

LUCIA M. VAINA

AKINETOPSIA, ACHROMATOPSIA AND BLINDSIGHT: RECENT STUDIES ON PERCEPTION WITHOUT AWARENESS

ABSTRACT. The neural substrate of early visual processing in the macaque is used as a framework to discuss recent progress towards a precise anatomical localization and understanding of the functional implications of the syndromes of blindsight, achromatopsia and akinetopsia in humans. This review is mainly concerned with how these syndromes support the principles of organization of the visual system into parallel pathways and the functional hierarchy of visual mechanisms.

1. INTRODUCTION

Studies of the visual system of the macaque provide the best model of the human visual system. Both humans and macaques are Old World primates, and the physiology and anatomy of the macaque visual system is well-studied. At subcortical levels, the anatomy of the macaque brain is very similar to that of man. At the cortical level a great deal is now known about the macaque, but very little is known about the human visual system. Although, in humans, well circumscribed lesions are rare and often not exactly where one would want them to be, it is important to study psychophysically the effect of these lesions for a number of reasons. First, it will help to map areas in the human visual cortex that are homologous to areas in the nonhuman primate, where we can learn much more about the cell physiology and connectional anatomy. Second, human verbal reports of damaged visual percepts are much more detailed and incisive than any type of data that animal psychophysics (coupled with specific lesions) could ever yield. When the link between human and monkey visual cortical areas can be made with greater confidence, it will be much clearer how to interpret properties of cells in a given functional or anatomical area, or both. Third, achieving a finer scale of understanding the underlying nature of visual deficits of central origin, is crucial to our ability to diagnose and treat them.

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Synthese is published monthly.

Subscription price per annum: USD 922.00.

Second-class postage paid at Rahway, N.J. USPS No. 043-250.

U.S. Mailing Agent: Mercury Airfreight International Ltd., 2323 Randolph Ave., Avenel, NJ 07001.

Published by Kluwer Academic Publishers, Spuiboulevard 50, P.O. Box 17, 3300 AA Dordrecht, The Netherlands, and 101 Philip Drive, Norwell, MA 02061, U.S.A.

Postmaster: Please send all address corrections to: *Synthese*, c/o Mercury Airfreight International Ltd., 2323 Randolph Ave., Avenel, NJ 07001, U.S.A.

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2. GENERAL FRAMEWORK OF THE STUDY

A wealth of anatomical and physiological studies concerned with the characterization of visual properties in the extrastriate regions of the macaque have led to the hypothesis that each of these areas is specialized in a particular aspect of vision. Two principles have been used in visual neuroscience as an explanatory framework for the anatomical and functional organization of the visual system in the primate brain. The first principle is that of hierarchical order among the visual areas. The second principle is the division of the many cortical visual areas into two major parallel processing systems, one directed ventrally into the temporal lobe that is specialized for object recognition and the other directed dorsally into the parietal lobe that is specialized for the appreciation of spatial perception and visuo-motor performance (Ungerleider and Mishkin, 1982). Recently, the principle of strict parallel organization of the visual system has been called into question, partly because of the discovery of abundant interconnections in the extrastriate areas (for a review, see Merigan and Maunsell, 1993). Similarly, the notion of strict hierarchical processing has been challenged and over the past year it has become the focus of an arduous debate about the "site of consciousness" which motivated revisiting the controversial syndrome of "blindsight" (discussed at the end of this review). The relationship between consciousness and visual perception appears to be among the hottest topics of the year and, accordingly, we will provide new evidence for "perception without awareness" of aspects of color in neurological patients with achromatopsia.

The review is strictly confined to a subset of neurological studies on color, motion and blindsight published during 1994–1995.

3. "WHAT" AND "WHERE" IN THE HUMAN VISUAL SYSTEM?

Most of the direct comparisons made between the parietal and temporal pathways primarily emphasize the contributions to visual analysis of the middle temporal area (MT) (referred to as V5 in the British literature) and V4. The major distinctive properties of these areas appear to be the lack of color selectivity but strong motion selectivity in MT, and conversely, V4 contains color and orientation selective neurons, but motion selective neurons are conspicuously absent. Selectivity for color persists along the occipital-temporal pathway (Gross, 1975), whereas the selectivity for motion related information persists along the occipito-parietal pathway.

