between the two is lost, although objective performance itself remains quite high (Kolb and Braun 1995). In these situations, observers report that they do not see the stimulus and that their forced-choice response is based on guessing.

This "blindsight in normal observers" is of interest in the present context because of the nature of the displays in question. These are ambiguous displays composed of conflicting stimuli in close spatial proximity, and single-unit results from nonhuman primates suggest that such displays stimulate extrastriate visual cortex much less than striate cortex (see below). The deficits of subjective experience observed in such displays thus bear directly on the functional anatomy of awareness.

One example of a situation involving conflicting information is dichoptic stimulation of the two eyes. In the display shown schematically in Figure 2a, each eye views an array of oriented stimuli, and in a small target region these stimuli are oriented orthogonally to the rest of the array. However, at each visual location, left and right eyes view stimuli of both orientations, so that neither orientation is clearly perceived. At viewing times of less than about 0.3 seconds, the array is reported to be subjectively uniform without any impression of a target region (at longer viewing times, "binocular rivalry" develops and the target region is perceived by the currently dominant eye). A similar failure to become aware of a target region can be obtained when conflicting information is delivered in other ways; for example, by a (binocularly viewed) dynamic texture of two sets of dots moving in opposite directions.

The surprising finding with these displays is that, although observers claim not to see the target and to guess its location, they report target position quite reliably in a forced-choice situation. To document lack of target awareness, observers comment on their subjective experience by rating their subjective confidence of having correctly located the target on a scale of 1 to 10. For the conflicting displays described above, no significant correlation between subjective confidence and objective performance is obtained, suggesting a complete lack of target awareness (Fig. 2c). In a control situation in which both eyes view consistent stimuli (Fig. 2b), a large positive correlation is observed, indicating normal target awareness (Fig. 2d). Importantly, the contrast between conflicting and consistent displays is not limited to the threshold regime but persists over the entire performance range. This can be demonstrated by varying either presentation time or luminance contrast. In short, consistent displays yield significant correlations even when performance approaches chance, and conflicting displays yield insignificant correlations even when performance approaches ceiling (Kolb and Braun 1995).

It seems clear that the neural response to conflicting displays is to a large extent shaped by "opponent mechanisms," that is, by reciprocal inhibition between conflicting visual features. An important indication as to the neural level of this opponency is that conflicting features must enter into close spatial proximity in order for visual awareness to be suppressed. In fact, the spatial separation of conflicting features must be comparable to a typical receptive field diameter in striate cortex (Qian and Andersen 1994; Kolb and Braun 1995; Braun and Kolb 1996). This is borne out by the single-unit studies with nonhuman primates described earlier. As mentioned previously, these studies suggest that conflicting orientation (viewed dichoptically) reduces orientation-selective responses far more in extrastriate area V4 than in striate cortex (Leopold and Logothetis 1996) and that conflicting motion (viewed binocularly) reduces direction-selective responses far more in extrastriate area MT than in striate cortex (Qian et al. 1994a). Overall, these single-unit results suggest that, at least to a first approximation, conflicting displays elicit a nearly normal response from striate cortex but a relatively poor response from extrastriate areas.

If these inferences are correct, then conflicting displays may create a situation where the major remaining inputs to extrastriate areas derive not from striate cortex but from subcortical nuclei (Fig. 1b), not at all unlike the situation after a striate cortex lesion! Of course, this assumes that subcortical responses are not attenuated by opponency. In contrast to the lesion, however, conflicting displays would still be processed by striate cortex, and responses in striate cortex could continue to reach extrastriate cortical areas after being relayed through subcortical areas. This would constitute strong evidence that a neuronal response in striate cortex is not sufficient for visual awareness. Whether or not this scenario corresponds to fact remains to be seen. Nevertheless, we find it intriguing...
that similar pathways may mediate blindsight in patients with striate damage and in normal observers viewing conflicting displays.

Additional psychophysical evidence that a striate response is insufficient for awareness comes from experiments on the perception of high-frequency gratings (He et al. 1995). Although the orientation of such gratings is not subjectively apparent, they nevertheless induce an orientation-specific aftereffect. The high spatial frequencies involved (>50 cps) are such that only neurons with the smallest receptive fields in striate cortex are likely to respond, and neurons with larger receptive fields in extrastriate cortex remain silent. The contrast between the presumed striate response and the failure to become aware of grating orientation in this experiment is especially glaring.

Yet another instance in which a striate response is not sufficient to ensure visual awareness seems to be provided by visual “crowding” (He et al. 1996). When a single patch of a luminance grating appears on a computer screen (Fig. 3a), the grating is clearly visible and it induces an orientation-specific aftereffect; that is, it raises detection thresholds for any faint grating of similar orientation that may appear afterward. However, when additional gratings appear at either side, the original grating is “masked” and ceases to be visible as a distinct entity (Toet and Levi 1992). Subjectively, one still sees “something” at the location of the original grating but cannot discern its orientation, even when given unlimited viewing time. Yet the aftereffect induced by this “invisible” grating remains strong and orientation-specific (Fig. 3b). The conclusion that can be drawn from these observations, which is foreshadowed by earlier experiments (Blake and Fox 1974), is that visual awareness arises at higher levels of visual processing than orientation-specific adaptation. Since adaptation is thought to be mediated by orientation-selective neurons in striate cortex (Blakemore and Campbell 1969), this implies that visual awareness must arise at a later stage.

