Chapter 8

VISUAL PERCEPTION AND VISUAL IMAGERY

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PATIENT-BASED APPROACHES TO COGNITIVE NEUROSCIENCE
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Primates are visual creatures, and humans are no exception to this generalization. If one surveys our cortex and asks which areas are either partially or exclusively devoted to processing information from our eyes, one finds that about half of the cortex is involved in vision. Vision is the main function of occipital cortex and occupies much of parietal and temporal cortex as well. Even the most anterior parts of the brain include areas dedicated to eye-movement programming and visual working memory. One consequence of having this far-flung visual network is that lesions to many different parts of the brain can affect vision. The nature of the visual disturbance depends on the particular contribution that the damaged area would normally have made to vision.

Several chapters in this book are devoted to specific visual disorders that result from damage to high-level visual areas—that is, visual areas that are several synapses past primary visual cortex. These disorders include visual object agnosia (Chap. 9); prosopagnosia (Chap. 10); certain disorders of reading (Chap. 20), neglect (Chaps. 14 and 15), and visuo, spatial disorders (Chap. 11). The goal of this chapter is to review cortical visual processing at stages prior to these high-level functions. The chapter also considers cognitive disorders of mental imagery, or the activation of these visual representations endogenously as a medium of thought. In each case, the disorders arising from damage to these stages of vision is reviewed with respect to their main behavioral features, associated lesion sites, and implications for our understanding of normal vision.

VISUAL PERCEPTION

Damage to Primary Visual Cortex and Its Afferents

Although considerable information processing is carried out in the retina and thalamus, the visual disorders relevant to behavioral neurology generally involve brain damage at the level of primary visual cortex and beyond. Because the vast majority of visual information is processed through primary visual cortex on its way to higher-level perceptual areas, destruction of primary visual cortex causes cortical blindness. Partial destruction causes partial blindness, and the location of the lesion within primary visual cortex corresponds to the location of the blind spot, or visual field defect, in a highly systematic way that reflects the retinotopy of primary visual cortex. With vascular lesions, it is common for some or all of the primary cortex in one hemisphere to be damaged while the opposite hemisphere is unaffected. This results in blindness restricted to one-half of the visual field, or *hemianopia*. It is sometimes called *homonymous hemianopia* to indicate that the blind regions are the same regardless of which eye is used to see.

As shown in Fig. 8-1, visual field defects can be used to deduce the location of the lesion and were regularly used in this way before the days of computed tomography (CT). Homonymous visual field defects imply that the lesion is posterior, because input from the two eyes merges anteriorly. Because the left optic radiation projecting to left visual cortex represent only the right visual field, and vice versa, visual field

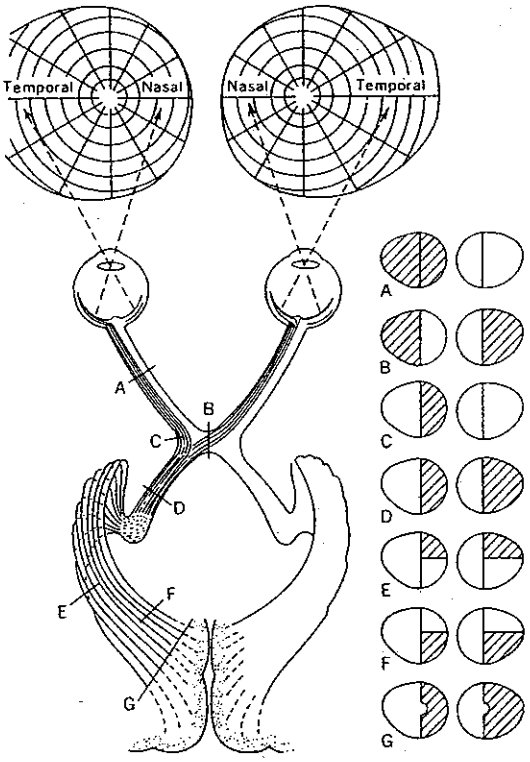

Figure 8-1
Correspondences between location of lesion within the visual system and pattern of visual field defects. (From Homans J: A Textbook of Surgery. *Springfield, IL: Charles C Thomas, 1945, with permission.)*

defects also reveal the side of the lesion. The altitude of the visual field defect is also informative, with lower-quadrant blindness, or *quadrantanopia,* suggesting a parietal or superior occipital lesion, because of the dorsal course of the pathways from thalamus to cortex, and upper quadrantanopia suggesting a temporal or inferior occipital lesion, because of the ventral course.

