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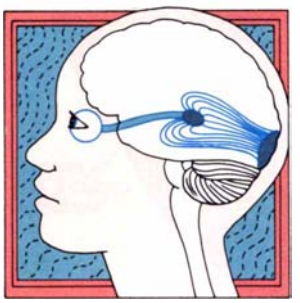
The Visual Image in Mind and Brain

In analyzing the distinct attributes of images, the brain invents a visual world. Unusual forms of blindness show what happens when specialized parts of the cortex malfunction

by Semir Zeki



The study of the visual system is a profoundly philosophical enterprise; it entails an inquiry into how the brain acquires knowledge of the external world, which is no simple matter. The visual stimuli available to the brain do not offer a stable code of information. The wavelengths of light reflected from surfaces change along with alterations in the illumination, yet the brain is able to assign a constant color to them. The retinal image produced by the hand of a gesticulating speaker is never the same from moment to moment, yet the brain must consistently categorize it as a hand. An object's image varies with distance, yet the brain can ascertain its true size.

The brain's task, then, is to extract the constant, invariant features of objects from the perpetually changing flood of information it receives from them. Interpretation is an inextricable part of sensation. To obtain its knowledge of what is visible, the brain cannot therefore merely analyze the images presented to the retina; it must actively construct a visual world. To do so, the brain has developed an elaborate neural mechanism, one so marvelously efficient that it took a century of study before anyone even guessed at its many components. Indeed, when studies of cerebral diseases began to reveal some secrets of the visual brain, neurologists initially dismissed the startling implications as improbable.

The hallmark of that machinery is a complex division of labor. It is manifested anatomically in discrete cortical areas and subregions of areas specialized for particular visual functions; it is manifested pathologically in an inability to acquire knowledge about some aspect of the visual world when the relevant machinery is specifically compromised. Paradoxically, none of this subdivision and specialization within the brain is normally evident at the perceptual level. The visual cortex thus presents us with the intellectual challenge of trying to understand how its components cooperate to give us a unified picture of the world, one that bears no trace of the division of labor within it. There is, to use an old phrase, a great deal more to vision than meets the eye.

This modern conception of the visual brain has evolved only within the past two decades. The early neurologists, starting with those who worked during the late 19th century, saw it very differently. Laboring under the false notion that objects transmitted visual codes in reflected or emitted light, they thought that an image was "impressed" on the retina, much as it would be on a photographic plate. These retinal impressions were subsequently transmitted to the visual cortex, which served to analyze the contained codes. This decoding process led to "seeing." Understanding what was seen—making sense of the received impressions and resolving them into visual objects—was thought to be a separate process that arose through the association of the received impressions with similar ones experienced previously.

This view of how the brain operates, which persisted into the mid-1970s, was therefore also deeply philosophical, although neurologists never acknowledged it as such. It divided sensing from understanding and gave each faculty a separate seat in the cortex. The origin of this dualistic doctrine is obscure, but it bears a resemblance to Immanuel Kant's belief in the two faculties of sensing and of understanding, the former passive and the latter active.

Neurologists saw evidence for their supposition in the fact that the retina connects overwhelmingly to one distinct part of the brain, the striate or primary visual cortex, also known as area V1. This connection is made with high topographic precision: V1 effectively contains a map of the entire retinal field. The retina and V1 are linked through a subcortical structure called the lateral geniculate nucleus, which contains six layers of cells. The four uppermost layers contain cells with small cell bodies and are referred to as the parvocellular layers; the two lowest layers, which have large cell bodies, are the magnocellular layers. Many years ago the late neurologist Salomon E. Henschen of Uppsala University supposed that the function of the large cells was "collecting

WHEN VIEWING AN IMAGE, distinct areas in the cortex analyze it for different visual attributes, such as color, shape and motion. "Seeing" and "understanding" occur simultaneously through the synchronized activities of these cortical areas. The world that one sees is an invention of the visual brain.

