Feature Article
On the Neural Correlates of Visual Perception

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Neurological findings suggest that the human striate cortex (V1) is an indispensable component of a neural substratum subserving static achromatic form perception in its own right and not simply as a central distributor of retinally derived information to extrastriate visual areas. This view is further supported by physiological evidence in primates that the finest-grained conjoined representation of spatial detail and retinotopic localization that underlies phenomenal visual experience for local brightness discriminations is selectively represented at cortical levels by the activity of certain neurons in V1. However, at first glance, support for these ideas would appear to be undermined by incontrovertible neurological evidence (visual hemineglect and the simultanagnosias) and recent psychophysical results on 'crowding' that confirm that activation of neurons in V1 may, at times, be insufficient to generate a percept. Moreover, a recent proposal suggests that neural correlates of visual awareness must project directly to those in executive space, thus automatically excluding V1 from a related perceptual space because V1 lacks such direct projections. Both sets of concerns are, however, resolved within the context of adaptive resonance theories. Recursive loops, linking the dorsal lateral geniculate nucleus (LGN) through successive cortical visual areas to the temporal lobe by means of a series of ascending and descending pathways, provide a neuronal substratum at each level within a modular framework for mutually consistent descriptions of sensory data. At steady state, such networks obviate the necessity that neural correlates of visual experience project directly to those in executive space because a neural phenomenal perceptual space subserving form vision is continuously updated by information from an object recognition space equivalent to that destined to reach executive space. Within this framework, activity in V1 may engender percepts that accompany figure–ground segregations only when dynamic incongruities are resolved both within and between ascending and descending streams. Synchronous neuronal activity on a short timescale within and across cortical areas, proposed and sometimes observed as perceptual correlates, may also serve as a marker that a steady state has been achieved, which, in turn, may be a requirement for the longer time constants that accompany the emergence and stability of perceptual states compared to the faster dynamics of adapting networks and the still faster dynamics of individual action potentials. Finally, the same consensus of neuronal activity across ascending and descending pathways linking multiple cortical areas that in anatomic sequence subserve phenomenal visual experiences and object recognition may underlie the normal unity of conscious experience.

Introduction

It may now be possible to discern the beginnings of a unified framework to delimit the neural correlates of at least one aspect of conscious vision. Tentatively, it appears helpful to subdivide the entirety of conscious vision into at least four components. Perhaps the most basic is that of phenomenal visual experience or 'phenomenal consciousness' as defined by Block (1995). Phenomenal qualities such as the raw sensations of brightness and color are sometimes referred to as 'qualia'. The neural correlates of such phenomenal visual experience may be considered to comprise a *phenomenal perceptual space* (PPS). Within this space, object localization is retinotopic and thus relative to the direct line of sight (Holmes, 1945). The term *executive space* may usefully apply collectively to all neuronal regions that participate in the planning and execution of voluntary motor acts including expressive speech. Executive space as so defined also includes neuronal assemblies that subserve *conscious access* (Block, 1995) to and utilization of working memories of objects and their locations as expressed by neurons as far anterior as the prefrontal cortices (Wilson *et al.*, 1993; Fuster, 1997). These distinctions between a phenomenal perceptual space and executive space are further justified by cumulative neurological experience since the 1930s (Brickner, 1936) that phenomenal visual experience survives extensive bilateral ablation or diverse injuries to prefrontal and frontal cortical areas (Eslinger and Damasio, 1985).

Between perceptual space and executive space, it may be convenient to assume the existence of an *object recognition space* within which we can surmise the existence of neural representations that can uniquely specify an object and serve as *concepts* and subsequently as working memories for further analysis within executive space. Later on, we may enquire as to whether such representations of concepts within object recognition space are purely symbolic or should be included within a phenomenal perceptual space. Finally, *extra-personal spaces* (Grüsser and Landis, 1991), i.e. mappings of objects in or locations of the external world in head-centered (Andersen *et al.*, 1985) or body-centered spatiotopic coordinate systems have long been recognized. Moreover, 'allocentric' coordinate systems provide mappings of the world independent of our actual percepts and spatial position (Grüsser and Landis, 1991). These representations, largely initially mediated by specialized regions within the parietal lobe (Critchley, 1953), subserve absolute location of objects in space (Holmes, 1945; Galletti *et al.*, 1995), mental imagery of spatial mappings (Grüsser and Landis, 1991), abstract representations of space that can be used to guide movements (Andersen *et al.*, 1985, 1997; Milner and Goodale, 1995), spatially referent binding of color and motion (Friedman-Hill *et al.*, 1995) and selective attention (Milner and Goodale, 1995). Some of these representations may modify but do not give rise independently to phenomenal visual experience.

I shall largely confine the present analysis to phenomenal visual experience; even here further restrictions to simplify the problem are helpful. Thus, I shall focus largely on static achromatic visual experience, leaving aside the equally important subjects of the experience of color and motion. I recognize that attempting to formulate a tentative but coherent framework for consideration of the neural correlates of even one type of phenomenal visual experience is hazardous. Nevertheless, I

believe that we have reached a point based on the cumulative experience of clinical neurology, the basic neurosciences and advances in neural network theory over the past decade that a unified framework for consideration of such correlates is now possible and, at the very least, opens the way for improved models based on refutation or confirmation of some of the present set of proposals.

The Neurology of Human Phenomenal Visual Experience

Not long after Henschen (1893) established that the human primary visual receptive area corresponded to what we now call the striate cortex or V1, neurologists began to discover disorders of higher visual function from brain injuries beyond the striate cortex that nevertheless left basic visual experience intact. Before the middle of the 20th century, Holmes (1945) concluded that human primary visual perception, including discriminations based on brightness and color, was subserved by the striate cortex. Subsequent work proved that Holmes was mistaken in assigning color perception to the striate cortex. We know now that certain brain lesions beyond V1 and V2 can eliminate perception of color (the achromatopsias) (Damasio *et al.*, 1980; Sacks and Wasserman, 1987; Zeki, 1990; Grüsser and Landis, 1991) or certain classes of motion (the akinotopsias) (Zihl *et al.*, 1990) while leaving perception of spatial detail intact. Whether the striate cortex subserves *any* direct role in static achromatic form perception remains an open issue and one that will be addressed here.

Holmes and his contemporaries knew that humans experiencing *alexia* (Holmes, 1945; Grüsser and Landis, 1991) as a consequence of certain occipital lobe lesions may lose the ability to read and even identify letters even though they see well enough to copy them accurately. Similarly, patients experiencing *associative visual agnosias* (Rubens and Benson, 1971; Damasio *et al.*, 1982; Grüsser and Landis, 1991) as a consequence of lesions often at the occipitotemporal junction may lose the ability to identify complex objects even when they can copy them accurately and their language ability has remained intact. Moreover, there remains longstanding evidence in both primates (Gross, 1976) and humans (Damasio, 1990) that bilateral ablation or injury to inferotemporal cortex impairs certain visual discriminations and some higher-level recognitions but leaves phenomenal visual experience essentially intact. Similarly, as noted above, phenomenal visual experience survives extensive or diverse injuries to prefrontal cortical areas, though no single reported case has had a complete bilateral ablation of all of prefrontal cortex.

Although neurons in MT/V5 may process certain classes of motion (Barbur *et al.*, 1993), integrate depth and motion cues (Bradley *et al.*, 1995) and derive three-dimensional structure from motion (Bradley *et al.*, 1998), other cells in dorsal MST are involved in the analysis of optic flow (for review, see Andersen *et al.*, 1996). I leave these motion-induced experiences largely aside in order to focus on static achromatic form vision which has long been considered a function of the occipitotemporal or 'ventral stream' (Mishkin *et al.*, 1983). Similarly, I will discuss only briefly the dorsal pathways within the parietal lobe that establishes multiple distinct spatial reference frames in parietal cortex that provide spatial representations for the guidance of selective actions with respect to the localization of visual targets (for review, see Colby, 1998). Within the ventral stream, V1 projects principally to V2 and thence predominantly to V4 with subsequent projections from V4 directly and through TEO to the multiple inferotemporal cortical areas (IT) within the lateral temporal lobe (Felleman and Van Essen, 1991) (see Fig. 1).

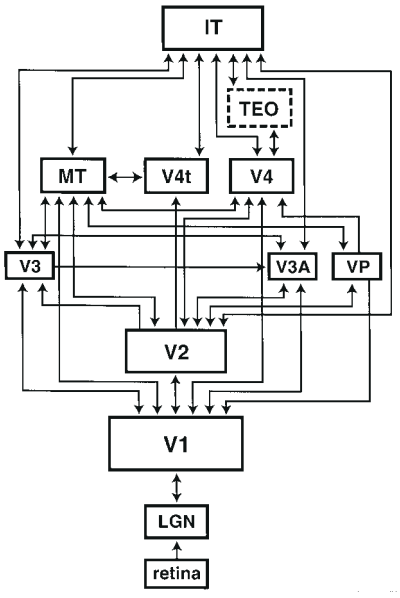

Figure 1. Simplified schematic of most currently known feedforward, feedback and cross-connections in the ventral loop as modified from the summary diagrams of Zilles and Clarke (1998, Figure 7), which, in turn, were based on review of over 50 studies. TEO is represented by broken lines because it is not considered in the above-cited summary diagrams. Not all feedback connections from IT and TEO are shown. For projections of anterior extrastriate areas to superior temporal, parietal and frontal cortices, see Zilles and Clarke (1998).

Restricted lesions within V4 in primates (Schiller, 1993; Merigan, 1996; Merigan and Pham, 1998) or in its human homologue (Rizzo *et al.*, 1992) produce relatively mild deficits for elemental visual stimuli presented in isolation although shape discriminations requiring the use of multiple cues are profoundly disrupted (Merigan and Pham, 1998). These latter results are consistent with earlier work that removal of V4 conspicuously impairs discrimination of form and pattern without impairing achromatic intensity thresholds (Heywood and Cowey, 1987). Despite these disruptions and the resultant impoverishment of the visual world necessary for complex two- and three-dimensional shape discrimination that requires comparison of information over different parts of the field, the perceptual experiences that psychophysicists test using isolated single patches of sinusoidal gratings apparently survives ablation of V4. Thus, such perceptual experiences are engendered either prior to V4 or pathways that bypass V4 are involved.

Visual field defects for static achromatic stimuli in humans invariably occur after injury to the corresponding retinotopic representation within contralateral V1 (Holmes, 1945). Field defects that strictly respect the horizontal meridian may occur after lesions of V2 that extend across the V2/V3 border (Horton and Hoyt, 1991; McFadzean and Hadley, 1997) which marks that meridian as well as after lesions within V1 (McFadzean and Hadley, 1997) (see Fig. 2). However, isolated lesions within V3 alone, sparing V2, or in *any* cortical area beyond V3 have not been reported to produce such field defects.

Based on the foregoing neurological results, V2 may seem to have an equal claim to that of V1 for subserving luminance-based visual experience. Indeed, although macaques with ablations of V1 are unable to detect sine-wave gratings above 12 c/d, they can detect the lower spatial frequencies, albeit only at very high

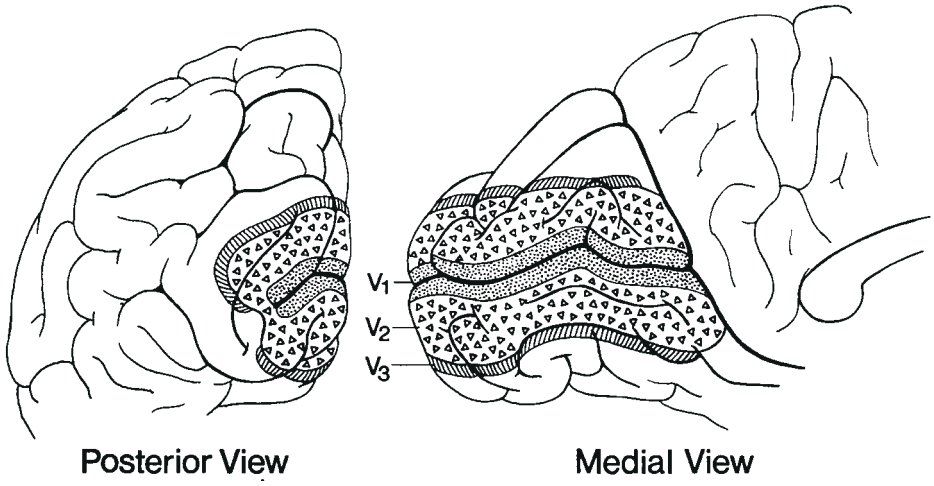

Figure 2. Schematic diagram from Horton and Hoyt (1991) (courtesy of Jonathan Horton) showing arrangement of V1, V2 and V3 along the medial and posterior occipital surface. The border between V1 and V2 marks the *vertical* meridian. However, the borders between V2 and V3 mark a split along the representation of the *horizontal* meridian into separate dorsal and ventral halves yielding spatially separated quandrantic representations of the upper and lower contralateral visual fields.

contrasts (Miller *et al.*, 1980). Even so, excitotoxic lesions of V2 (Merigan *et al.*, 1993) in macaques, which destroy neurons within V2 but leave connections from V1 to and from higher cortical areas intact, caused no change in visual acuity and little or no change in contrast sensitivity. The monkeys with lesions in V2 discriminated whether the test patch of a sinusoidal grating was horizontal or vertical by processing either a left or a right push button.