Functional segregation between the properties of the parietal and temporal pathways in humans has been demonstrated in positron emission

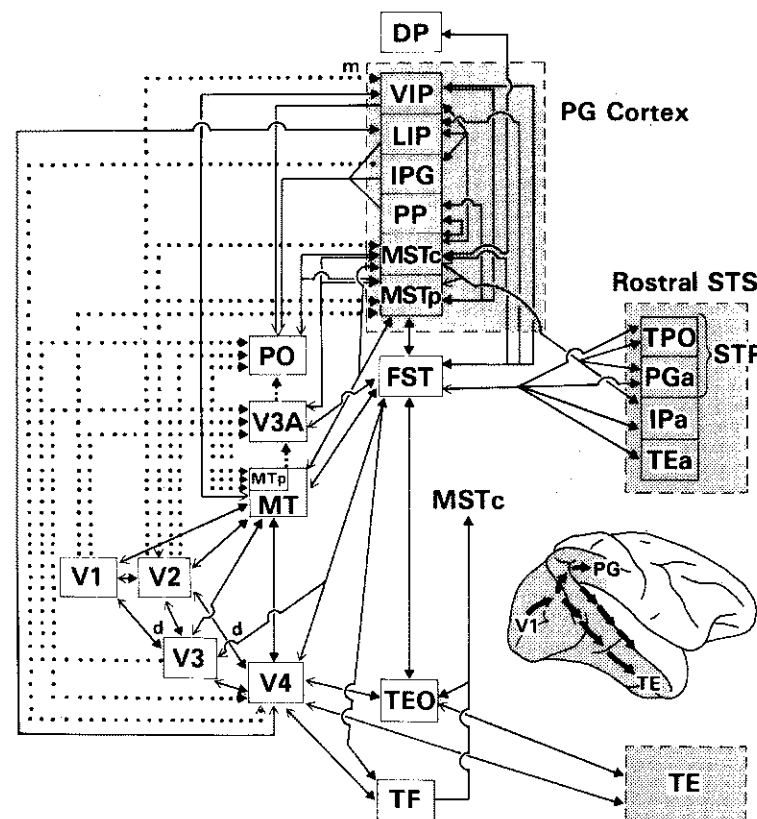


Figure 1. Summary of visual cortical areas and their connections (from Boussaoud et al., 1990). The diagram shows some of the major visual areas along the dorsal and ventral cortico-cortical pathways represented in the shaded area on the schematic drawing of a lateral view of the monkey brain. In addition to the occipito-temporal (ventral) stream for object vision and the occipito-parietal (dorsal) stream for spatial vision, there may be a third processing stream which relays motion information from MST and FST into the anterior portion of the superior temporal sulcus (STS).

tomography studies (PET) studies of normal individuals. Most notably, Zeki and his London group (Watson et al. 1993; Zeki, 1991; Zeki et al., 1991) showed differential cortical activation when subjects view color or moving stimuli, and Haxby et al. (1991a,b) showed differential activation when subjects performed a spatial task or a face recognition task. Consistent with neurophysiological data, these activation studies support the model of a coarse organization of the visually responsive cortex into parallel pathways. This segregation is also reinforced by studies of lesions in

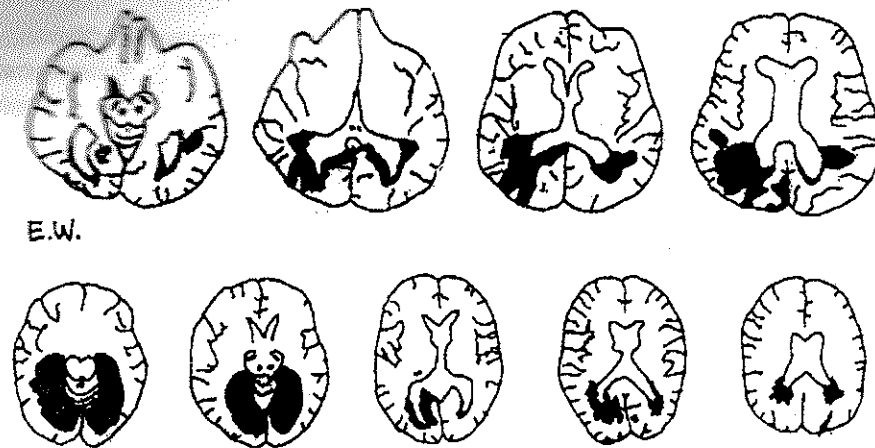


Figure 2. Schematic drawings of the relevant axial slices of patients' A.F. and E.W. brains showing (in black) the locations of their bilateral lesions.

restricted regions of the human visual cortex, leading to specific deficits in visual perception.

A recent study (Vaina, 1994) reported the results of detailed psychophysical and neuropsychological tests from two patients (E.W. and A.F.) with bilateral lesions showing particularly selective visual deficits.