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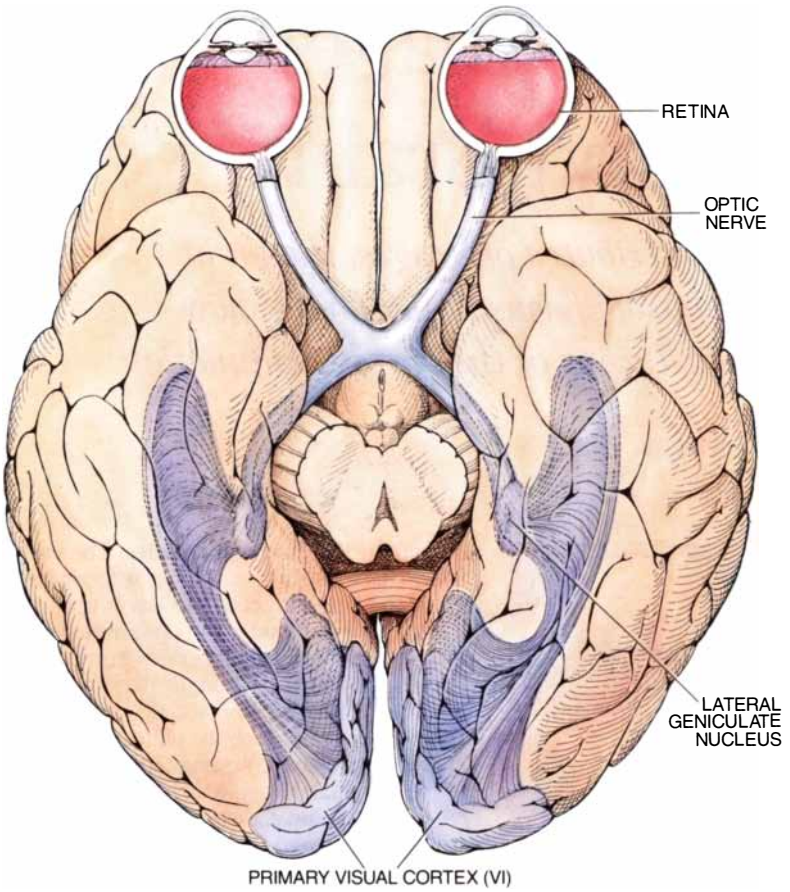
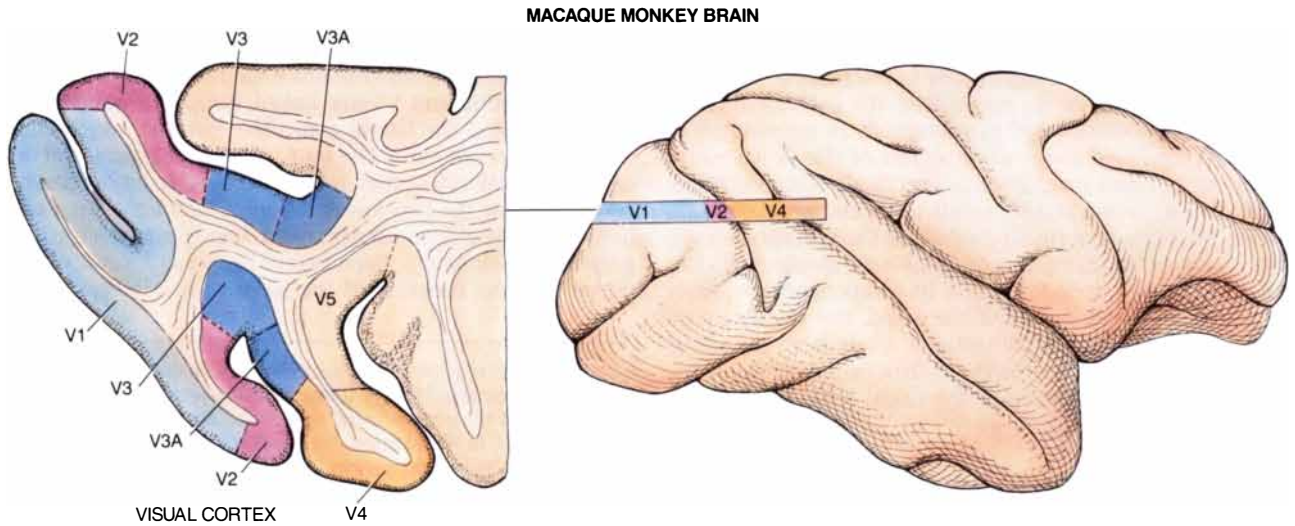


ANATOMIC AND FUNCTIONAL DIVISIONS within the visual system are the physical foundation for vision. Most connections between the retina and the visual cortex at the back of the brain pass through the lateral geniculate nucleus. In cross section, this subcortical structure has six cell layers: two in the magnocellular pathway (*M*) and four in the parvocellular pathway (*P*).

light" and that of the small cells was registering colors; his basic insight, that the anatomic subdivisions had functional implications, has assumed increasing importance in recent years.

Neurologists at the time found that lesions anywhere along the pathway connecting the retina with V1 created a field of absolute blindness, the extent and position of which corresponded precisely with the size and location of the lesion in V1. That observation led Henschen to conceive of area V1 as the "cortical retina"—the place where "seeing" occurred.

Moreover, the German psychiatrist Paul Emil Flechsig of Leipzig University had shown during the late 19th century that certain regions of the brain, among them V1, had a mature appearance at birth, whereas others, including the cortical regions surrounding V1, continued to develop, as though their maturation depended on the acquisition of experience. For Flechsig and most other neurologists, this observation implied that V1 was "the enter-



VISUAL CORTEX of the macaque monkey has been studied in detail. A cross section through the brain (*left*) at the level indicated (*right*) shows part of the primary visual cortex (V1) and some of the other visual areas in the prestriate cortex (V2–V5).

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ing place of the visual radiation into the organ of the psyche," and the areas around it were the repositories of higher "psychic" functions (*Cogitationzentren*) related to sight. Flechsig's theory found support in rather questionable evidence purporting to show that lesions in this so-called visual association cortex, unlike those in V1, might lead to "mind blindness" (*Seelenblindheit*), a condition in which subjects were thought to see but not to comprehend what they saw.

Surprisingly, it was research on the visual association cortex that ultimately compromised this dualistic concept of visual brain organization. Work undertaken in the 1970s by John M. Allman and Jon H. Kaas of the University of Wisconsin in the owl monkey and by me in the macaque monkey showed that the visual association cortex—now better referred to as the prestriate cortex—consists of many different cortical areas separated from V1 by another area, V2. A turning point in our understanding of how the brain constructs the visual image came subsequently, with my demonstration that these areas are individually specialized to undertake different tasks.

In my physiological studies, I presented macaques with a range of stimuli (colors, lines of various orientations and dots moving in different directions) and, using electrodes, monitored the activity of cells in the prestriate cortex. The results showed that all the cells in a prestriate area called V5 are responsive to motion, that most are directionally selective and that none is concerned with the color of the moving stimulus. These facts suggested to me that V5 is specialized for visual motion. (Neuroanatomic terminology is not always uniform; some investigators prefer the label MT to V5.)