It might be argued that Merigan *et al.* (1993) did not exclude 'blindsight', i.e. 'visual capacity in the absence of acknowledged awareness' (Weiskrantz, 1995), as an explanation for their results because they did not also employ the paradigm of Cowey and Stoerig (1995), who trained monkeys to discriminate between real world events and no-stimulus blanks, thus permitting these authors to determine whether the monkeys perceived stimuli or treated them as blanks. However, Merigan *et al.* (1993) placed lesions in V1 for comparison and these devastated vision. Moreover, the conditions for eliciting 'blindsight' and the attendant stimulus parameters may be rather stringent (Weiskrantz, 1995). Thus, it is much more likely, although not entirely certain, that the macaques with lesions in V2 as studied by Merigan *et al.* (1993) actually perceived the test stimuli. However, tasks involving complex spatial discriminations were impaired after lesions of V2.

Lesions within either V2 or V4 do, however, interfere with the ability of an animal to distinguish test stimuli, especially weak stimuli (Schiller, 1993), embedded in a dense array of 'competing' stimuli. Analogous results have been found in humans with lesions in extrastriate areas (Rizzo and Robin, 1990). Thus, even though extrastriate cortices beyond V2 remain essential for the discrimination of complex patterns, the most elemental phenomenal experience and isolated visual discriminations based on brightness discriminations for stimuli of medium and fine-grained detail in higher primates as well as humans seem to depend on the structural integrity of the striate cortex as long as this area is not isolated from the rest of the cerebral cortex (Bodis-Wollner *et al.*, 1977).

The lateral geniculate nucleus (LGN), however, does not appear to be essential for phenomenal visual experience. For example, elemental visual experiences of punctate white or colored lights called 'phosphenes' can be evoked in man by direct electrical stimulation of densely hemianopic striate cortex after severance of its connections to and from the LGN (Brindley and Lewin, 1968; Dobelle and Miladejovsky, 1974). Even so, these results do not necessarily exclude the LGN as a substratum for visual experience – as opposed to simply a conveyor of information from retina to visual cortex – under normal conditions. However, if we accept – as I do – the premise of Crick and Koch (1995a) that the brain must construct an *explicit* representation of any particular visual feature as a *necessary* condition before that feature can be perceived, then it appears less likely that the LGN is directly involved in visual experience. For example, the 'narrow-band' representations for orientation and spatial frequency in the luminance domain that match the 'channels' revealed psychophysically by adaptation studies (Blakemore and Campbell, 1968) are not found in the macaque LGN (Derrington and Lennie, 1984) but are achieved at the level of the striate cortex. Moreover, explicit representations for color are computed well beyond the LGN and even beyond V1 (Damasio *et al.*, 1980; Zeki, 1990) in an area in the fusiform gyrus variously identified as V4 (McKeefry and Zeki, 1997) or V8 (Hadjikhani *et al.*, 1998). See also Zeki *et al.* (1998) and Tootell and Hadjikhani (1998). Thus, there is no obvious evidence for the construction of explicit representations for either form or color vision within the LGN.

However, evidence that V1 is indispensable for at least certain types of visual experience, apart from its role as a central distributor of retinally derived information to extrastriate cortices, derives from results that static achromatic visual experience and luminance-based form discrimination have remained intact despite a multiplicity of diverse lesions to extrastriate, parietal, temporal and frontal cortical areas, and the fact that the LGN is not essential for humans to experience phosphenes. This interpretation of the neurological and behavioral literature is essentially in agreement with the views of Damasio (1989) and Stoerig and Cowey (1995, 1997). Admittedly, such lesions beyond V1 and V2 are often incomplete and/or unilateral, but a finding of a visual field cut as a consequence of any cortical lesion more anterior than that studied by Horton and Hoyt (1991) would have been such an extraordinary finding that it could scarcely have escaped notice in the neurological literature. Moreover, for Stoerig and Cowey, the significance of 'blindsight' in humans and monkeys after unilateral striate cortex injury or ablation is not simply that such

subjects possess some evidence of residual visual processing in the form of pointing to a stimulus above chance level, but that phenomenal vision is absent. Thus, for these authors, the existence of blindsight provides further evidence that the striate cortex is indispensable for at least certain aspects of phenomenal vision. Physiological studies provide further support for these conclusions. However, the striate cortex does not appear to be indispensable for the phenomenal experience of certain types of motion (Blythe *et al.*, 1987; Ceccaldi *et al.*, 1992; Barbur *et al.*, 1993; Zeki and ffytche, 1998).

The 'Grain' Problem

We simultaneously perceive the finest detail and retinotopic localization of visual signals in the frontoparallel plane with great precision. That such experiences are conjoined is not trivial because vast territories of the cortical mantle are involved in the disparate tasks of identifying individual objects independently of size, position and location (Lashley, 1942), whereas other regions are dedicated to localizing objects in various coordinate frames independently of object identity (Grüsser and Landis, 1991).

The conjoined optimal localization of signals in both the two-dimensional spatial and spatial frequency domains (Daugman, 1985) is best expressed by sets of phase-specific simple cells in V1 (Pollen and Ronner, 198l; Foster *et al.*, 1983). The subzones of the receptive fields of these cells are selectively sensitive to either increments or decrements of light (Hubel and Wiesel, 1962) and spatial processing across such receptive fields is largely linear (Jacobson *et al.*, 1993). The two-dimensional joint optimalization for preferred orientation and spatial frequency in the frequency domain and for the x and y coordinates in the spatial domain follows from results that the largely linear receptive field line-weighting functions of these cells are well-described as Gaussian-attenuated sinusoids and cosinusoids (Marcelja, 1980). The Gaussian weighting renders the signal as the most compact to specify jointly spatial frequency and space (Gabor, 1946). The Fourier transform of these 'Gabor functions' in the space domain yields an equally compact function in the spatial frequency domain (Gabor, 1946; Marcelja, 1980). The products of uncertainties within the two domains approaches a theoretical minimum (Marcelja, 1980). Simple cells with corresponding properties, at least for analyses of brightness distributions within frontoparallel planes, are found within both V1 and V2 (Foster *et al.*, 1985), but not within V3A (Gaska *et al.*, 1988) nor apparently in V4 (Desimone and Schein, 1987).

Thus, neuronal ensembles that subserve both fine spatial detail and spatial position together within the same cortical areas appear to be localized to V1 and V2. Both cortices also abound in complex cells, i.e. neurons that are insensitive to local sign for luminance (Hubel and Wiesel, 1962), responding to either increments or decrements of light. The response properties of such neurons, which follow second-order statistics in V1 (Gaska, *et al.*, 1994), seem unsuited to convey precise information about the direction of brightness changes, and seem not to permit these neurons to interact linearly with other neurons across a cortical area to compute precise retinotopically specified brightness discriminations. Thus, the simple cells of V1 and V2 are more apt to subserve phenomenal vision for luminance discriminations than neurons that are non-selective to local sign.

There are also other neurons in V1, the non-orientation-selective 'blob' cells (Livingstone and Hubel, 1982), that are selective to local sign. However, these lack the orientation and spatial frequency selectivity required for humans to distinguish square-wave from sine-wave gratings only when the contrast of the third harmonic of the square-wave grating has reached its own independent threshold (Campbell and Robson, 1968).

Thus, based solely on the above discussion, the simple cells of V1 and V2 might lay equal claim for a privileged role in phenomenal vision based upon luminance discriminations. There are, however, certain differences in the properties of simple cells in the two cortical areas. Spatial frequencies of neurons in V1 are higher than those in V2 at the same retinal eccentricity by at least an octave (Foster *et al.*, 1985; Levitt *et al.*, 1994). Thus, the finest *conjoined* representation for both spatial detail and retinotopic localization appears to be subserved by sets of simple cells within but not beyond V1. Moreover, the spatiotemporal pattern of the most efficient human contrast detector corresponding to 'what the eye sees best' well approximates the receptive field profiles of simple cells in V1 (Watson *et al.*, 1983). This result strengthens the case for identifying the ensemble of phase-specific simple cells within V1 as part of an explicit representation for the detection and perception of localized achromatic stimuli.

Conversely, conjoined explicit representations for fine detail over two-dimensional space and spatial frequency are partially *decoupled* during recoding beyond V1 and V2 and irrevocably so beyond V4 such that spatial information for *object localization* in specific coordinate systems projects largely to the parietal lobe, whereas two-dimensional spatial frequency data for *object recognition* projects largely to the temporal lobe (Mishkin *et al.*, 1983). For example, posterior parietal neurons, apart from showing some limited spatial summation properties with respect to target size and luminance, are remarkable for their lack of specificity for object shape (Robinson *et al.*, 1978). Some such cells encode locations dependent upon eye position in head-centered (Andersen *et al.*, 1985) or other (Colby, 1998) coordinate spaces. There are neurons within area PO (V6) that can encode the absolute or 'real position' of an attended object independent of direction of gaze (Galletti *et al.*, 1996), and such neurons may provide motor areas with visuospatial information required for arm-reaching movements with respect to the location of a specific target. This computation does not appear to be confounded by the shape or intrinsic detail of the attended object. However, some sensitivity for simple two-dimensional geometric shape has been reported for neurons with typically large receptive fields in the lateral intraparietal cortex (Sereno and Maunsell, 1998) presumably serving to facilitate the manipulation and grasping of objects (Logothetis, 1998).

Moreover, the enormously large receptive fields of inferotemporal neurons do not appear to undertake an analysis of fine spatial position in addition to that of object recognition, though there are three important qualifications. First, such neurons show increased sensitivity over foveal and parafoveal regions (Gross, 1976), and selective attention within a spatial window can differentially enhance signal to noise within the aperture of interest compared to that within ignored regions (Moran and Desimone, 1985). Moreover, a small percentage of inferotemporal neurons show some sensitivity for encoding both object size and retinal location (Lueschow *et al.*, 1994). However, none of these mechanisms provide more than coarse localization for individual objects as opposed to the fine spatial representation across the entire visual panorama subserved by neurons in V1 and V2.

There are cross-connections between dorsal and ventral streams (Merigan and Maunsell, 1993), and perhaps they

coarsely bind identification and localization of attended objects. Even so, the cross-connections cannot restore information if it has been lost pursuant to generalization processes within the two streams. For example, suppose that those neurons in V6 that encode the 'real position' of an object (Galletti *et al.*, 1995) could somehow convey such information to IT by cross-connections. Even if possible, such cells would likely be conveying only the central coordinates of the target but not that of its fine structure nor that of the panorama of the associated visual scene. Thus, at the cortical level, if we are to look for neurons that conjointly specify both fine spatial detail and precise retinotopic localization we must look to the simple cells in V1 and V2, and for those neurons that subserve the finest-grain conjoined representations we must, based on present information, look exclusively to the simple cells in V1.

Other evidence supports the role for these early cortices in phenomenal vision. For example, the representation of surfaces (Nakayama and Shimojo, 1992), which also requires specification of both fine spatial detail and spatial position, can be instantiated prior to the evocation of selective attention (Mattingley *et al.*, 1997), likely placing such implementation prior to stages of object recognition, and thus at least in part within early visual cortical areas such as V1 and V2. However, although the case for a primary role of neurons in V1 in phenomenal visual experience appears quite strong based on both neurological and physiological results, there remains equally strong evidence that activation of neurons in V1 may not be sufficient to activate a visual percept.

Afferent Activation of Neurons in V1 May Not Generate a Visual Experience

Many patients with structural damage to the right parietal lobe fail to attend to complex visual stimuli in the left hemifield even when tests with individual stimuli show that visual fields are intact and that such patients are not hemianopic (Critchley, 1953). Other such patients may identify a test object in the left hemifield when it is presented in isolation but not when a competing stimulus is simultaneously shown within the right hemifield (Critchley, 1953).