The patient E.W. displayed deficits in all basic aspects of color vision, but his ability to perceive motion remained intact. Patient A.F. showed the opposite pattern of deficits, he was severely impaired on motion perception, especially on speed discrimination and on the discrimination of direction in displays containing distracting dynamic visual noise, but not on color. Although other perceptual deficits were found concurrently, these results provided further support for the idea that different visual cortical streams process color and motion aspects of visual stimuli. The location of these human cortical areas are in general agreement with a magnified version of the "what" and "where", or the parietal and temporal models of information processing, proposed in the macaque visual cortex.

We will briefly discuss recent data supporting the view that the functional segregation into strictly defined parallel pathways is not absolute. For example, neurological evidence for interaction of form and motion cues was provided in a recent study of the performance of 28 stroke patients with single, unilateral infarcts, on a large number of form and motion psychophysical tasks. By and large, the results are consistent with the two parallel pathways model (Vaina, 1995 – in press): Patients with occipital-

temporal lesions were mostly impaired on the discrimination of *form*, but their performance was normal on a large class of motion discrimination tasks. But this is not the whole story. These patients' performance on form discrimination tasks when the form was defined solely by motion cues (speed or direction) was significantly better than their performance at chance level when the form was defined by luminance, color or global stereopsis cues. The converse holds for patients with occipital-parietal lesions, who were impaired on *motion and stereopsis*, but their form discrimination ability was generally unaffected when the form was defined by color or luminance cues. However, their performance was close to chance when the form was defined by motion or depth cues. These results suggested that form discrimination is strongly linked to the specific cues that define it, and is not entirely the responsibility of specific areas in the "what" pathway. This view is supported by a series of carefully designed functional neuroanatomical studies by PET (Gulyas et al., forthcoming, 1994) in which changes in regional blood flow were measured in normal individuals who performed form discrimination tasks, when the form was defined solely by luminance, disparity, motion or color cues. Using the subtraction technique, the contribution of each component (e.g. luminance, disparity, motion, or color) was eliminated. Subsequent comparison of the localization of the regions specifically activated during each kind of form discrimination revealed that there was almost no overlap between them, for example the discrimination of form defined by motion activated functional fields bilaterally in the inferior occipital gyri, in the lingual, anterior cingulate, middle frontal and orbitofrontal gyri, and in the left fusiform and right inferior temporal gyri. The discrimination of form by color cues produced bilateral activation in the inferior temporal, lateral occipital, and orbito-frontal gyri, the left precuneus and intraparietal sulcus and in the right precentral gyrus (Figure 3). Taken together, these studies demonstrate that the dogma of a "form center" in the visual cortex is probably not correct. Instead, discrimination of visual form is mediated by different, non-overlapping, networks of cortical areas when different visual cues are used to build up the internal representation on which the decision (discrimination) is based.

4. MOTION VISION AND AKINETOPSIA

The analysis of motion plays a central role in the visual systems and arguably it is the one for which we now have the best insight into its neural underpinnings. The dorsal pathway (the "where" system) of non human primates contains a complex system of cortical areas that play a major role

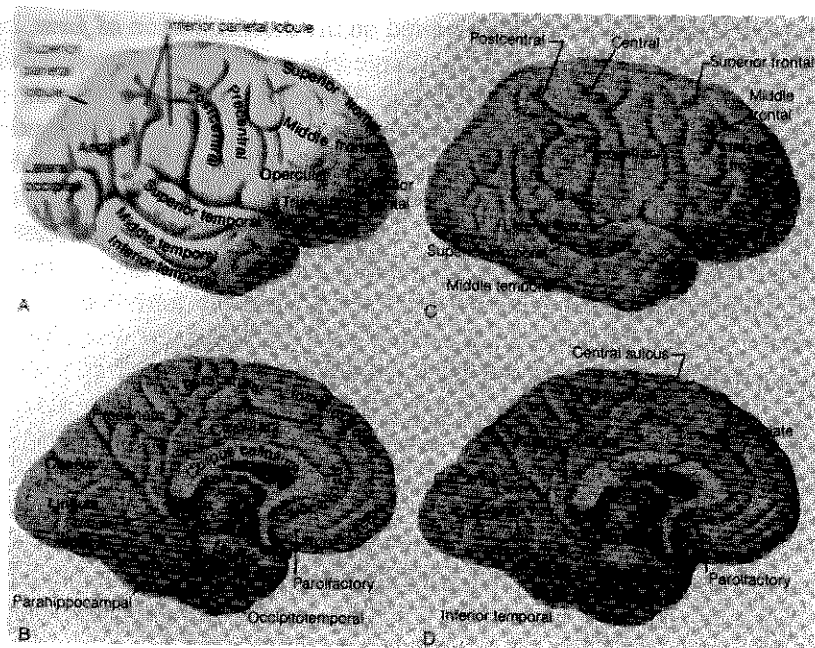


Figure 3. Gyri and sulci. Lateral (A) and medial (B) views of the gyri. Lateral (C) and Medial (D) views of the sulci.