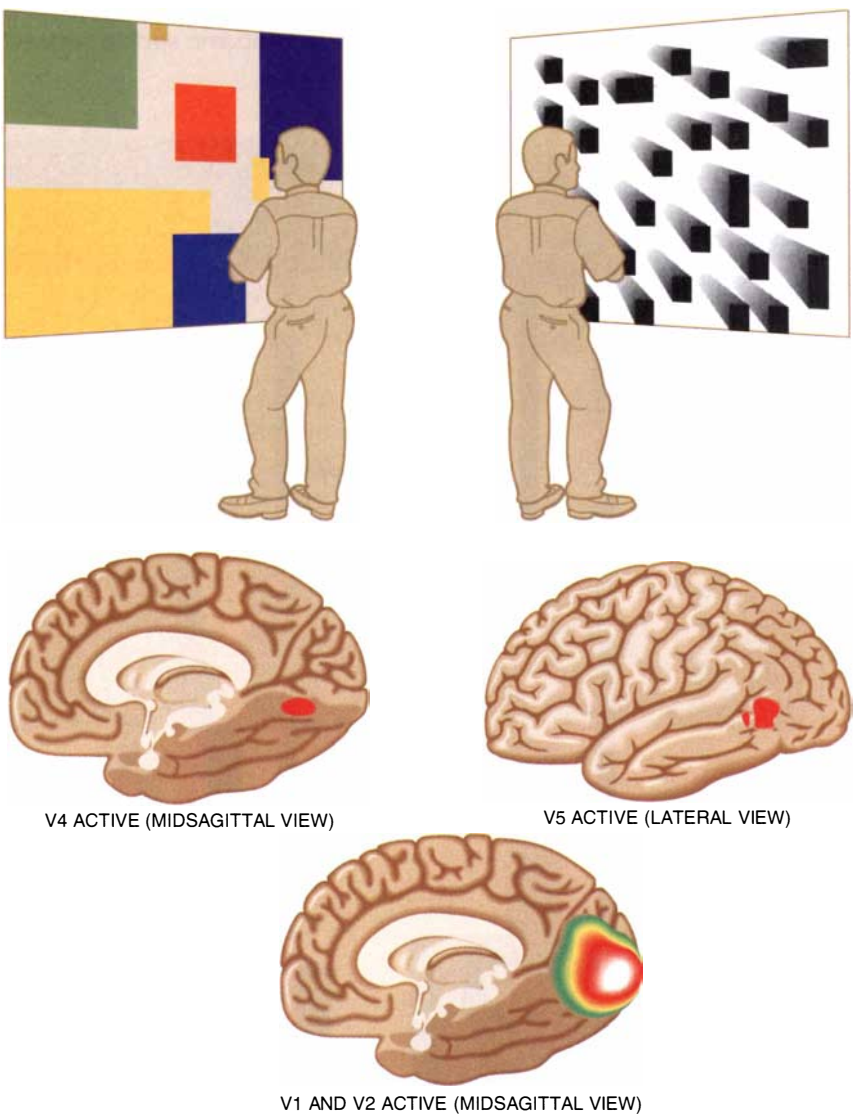
In contrast, I found that the overwhelming majority of cells in another area, V4, are to some extent selective for specific wavelengths of light and that many are selective for line orientation, the constituents of form, as well. By far most of the cells in two further adjoining areas, V3 and V3A, are also selective for form but like the cells of area V5 are largely indifferent to the color of the stimulus.

These studies led me to propose in the early 1970s the concept of functional specialization in the visual cortex, which supposes that color, form, motion and possibly other attributes of the visible world are processed separately. Because the preponderance of input to the specialized areas comes from V1, a corollary of this finding was that V1 must also show a functional specialization, as must area V2, which receives input from V1 and connects with the same specialized areas. These two areas must, in a sense, act as a kind of post office, parceling out different signals to the appropriate areas.

In recent years, new tissue-staining techniques in combination with physiological studies have provided a startling confirmation of that theory. They have also allowed us to trace these specializations from V1 throughout the prestriate cortex.

With the advent of positron emission tomography (PET), which can measure increases in regional cerebral blood flow when people perform specific tasks, my colleagues at the Hammersmith Hospital in London and I have begun to apply these findings, which were derived from experiments on monkeys, to a direct study of the human brain. We found that when normal-seeing humans view a Land color Mondrian (an abstract painting containing no recognizable objects), the highest increase in regional cerebral blood flow occurs in a structure named the fusiform gyrus. By analogy with a similar region in macaque monkeys, we refer to this cortical area as human V4. The results are very different when subjects view a pattern of moving black-and-white squares: the highest cerebral blood flow then occurs in a more lateral area, quite separate from V4, which we call human V5.

This demonstration of the separation of motion and color processing constitutes direct evidence that functional



DISSIMILAR IMAGES stimulate different regions of the visual cortex. A brightly colored Mondrian causes area V4 to become highly active, as shown by tests of regional cerebral blood flow. Black-and-white moving images trigger activity in area V5. Both types of images lead to activity in areas V1 and V2, which have less specialized functions and distribute signals to other cortical areas.

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specialization is also a feature of the human visual cortex. The PET studies reveal another interesting feature: under both conditions of stimulation, area V1 (and probably the adjoining area V2) also showed marked increases in regional cerebral blood flow. As in the monkeys, these regions, too, must be distributing signals to different areas of the prestriate cortex.