Equally informative are case presentations of the *simul tanagnosias* (Critchley, 1953; Rizzo and Robin, 1990), wherein patients with lesions of extrastriate cortices may at any one instant see only fragmentary components of the visual field. Luria (1959) described a patient who could perceive a 3 × 2 array of points when asked to search for a rectangle but could experience only a single point when asked to count the dots. Rizzo and Robin (1990) explain simultanagnosia as the inability to sustain visuospatial attention simultaneously across all the elements in an array. Their clinical experiences are matched by behavioral studies in primates showing that animals with V1 intact but with lesions within V4 may identify individual stimuli very well but fail to make correct identifications when a particular stimulus is embedded within a complex array of competing stimuli (Schiller, 1993; Merigan, 1996). In these cases, there is every reason to suspect that the non-perceived visual stimuli have excited neurons within the striate cortex and that suppression has initially occurred at a higher level, although no direct test has yet been made. However, two recent psychophysical studies have provided incontrovertible evidence that neurons in V1 can be activated in the absence of a visual percept.

He *et al.* (1995) used laser interferometry to produce sinusoidal gratings of extremely high spatial frequency close to or just above the foveal resolution limit of 60 c/d (Campbell and Green, 1965). When they presented an interference pattern at a slightly higher spatial frequency of 67 c/d, the subjets could no longer perceive the grating, although they could detect an orientation-specific loss of sensitivity at 48 c/d, indicating that their non-perceived test stimulus had activated orientation-selective neurons in the primary visual cortex before its trace was 'subsequently obliterated by subsequent spatial filtering within the cortex' (S. He, personal communication, provided the specific test values).

In a second experiment, He *et al.* (1996) found that human observers can identify the orientation of a single small grating patch presented to the periphery of the superior visual field when the patch is viewed in isolation but not when the patch is flanked or 'crowded' by similar patches. Orientation-specific adaptation was only minimally reduced by the crowding, suggesting that neurons in the first orientation-selective stage — of necessity within V1 — were still active. The authors interpret their results as implying that spatial resolution was limited by an attentional filter acting beyond the striate cortex to restrict the availability of visual information to conscious awareness.

In a sense, the second experiment of He *et al.* represents an example of 'asimultanagnosia in normally sighted individuals' and together with the above-cited neurological studies suggest that neurons in V1 either are not directly involved in phenomenal visual experience or alternatively that something more than initial excitation of neurons in V1 by afferent activity may be necessary to produce a visual percept. The first alternative is preferred by Crick and Koch (1998), who have suggested other reasons to doubt the involvement of V1 in any kind of visual awareness.

The Crick–Koch Conjecture

Crick and Koch (1995a) postulated that only those visual areas that project directly to anterior or 'frontal' brain regions that 'contemplate, plan and execute voluntary motor outputs' can participate directly in visual awareness. Their conjecture is based initially on an unchallenged assumption that 'in going from one visual area to another further up in the visual hierarchy . . . the information is recoded at each step'. However, they further assume that such recoding, which effectively isolates those neurons that participate in early cortical visual processing from *direct* access to executive space, automatically precludes these same neurons from participation in visual awareness. In their view, it then follows that we cannot be directly aware of activity in our striate or primary visual cortex (V1) because this region does not project directly to frontal areas.

Crick and Koch also proposed that *explicit representations* of visual features, coarse-coded neural representations that correlate with percepts or objects, are a necessary but not sufficient condition for visual experience. I find no reason to disagree with this premise. However, within their model there is either an inference that explicit neuronal representations do not exist within V1 because their content would be altered during recoding beyond V1 prior to their projection to planning stages, or a conviction that even if explicit representations exist in V1 that we are unaware of them because of the absence of projections from V1 to planning stages. In any case, an absence of explicit representations within V1 would argue against a direct role for this cortex in visual perception.

Elsewhere, I cited evidence that at least some explicit representations are achieved in V1, and briefly noted that the

involvement of the striate cortex in static achromatic form vision could not be excluded based upon neurological experience in brain damaged subjects (Pollen, 1995). Crick and Koch (1995b) responded to my critique, but several areas of disagreement have remained outstanding. Moreover, hitherto I had not proposed a comprehensive plausible alternative to their conjecture that neurons within a phenomenal perceptual space must project directly to those within executive space.

Can we reconcile the large body of neurological and physiological evidence suggesting that the striate cortex is indispensable for at least one type of phenomenal visual experience with the equally impressive evidence suggesting that excitation of neurons within V1 by afferent activity may at times be insufficient to generate a visual percept? A common solution to this dilemma and a counter-argument to the Crick–Koch conjecture may be possible within a framework that arose from early insights of Locke (1690/1976) and Helmholtz (1860/1962).

Origins of Adaptive Resonance Theories

Locke (1690) surmised that 'our mind should often change the *idea* of its sensation into that of its judgment, and make one serve only to excite the other, without our taking notice of it'. Helmholtz (1860) agreed and emphasized that 'it may often be rather hard to say how much of our perceptions (Anschauungen) as derived by the sense of sight is due directly to sensation, and how much of them, on the other hand, is due to experience and training'. He used the term 'Vorstellung' or *idea* 'to mean the image of visual objects as retained in the memory, without being accompanied by any present sense-impressions', and the term 'Perzeption' or *immediate perception* to denote an awareness in which 'there is no element whatever that is not the result of direct sensation'. For the vast majority of perceptual experience involving spatial structure, he assumed a meld in which idea and immediate perception are combined in different proportions. For Helmholtz, it was 'the unconscious processes of association of ideas going on in the dark background of our memory' functioning as 'inductive conclusions unconsciously formed' that played upon sense data to produce actual visual experience.

Counterviews have at times prevailed. Skinner (1957) adhered to a rigid 'bottom-up' conditioned reflex approach to behavior which Chomsky (1959) assailed in his review of Skinner's book, concluding that 'we must attribute an overwhelming influence on actual behavior to ill-defined factors of attention, set, volition, and caprice'.

Citing Chomsky's review and strongly influenced by *Cybernetics* (Wiener, 1948), Miller *et al.* (1960) proposed that *recursive* or feedback loops were the fundamental unit of neuronal activity: such loops allow sensory inputs to be compared against some criteria established within the nervous system and are set up either to match the input to a template or alternatively to recognize incongruities between the two in which case the network would continue to respond recursively until the incongruity vanished. Subsequently, Pribram (1974), now well aware of both the cortico-cortical back-projections (Kuypers *et al.*, 1965; Pandya and Kuypers, 1969) and the by then well-documented corticofugal projections from V1 to the dorsal LGN (Guillery, 1966; Jones and Powell, 1969), envisioned a progressively differentiating self-organizing feedback loop from active templates (referred to as programmed filters or programmed tapes) within inferotemporal cortex projecting back to striate cortex and to subcortical structures including the LGN. Similarly, Milner (1974) proposed an iterative process for pattern recognition wherein the ascending and descending visual pathways leave mutually consistent trails of facilitated synapses in the complementary pathway.

In reformulating and extending Helmholtz's concepts in current terms, Grossberg (1980) surmised, as had Pribram and Milner, that 'sensory data activate a feedback process wherein a learned template, or expectancy, deforms the sensory data until a consensus is reached between what the data "are" and what we "expect" them to be'. For Grossberg (1976) select groups of neurons in a series of visual areas can establish a steady-state *adaptive resonance*, or reverberation, between regions if their patterns match, and suppress the reverberation if their patterns do not match. Models based upon these ideas for the occipito-temporal pathways have been independently developed or extended by many others, notably Harth (1976, 1987), Edelman (1978, 1987), Carpenter and Grossberg (1987), Finkel and Edelman (1989), Fukushima (1986), Koch (1987), Deacon (1988), Damasio (1989, 1990, 1994), Rolls (1990), Okajima (1991), Mumford (1991, 1992), Humphrey (1992) and Ullman (1995). Grüsser and Landis (1991) have proposed an analogous model for the occipito-parietal pathways suggesting that a continuous updating of information between the retinotopic and spatiotopic coordinates is essential for the biologically relevant perception of extrapersonal space. Quantitative mathematical adaptive models now exist to analyze how brain networks can establish stable sensory and cognitive recognition codes in response to arbitrary sequences of input patterns, to resolve the 'stability–plasticity dilemma' so that the brain can keep old memories stable yet remain plastic enough for new learning, and to show how such models can account for a myriad of perceptual phenomena (for reviews, see Carpenter and Grossberg, 1992; Grossberg, 1995).

Proposed Functions for Adaptive Resonant Loops

Evolutionary pressures to develop such feedback loops have probably been based in part upon the need of an organism to discriminate and interpret sensory data on the basis of its past experience and motivational state (Pandya and Yeterian, 1995). At the basic level of object recognition, Grossberg (1994) and Mumford (1994) recognized that, on the one hand, it is difficult to segregate an object from background without prior recognition of that object. On the other hand, recognition often follows only after the representation of an object has been segregated from its background and its boundaries defined. Thus, figure–ground segregation and object recognition cannot progress in a simple bottom-up serial fashion, but have to occur concurrently and interactively within recursive loops (Grossberg, 1994; Mumford, 1994; Lee *et al.*, 1998).

Thus, such loops have been proposed to employ active use of higher-level knowledge to disambiguate lower-order percepts (Cavanagh, 1991; Grossberg *et al.*, 1994; Mumford, 1994; Lee *et al.*, 1998) to mediate the play of selective attention upon early image representations (Milner, 1974; Fukushima, 1986; Koch, 1987; Gove *et al.*, 1995), to correlate and synchronize the activity of interrelated groups thereby facilitating the continual updating of the perceptual image (Edelman, 1978; Grossberg and Somers, 1991), to permit parallel exploration and selection of multiple alternatives (Mumford, 1992; Carpenter and Grossberg, 1993; Ullman, 1995), to facilitate binocular fusion by suppression of non-corresponding retinal images in the LGN (Singer, 1977) to provide spatial 'shifter circuits' for the computation of fine stereo vision and disparity hyperacuity (Anderson and Van Essen, 1987; Mumford, 1994) to pre-attentively separate figure from ground (Okajima, 1991; Mumford, 1994; Lee *et al.*,

1998), to modulate cortical output across cortical areas (Sandell and Schiller, 1982), to sustain 'temporal buffering' when there must be integration of clues otherwise 'hidden' over immediately preceding and succeeding spatial or temporal events (Mumford, 1992; Ullman, 1995), and to mediate control of contrast gain of LGN neurons (Grossberg, 1980; Koch, 1987).

These functions are not, in general, mutually exclusive because all imply that the *expectancy* facilitates activity evoked by sensory afferents within the classical receptive field of target cells and/or a suppresses activity by irrelevant features in the surround. Despite some unresolved issues as to how *residua* (Mumford, 1992), i.e. incongruities or mismatches between learned expectations and sensory inputs are handled, all theorists share an implicit and usually explicit view that once resonance is achieved, activity in the principal ascending and descending loops represents successive transforms that become largely *complementary* within each successive stage once resonance has been achieved.

With resonance established, activity at corresponding levels of the afferent and efferent pathways *within* each cortical area is roughly *complementary* in the sense that sensory input conveyed in ascending, largely supragranular, pathways matches that of expectations conveyed in descending pathways of largely infragranular origin. Thus, activity represented in 'higher', i.e. 'anterior', cortices bears an unique relationship to activity in 'lower', i.e. 'posterior', cortices.

The type of unique relationship suggested is not that of a one-to-one arrangement between neurons at lower and higher levels. Rather, the relationship is many-to-one or convergent in the feed-forward pathways to allow for generalization and abstraction as, for example, carried out by inferotemporal neurons (Gross, 1976). Conversely, the back-projecting pathways support a one-to-many or divergent arrangement so that the neural representations of generalizations may be projected back to lower levels to search for matches over multiple apertures simultaneously consistent with anatomical and neurological evidence that will be presented later. Thus, neurons in posterior cortices may be continuously updated as to output directly from recognition space. Reports to executive space may emanate from anterior levels of the resonant loop without depriving neurons in early visual cortices of copies of roughly corresponding content. Moreover, executive space may indirectly selectively attend to targets within phenomenal perceptual space by means of projections back to object recognition space, which, in turn, may modify search strategies over phenomenal perceptual space.