in visual motion processing, starting with V1 and coursing through V2, V3, MT and MST (the medial superior temporal area) before entering the more anterior parietal and anterior temporal areas which are also involved higher aspects of motion processing (Figure 1). The abundant representation of motion in the primate visual system is not surprising as motion vision is used for many tasks such as establishing the three-dimensional structure of the visual scene, guiding balance and postural control, estimating the direction of the observer's own path of movement, determining the time to collision with objects in the environment and segmenting the scene into different objects. In spite of the importance of motion in visual perception and in spite of the availability of a detailed unified theoretical and experimental framework for exploring motion deficits in neurological patients, neurological studies specifically devoted to describing acquired motion deficits after brain damage, a condition called *cerebral akinetopsia* (Zeki, 1991), are scarce. This is in striking contrast with the relatively larger number of reports describing *cerebral achromatopsia* a clinical condition where brain damage abolishes color vision.

4.1. Where Is MT in the Human Brain?

In the tradition of classical neurology, one is naturally interested in the precise anatomical localization of cortical regions involved in different aspects of motion processing in humans. The approach has been to search for human homologues of those motion-selective cortical areas that are most completely characterized in the macaque visual system. Lesions of the MT in the macaque produce transient but selective behavioral impairments on both perceptual and motor skills requiring the analysis of visual motion (for a review see Maunsell and Newsome, 1987). MT in the monkey is the first level of visual processing where the perception of motion is directly useful for behavior, and thus it marks a specialized anatomical beginning of the branch of the dorsal parallel pathway defined by a chain of cortical areas underlying the analysis of motion and spatial localization. So selective to direction and speed of motion is this area, that it earned the nickname of "the motion area" and to date is probably the most studied region of the dorsal pathway in the macaque cortex.

Combined psychophysical and neuroimaging methods have been used to delineate the human homologue of MT in patients with cerebral lesions. The first detailed studies are the cases L.M. (described extensively by Zihl and various collaborators (Baker et al., 1990; Hess et al., 1989; Zihl et al., 1983, 1989, 1991)), and A.F. (studied in detail by Vaina and collaborators (Vaina 1989; Vaina et al., 1990b,c,d; Vaina et al., 1991)), both with bilateral extrastriate lesions of vascular origin. In both the primary visual cortex (V1) which in the macaque provides the major cortical input to MT, was spared. Although L.M. has some residual motion-vision she is so impaired on motion perception that in the original paper she was described as "motion blind". The almost total and selective loss of motion perception is so rare, and so unusual, that many researchers have set out to evaluate a wide range of aspects of L.M.'s motion perception. L.M. is the most detailed study of deficits in motion vision to date. Although both L.M. and A.F. had a large spectrum of motion deficits, the most relevant ones for this discussion are the severe impairment of speed discrimination and discrimination of direction of motion in dynamic stochastic random dot displays, in which a variable proportion of dots moved coherently in one direction, the rest providing distracting (static or dynamic) noise (Figure 4, top). The latter visual deficit indicates an inability to cope with masking dynamic visual noise, which is very similar to the deficits displayed by monkeys with ibotenic acid lesions in the area MT (Newsome and Wurtz, 1988).

Based on several neuroanatomical maps of humans and on putative anatomical analogy with the macaque, the characterization of L.M. and A.F.'s lesions by computerized tomography (CT) and magnetic resonance