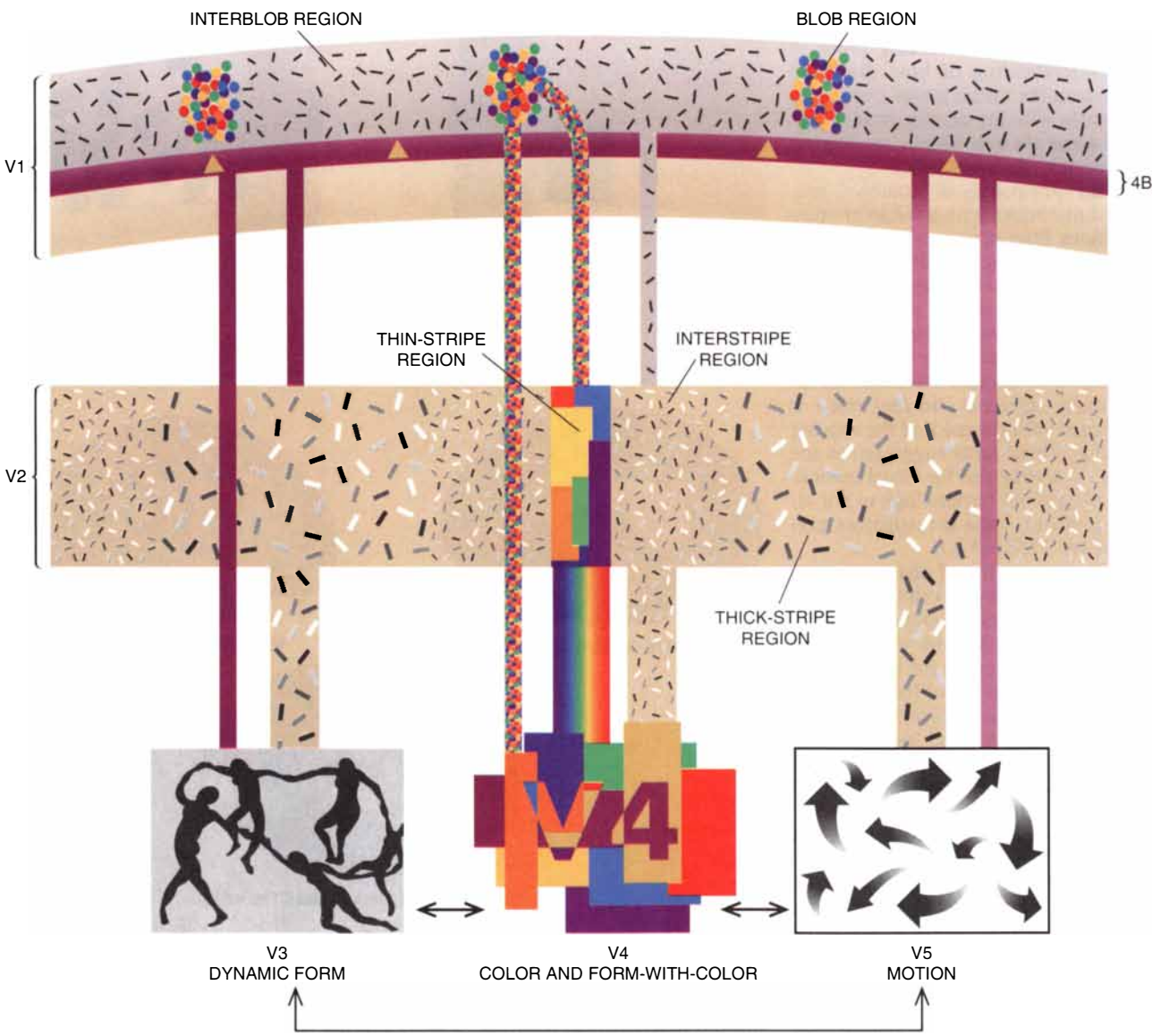
The key to the distribution system in these areas lies in their structural and functional organization. Area V1 is unusually rich in cell layers, yet it reveals an even richer architecture if one examines it with a staining technique first applied by Margaret Wong-Riley of the Medical College of Wisconsin in Milwaukee. The organelles known as mitochondria contain a metabolic enzyme called cytochrome oxidase that makes energy available to a cell. By staining a region of the brain for that enzyme, researchers can identify which cells have the greatest metabolic activity.

When so stained, the metabolic architecture of V1 is characterized by columns of cells that extend from the cortical surface to the underlying nerve tissue called white matter. If viewed in sections cut parallel to the cortical surface, these columns appear as heavily stained blobs or puffs, separated from one another by more lightly stained interblob regions. At Harvard Medical School, Margaret Livingstone and David H. Hubel found that wavelength-selective cells are concentrated in the blobs of V1, whereas form-selective cells are concentrated in the interblobs.

The columns are especially prominent in the second and third layers of V1, which receive input from the parvocellular layers of the lateral geniculate nucleus. The cells in those parts of the lateral geniculate nucleus respond in a strong, sustained way to visual stimuli, and many of them are concerned with color.

A distinct set of structures can be seen in layer 4B of V1, which receives input from the magnocellular layers of the lateral geniculate nucleus, whose cells respond transiently to stimuli and are mostly indifferent to color. Layer 4B projects to areas V5 and V3. The cells in layer 4B that connect with V5 are clustered into small patches that are isolated by cells connected to other visual areas. In short, the organization of layer 4B in V1 suggests that certain parts of it are specialized for motion perception and are segregated from regions that handle other attributes.

Like V1, area V2 has a special metabolic architecture. In the case of V2, however, that architecture takes the shape of thick stripes and thin stripes separated from one another by more



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lightly staining interstripes. As work done by Edgar A. DeYoe and David C. Van Essen of the California Institute of Technology, by Hubel and Livingstone, and by Stewart Shipp of University College, London, and me has shown, cells selective for wavelength congregate in the thin stripes, and cells selective for directional motion are found in the thick stripes. Cells sensitive to form are distributed in both the thick stripes and the interstripes.

V1 and V2 might therefore be said to contain pigeonholes into which the different signals are assembled before being relayed to the specialized visual areas. The cells in these pigeonholes have small receptive fields; that is, they respond only to stimuli falling on a small region of the retina. They also register information about only a specific attribute of the world within that receptive field. It is as though V1 and V2 were undertaking a piecemeal analysis of the entire field of view.

These facts allow us to delineate four parallel systems concerned with different attributes of vision—one for motion, one for color and two for form. The two that are computationally most distinct from each other are the motion and color systems. For the motion system, the pivotal prestriate area is V5; its inputs run from the retina, through the magnocellular layers of the lateral geniculate nucleus, to layer 4B of V1. From there the signals pass to V5, both directly and through the thick stripes of V2. The color system depends on area V4; its inputs pass through the parvocellular layers of the lateral geniculate nucleus to the blobs of V1, then proceed to V4 directly or through the thin stripes of V2.