The concept of bidirectional and complementary transforms is implicit in the concept of adaptive resonant loops but became especially explicit in the models of Okajima (1991) and Ullman (1995). Koch (1987), in proposing a role for the cortico-geniculate projection system in selective attention and gain control, recognized the consequences of strong complementarity; 'In the more radical version of this theory, the entire input to striate cortex from the thalamus would be limited to those locations where retinal and cortical-thalamic inputs coincide.' Such a model for strong complementarity between LGN and V1 is not difficult to envision given the tight anatomical coupling in both directions between neurons in the LGN and in layer 6 of V1 (Lund *et al.*, 1975). However, any attempt to maintain that there is reasonable complementarity between ascending and descending pathways along the cortico-cortical loops requires a brief account of pertinent results from anatomical, physiological, psychophysical and functional brain imaging studies.

Anatomical Studies of the Back-projecting Pathways

Pandya and Yeterian (1985) have reviewed the system of reciprocal projections originating from limbic structures, proceeding through the proisocortex in anterior temporal lobe back through inferotemporal cortices (IT) and thence back serially through a succession of extrastriate visual areas to striate cortex (V1) and the LGN. For present purposes it is sufficient to summarize the origins of the back-projections from V1 to LGN and from V2 to V1 because serial projections feeding back from still high areas follow similar general principles. Moreover, V3 and V4 (Rockland and Van Hoesen, 1994), V5 (MT) (Ungerleider and Desimone, 1986; Shipp and Zeki, 1989) as well as minor connections from IT (Kennedy and Bullier, 1985; Rockland and Van Hoesen, 1994; Rockland and Drash, 1996) project directly back to V1. See also reviews by Felleman and Van Essen (1991), Salin and Bullier (1995), and Zilles and Clarke (1998).

Distinct sublaminae of layer 6 in V1 of the primate project back to parvocellular, magnocellular, and perhaps to intralaminar neurons within the dorsal LGN, thereby complementing direct ascending pathways to these same sublaminae (Lund *et al.*, 1975; Fitzpatrick *et al.*, 1994). The predominant back-projecting pathway from V2 to V1 originates in layer 6 and the bottom-most tier of layer 5 of V2 and projects in a bifurcating manner to supragranular and infragranular laminae in V1 bypassing the input layers (Rockland and Pandya, 1979; Rockland and Virga, 1987).

The infra- to infragranular terminals end on dendrites relatively close to cell bodies, whereas the back-projections to supragranular layers are especially divergent and terminate on distal dendrites in layers 1 and 2 and inconstantly in layer 3 (Rockland and Virga, 1989). The pattern of terminations might seem to suggest that the infra- to infragranular projections are stronger and may excite neurons, whereas the more distant connections on superficial dendrites might seem to suggest a milder modulatory role. However, the vastly greater number of distal terminals may compensate for this more remote location and it remains possible that many such distal terminals involve active dendrites conductances (Cauller and Connors, 1994; Hoffman *et al.*, 1997). A second back-projecting pathway from V2 to V1 comprises <10% of the total of back-projecting neurons (Rockland and Virga, 1989). This pathway emanates from layer 3A of V2 and has a similar pattern of terminations in V1.

Physiological Studies of Back-projecting Pathways

Physiological studies of LGN neurons have demonstrated various examples of cortically mediated binocular *suppression* when the extended surrounds beyond the classical LGN receptive field are stimulated (Schmeliau and Singer, 1977; Singer, 1977). Monocular effects are generally specific to the orientation, direction and spatial frequency content of stimuli in the extended surrounds relative to the corresponding parameters for stimuli presented over the classical receptive field (Cudeiro and Sillito, 1966; Sillito *et al.*, 1993).

Much less is known about center-to-center responses than about suppressive effects from the surround. Early studies showed direct excitatory projections from V1 to neurons in the LGN when their cell bodies are in topographic registration (Schmielau and Singer, 1977; Tsumoto *et al.*, 1978; Ahlsen *et al.*, 1982). However, other studies that attempted to demonstrate such effects in other paradigms have given variable and inconstant results, raising doubt as to how the corticofugal excitatory influence is conveyed to LGN neurons (Baker and Malpeli, 1977). More recently, however, *stimulus-dependent*

synchronization of LGN neurons with non-overlapping receptive fields was demonstrated when bar stimuli set to jointly stimulate these fields over distances comparable to the preferred lengths of neurons in layer 6 of V1 were tested (Sillito *et al.*, 1994). Such synchronization can lead to enhanced spatio-temporal summation at a cortical level and thereby reinforce the LGN → V1 → LGN loop (Sillito *et al.*, 1994).

Recently, we discovered a general principle mediating the corticofugal control of macaque LGN neurons (Przybyszewski *et al.*, 1998). The gain of the contrast–response function of LGN neurons is substantially reduced by reversible inactivation of V1 in a manner that implies a robust role for a multiplicative or non-linear control of the contrast gain of LGN neurons by corticofugal projections under normal conditions. Thus, the activity of LGN neurons is generally substantially enhanced when their retinal inputs match the corticofugal output of the striate cortex. These effects apply to luminance processing by both magnocellular and parvocellular neurons and the processing of isoluminant chromatic stimuli by parvocellular neurons.

There are fewer direct demonstrations of the effect of backprojecting activity to V1. Sandell and Schiller (1982) found decreased activity of neurons in the infragranular layers of V1 following selective inactivation of V2 by cooling, implying the possibility of robust excitatory effects on V1 neurons under normal conditions. Payne *et al.* (1996) have shown that the response of the center of the receptive field of V1 neurons in the macaque to visual stimulation decreases during GABA-induced selective inactivation of the retinotopically corresponding region of V2, while the response to stimulation of the surround increases. Thus, the normal distinctions between stimulations of the center and surround are blurred during inactivation of V2. Payne *et al.* (1996) suggest that the main effect of feedback from V2 to V1 is to increase the selectivity of neurons in V1 for small stimuli activating the receptive field center, a conclusion in keeping with adaptive filtering and our own results on feedback from V1 to LGN (Przybyszewski *et al.*, 1998). Facilitory modulations of neurons in V1 and V2 during selective attention, which must necessarily be mediated by back-projecting pathways, have also been demonstrated (Motter, 1993; Press *et al.*, 1994; Roelfsema *et al.*, 1998).

Psychophysical, Functional Imaging and Neurological Studies of Visual Imagery

Visualization of a previously viewed grating pattern at an appropriate distance from the target can lower the threshold for detecting a similar grating pattern within the target area (Ishai and Sagi, 1995). These effects are specific for position, orientation and spatial frequency and are binocularly mediated as well suggesting to these authors that these priming effects, though small, can extend backwards at least as far as V1. Similarly, functional MRI (LeBihan *et al.*, 1993) or positron emission tomography (Kosslyn *et al.*, 1995) studies in man have demonstrated activation of widespread regions of the occipital lobe including prestriate areas, V2 and V1 during visual imagery.

Whether such activation of early visual cortical areas, which is sometimes but not invariably found, is an essential (Kosslyn and Ochsner, 1994) or perhaps incidental (Moscovitch *et al.*, 1994; Roland and Gulyas, 1994) component of visual imagery remains controversial. The neurological literature has not yet resolved the issue. For example, Goldenberg *et al.* (1995) described a patient with apparently preserved visual imagery despite severe but not complete damage to V1. The patient initially appeared blind after bilateral posterior cerebral artery occlusions. However, subsequent MRI examination demonstrated islands of intact cortex at the occipital tip of the upper left calcarine lip, and the patient eventually recovered her sight within the central 5° of the right inferior quadrant. Thus, Goldenberg *et al.* (1995) conclude that 'our case can neither confirm nor ultimately discard the possibility that the preservation of at least small islands of primary visual cortex is necessary for the preservation of visual imagery'. In any case, for present purposes the next key issue is to try to determine how the neural correlates of visual imagery may differ from those of a number of infrequent states in which phenomenal visual experience exists in the absence of concurrent retinal stimulation.

Phenomenal Visual Experience in Hemianopic Fields

Palinopsia (Critchley, 1953), which literally means 'to see again', is the persistence or recurrence of visual images in a defective field of vision after a visual stimulus has been removed (Bender *et al.*, 1968). Many such cases occur after lesions of the right parieto-occipital region, well beyond the striate cortex (Bodis-Wollner *et al.*, 1984). Some are overtly associated with focal seizure activity and such visual experiences disappear if a relative hemianopsia becomes absolute due to direct damage to the striate cortex (Bender *et al.*, 1968).

Colored patterns or isolated bright or colored elemental spots of light or 'phosphenes' occur as perceptual correlates of irritative phenomena and may be experienced by subjects within hemianopic fields (Kölmel, 1984). Evidence suggests that the irritative stimulus originates in prestriate areas but that the phenomenal experience depends upon the integrity of striate cortex (Kölmel, 1984). Patients who experience such phosphenes within hemianopic fields usually have associated pathology characterized by lesions in the subcortical white matter or optic radiations rather than within V1 itself (Kölmel, 1984).

Anderson and Rizzo (1994) documented anatomically and functionally a case of visual hallucinations within a visual field rendered hemianopic due to destruction of the optic radiation rather than to the striate cortex. Pathways to and from preserved portions of V1 and V2 to higher cortical regions were spared. These authors concluded that phenomenal visual experience is possible on the basis of activation of the striate cortex by back-projections from higher cortical areas even in the absence of direct links from retina to LGN to visual cortex.

Moreover, complex visual hallucinations associated with temporal lobe lesions disappear when a severe visual defect develops (Kölmel, 1985, 1993). Gloning *et al.* (1967) described a patient who underwent a partial right occipital lobectomy for a brain tumor which extended into the temporal lobe and triggered complex visual hallucinations. Eight days post-operatively the patient developed a hallucinatory experience of seeing a man moving slowly across the field from the left visual periphery, then disappearing as if behind a wide pole that corresponded to the hemianopic field; the human figure subsequently reappeared within the normal right visual field and moved out to the far periphery. The sequence of motion then reversed, producing yet another gap in phenomenal visual experience when that part of the visual field rendered hemianopia, presumably due to damage to V1 and perhaps V2 as well, was re-entered. (Recall from prior discussion that cortical lesions beyond V2 do not produce visual field defects for static achromatic form vision.) Such results suggest that V1, and perhaps V2 as well, subserve at least some types of phenomenal

visual experience quite apart from their role in transmitting retinally derived information to higher extrastriate cortices.

Presumably, the seizure activity that originated in the temporal lobe continued even when it reached the temporal locus projecting back to those regions of early visual cortex rendered hemianopic inasmuch as the same seizure-driven hallucinations soon crossed to the other side of the blind region. If so, these results also suggest that hyperactive clusters of neurons within the temporal lobe that trigger complex, well-formed visual hallucinations referable to intact but not to hemianopic regions of early visual cortices are not sufficient on the basis of their own activity within the temporal lobe or feed-forward connections beyond the temporal lobe to generate phenomenal visual experience because the critical co-requirement for the activation of visual experience was the preservation of early visual cortical areas. Whether the patient of Gloning *et al.* had an explicit *idea* or *concept* – as distinct from a phenomenal experience or *percept* – of the man disappearing behind the visual gaps symbolized as a pole, or made the assumption of continuity based on the logic of previous experiences, would be of great interest but seems not resolvable from the details reported.

Rows of multiple identical images have been experienced within hemianopic fields in other cases of complex visual hallucinations. Kölmel (1993) suggests that these represent projections of stored engrams back onto the disturbed visual field. Such results suggest a divergent projection system from higher cortices back to V1 which under normal conditions might permit the memory to search for a match over a wide expanse of the visual field and thereby compensate for the loss of precise spatial localization that occurs when inferotemporal neurons generalize for object identity independently of precise spatial position.

The above-mentioned results that phenomenal visual experience can occur when the striate cortex is excited by back-projections while cut off from input from the LGN further suggests that this nucleus is not essential for phenomenal visual experience. These results are consistent with evidence that phosphenes can be elicited by direct electrical stimulation of hemianopic striate cortex either long cut off from (Brindley and Lewin, 1968) or recently deprived (Dobelle and Miladejovsky, 1974) of LGN input.

Phenomenal Visual Experience, Visual Imagery and Recursive Processing

Grossberg (1995), citing examples of phonemic restoration wherein a missing first letter of the first word of a phrase was actually filled in and experienced depending upon the context of the subsequent message (Warren, 1970), suggested that phenomenal auditory experience may require recursive processing. Strong theoretical reasons for analogous processes in the visual system – at least when segmentation and grouping are required – have already been discussed within the context of adaptive resonance theories which, at their core, require iterative processes to achieve figure–ground separation and object recognition contemporaneously. Whereas, as noted in the previous section, especially strong excitation of early visual cortices by a back-projecting systems may generate phenomenal visual experience under exceptional circumstances, this is not generally the case. Therefore, it may be useful to enquire further as to why feedback activity engenders experience in some cases but not others.