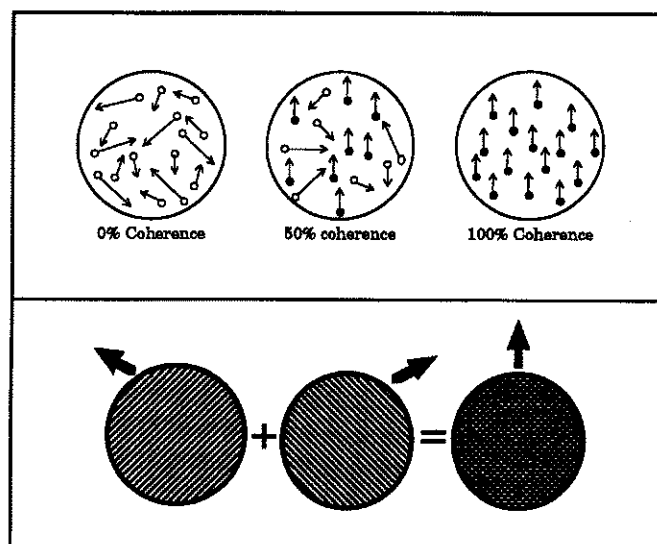


Figure 4. A schematic representation of two major visual stimuli used to demonstrate visual processing at the level of the area MT. **MOTION COHERENCE:** On top, the stimuli show patterns of dynamic random dots. In the left hand aperture the dots are shown schematically moving in random directions (0% coherence), that is the display consists of dynamic noise with no net motion in any single direction. In the right panel, all the dots move uniformly in the same direction (100% coherence). The middle panel illustrates 50% coherence, that is 50% of the dots carry the uniform motion signal and the remaining dots provide masking noise. **PLAIDS:** On the bottom, the figure shows a moving plaid pattern (on the right) that results from the superimposition of two drifting gratings moving in different directions.

imaging (MRI) studies suggests an involvement of the human homologue of the macaque MT, roughly thought to be a region encroaching the junction of the occipital-parietal and temporal areas, equated by Tusa and Ungerleider (1985) with the area PTOF of Polyak (1957) (Figure 3c). But in all the neurological cases reported thus far, including L.M. and A.F., the brain lesions extend significantly beyond MT and often involve several brain structures, and thus one cannot sort out what, if anything, those other areas contribute to motion deficits, and what is the specific contribution of the MT lesion.

The first precise localization of the human homologue of the macaque MT was reported by Zeki's group at the Hammersmith hospital, London in a series of elegant functional neuroanatomical studies in normal subjects, using PET activation images co-registered with MRI (Watson et al., 1993; Zeki, 1993). Studying selective brain activation to specific motion stimuli, Zeki and collaborators proposed that the MT (V5) in humans is situated laterally and ventrally in the occipital lobe, near to the intersection of the

ascending limb of the sulcus and the lateral occipital sulcus (Figure 3). Recently, using noninvasive functional MRI techniques based on blood flow oxygenation, Tootell's group (Tootell et al., 1995) studied the foci of activation when normal observers viewed radially moving random dots alternated with stationary random dot arrays. Comparing moving versus stationary dots they found an area of highest selectivity in the same location as determined by the PET studies based on the Talarich coordinates. A detailed analysis of the functional activation of this area revealed that, functionally, it is identical to the MT (V5) in the monkey.

Naturally it is of great neurological interest to use the functional-anatomical localization of the human MT and explore what may be the cortical areas responsible for the residual motion vision in patient L.M. This is what Zeki's group did in a recent PET study of the pattern of activation in L.M. while she was performing several motion discrimination tasks (Shipp et al., 1994). The study revealed several foci of activation, the strongest being in the parietal cortex bilaterally. There was no evidence of the focus of activation of MT (V5) which is a definitive proof that this area was entirely destroyed by the lesion bilaterally. Interestingly, little or no activation was seen in the lower segment of the motion pathway (the equivalent of V1 and V2), although these areas were not directly involved in L.M.'s lesion. In contrast, prominent activation was found in the upper segment of V3 and the superior parietal cortex, the Brodman's area 7a. In spite of their sensitivity to motion, the pattern of L.M.'s visual motion deficits indicate that these areas do not mimic the function of the MT in visual motion perception and, in fact, they may depend on the MT in this respect which highlights the marked specialization of MT for motion.

4.2. What Do Motion Deficits Reveal about Motion Vision?

Cortical motion processing appears to be a two stage process. First, are local measurements of motion which in a second stage are integrated to reconstruct the global velocity field. The problem is complex because multiple motion signals can arise from the same object or from different moving objects, and it is critical for a reliable motion system to integrate only those signals that define one object, and to not integrate those motion signals that belong to different objects. Reliable motion perception requires both *segmentation* processes capable of detecting differential properties of optic flow associated with moving objects, and *integration* processes that combine motion information within single objects to compute their overall two dimensional and three dimensional motion and structure.

The visual mechanisms underlying the integration of motion have been studied for almost 15 years, using *plaid patterns* (Figure 4, bottom) com-