Of the two form systems, one is intimately linked to color, and the other is independent of it. The first is based on V4 and derives its inputs from the parvocellular layers of the lateral geniculate nucleus by way of the interblobs of V1 and the interstripes of V2. The second is based on V3 and is more concerned with dynamic form—the shapes of objects in motion. It derives its inputs from the magnocellular layers of the lateral geniculate nucleus through layer 4B of V1; the signals then proceed to V3 both directly and through the thick stripes of V2.

Although these four systems are distinct, the anatomy of areas V1 and V2 offers many opportunities for the pigeonholes to communicate with one another, as do the direct connections between the specialized visual areas. Hence, there is an admixture of the parvocellular and magnocellular signals, which the prestriate areas use in different ways to execute their functions.

This remarkable segregation of functions is reflected in some of the pathologies afflicting the visual cortex. Lesions in specific cortical areas produce correspondingly specific visual syndromes that may be far less debilitating than total blindness yet still severe enough to drive patients to distraction and despair. Lesions in area V4 lead to achromatopsia, in which patients see only in shades of gray. This syndrome is different from simple color blindness: not only do such patients fail to see or know the world in color, they cannot even recall colors from a time before the lesion formed. Nevertheless, if their retinas and V1 regions are healthy, their knowledge of form, depth and motion remains intact.

Similarly, a lesion in area V5 produces akinetopsia, in which patients neither see nor understand the world in motion. While at rest, objects may be perfectly visible to them, but motion relative to them causes the objects to vanish. The other attributes of vision remain unscathed—a specificity that results directly from functional differentiation in the human visual cortex.

Given the separation of form and color in the cortex, it is perhaps a little surprising that no one has ever reported a complete and specific loss of form vision. A partial explanation is that such a deficit would require the obliteration of areas V3 and V4 to eliminate both form systems. Area V3 forms a ring around V1 and V2. Consequently, a lesion large enough to destroy all of V3 and V4 would almost certainly destroy V1 as well and thus cause total blindness.

Some patients with lesions in the prestriate cortex do suffer from a degree of form imperception, often coupled with achromatopsia. These people commonly experience far greater difficulty when identifying stationary forms than when the same forms are in motion. They frequently prefer watching television to watching the real world, because television is dominated by moving images. When faced with stationary objects, these patients often resort to the strategy of moving their heads to simplify the task of identification. These observations suggest that they acquire their knowledge of forms through the dynamic form system based in area V3.

The functional specialization in the visual cortex also manifests itself in a syndrome that I have called the chromatopsia ("color vision") of carbon monoxide poisoning. This condition has been described sporadically but not infrequently in the medical literature, although it was never taken seriously until the functional specialization was discovered. Some people who survive the lethal effects of smoke inhalation during fires often suffer diffuse cortical damage from carbon monoxide poisoning, which deprives tissues of oxygen. As a result, these patients often have vision that is severely compromised in all respects except one: their color vision is affected only mildly if at all. Because color is the only kind of visual knowledge available to them, the patients try—often unsuccessfully—to identify all objects solely on the basis of color. They may, for example, misidentify all blue objects as "ocean."

The precise cause of this strange chromatopsia is unknown. The metabolically active blobs of V1 and the thin stripes of V2, both of which are concerned with color, do have unusually high concentrations of blood vessels nourishing them. It is therefore probable that these regions are relatively spared from damage because their rich blood supply renders them less vulnerable to oxygen deprivation.

In summary, then, we know that a total lesion in area V1 produces a complete inability to acquire any visual information and that a lesion in one of the specialized areas makes a corresponding attribute of the visual world inaccessible and incomprehensible. What would happen, we might ask, if signals from the lateral geniculate nucleus were routed directly to the specialized areas, thus bypassing V1 altogether? Nature has actually done that experiment for us, and the resulting phenomenon provides more important insights into the functioning of the visual cortex.

The phenomenon is known as blindsight; it was first described by Ernst Pöppel of the University of Munich and his colleagues and later studied in great detail by Lawrence Weiskrantz of the

FOUR PERCEPTUAL PATHWAYS within the visual cortex have been identified. Color is seen when wavelength-selective cells in the blob regions of V1 send signals to specialized area V4 and also to the thin stripes of V2, which connect with V4. Form in association with color depends on connections between the interblobs of V1, the interstripes of V2 and area V4. Cells in layer 4B of V1 send signals to specialized areas V3 and V5 directly and also through the thick stripes of V2; these connections give rise to the perception of motion and dynamic form.

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University of Oxford and his colleagues. People with this condition are totally blind because of lesions in area V1. Yet if they are forced to guess, they can discriminate correctly among a wide variety of visual stimuli. They can, for example, distinguish between motion in different directions or between different wavelengths of light. Their abilities are imperfect and not completely reliable, but they are better than random guessing. Nevertheless, blindsight patients are not consciously aware of having seen anything at all, and they are often surprised that their "guesses" should have been so accurate.

The basis for this discrimination almost certainly resides in a small but direct connection between the lateral geniculate nucleus and the prestriate cortex, as uncovered by Masao Yukie of the Tokyo Metropolitan Institute for Neurosciences and by Wolfgang Fries of the University of Munich. Alternatively, some other as yet undiscovered subcortical connection to the specialized areas may be responsible. In any event, neurologists have good reason to suppose that in blindsight patients, visual signals reach the prestriate cortex.

Blindsight patients are people who "see" but do not "understand." Because they are unaware of what they have seen, they have not acquired any knowledge. In short, their "vision," which can be elicited only in laboratory situations, is quite useless. Thus, for the visual cortex to do its job of acquiring a knowledge of the world, a healthy V1 area is essential. V1 (and, by extension, V2) may be necessary because it begins to process information for further refinement by the specialized areas or because the results of the processing performed by the specialized areas are referred back to it.