For example, visual imagery may remain distinct from visual experience except when 'spill-overs' occur as in eidetic imagery (Jaensch, 1930), hallucinations and perhaps vivid dreams. Some normal children and rare young adults possess such vivid capacity for imagery that they can recall an eidetic image of a specified scene, superimpose it onto a second picture and report details only possible based upon superimposition of the two images (Haber, 1969; Stromeyer and Psotka, 1970). Eidetic images are scanned by central attentive mechanisms that need not require saccadic eye movements (Pollen and Trachtenberg, 1972a) (presumably such representations are projected back from spatiotopic to retinotopic spaces), contrary to those that may be involved in the scanning of especially detailed mental imagery, where, for example, visualized chessboards in 'blindfold chess' are saccadically scanned (Pollen and Trachtenberg, 1972b).

Yet ordinarily, mental imagery does not engender phenomenal experience despite activating multiple visual cortical areas including V1 and V2 (LeBihan *et al.*, 1993; Ishai and Sagai, 1995). Thus, visualization and visual experience generally remain distinct, except perhaps for those possessing eidetic imagery. The cascade begun by the simultaneous absorption by rods of only 5–8 photons leads to a visual percept (Hecht *et al.*, 1942), whereas activity evoked through descending pathways by mental imagery, memory or selective attention does not in general lead to a phenomenal visual experience. Granted that the absorption of these few photons sets off an amplification process within the cascade that follows, there nevertheless seems something unique or different about networks subserving even the most minimal percept compared to those subserving extensive mental imagery.

While the strength of visual imagery may be weak in some subjects and strong in others, all normal subjects can generally distinguish whether they are experiencing visual imagery or experiences driven by external stimuli. Could they not do so, they would suffer the tragic and often life-threatening consequences of hallucinations that appear as real events to its victims. Thus, there must be intense evolutionary pressures to maintain the strength of feedback projections during the waking state within a modulatory range – at least for those projecting to the early visual cortical areas and the LGN – so that they can enhance the search for relevant objects in the external world without reaching a strength that would spuriously drive neurons in the input layers that are normally activated by external stimuli. For independent reasons, Crick and Koch (1998b) have also suggested that the feedback projections are more likely to be modulatory than driving.

Thus, a key distinction between imagery and phenomenal experience may be that sensory input which engenders the latter does so by initial neural activation within the input layers of early cortical areas and subsequent bidirectional recursive processing. Ordinary visual imagery (Farah, 1989) may correlate with central activation of exclusively descending pathways. Within these limiting cases, the 'spill-over' states (eidetic imagery and hallucinations) may activate recursive processing by sufficiently strong activation of ascending pathways by descending activity. Phenomenal visual experience, as opposed to ordinary mental imagery, seems to require the activation of at least some minimal resonant loop. It remains then to try to discern what may be unique about recursive rather than solely afferent or solely efferent processing.

The issue as to whether the striate cortex may be activated during dreams seems not yet resolved. Taking regional cerebral blood flow as an indicator of cortical activity and rapid eye

movement (REM) sleep as an indicator of intense visual imagery/dream-laden activity, Braun *et al.* (1998) found activation of extrastriate cortices but no change in striate cortices in REM sleep compared to wakefulness, although the REM density negatively correlated with blood flow in striate cortex. Blood flow in striate cortex was less during REM sleep than during deep, slow-wave sleep (SWS) – a state in which dreams are less likely to occur. If we assume, as the authors do, that all the changes in regional blood flow during wakefulness, REM sleep and SWS are due exclusively to the presence or absence of dreaming, then the striate cortex is apparently deactivated during dreaming. However, if changes in blood flow are confounded by changes dependent upon sleep stage that are independent of dreaming, then the issue of the effect of dreaming alone on striate activity is less clear. However, their conclusion is supported by other evidence. Based on studies of human brain-injured subjects, Solms (1997) concludes that the critical cortical components for visual dreaming include V3, V3A, V4 but not V1, V5 or V6. Even so, it is not yet certain whether visual dreaming is more akin to vivid visual imagery or to phenomenal vision.

Are the Neural Correlates of Phenomenal Visual Experience Referable to Individual Cortical Areas or Only to Adaptive Resonant Loops?

Studies in the somatosensory system suggest that the strength of the first surface negative response (N1) in response to a punctuate touch stimulus is the best objective measure of a monkey's subjective experience to that stimulus (Kulics, 1982). The N1 component is observed only during the conscious state. Cauller (1995) provided evidence that the N1 component may be derived from the depolarization of superficial dendrites of neurons in the primary somatosensory area in response to activation by descending fibers. Noting that as many as seven higher-order areas distribute their descending projections across layer I of the striate cortex, Cauller, supporting the 'retro-activation' hypothesis of Damasio (1989, 1990), envisions an analogous convergence zone for visual perception in V1. The key issue, nevertheless, remains whether there is anything fundamental to phenomenal experience resulting from the interface between descending and ascending systems within a modular framework, or whether the adaptive resonant loop itself is the perceptual correlate.

Several workers have already proposed that it is the recursive or adaptive resonance loops that embody phenomenal experience (Miller *et al.*, 1960; Milner, 1974; Grossberg, 1976; Edelman, 1978). At first glance, the idea appears heuristically appealing because it seems to free us from confronting the idea that any particular cortical area, type of neuron or neuronal component serves as a correlate for phenomenal experience. However, upon further scrutiny, the suggestion does not seem to offer promise of any deeper explanation of phenomenal experience than does the concept of modularity of visual function as pioneered by Zeki (1997), Allman (1987) and Van Essen (see Felleman and Van Essen, 1991), and extended by many others over the past thirty years.

The modular organization and specialized function of multiple visual areas is a cardinal consideration for any model of visual function not only because each successive specialized area carries out a new computation, which may or may not engender a phenomenal experience, but because each successive computation may eliminate at least some of the information inherent at the previous stage as Crick and Koch (1995a) have noted. Thus, it seems necessary for each cortical area that processes visual form to send forward at least two types of projections: one that is useful for subsequent computations and a second that creates the opportunity for a subsequent cortical area to achieve some generalization or summary statement *about* prior computations through either transcortical or subcortical processing.

Perceptual Transitions

It would seem then that there may be neural networks or particular neurons *within* distinct cortical areas – at least for form vision – that are somehow modified by recursive pathways to engender phenomenal experience as uniquely specified by activity within that region after modification by recursive processing. There are already several hints as to how such modifications may occur. Perceptual transitions in perspective (Necker cube reversals) for figure–ground reversals (Rubens vase) and bistable states occur abruptly without perception of intermediate states. Phenomenal experiences seem to occur when an hypothesis consistent with sensory data and memory has been achieved and confusion has been eliminated. We do not perceive the dynamic incongruities between ascending and descending streams that must occur during the computations that accompany transitional states. Thus, the time constant of the phenomenal process must be longer than the time constant of the dynamic process required to reach steady state. Anderson *et al.* (1977), studying positive feedback systems, developed some of the earliest models suggesting how brain states move from stable to other stable states with abrupt transitions between them. Apparently then, transitional states are too rapid to engender phenomenal experience.

Examples of such abrupt transitions have been demonstrated in studies of binocular rivalry. Scheinberg and Logothetis (1997) trained monkeys to report their percepts when viewing rivalrous and thus ambiguous stimuli; they found that the activity of almost all neurons in inferotemporal and adjacent temporal cortex was contingent upon one of the stimuli achieving perceptual dominance. Neurons that so correlate with the perceptually dominant stimuli are much less frequent percentage-wise, though not inconsiderable in V1/V2 and V4 where they comprise 18 and 25% of neurons tested respectively (Leopold and Logothetis, 1996). Since V1 and V2 comprise a vast expanse of the occipital lobe, even these lower percentages translate into an immense number of neurons in early vision that correlate with the perceptually dominant stimuli. Scheinberg and Logothetis (1997) suggest that the different response patterns within the ventral loop may be the result of feedforward and feedback activity that underlies the processes of grouping and segmentation. They further suggest that the temporal areas represent a stage of processing beyond the resolution of ambiguities and 'where neural activity reflects the integration of constructed visual percepts into those subsystems responsible for object recognition and visually guided action'.

The Timescale for Phenomenal Visual Experience

Various studies suggest that synchronous neural activity on a fast timescale within and across cortical areas is necessary for phenomenal experience. These views have strong theoretical underpinning (Milner, 1974; Crick and Koch, 1990; von der Malsburg, 1995) and some experimental support (Echhorn *et al.*, 1988; Roefsema *et al.*, 1997). However, such synchronous neural activity may simply serve as a marker that a steady state has been achieved, which, in turn, may be the requirement for the longer

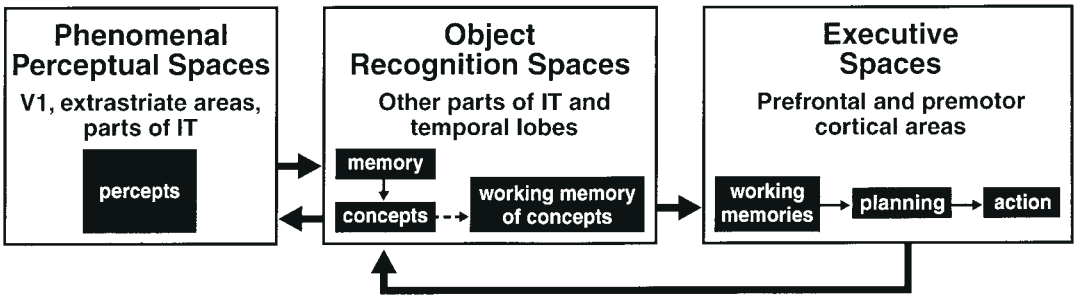

Figure 3. Very simplified schematic diagram illustrating feedforward and feedback pathways between phenomenal perceptual spaces and object recognition spaces and direct projection from the latter to executive spaces. The connections from executive spaces back to object recognition spaces are not likely to be feedback connections in any traditional sense because the former spaces can presumably address the latter independently of the content of ongoing visual processing.

time constants that accompany the emergence and stability of perceptual states compared to the faster dynamics of adapting networks and the still faster dynamics of individual action potentials.

Evidence that a sufficiently long duration of steady state activity is an essential requirement for the emergence of phenomenal experience has been provided by Libet (1991), who has shown that trains of repetitive stimuli which engender phenomenal experience require a longer duration than those that evoke unconscious detection above chance levels. Although earlier work by Libet (1964), based in part upon direct electrical stimulation of the human somatosensory cortex near threshold, suggested that several hundreds of milliseconds of repetitive stimulation was required before phenomenal experience was achieved, other work on direct electrical stimulation of the feline striate cortex (Pollen, 1977) has shown that most striate neurons are silent during the first several hundred milliseconds of stimulation. Thus, the extremely long latencies originally suggested by Libet may be attributed to inhibitory processes prior to the activation of those neuronal assemblies that engender percepts. Even so, his major conclusion regarding the longer stimulus durations required to engender phenomenal experience compared to those for unconscious detection seems secure.

Whether the longer time duration required for the emergence of the phenomenal state is itself the key requirement for the emergence of visual experience or is a marker for more intense levels of activity within some neuron, neuronal component or local circuit as a consequence of the greater opportunities for temporal summation that accompany such steady conditions remains an open question. In either case, the fundamental mechanism by which these changes may be achieved may be multiplicative or non-linear effects on the control of contrast gain of afferent activity by efferent activity as has been recently demonstrated for the effects of V1 on LGN neurons (Przybyszewski *et al.*, 1998). If such a similar mechanism holds at cortical levels, then the next steps at further discerning the *necessary* neural correlates of phenomenal experience may depend upon resolving what the differences are between states accompanying phenomenal experience and those that are subthreshold for it within individual cortical modules. It is not yet obvious that it will be possible to define *sufficient* conditions for phenomenal experience unless and until we achieve an understanding of such perception itself.

An Emerging Viewpoint

The viewpoint that has emerged here suggests the independent structural identity of neuronal spaces in early visual cortices including V1 for *percepts* or phenomenal visual experience (Fig. 3). In the next stage neural representations for *concepts* emerge after correlation of sensory data with the memory. Such representations retain the essence of an object but are divested of particulars extraneous to identification. Subsequent object recognition spaces feed backwards to provide functional modifications of activity within phenomenal perceptual spaces and feed forward to executive spaces to provide summary statements or concepts *about* approximately simultaneously occurring phenomenal visual experiences. Whether the same representations of concepts can immediately also serve as working memories or require further stages of processing to do so is unclear. In any case, such an interposition of an object recognition space obviates the need for the brain to convey copies of neural correlates of each and every phenomenal experience to those neural assemblies that participate in executive space.