posed of two superimposed gratings usually of similar contrast and spatial frequency but differing in orientation and moving independently (Adelson and Movshon, 1982). While the motion of each component grating is ambiguous as only the orthogonal components of the oriented patterns can be measured, combined movement of the two superimposed gratings can yield a coherently moving plaid (pattern motion). It has been suggested that the motion of individual gratings (locally oriented components) is detected at the first cortical stage. The signals are then integrated at a second stage, providing a true pattern motion that is consistent with our perceptual experience. These two stages of motion processing are expressed by properties of cells in the visual cortex. The first stage is mediated by directionally selective neurons in V1 and the second by a population of neurons in the MT that can respond selectively to pattern motion. This strict hierarchical organization has been challenged (Victor and Conte, 1994) in a report of a patient severely impaired in direction and orientation judgments, that is, he was impaired in the first stage of motion analysis, but he performed much better on plaids motion (the integration stage). This result is at odds with the two stage model in which the component motion is first extracted and subsequently used as an input to the plaid motion extraction. When the two components differ sufficiently in their defining characteristics (e.g. spatial frequency, color, stereoscopic depth) the perception of a coherently moving pattern is reduced. When integration does not occur, the two component gratings appear transparent. Perceptually this means that they arise from different objects in the scene and, instead of *integration*, the visual system will produce a *segmentation* of signals. For example we do not combine the motion of shadows with the motion of objects which move over them, or of the tree leaves moved by the wind with the bird flying to its nest. We have recently observed a patient, G.T., who for stimuli presented in the visual field contralateral to her lesion always perceived a coherently moving plaid, even when the two component gratings differed three-fold in spatial frequency (Vaina, unpublished observation). Her perception in the ipsilateral field was somewhat more accurate. The patient said that the world looked "not right", things appearing to blend one into the other without making any sense.

Segmentation processes are also involved in computing motion discontinuity. In plaid stimuli, when subjects do not perceive a coherently moving plaid but see two gratings sliding one over another, the process is one of *discontinuity at a point*. We reported results from an experiment using random dot displays, similar to the motion coherence test (Figure 4, top), in which the discontinuity occurred along a border. Detection of motion discontinuities along the border is perceptually important

as it underlies human ability to carve the scene into several moving and stationary objects, used in detecting camouflage and different forms of relative motion. Computational theories of motion discontinuities (Koch et al., 1989) maintain that coherence and discontinuity are computed in parallel by the visual system. Our data do not support this fashionable theory (Vaina et al., 1990d, 1994; Vaina, 1993). We showed a double dissociation of deficits in motion coherence and motion discontinuity for stimuli presented in the visual field contralateral to the patient's lesion. One of the patients impaired on motion discontinuity, A.M.G., was so impaired on motion tasks such as speed, direction discrimination and form from relative motion that she described her perceptual problems as "I almost don't see how things are moving". However, her perception of motion coherence was normal (Vaina et al., 1994). A possible explanation for the dichotomy between motion discontinuity and motion coherence is that basic motion measurements feed into two mechanisms: one devoted to the computation of discontinuities and the other devoted to motion coherence. Both send information to higher level motion processes, such as the mechanisms for recovering 3-dimensional structure, for example. However, the motion discontinuity mechanism would also send border information to the motion coherence mechanism to set boundary conditions for spatial integration over the field, necessary for perceiving motion of objects in the scene. Anatomically, A.M.G.'s lesion appears to spare the MT, and it involves V3 which, like MT is suited for detecting moving objects and it may play a role in dynamic form perception (Zeki, 1990).

The patient A.F. provides another excellent example against a strict hierarchical organization of the motion perception system and for the existence of multiple motion pathways. A.F. was very impaired on low level motion tasks, such as speed discrimination, motion coherence and 2-dimensional form from relative motion cues (but not from static cues), yet his performance was normal on 3-dimensional structure from motion and on biological motion. How could A.F. perform well on 3-D structure from motion with severely impaired elementary motion mechanisms? Previous studies in monkeys (Siegel and Andersen, 1988) indicate that MT is crucial for the perception of 3-D structure from motion. The most popular theories of recovery of 3-D structure from motion require either the ability to precisely discriminate speed of motion or depth, but neither were available to A.F. It is possible that precise computations of speed are not necessary for this task, or that several cues fused together may be helpful to recover 3-D structure from motion (Vaina et al., 1990a) How could A.F. detect 3D structure from motion without a functioning MT? Physiological and anatomical evidence (Figure 1) suggest that the areas MST and FST supply

information to the cortex within the superior temporal sulcus (STS), which has been shown to be sensitive to the more cognitive stages of motion analysis, such as "biological motion" (Bruce et al., 1981). Indeed, A.F. had no difficulties in recognizing "biological motion" in the Johansson movie in which lights were attached to the major joints of a human actor doing several simple actions, such as walking, riding a bicycle, etc. Filmed in the dark, the only source of information was provided by the pattern of the moving lights. Normal observers, and A.F., require less than 200 msec to detect that the pattern of lights portrays a man performing some simple actions. L.M., was also normal on the recognition of biological motion (Marcar and Cowey, in press). The opposite pattern was reported in two patients, J.R. and R.J. with lesions to the anterior portion of the temporal lobe (involving the area STS) who, presented with the biological motion displays reported seeing "just dots moving" or "pool balls", but whose earlier motion mechanisms, such as speed, direction and motion coherence were intact (Vaina and LeMay, 1992; Vaina and Gross, in press).