The clinical literature holds many other examples that illuminate how the preprocessing in areas V1 and V2 may contribute directly and explicitly to perception. Damage to V5 can destroy the ability to discriminate the direction or coherence of motions. Yet as Robert F. Hess of the University of Cambridge and his colleagues found, such akinetopsic patients may still be aware that motion of some type is taking place, presumably because of signaling by cells in V1 and V2 (and possibly in other areas that receive magnocellular pathway signals). Similarly, an achromatopsic patient with a V4 lesion, whom I studied with Fries, could discriminate between different wavelengths because of his largely intact V1 even though he could no longer interpret the wavelength information as color.

Further insights come from a comparison of the residual form-vision capacities in two other patients who have cortical lesions that are, in a way, complementary. The first patient has a diffuse cortical lesion, caused by carbon monoxide poisoning, that affects area V1. He has terrific difficulty copying even simple forms, such as geometric shapes or letters of the alphabet, because the form-detecting system in his V1 area is so severely compromised.

The second patient has an extensive prestriate lesion from a stroke that has generally spared area V1. He can reproduce a sketch of St. Paul's Cathedral with greater skill than many normal people, although it takes him a great deal of time to do so. Yet this patient has no comprehension of what he has drawn. Because his V1 system is largely intact, he can identify the local elements of form, such as angles and simple shapes, and accurately copy the lines he sees and understands. The prestriate lesion, however, prevents him from integrating the lines into a complex whole and recognizing it as a building. The patient sees and understands only what the limited capacity of his intact system allows.

The residual capacity in such patients unmasks an important feature of the organization of the visual cortex, namely, that none of the visual areas—not even "post office" areas V1 and V2—serves merely to relay signals to other areas. Instead each is part of the machinery that actively transforms the incoming signals and may contribute explicitly, if incompletely, to perception.

The profound division of labor with-

The World Seen through a Damaged Cortex

Damage to specialized regions of the cortex can cause strange types of blindness in which patients lose the ability to see just one attribute of the visual world, such as color, form or motion. Artwork produced by some of these patients offers glimpses into their view of the world, as well as into the workings of the visual cortex itself.

A patient with damage to the color pathways in the cortex lost all color vision. In his drawings, a banana, a tomato and green leaves all have similar colors.

This stroke patient suffered damage to the prestriate cortex that impaired his perception of form. He could copy a drawing with great skill, but he was unable to understand that the lines he had drawn produced an image of St. Paul's Cathedral.

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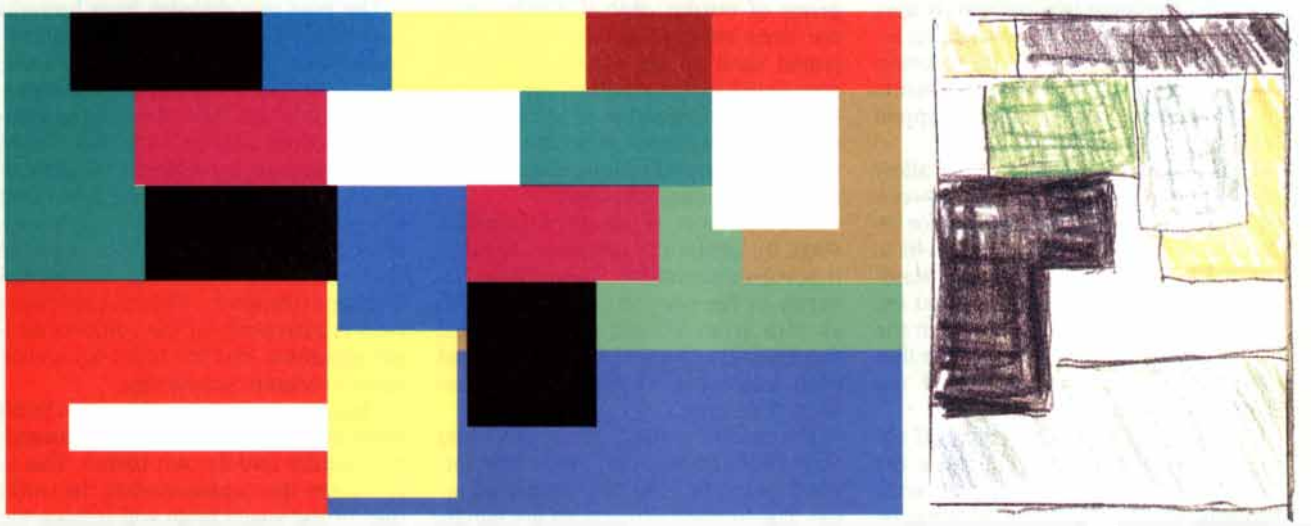
in the visual cortex naturally raises the question of how the specialized areas interact to provide a unified image. The simplest way would be for all the specialized areas to communicate the results of their operations to one master area, which would then synthesize the incoming information. Philosophically, that solution begs the question, because one must then ask who or what looks at the composite image, and how it does so. That problem is beside the point, however, because the anatomic evidence shows no single master area to which all the antecedent areas exclusively connect. Instead the specialized areas connect with one another, either directly or through other areas.