All visual cortices in the ventral loop from V1 through the inferotemporal cortex and still unspecified temporal areas serve as possible substrates for different aspects of phenomenal visual experience, consistent with views on the modularity of visual perception by Zeki (1997) and also those of Damasio (1989, 1990) on multiple convergence zones for visual perception which are functionally sharpened by retroactivation. Indeed, the demonstration by Moutoussis and Zeki (1998) that color, motion and luminance-based form are perceived at slightly different latencies offers robust support for such modularity.

In the present model, V1 and V2 provide respectively the fine-grained and medium-grained representations in the luminance domain that equate with psychophysical performance tested over localized spatial domains and subject to segmentation in grouping processes iteratively achieved based on feedforward and feedback connections throughout the ventral loop. However, these cortices do not provide the explicit representations required to discriminate and identify complex objects over wider expanses of two-dimensional space or to appreciate vivid three-dimensional representations of the visual world that can be experienced even on the basis of appropriate monocular clues (Gibson, 1950). Phenomenal experience of such a three-dimensional visual world may well depend upon computations begun in V4 (Merigan and Pham, 1998) and perhaps completed within the temporal lobe.

Of course, if we accept the existence of a representation of a three-dimensional visual world well beyond V1/V2, then why need we assume that V1/V2 make any contribution to *normal* phenomenal experience although seeming to subserve more basic perceptual functions when the temporal lobe is damaged or ablated? At least one explanation may be plausible. Spatial

gradients in *size* are a powerful stimulus for the experience of three-dimensional structure but the spatial extent of the field expressing the gradient may be relatively large compared to windows of fine detail within that field (Gibson, 1950). By the level of V4, receptive field sizes in V4 are roughly 16–50 times greater than at corresponding eccentricities in V1 (Desimone and Schein, 1987) and often >1000 times greater in IT than in V1 (Gross, 1976). Given the limited dynamic range of cortical neurons, which even in attentive animals often fire at <100 Hz, there is little dynamic range available to specify both the finest localized detail within a large field as well as encoding some measure of the overall spatial content over a given size gradient.

Thus, the totality of phenomenal experience may require multiple, near-simultaneously experienced percepts in different cortical areas even *within* a common modular function such as luminance processing. Which aspect of phenomenal experience we wish to correlate depends upon which we test. For example, Leopold and Logothetis (1998) report that the decrease in visual sensitivity associated with saccadic suppression may be directly related to decreased activity in V1 during microsaccades. However, they also suggest that the continuous, stable perception of the stimulus is better correlated with activity in IT wherein most neurons show no change during microsaccades.

Eventually some match of sensory data with the memory must occur so that a neural representation of some sort of summary statement, a *concept* or a working memory *about* an object, can achieve an independent but non-phenomenal existence apart from the sensory data from which it was derived. Neurons that can represent such a working memory of an object – even when it is no longer present – and make the representation invariant of size and color versus black and white are found in both IT and prefrontal cortex (Wilson *et al.*, 1993; Fuster, 1997; Scalaidhe *et al.*, 1997). Indeed, Wilson *et al.* suggest that the activity of these neurons represents 'objects' rather than their local features. Thus, the present model (Fig. 3) interposes a non-phenomenal object-recognition stage between phenomenal perceptual space and executive space, whereas the model of Crick and Koch (1995a, 1998a) does not.

The present model was based on an interpretation of existing neurological data, but a heuristic example may further illustrate distinctions between the two models noted above. In normal vision, the perception of the visual world and the identification of objects within it appear so seemlessly bound that it may be difficult to distinguish the one process from the other. However, consider olfaction, where we may savor the aroma of wild roses and but strain our vocabularies to describe something about the scent to another person. In my view, we do not report the essence of the phenomenal experience of the aroma but rather some idea or concept about it. Similarly, the phenomenal experience of vision may be equally private. We speak and act based on information *about* what we have experienced. Thus, I find no necessity for neurons in phenomenal perceptual space to communicate directly with those in executive spaces.

Testing the Proposed Model

One critical test of the present model would require studies in man analogous to those reported by Gloning *et al.* (1967) on the patient with visual hallucinations on the basis of a temporal lobe seizure whose phenomenal experience ceased when the march of the seizure focus projected back to ablated areas in the occipital lobe. The present model would predict that phenomenal experience of detailed spatial form evoked by temporal lobe seizures in man would cease after permanent or temporary inactivation of V1/V2. If another case like that of Gloning *et al.* (1967) is ever found, that would provide the opportunity to use MRI scanning to define more precisely the cortical areas involved and to enquire also as to what, if anything, the patient sensed when the percept vanished. For example, suppose the hallucinations evoked the vivid image of a particular bright red barn. Assuming that the temporal lobe seizure continued when the phenomenal experience ceased, would the patient still preserve the idea of a bright red barn in a working memory but one devoid of phenomenal experience?

Other aspects of the proposed model are more readily testable. The model predicts that physiological correlates in alert monkeys for segmentation and grouping in V1/V2 and perceptual transitions such as those that are accompanied by binocular rivalry would be less frequent in V1/V2 after temporary inactivation of V4 or IT. Similarly, humans with lesions in V4 and IT would be expected to show altered performance on similar tasks and on the perception of a three-dimensional world from two-dimensional cues even apart from known defects in object recognition.

Note, however, that the present model makes no claim that V1 is required for the existence of all phenomenal visual experience. Certain types of motion can be perceived by neural pathways that bypass V1 and access MT (Blythe *et al.*, 1987; Ceccaldi *et al.*, 1992; Barbur *et al.*, 1993; ffytche *et al.*, 1996; Zeki and ffytche, 1998). Nor does the present model suggest that there is anything unique about the back-projecting pathways in the emergence of perceptual experience apart from their imposing a consensus between computations achieved within modules at higher and lower levels – each responsible for different functions. Even so, this linking or updating function is scarcely trivial, especially when figure–ground segregation and object recognition must occur concurrently and interactively (Mumford, 1994). Such updating may be essential for the perception and discrimination of luminance-based spatial form, especially that based on prior learning.

However, there is no evidence that such feedback loops are essential for the raw perceptions of motion and color that might be independent of prior experience (von Senden, 1960). As noted above, perception of certain types of motion does not depend on the integrity of the striate cortex and thus on projections back to the striate cortex. Similarly, subjects with extensive extrastriate lesions may perceive color and orientation-specific induced color aftereffects even when they cannot distinguish difference in the orientation of the inducing gratings (Humphrey *et al.*, 1995). Thus, surviving feed-forward pathways in such patients may suffice to convey information for color discrimination even when damage to extrastriate cortices – and presumably their projections back to V1 as well – is too extensive to support form vision. Finally, the failure of subjects in the experiments of He *et al.* (1995, 1996) to perceive certain marginal stimuli even when V1 was activated may well be due to extinction of neural traces at higher levels of the visual system with subsequent failure of these traces to activate a loop back to V1/V2.

Conclusion

In summary, the present model is based on the primacy of phenomenal experience (Humphrey, 1992; Nelkin, 1995; Raffman, 1995) and supports views that any possible explanation of phenomenal experience cannot be formulated solely in terms of its reportability (Chalmers, 1995). Finally, the evidence reviewed here also suggests that it may be the consensus of

neuronal activity across ascending and descending pathways linking multiple cortical areas that in anatomical sequence subserves phenomenal visual experience and object recognition and that may underlie the normal unity of conscious experience.

Notes

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References

Ahlsen G, Grant K, Lindstrom S (1982) Monosynaptic excitation of principal cells in the lateral geniculate nucleus of corticofugal fibers. Brain Res 234:454–458.

Allman J (1987) Evolution of the brain in primates. In: Oxford companion to the mind (Gregory R, ed.). New York: Oxford University Press.

Andersen RA, Essick GK, Siegel RM (1985) Encoding of spatial location by posterior parietal neurons. Science 230:456–458.

Andersen RA, Bradley D C, Shenoy KV (1996) Neural mechanisms for heading and structure-from-motion perception. Cold Spring Harbor Symp Quant Biol 61:15–25.

Andersen RA, Snyder LH, Bradley DC, Xing J (1997) Multimodal representation of space in the posterior parietal cortex and its use in planning movements. Annu Rev Neurosci 20:303–330.

Anderson CH, Van Essen DC (1987) Shifter circuits: a computational strategy for dynamic aspects of visual processing. Proc Natl Acad Sci USA 84:6297–6301.

Anderson JA, Silverstein JW, Ritz SA, Jones RS (1977) Distinctive features, categorical perception, and probability learning: some applications of a neural model. Psychol Rev 84:413–451.

Anderson SW, Rizzo M (1994) Hallucinations following occipital lobe damage: the pathological activation of visual representations. J Clin Exp Neuropsychol 16:651–663.

Baker FH, Malpeli JG (1977) Effects of cryogenic blockade of visual cortex on the responses of lateral geniculate neurons in the monkey. Exp Brain Res 29:433–444.

Barbur JL, Watson JDG, Frackowiak RSJ, Zeki S (1993) Conscious visual perception without V1. Brain 116:1293–1302.

Bender MB, Feldman M, Sobin A J (1968) Palinopsia. Brain 9:321–338.

Blakemore C, Campbell FW (1969) On the existence of neurones in the human visual system selectively sensitive to the orientation and size of retinal images. J Physiol 203:237–260.

Block N (1995) On a confusion about a function of consciousness. Behav Brain Sci 18:227–247.

Blythe IM, Kennard C, Ruddock KH (1987) Residual vision in patients with retrogeniculate lesions. Brain 110:887–905.

Bodis-Wollner I, Atkin A, Raab E, Wolkstein M (1977) Visual association cortex and vision in man: pattern-evoked occipital potentials in a blind boy. Science 198:629–631.

Bodis-Wollner I, Bender MB, Diamond SP (1984) Palinopsia: anomalous mapping from retinal to nonretinal coordinates of vision. In: Sensory experience, adaptation, and perception (Spillmann L, Wooten BR, eds), pp. 659–676. Mahwah, NJ: Lawrence Erlbaum.

Bradley DC, Qian N, Andersen RA (1995) Integration of motion and stereopsis in middle temporal cortical area of macaques. Nature 373:609–611.

Bradley DC, Chang GC, Andersen RA (1998). Encoding of three-dimensional structure-from-motion by primate area MT neurons. Nature 392:714–717.

Braun AR, Balkin TJ, Wesensten NJ, Gwadry F, Carson RE, Varga M, Baldwin P, Belenky G, Herscovitch P (1998) Dissociated pattern of activity in visual cortices and their projections during human rapid eye movement sleep. Science 279:91–95.

Brickner RM (1936) The intellectual function of the frontal lobes. New York: Macmillan.

Brindley GS, Lewin WS (1968) The sensations produced by electrical stimulation of the visual cortex. J Physiol 196:479–493.

Campbell FW, Green DG (1965) Optical and retinal factors affecting visual resolution. J Physiol 181:576–593.

Campbell FW, Robson JG (1968) Application of Fourier analysis to the visibility of gratings. J Physiol 197:551–566.

Carpenter GA, Grossberg S (1993) Normal and amnesic learning, recognition, and memory by a neural model of cortico-hippocampal interactions. Trends Neurosci 16:131–137.

Carpenter GA, Grossberg S (1987) A massively parallel architecture for a self-organizing neural pattern recognition machine. Comput Vis Graphics Image Process 37:54–115.

Carpenter GA, Grossberg S (1992) Adaptive resonance theory. In: Encyclopedia of artificial intelligence, 2nd edn New York: Wiley.

Cauller LJ (1995) Layer I of primary sensory neocortex: where top-down converges upon bottom-up. Behav Brain Res 71:163–170.

Cauller LJ, Connors BW (1994) Synaptic physiology of horizontal afferents to layer I in slices of rat SI neocortex. J Neurosci 14:751–762.

Ceccaldi M, Mestre D, Brouchon M, Balzamo M, Poncet M (1992). Ambulatory autonomy and visual motion perception in a case of almost total cortical blindness. Rev Neurol 148:343–349.

Cavanagh P (1991) What's up in top-down processing? In: Representations of vision: trends and tacit assumptions in vision research (Gorea A, ed.), pp. 295–304. Cambridge: Cambridge University Press.

Chalmers D (1995) The conscious mind: in search of a fundamental theory. Oxford: Oxford University Press.

Chomsky N (1959) Review of *Verbal Behavior*. Language 35:26–58.

Colby CL (1998) Action-oriented spatial reference frames in cortex. Neuron 20:15–24.

Cowey A, Stoerig P (1995). Blindsight in monkeys. Nature 373:247–249.