These studies, taken together illustrate that motion perception is mediated by a complex system with several pathways and that the different motion mechanisms are not organized in a strict hierarchy.

5. COLOR VISION AND CEREBRAL ACHROMATOPSIA

Color vision is described as the ability to discriminate between lights differing in spectral composition. Together with his London collaborators Zeki was among the first to show, in functional anatomy studies by PET scan, that in normal human observers a *color sensitive center* is found in the fusiform and lingual (occipito-temporal) gyri situated in the ventromedial extrastriate cortex. This area corresponds well with the anatomical locus of damage in the cortex of neurological patients with color vision deficits, which has been described in detail in postmortem studies, as well as by structural and functional neuroimaging. This neurological condition is called cerebral achromatopsia.

Zeki was the first to report that a high proportion of neurons in area V4 of the macaque prestriate cortex respond to stimuli of a specific color, regardless of spectral composition (Zeki, 1980). This area has been proposed as a likely analogue and homologue of the human "*color center*". If this were true, functional anatomical analogies between the two species would be much more straightforward, because V4 is as much a pivotal area in the ventral pathway as the MT (V5) is in the dorsal pathway. Anatomically it would indicate that the lingual gyrus in humans whose damage leads to cerebral achromatopsia, is the homologue of the macaque V4. However,

several recent physiological and behavioral studies do not support such a homology (Merigan and Maunsell, 1993, for a review), and in fact during the last 20 years there have been scattered observations suggesting that inferotemporal cortical ablations severely disrupt color perception (Gross et al., 1972). The role of the temporal lobe in color vision was recently reassessed by Heywood and Cowey (Heywood et al., 1995) in a behavioral study of color vision of macaque monkeys after cortical ablations sparing the area V4. One group of monkeys received ablations in the temporal lobe anterior to V4, and the other to the medial occipital-temporal area corresponding to the location of lesions that, in humans, produce cerebral achromatopsia. They found that the latter group showed no impairment in color vision. In contrast, temporal lobe lesions anterior to V4 produce the same color deficits of achromatopsic patients. This study is definitive proof that the area V4 in the macaque is *not* analogous to the human *color center*, a conclusion also supported by anatomical studies in humans (Clarke and Miklossy, 1990). It also suggests that the monkey analogue of the color center in humans is in the temporal cortex anterior to the area V4.

5.1. What Do Color Deficits Reveal about Color Vision?

The patients B.L. (Kennard et al., 1995) and E.W. (Vaina, 1994), with bilateral lesions in the occipital-lobes extending into the occipital-temporal gyri and parahippocampal gyri, have been the subject of recently published cases that illustrate in detail the specific and selective deficits of color vision associated with achromatopsia. Both had upper visual field loss (E.W. more pronounced than B.L.), and E.W. was significantly more impaired on than B.L. on color discrimination (tested with the Farnsworth-Munsell 100 Hue test), color matching (tested with a tristimulus colorimeter (Wright, 1946), and color naming. Like B.L., E.W. was unable to name, upon confrontation or from memory, colors of familiar objects and also could not recognize (by name or function) any visually presented objects. Both had prosopagnosia (E.W.'s was total, compared with B.L. whose deficit was only mild) and topographic agnosia. Like other achromatopes, these patients preserved normal motion and spatial perception and binocular vision.

As B.L. exhibited partial cerebral achromatopsia, his color vision abilities were further investigated. The most interesting and novel findings were that B.L.'s ability to identify surface colors varied with changes in illumination, and these variations were predictable on the basis of the spectral composition of the light reflected by the colored samples. This is the first report of failure of color constancy in humans as a result of central achromatopsia. Simply put, color constancy implies that surface color is judged the same in spite of changes in illumination.

What else is color information used for in human vision? A clever investigation of this question by Barbur et al. (1994) revealed two possible uses of chromatic signals: the generation of perceived color or the construction and spatial representation of object structure. Subjects with normal color vision and dichromats scored very similarly on task measuring thresholds for each of the above color uses. In contrast, patients with cerebral achromatopsia displayed selective deficits. Case C. obtained higher thresholds for color discrimination than structure discrimination, the opposite being true for Case I., who had smaller color thresholds but larger structure thresholds. These authors suggest that chromatic signals can have at least two distinct functions that can be affected differentially by lesions typical of cerebral achromatopsia. It is hoped that with the rapid increase in resolution of structural neuroimaging and the increased precision of anatomical localization of function in the human brain by functional MRI, one will soon be able to determine the specific neural substrate of these two functions of the chromatic signals.