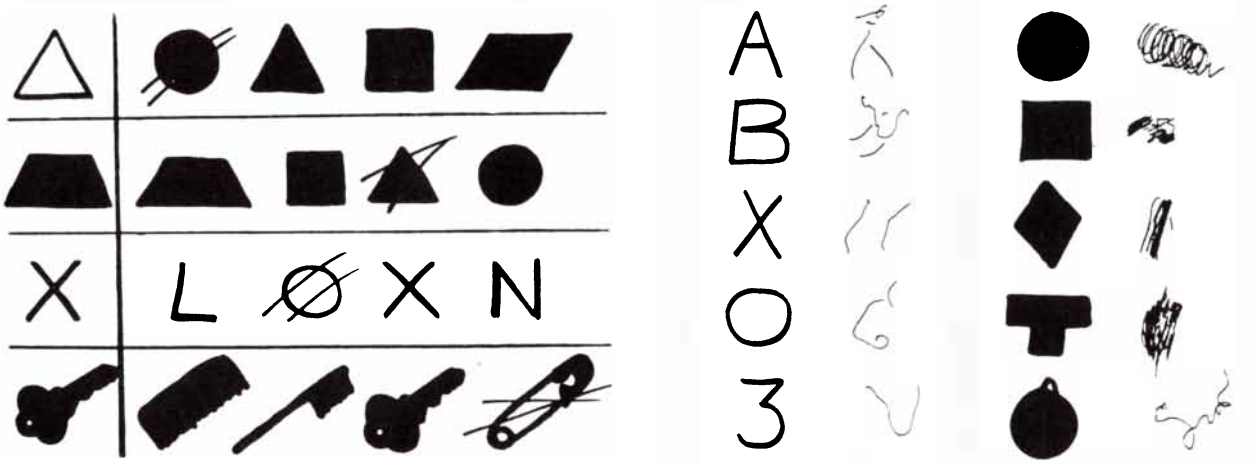
For example, areas V4 and V5 connect directly and reciprocally with each other. Both of them also project to the parietal and temporal regions of the brain, but as my work with my colleagues has shown, the outputs from each area occupy their own unique territory within the receiving region. Direct overlap between the signals from V4 and V5 is minimal. It is as if the cortex wishes to maintain the separation of the distinct visual signals—a strategy it also employs in memory and other systems [see "Working Memory and the Mind," by Patricia S. Goldman-Rakic, page 110]. Any integration of the signals within the parietal or temporal regions must occur through local "wiring" that connects the inputs.

In fact, integration of the visual information is a monumental task that necessitates a vast network of anatomic links between the four parallel systems at every level, because each level contributes explicitly to perception. Integration also creates some formidable problems. To understand coherent motion, for example, the brain must determine which features in the field of view are moving in the same direction and at the same speed. The motion-sensing cells in the specialized areas are able to make those comparisons because they have larger receptive fields than do their antecedent counterparts in V1.

Yet because their receptive fields are larger, these cells are inherently less efficient at pinpointing the position of any one stimulus within the visual field. For the brain to make spatial sense of the integrated information, the information must somehow be referred to an area that has a more precise topographic map of the retina and hence of the visual field. Of all the visual areas, the



When an achromatopsic (color-blind) patient was shown a Land color Mondrian (*left*) and asked to reproduce it, he was able to copy the shapes in the painting successfully. The colors within the blocks eluded him (*right*).



This patient sustained damage from carbon monoxide poisoning and lost his perception of form. At the left, he was asked to choose which of four selections was identical to a given shape; as the marks on his choices reveal, he could not identify them correctly. At the right, his inability to copy simple shapes is displayed.

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KANIZSA TRIANGLE consists of illusory contours. A normal visual cortex sees a triangle even though interconnecting lines are missing. Such illusions show that the visual cortex must resolve conflicts between different functional areas.

one with the most precise map is area V1, followed by V2. The specialized areas must therefore send information back to V1 and V2 so that the results of the comparisons can be mapped back onto the visual field.

Reentrant connections, which allow information to flow both ways between different areas, are also essential for resolving conflicts between cells that have different capabilities and are responding to the same stimulus. A good example of such conflicts is found in the responses of cells in V1 and V2 to illusory contours, such as those of the Kanizsa triangle.

In this famous illusion, a normal observer perceives a triangle among the presented shapes even though the lines forming the triangle are incomplete; the brain creates lines where there are none. As Rudiger von der Heydt and Esther Peterhans of the Zurich University Hospital have demonstrated, the form-selective cells in V1 do not respond to the illusion and do not signal that a line is present. The V2 cells receive their inputs from V1, but because the V2 cells have larger receptive fields and more analytic functions, they do respond to the illusion by "inferring" the presence of a line. To settle the conflict, the V2 cells must have reentrant inputs to their counterparts in V1.

Another difficulty that arises from the process of integration is the binding problem. Cells responding to the same object in the field of view may be scattered throughout V1. Something must therefore bind together the signals from those cells so that they are treated as belonging to the same object and not separate ones. The problem becomes even more thorny when cells in two or more visual areas respond to different attributes of the same object.

One way to resolve the problem is for the cells to fire in temporal synchrony. In practice, such synchrony does occur, at least to some extent, among cells that are anatomically connected to one another, as work by Wolf J. Singer of the Max Planck Institute for Brain Research in Frankfurt and his colleagues has shown. We must then face the problem, however, of who or what determines that the firing should be synchronous. Reentrant inputs provide at least a partial solution by linking the output of one area to other areas that sent it information.

These problems have led me and my colleagues to develop a theory of multistage integration. It hypothesizes that integration does not occur in a single step through a convergence of output onto a master area, nor does integration have to be postponed until all the visual areas have completed their individual operations. Instead the integration of visual information is a process in which perception and comprehension of the visual world occur simultaneously.

The anatomic requirement for multistage integration is immense, because it involves reentrant connections between all the specialized areas as well as with areas V1 and V2, which feed them signals. Our studies indicate that such a network of reentrant connections does exist.

The reentrant inputs to areas V1 and V2 differ fundamentally from their forward connections to the specialized areas. The forward projections are patchy and discrete, because segregated groups of cells in V1 and V2 send their outputs to specialized areas with corresponding visual attributes. The return projections, however, are diffuse and fairly nonspecific. For example, whereas V5 receives input only from select groups of cells in layer 4B of V1, the return input from V5 to layer 4B is diffuse and encompasses the territory of all the cells in the layer, including ones that project into V3. This reentrant system can thus serve three purposes simultaneously: it can unite and synchronize the signals for form and motion found in two different visual pathways; it can refer information about motion back to an area with an accurate topographic map; it can integrate motion information from V5 with form information on its way to V3.