Crick F, Koch C (1990) Towards a neurobiological theory of consciousness. Semin Neurosci 2:263–275.

Crick F, Koch C (1995a) Are we aware of neural activity in primary visual cortex? Nature 375:121–123.

Crick F, Koch C (1995b) Cortical areas in visual awareness — reply. Nature 377:294–295.

Crick F, Koch C (1998a) Consciousness and neuroscience. Cereb Cortex 8:97–107.

Crick F, Koch C (1998b) Constraints on cortical and thalamic projections: the no-strong-loops hypothesis. Nature 391:245–250.

Critchley M (1953) The parietal lobe. New York: Hafner.

Cudeiro J, Sillito AM (1996) Spatial frequency tuning of orientation-discontinuity-sensitive corticofugal feedback to the cat lateral geniculate nucleus. J Physiol 490:481–492.

Damasio AR (1989) The brain binds entities and events by multiregional activation from convergence zones. Neural Comput 1:123–132.

Damasio AR (1990) Synchronous activation in multiple cortical regions: a mechanism for recall. Neurosciences 2:287–296.

Damasio AR, Damasio H (1994) Cortical systems for retrieval of concrete knowledge: the convergence zone framework. In: Large-scale theories of the brain (Koch C, Davis JL, eds). Cambridge, MA: MIT Press.

Damasio A, Yamada T, Damasio H, Corbett J, McKee J (1980) Central achromatopsia: behavioral, anatomic, and physiological aspects. Neurology 30:1064–1071.

Damasio AR, Damasio H, Van Hoesen GW (1982) Prosopagnosia: anatomic basis and behavioral mechanisms. Neurology 32:331–341.

Daugman JG (1985) Uncertainty relation for resolution in space, spatial frequency, and orientation optimized by two-dimensional visual cortical filters. J Opt Soc Am A2:1160–1169.

Deacon T (1988) Holism and associationism in neuropsychology: an anatomical synthesis: In: Integrating theory and practice in clinical neuropsychology (Perecman E, ed.). Hillsdale NJ: Erlbaum.

Derrington AM, Lennie P (1984) Spatial and temporal contrast sensitivities of neurones in lateral geniculate nucleus of macaque. J Physiol 357:219–240.

Desimone R, Schein SJ (1987) Visual properties of neurons in area V4 of the macaque: sensitivity to stimulus form. J Neurophysiol 57:835–868.

Dobelle WH, Miladejovsky MG (1974) Phosphenes produced by electrical stimulation of human occipital cortex, and their application to the development of a prosthesis for the blind. J Physiol 243:553–576.

Eckhorn R, Bauer R, Jordan W, Brosch M, Kruse W, Munk M, Reitboeck H J (1988) Coherent oscillations: a mechanism of feature linking in the visual cortex. Biol Cybernet 60:121–130.

Edelman GM (1978) Group selection and phasic reentrant signalling: a theory of higher brain function. In: The mindful brain (Edelman GM, Mountcastle VB, eds), pp. 51–100. Cambridege MA: MIT Press.

Edelman GM (1989) The remembered present: a biological theory of consciousness. New York: Basic Books.
Eslinger PJ, Damasio AR (1985) Severe disturbance of higher cognition after bilateral frontal lobe ablation: patient EVR. Neurology 35:1731–1741.
Farah M (1989) The neural basis of mental imagery. Trends Neurosci 12:395–399.
Felleman DJ, Van Essen C (1991) Distributed hierarchical processing in the primate cerebral cortex. Cereb Cortex 1:1-47.
ffytche OH, Guy GN, Zeki S (1996) Motion specific responses from a blind hemifield. Brain 119:1971–1982.
Finkel LH, Edelman GM (1989) Integration of distributed cortical systems by reentry: a computer simulation of interactive functionally segregated visual areas. J Neurosci 9:3188–3208.
Fitzpatrick D, Usrey WM, Schofield BR, Einstein G (1994) The sublaminar organization of corticogeniculate neurons in layer 6 of macaque striate cortex. Vis Neurosci 11:307–315.
Foster KH, Gaska JP, Marcelja S, Pollen DA (1983) Phase relationships between adjacent simple cells in the feline visual cortex. J Physiol 345:26P.
Foster KH, Gaska JP, Nagler M, Pollen DA (1985) Spatial and temporal frequency selectivity of neurones in visual cortical areas V1 and V2 of the macaque monkey. J Physiol 365:331–363.
Friedman-Hill SR, Robertson LC, Treisman A (1995) Parietal contributions to visual feature binding: evidence from a patient with bilateral lesions. Science 269:853–855.
Fukushima K (1986) A neural network model for selective attention in visual pattern recognition. Biol Cybernet 55:5-15.
Fuster JM (1997) Network memory. Trends Neurosci 20:451–459.
Galletti C, Battaglini PP, Fattori P (1995) Eye position influence on the parieto-occipital area PO (V6) of the macaque monkey. Eur J Neurosci 3:452–461.
Gabor D (1946) Theory of communication. J IEE 93:429–459.
Gaska JP, Jacobson LD, Pollen DA (1988) Spatial and temporal frequency selectivity of neurons in the third visual cortical area of the macaque monkey. Vis Res 28:1179–1191.
Gaska JP, Jacobson LD, Chen HW, Pollen DA (1994) Space–time spectra of complex cell filters in the macaque monkey: a comparison of results obtained with pseudowhite noise and grating stimuli. Vis Neurosci 11:805–821.
Gibson JJ (1950) The perception of the visual world. Boston, MA: Houghton Mifflin.
Gloning I, Gloning K, Hoff H (1967) Über optische Halluzinationen. Wiener Z Nervenheil 25:1–19.
Goldenberg G, Müllbacker W, Nowak A (1995) Imagery without perception – a case study of anosagnosia for cortical blindness. Neuropsychologia 33:1373–1182.
Gove A, Grossberg S, Mingolla E (1995) Brightness perception, illusory contours, and corticogeniculate feedback. Vis Neurosci 12: 1027–1052.
Gross CG (1976) Inferotemporal cortex and vision. In: Progress in psychobiology and physiological psychology (Sprague JM, Epstein AN, eds), pp. 77–123. New York: Academic Press.
Grossberg S (1976) Adaptive pattern classification and universal reading: II. Feedback, expectation, olfaction, illusions. Biol Cybernet 23: 187–202.
Grossberg S (1980) How does the brain build a cognitive code? Psychol Rev 87:1–51.
Grossberg S (1994) 3-D vision and figure–ground separation by visual cortex. Percept Psychophys 55:48–120.
Grossberg S (1995) The attentive brain. Am Scient 83:438–449.
Grossberg S, Somers D (1991) Synchronized oscillations during cooperative feature linking in a cortical model of visual perception. Neural Networks 4:453–466.
Grossberg S, Mingolla E, Ross WD (1994) A neural theory of attentive visual search: interactions of boundary, surface, spatial, and object representations. Psychol Rev 101:470–489.
Grüsser OJ, Landis T (1991) Visual agnosias and other disorders of visual perception and cognition. In: Vision and visual dysfunction (Cronly-Dillon JR, ed.). Boca Raton, LA: CRC Press.
Guillery RW (1966) A study of Golgi preparations from the dorsal lateral geniculate nucleus of the adult cat. J Comp Neurol 128:21–50.
Haber RN (1969) Eidetic images. Scient Am 220:36–44.
Hadjikani N, Liu AK, Dale A, Cavanagh P (1998) Retinotopy and color sensitivity in human visual cortical area V8. Nature Neurosci 1:235–241.
Harth E (1976) Visual perception: a dynamic theory. Biol Cybernet 22:169–180.
Harth E, Unnikrishnan KP, Pandya AS (1987) The inversion of sensory processing by feedback pathways: a model of visual cognitive functions. Science 237:184–187.
He S, Smallman HS, MacLeod DIA (1995) Neural and cortical limits on visual resolution. Invest Ophthalmol Vis Sci (Suppl) 36:S438.
He S, Cavanagh P, Intriligator J (1996) Attentional resolution and the locus of visual awareness. Nature 383:334–337.
Hecht S, Shlaer S, Pirenne M (1942) Energy, quanta and vision. J Gen Physiol 25:819–840.
Helmholtz H (1860/1962). Handbuch der physiologischen optik. (English trans., Southall JPC, ed.), Vol 3. New York: Dover.
Henschen SE (1893) On the visual path and center. Brain 16:170–180.
Heywood CA, Cowey A (1987) On the role of cortical area V4 in the discrimination of hue and pattern in macaque monkeys. J Neurosci 7:2601–2617.
Holmes G (1945) Ferrier Lecture. The organization of the visual cortex in man. Proc R Soc Lond B 132:348–361.
Hoffman DA, Magee JC, Colbert CM, Johnston D (1997) K^+ channel regulation of signal propagation in dendrites of hippocampal pyramidal cells. Nature 387:869–875.
Horton JC, Hoyt WF (1991) Quadrantic visual field defects. A hallmark of lesions in the extra-striate (V2/V3) cortex. Brain 114:1703–1718.
Hubel DH, Wiesel TN (1962) Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. J Physiol 160:106–154.
Humphrey GK, Goodale MA, Corbetta M, Aglioti S (1995) The McCollough effect reveals orientation discrimination in a case of cortical blindness. Curr Biol 5:545–551.
Humphrey N (1992) A history of the mind. New York: Simon & Schuster.
Ishai A, Sagi D (1995) Common mechanisms of visual imagery and perception. Science 268:1772–1774.
Jacobson LD, Gaska JP, Chen HW, Pollen DA (1993) Structural testing of multi-input linear–nonlinear cascade models for cells in macaque striate cortex. Vis Res 33:609–626.
Jaensch ER (1930) Eidetic imagery and typological methods of investigation, 2nd edn (trans. Oeser O). New York: Harcourt, Brace.
Jones EG, Powell TPS (1969) An electron microscopic study of the mode of termination of cortico-thalamic fibres within the sensory relay nuclei of the thalamus. Proc R Soc Lond B 172:173–185.
Kennedy H, Bullier J (1985) A double-labeling investigation of the afferent connectivity to cortical areas V1 and V2 of the macaque monkey. J Neurosci 5:2815–2830.
Kölmel HW (1984) Coloured patterns in hemianopic fields. Brain 107:155–167.
Kölmel HW (1985) Complex visual hallucinations in the hemianopic field. J Neurol Neurosurg Psychiat 48:29–38.
Kölmel HW (1993) Visual illusions and hallucinations. Bailliere's Clin Neurol 2:243–246.
Koch C (1987) The action of the corticofugal pathway on sensory thalamic nuclei: a hypothesis. J Neurosci 23:399–406.
Kosslyn SM, Ochsner KN (1994) In search of occipital activation during visual mental imagery. Trends Neurosci 17:290–292.
Kosslyn SM, Thompson WL, Kim IJ, Alpert NM (1995) Topographical representations of mental images in primary visual cortex. Nature 378:496–498.
Kulics AT (1982) Cortical neural evoked correlates of somatosensory stimulus detection in the rhesus monkey. EEG Clin Neurophysiol 53:78–93.
Kuypers HGJM, Szwarcbart MK, Mishkin M, Rosvold HE (1965) Occipitotemporal corticocortical connections in the rhesus monkey. Exp Neurol 11:245–262.
Lashley KS (1942) The problem of cerebral organization in vision. Biol Symp 7:301–322.
LeBihan D, Turner R, Zeffiro TA, Cuénod CA, Jezzard P, Bonnerot V (1993) Activation of human primary visual cortex during visual recall. Proc Natl Acad Sci USA 90:802–811.
Lee TS, Mumford D, Romero R, Lamme V (1998) The role of primary visual cortex in object representation and recognition. Vis Res 38:2429–2454.
Levitt JB, Kiper DC, Movshon JA (1994) Receptive fields and functional architecture of macaque V2. J Neurophysiol 71:2517–2542.