Of course, the weakness of purely psychophysical experiments is that the actual anatomical distribution of neuronal activity is unknown and must be inferred from other evidence. Fortunately, the precise mapping

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Figure 2. Psychophysical displays (schematic) and results pertaining to blindsight in normal observers. (a) Left and right eyes view conflicting displays consisting of oriented stimuli. In a small target region these stimuli are oriented orthogonally to the rest of the array. At each visual location, left and right eyes view stimuli of orthogonal orientations. As a result, the target region is not subjectively apparent. A fixation task (central C) ensures that both eyes fixate the same point of the display. Observers guess the location of the target region, choosing between four possible locations, and rate their confidence on a scale of 1 to 10. (b) Control situation in which left and right eyes view identical, consistent, displays. Here the target region is subjectively seen. (c) Results from three observers for conflicting displays (Kolb and Braun 1995). Performance is plotted separately for trials with different confidence ratings (e.g., when subjects give the lowest possible confidence rating of 1, performance still is near 65%, far above the chance level of 25%). No correlation between confidence and performance is observed, indicating complete lack of visual awareness of the target region (roughly flat bar graph). The thick line represents the frequency of different confidence ratings, the arrow being the distribution mean. (d) Results from the same observers for consistent displays. Confidence and performance are strongly correlated, indicating normal visual awareness of the target region (rising bar graph). (Reprinted, with permission, from Koch and Braun 1996.)
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Figure 3. Psychophysical displays (schematic) and results pertaining to adaptation by crowded grating patches. (a) Adaptation followed by contrast threshold measurement for a single grating (left) and a crowded grating (right). In each trial, the orientation of the adapting grating was either the same as or different from the orientation of the test grating. Observers fixated at a distance of approximately 26° from the adapting and test gratings. (b) Threshold contrast elevation after adaptation relative to baseline threshold contrast before adaptation. Data are averaged across four subjects. The difference between same and different adapt-test orientations reflects the orientation-selective aftereffect of the adapting grating. The data show that this aftereffect is comparable for a crowded grating (whose orientation is not perceived) and for a single grating (whose orientation is readily perceived). (Reprinted, with permission, from He et al. 1996 [copyright Macmillan].)

of the human visual cortical areas with functional magnetic resonance imaging (Sereno et al. 1995) opens up the very real possibility of repeating these psychophysical experiments while the observer's visual cortex is being scanned.

Experiments on visual imagery are sometimes cited (we think misleadingly) in support of a striate contribution to visual awareness. A recent brain imaging study (Kosslyn et al. 1995) has resolved the debate whether visual imagery can lead to an activation of striate cortex (Roland and Gulyas 1994). Using PET, Kosslyn and colleagues showed that visual imagery can activate visual cortical areas as early as striate cortex (Kosslyn et al. 1995). However, this finding does not address the question of whether V1 activity expresses the phenomenological aspects of visual imagery (indeed, V1 activation may simply be necessary for activity in extrastriate visual cortical areas that would give rise to the phenomenal aspects).

CONCLUSION

The picture that emerges is that neurons in striate cortex, although necessary for almost all forms of phenomenal vision (as are retinal ganglion cells), do not directly contribute toward the neuronal correlate of conscious visual experience. Whether or not this has anything to do with the connectivity of area V1, especially its lack of a direct projection to frontal lobe (Crick and Koch 1995), remains an open question. Of course, it would be difficult to establish that none of the neuronal activity in area V1 correlates with the current contents of visual awareness. However, the evidence reviewed here uniformly points to the conclusion that visual awareness correlates far better with extrastriate than with striate neuronal activity. An even more specific hypothesis—that visual awareness correlates with activity in areas of the occipito-temporal rather than the occipito-parietal pathway—is not as well supported at present, but may well be confirmed by future evidence.

Further progress in understanding visual (and indeed all forms of) awareness can only come from precisely localizing the “neural correlate of consciousness” to specific brain areas and neuronal populations. That activity correlated with the changing perception in binocular rivalry in area MT occurs mostly in the deeper layers of cortex (Leopold and Logothetis 1996) constitutes very encouraging news on this front. It is conceivable that the correlate of visual awareness is expressed by a subset of neurons with very specific biophysical, biochemical, and morphological properties, rather than by all neurons in a given cortical area. This raises the fascinating, if distant, prospect of molecular tools that specifically label and manipulate such neurons (Koch 1996). It is by studying the dynamics and projection patterns of these neurons that we will begin our final assault on the age-old puzzle of the relationship between mind and brain.

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