Similarly, whereas the output from V2 to the specialized areas is highly segregated, the return input from those areas to V2 is diffuse. V4 projects back not only to the thin stripes and the interstripes, from which it receives its input, but also to the thick stripes, from which it does not. This reentrant system can therefore help unite signals dealing with form, motion and color.

It is becoming increasingly evident that the entire network of connections within the visual cortex, including the reentrant connections to V1 and V2, must function healthily for the brain to gain complete knowledge of the external world. Yet as patients with blindsight have shown, knowledge cannot be acquired without consciousness, which seems to be a crucial feature of a properly functioning visual apparatus. Consequently, no one will be able to understand the visual brain in any profound sense without tackling the problem of consciousness as well [see "The Problem of Consciousness," by Francis Crick and Christof Koch, page 152].

The past two decades have brought neurologists many marvelous discoveries about the visual brain. Moreover, they have led to a powerful conceptual change in our view of what the visual brain does and how it accomplishes its functions. It is no longer possible to divide the process of seeing from that of understanding, as neurologists once imagined, nor is it possible to separate the acquisition of visual knowledge from consciousness. Indeed, consciousness is a property of the complex neural apparatus that the brain has developed to acquire knowledge.

Thus, our inquiry into the visual brain takes us into the very heart of humanity's inquiry into its own nature. This is not to say that understanding the workings of the visual brain will resolve the problem of consciousness—far from it. But it is a good beginning.

FURTHER READING

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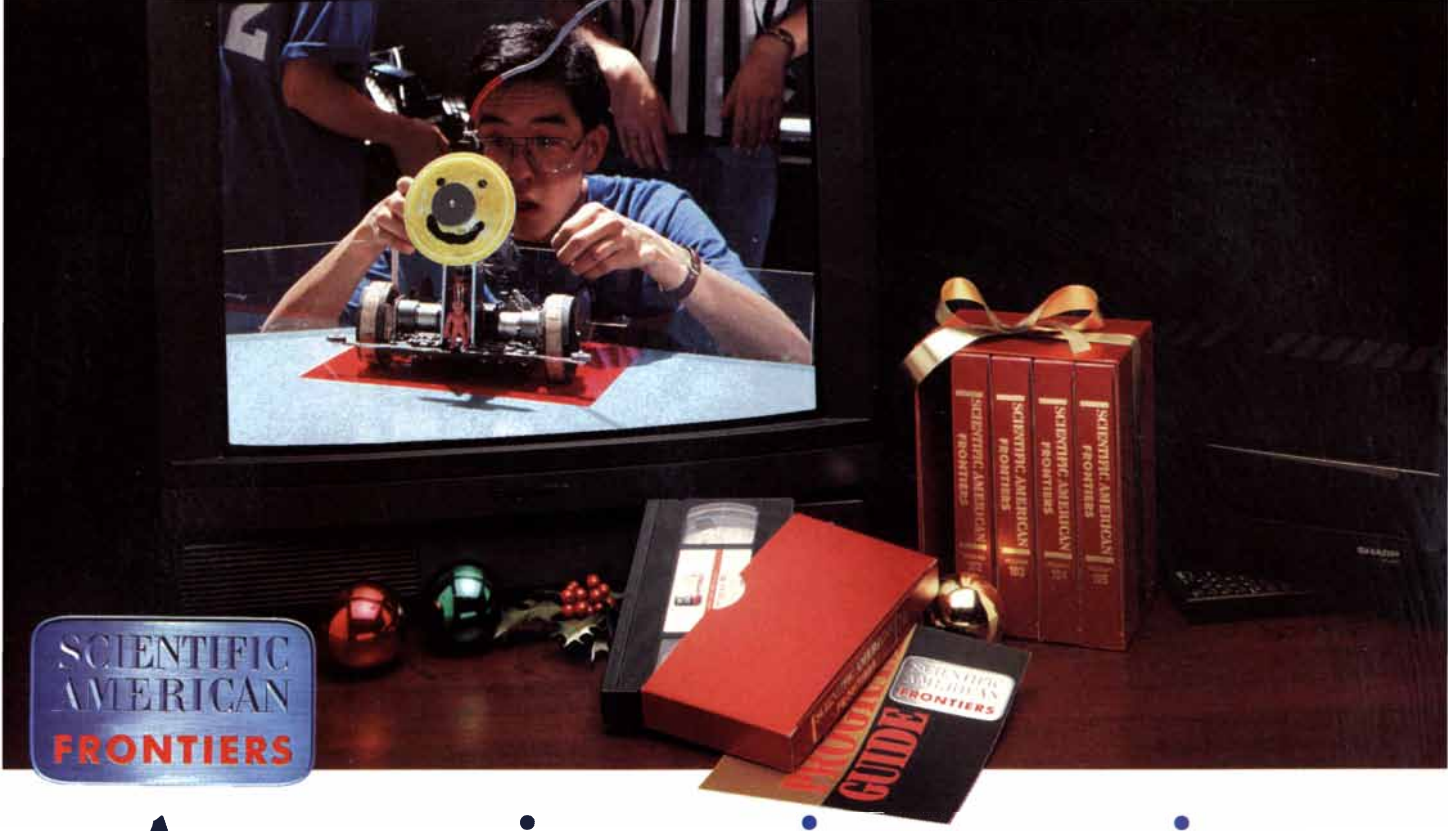

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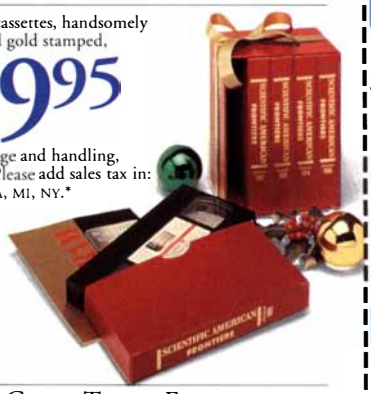

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