Leopold DA, Logothetis NK (1996) Activity changes in early visual cortex reflect monkeys' percepts during binocular rivalry. Nature 274:549–553.
Leopold DA, Logothetis NK (1998) Microsaccades differentially modulate neural activity in the striate and extrastriate visual cortex. Exp Brain Res (in press).
Libet B, Alberts WW, Wright EW, Delattre LD, Levin G, Feinstein B (1964) Production of threshold levels of conscious sensation by electrical stimulation of human somatosensory cortex. J Neurophysiol 27:546–578.
Libet B, Pearl DK, Morledge DE, Gleason CA, Hosobuchi Y, Barbaro NM (1991) Control of the transition from sensory detection to sensory awareness in man by the duration of a thalamic stimulus. The cerebral 'time-on' factor. Brain 114:1731-1757.
Livingstone MS, Hubel DH (1982) Thalamic inputs to cytochrome oxidase-rich regions in monkey visual cortex. Proc Natl Acad Sci USA 79:6098–6101.
Locke J (1690/1976) An essay concerning human understanding. Dent: London.
Logothetis NH (1998) See and grasp. Nature 395:444–445.
Lueschow A, Miller EK, Desimone R (1994) Inferior temporal mechanisms for invariant object recognition. Cereb Cortex 5:523–531.
Lund JS, Lund RD, Hendrickson AE, Bunt AH, Fuchs AF (1975) The origin of efferent pathways from the primary visual cortex, area 17, of the macaque monkey as shown by the retrograde transport of horseradish peroxidase. J Comp Neurol 164:287–304.
Luria AR (1959) Disorders of 'simultaneous perception' in a case of bilateral occipito-parietal brain injury. Brain 82:437–449.
Marcelja S (1980) Mathematical description of the responses of simple cortical cells. J Opt Soc Am 70:1297–1300.
Mattingley JB, Davis G, Driver J (1997) Preattentive filling-in of visual surfaces in parietal extinction. Science 275:671–674.
McFadzean RM, Hadley DM (1997) Homonymous quadrantanopia respecting the horizontal meridian. A feature of striate and extrastriate cortical disease. Neurology 49:1741-1746.
McKeefry DJ, Zeki S (1997) The position and topography of the human colour centre as revealed by functional magnetic resonance imaging. Brain 120:2229–2242.
Merigan WH (1996) Basic visual capacities and shape discrimination after lesions of extrastriate area V4 in macaques. Vis Neurosci 13:51–60.
Merigan WH, Maunsell JHR (1993) How parallel are the primate visual pathways? Annu Rev Neurosci 16:369–402.
Merigan WH, Pham HA (1998) V4 lesions in macaques affect both single and multiple-viewpoint shape discriminations. Vis Neurosci 15:359–369.
Merigan WH, Nealey TA, Maunsell HR (1993) Visual effects of lesions of cortical area V2 in macaques. J Neurosci 13:3180–3191.
Miller GA, Galanter E, Pribram KH (1960) Plans and the structure of behavior. New York: Henry Holt.
Miller M, Pasik P, Pasik T (1980) Extrageniculostriate vision in the monkey. VII. Contrast sensitivity functions. J Neurophysiol 43: 1510–1526.
Milner AD, Goodale MA (1995) The visual brain in action. Oxford: Oxford University Press.
Milner PM (1974) A model for visual shape recognition. Psychol Rev 81:521–535.
Mishkin M, Ungerleider LG, Macko KA (1983) Object vision and spatial vision: two cortical pathways. Trends Neurosci 6:414–417.
Moran J, Desimone R (1985) Selective attention gates visual processing in the extrastriate cortex. Science 229:782–784.
Moscovitch M (1994) Do PETS have long or short ears? Mental imagery and neuroimaging. Trends Neurosci 17:292–294.
Motter BC (1993) Focal attention produces spatially selective processing in visual cortical areas V1, V2, and V4 in the presence of competing stimuli. J Neurophysiol 70:909–919.
Moutoussis K, Zeki S (1997) Functional segregation and temporal hierarchy of the visual perceptive systems. Proc R Soc Lond B 264:1407–1414.
Mumford D (1991) On the computational architecture of the neocortex. I. The role of the thalamo-cortical loop. Biol Cybernet 65:135–145.
Mumford D (1992) On the computational architecture of the neocortex. II. The role of cortico-cortical loops. Biol Cybernet 66:241–251.
Mumford DM (1994) Neuronal architectures for pattern-theoretic problems. In: Large-scale neuronal theories of the brain (Koch C, Davis JL, eds), pp. 125–152. Boston, MA: MIT Press.
Nakayama K, Shimojo S (1992) Experiencing and perceiving visual surfaces. Science 257:1357–1363.
Nelkin N (1995) The dissociation of phenomenal states from apperception. In: Conscious experience (Metzinger T, ed.), pp. 373–383. Schningh, Paderborn: Imprint Academic.
Okajima K (1991) A recurrent system incorporating characteristics of the visual system: a model for the function of backward neural connections in the visual system. Biol Cybernet 65:235–241.
Pandya DN, Kuypers HGJM (1969) Cortico-cortical connections in the rhesus monkey. Brain Res 13:13–36.
Pandya DN, Yeterian EH (1995) Architecture and connections of cortical association areas. Cereb Cortex 4:3–61.
Payne BR, Lomber SG, Villa AE, Bullier J (1996) Reversible deactivation of cerebral network components. Trends Neurosci 12:535–542.
Pollen DA (1977) Responses of single neurons to electrical stimulation of the surface of the visual cortex. Brain Behav Evol 14:67–86.
Pollen DA (1995) Cortical areas in visual awareness. Nature 377:293–294.
Pollen DA, Trachtenberg MD (1972a) Alpha rhythm and eye movements in eidetic imagery. Nature 237:109–112.
Pollen DA, Trachtenberg MC (1972b) Some problems of occipital alpha block in man. Brain Res 41:303–314.
Pollen DA, Ronner SF (1981) Phase relationships between adjacent cells in the visual cortex. Science 212:1409–1411.
Press WA, Knierim JJ, Van Essen DC (1994) Neuronal correlates of attention to texture patterns in macaque striate cortex. Soc Neurosci Abstr 20:838.
Pribram KH (1974) How is it that in sensing so much we can do so little? In: The neurosciences: third study program (Schmitt FO, Worden FG, eds). Cambridge, MA: MIT Press.
Przybyszewski AW, Foote W, Pollen DA (1998) Contrast gain control of LGN neurons by V1. Invest Ophthalmol Vis Sci 39:S238.
Raffman D (1995) On the persistence of phenomenology. In: Conscious experience. (Metzinger T, ed.), pp. 293–308. Schöningh, Paderborn: Imprint Academic.
Rizzo M, Robin DA (1990) Simultanagnosia: a defect of sustained attention yields insights on visual information processing. Neurology 40:447–455.
Rizzo M, Nawrot M, Blake R, Damasio AR (1992) A human disorder resembling area V4 dysfunction in the monkey. Neurology 42: 1175–1180.
Robinson DL, Goldberg ME, Stanton GB (1978) Parietal association cortex in the primate: sensory mechanisms and behavioral modifications. J Physiol 41:910–932.
Rockland KS, Pandya DN (1979) Laminar origins and terminations of cortical connections of the occipital lobe in the Rhesus monkey. Brain Res 179:3–20.
Rockland KR, Virga A (1989) Terminal arbors of individual 'feedback' axons projecting from area V2 to V1 in the macaque monkey. J Comp Neurol 285:54–72.
Rockland KS, Van Hoesen GW (1994) Direct temporal-occipital feedback connections to striate cortex (V1) in the macaque monkey. Cereb Cortex 4:300–313.
Rockland KS, Drash GW (1996) Collateralized divergent feedback connections that target multiple cortical areas. Comp Neurol 373: 529–548.
Roelfsema PR, Engel AK, König P, Singer W (1997) Visuomotor integration is associated with zero time-lag synchronization among cortical areas. Nature 385:157–161.
Roelfsema PR, Lamme VAF, Spekreijse H (1998) Object-based attention in the primary visual cortex of the macaque monkey. Nature 395: 376–381.
Roland PE, Gulyás B (1994). Visual representations of scenes and objects: retinotopical or non-retinotopical? Trends Neurosci 17:294–297.
Rolls ET (1990) The representation of information in the temporal lobe visual cortical areas of macaques. In: Advanced neural computers (Eckmiller R, ed.), pp. 69–78. New York: Elsevier.
Rubens AB, Benson DF (1971) Associative visual agnosias. Arch Neurol 24:305–316.
Sacks O, Wasserman R (1987) The case of the colorblind painter. Rev Books NY 34:25–34.
Salin PA, Bullier J (1995) Corticocortical connections in the visual system: structure and function. Physiol Rev 75:107–154.

Sandell JH, Schiller PH (1982) Effect of cooling area 18 on striate cortex cells in the squirrel monkey. J Neurophysiol 48:38–48.
Scalaidhe SPO, Wilson FAW, Goldman-Rakic PS (1997) Areal segregation of face-processing neurons in prefrontal cortex. Science 278: 1135–1138.
Scheinberg DL, Logothetis NK (1997) The role of temporal cortical areas in perceptual organization. Proc Natl Acad Sci USA 94:3408–3413.
Schiller P (1993) The effects of V4 and middle temporal (MT) area lesions on visual performance in the rhesus monkey. Vis Neurosci 10:717–746.
Schmielau F, Singer W (1977) The role of visual cortex for binocular interactions in the cat lateral geniculate nucleus. Brain Res. 120:359–361.
Sereno AB, Maunsell JHR (1998) Shape selectivity in primate lateral intraparietal cortex. Nature 395:500–503.
Shipp S, Zeki S (1989) The organization of connections between areas V5 and V1 in the macaque monkey. Eur Neurosci Assoc 1:309–332.
Sillito AM, Cudeiro J, Murphy PC (1993) Orientation sensitive elements in the corticofugal influence on centre–surround interactions in the dorsal lateral geniculate nucleus. Exp Brain Res 93:6–16.
Sillito AM, Jones HE, Gerstein GL, West DC (1994) Feature-linked synchronization of thalamic relay cell firing induced by feedback from the visual cortex. Nature 369:479–482.
Singer W (1977) Control of thalamic transmission by corticofugal and ascending pathways in the visual system. Physiol Rev 57:386–420.
Skinner BF (1957) Verbal behavior (Century Psychology Series). New York: Appleton-Century-Crofts.
Solms M (1997) The neuropsychology of dreams; a clinico-anatomical study. Hillsdale, NJ: Lawrence Erlbaum.
Stoerig P, Cowey A (1995) Visual perception and phenomenal consciousness. Behav Brain Res 71:147–156.
Stoerig P, Cowey A (1997) Blindsight in man and monkey. Brain 12:535–559.
Stromeyer CF III, Psotka J (1970) The detailed texture of eidetic images. Nature 225:346–349.
Tootell RBH, Hadjikhani N (1998) Has a new color area been discovered ? Reply. Nature Neurosci 1:335–336.
Tsumoto T, Creutzfeldt OD, Legendy CR (1978) Functional organization of the corticofugal system from visual cortex to lateral geniculate nucleus in the cat (with an appendix on geniculo-cortical monosynaptic connections). Exp Brain Res 32:345–364.
Ullman S (1995) Sequence seeking and counter streams: a computational model for bidirectional information flow in the visual cortex. Cereb Cortex 1:1–11.
Ungerleider LG, Desimone R (1986) Cortical connections of visual area MT in the macaque. J Comp Neurol 248:190–222.
von der Malsburg C (1995) Binding in models of perception and brain function. Curr Opin Neurobiol 5:520–526.
von Senden M (1960) Space and sight; the perception of space and shape in the congenitally blind before and after operation. Glencoe, IL: Free Press.
Warren RM (1970) Perception restoration of missing speech sounds. Science 167:393–395.
Watson AB, Barlow HB, Robson JG (1983) What does the eye see best? Nature 302:419–422.
Weiskrantz L (1995) The problem of animal consciousness in relation to neuropsychology. Behav Brain Res 71:171-175.
Weiskrantz L (1995) Blindsight — not an island unto itself. Curr Dir Psychol Sci 4:146–151.
Wiener N (1948) Cybernetics. New York: Wiley.
Wilson FA, Scalaidhe SP, Goldman-Rakic PS (1993) Dissociation of object and spatial processing domains in primate prefrontal cortex. Science 260:1955–1958.
Zeki S (1990) A century of cerebral achromatopsia. Brain 113:1721–1777.
Zeki S (1997) The color and motion systems as guides to conscious visual perception. Cereb Cortex 12:777–809.
Zeki S, ffytche DH (1998) The Riddoch syndrome: insights into the neurobiology of conscious vision. Brain 121:25–45.
Zeki S, McKeefry DJ, Bartels A, Frackowiak RSJ (1998) Has a new color area been discovered? Nature Neurosci 1:335.
Zihl J, Von Cramon D, Mai N, Schmid CH (1991) Disturbance of movement vision after bilateral posterior brain damage. Brain 114:2235–2252.
Zilles K, Clarke S (1998) Architecture, connectivity and transmitter receptors in human extrastriate visual cortex. Comparison with non-human primates. In: Cerebral cortex, Vol. 12: Extrastriate cortex in primates (Rockland KS, Kaas JH, Peters A, eds). New York and London: Plenum.