6. THE BLINDSIGHT CONUNDRUM: COVERT RECOGNITION WITH STRIATE CORTEX LESIONS?

Patients with damage to the primary visual cortex often deny any visual sensation in the scotoma corresponding to the lesion, but nonetheless they can detect and localize stimuli within the blind region when they are forced to guess. This phenomenon, termed "blindsight" (Weiskrantz et al., 1974), is a form of covert knowledge where consciousness of visual events in the scotoma is abolished. It is not at all surprising that the reports of blindsight have been met with active skepticism. Among the most frequent objections are that the phenomenon is nothing more than an artefact of scattered light, of poor control of the patient's fixation, or of using a lax criterion for the patient's guess, or that it might possibly result from the activity of the residual striate cortex. In the past, all these objections but the last have been responded to satisfactorily over the last twenty years (Marcar et al., 1995; Stoerig and Cowey, 1993). A recent study of a patient (the case G.Y.) in whom MRI scan provides no evidence for residual intact tissue in the left striate cortex, documented that G.Y. could reliably detect transient stimuli at all points in his blind hemifield along all radial directions and extending to the very periphery of the field. Thus localized sparing of the striate cortex is not a fit explanation of this patient's residual vision, and the visual localization abilities are accounted for in terms of "blindsight". Furthermore, behavioral studies in monkeys demonstrated that (Cowey and Stoerig 1995; Moore et al., 1995) complete unilateral ablation of the

striate cortex results in a selective deficit that parallels human blindsight: animals exhibited a dissociation between the ability to experience visual stimuli in the blind field and the ability to respond to those same stimuli (at the same locations in the visual field). Interestingly the response to the location of the stimuli is normal only in "forced-choice" conditions (Moore et al., 1995), that is when the monkey is trained to initiate an eye movement to locate the target. These experiments showed that, in the forced choice conditions, the monkeys made appropriate saccades to targets presented exactly in the same location in the visual field as in the standard condition in which they failed to initiate eye movements away from the fixation mark during the target presentation. This suggests that, although the information about target location was available, the signals were weak and insufficient to disengage fixation from an actively fixated target. Thus it seems that there is "real vision" in blindsight, but it is too weak to evoke explicit response; and external signals release from the contralateral inhibition are necessary to demonstrate spared capacities. Blindsight has been also reproduced in normal observers (Kolb and Brown, 1995). In a series of elegant experiments Kolb and Braun showed briefly presented stimuli in one of the four quadrants embedded in a similar background. The stimuli were either easy to see (awareness) or impossible to see (unawareness). By coupling the detection task with a measure of confidence of the choice, it was possible to correlate performance with subjective perceptual experience. Thus, for example, for displays in which observers reported no perceptual awareness, there was no correlation between confidence rating and performance, in spite of the similar good detection ability on the conditions of awareness and non awareness. The data indicate that subjects' ability to localize faint visual stimuli was significantly better than their ability to detect the stimuli (which was no better than guessing). The authors suggested that this dissociation is probably restricted to performance and not to awareness. This result is important as it shows that the dissociation between performance and awareness is neither a phenomenon restricted to vision near threshold, nor to blindsight and it "cautions against using blindsight as a model for rehabilitation by attempting to train observers to use unacknowledged visual stimuli in their blind field". Effective methods of rehabilitation in blindsight patients are required to first restore awareness, since when lacking awareness, even practiced subjects cannot gauge anything cognitively useful about the visual display (Cowey, 1995).

6. CONCLUSION

In this review, the past year's neurological studies of those visual deficits for which good theoretical and neurobiological models are available were discussed. This constrained us to focus on akinetopsia, achromatopsia and blindsight. We were interested to emphasize the contribution of the neurological studies of these deficits to the recent questioning of the parallel and hierarchical organization of the visual system in humans and monkeys. The more cognitive aspects of vision, such as prosopagnosia, visual object agnosia and deficits of spatial awareness are increasingly the focus of neurophysiological and behavioral research as well as of computational and psychophysical models and their anatomical site in humans is now being investigated by functional neuroimaging methods in normal subjects. We expect that, before long, an integrated discussion of these classical neurological vision deficits will shed light on their neural substrate and the mechanisms mediating the higher level neurological syndromes.

ACKNOWLEDGEMENTS

This research has been supported by NIH-National Eye Institute grant 2RO1 EY7861-06. Thanks are due to Dr. Shirley Wray who motivated me to write this review and to Liz Ferguson for editorial help.

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Department of Biomedical Engineering & Neurology
Boston University
and
Department of Neurology
Brigham and Women's Hospital
and Harvard Medical School
Boston, MA
U.S.A.