Prosopagnosia (see Chap. 10) was first localized on the basis of the visual field defects reported in a large set of cases (Meadows, 1974). Most cases reported a upper-left quadrantanopia, some with defects in the upper right as well. From this, Meadows was able to infer that the critical substrates for face recognition are in the right temporal cortex or bilateral temporal cortices in most people—a conclusion that has withstood the tremendous increase in localizing capability as structural and functional brain imaging became available.

Although hemianopia and quadrantanopia are, by definition, blindness in the regions of the visual field represented by damaged visual cortex or its afferents, there are patients who retain some visual functions in these regions. *Blindsight* is the appropriately oxymoronic term applied to this puzzling phenomenon. The preserved perceptual abilities may be limited to localization of light and movement, but in some cases the limitations go well beyond this.

In one very thoroughly studied case of blindsight, Weiskrantz and colleagues (1986) found relatively preserved ability to point to the locations of visual stimuli, to detect movement, to discriminate the orientations of lines and gratings, and to discriminate large shapes such as X and O despite the patient's denial that he could see anything. The mechanism of blindsight has been a controversial topic. One possibility is that it simply reflects incomplete damage to visual cortex (see, e.g., Fendrich and coworkers, 2001), although this seems unlikely given that hemidecorticate patients have shown blindsight. Other explanations involve pathways to visual association cortex that bypass primary visual cortex. One such possibility is that blindsight is mediated by the subcortical visual system, which consists of projections from the retina to the superior colliculus and pulvinar and its projections to secondary cortical visual areas (e.g., Rafal et al., 1990). Alternatively, there may be sparse projections within the cortical visual system, from the lateral geniculate nucleus directly to visual association cortex (Stoerig and Cowey, 1997).

Damage to Surrounding Association Cortex

Surrounding primary visual cortex is additional modality-specific visual cortex that receives its input principally from primary visual cortex. From single-cell recording and other invasive measures used in animals, it is known that this region is functionally a mosaic of areas, each of which represents the visual field with some degree of retinotopy and has largely reciprocal projections to particular sets of other visual areas (see Zigmond et al., 1999). It is assumed that this multiplicity of areas is there for some purpose and that

each area probably analyzes different aspects of the input, although this assumption has been fully validated in only a couple of cases—areas that subserve color vision and motion vision.

Primate neurophysiology has shown that neurons in area V4 of the monkey brain are highly selective for color and indeed respond to color per se rather than wavelength (Zeki, 1983). (The difference can be appreciated by considering that the green color of a plant, for instance, remains at least roughly constant across ambient lighting conditions containing widely differing wavelengths, which result in different wavelengths reflecting off the plant's surface and stimulating the retina.) That a homologous area exists in humans and is vulnerable to damage is suggested by the disorder *cerebral achromatopsia,* color blindness due to brain damage. Achromatopsic patients report that the world seems drained of color, like a black-and-white movie. In other respects, their vision may be at least roughly normal. For example, they may have good acuity, motion and depth perception, and object recognition. It should be added that problems with face, object and printed word recognition do sometimes accompany achromatopsia, but they are often transient and are likely to be caused by impairment to areas neighboring the color area. Cases in which the color vision impairment is truly selective imply that there is a brain region dedicated to color perception—that is, necessary for color perception and not for other aspects of vision.

In some cases, a unilateral lesion will result in color loss in just one hemifield, consistent with retinotopic mapping of the area responsible for color vision. A particularly selective and well-studied case of this was described by Damasio et al. (1980). Although acuity, depth perception, motion perception, and object recognition was normal in both hemifields, they differed strikingly for color perception:

> *He was unable to name any color in any portion of the left field of either eye, including bright reds, blues, greens and yellows. As soon as any portion of a colored object crossed the vertical meridian, he was able instantly to recognize and accurately name its color. When an object such as a red flashlight was held so that it bisected the vertical meridian, he reported that the hue of the right half appeared normal while the left half was gray.*

He was also unable to match colors in the left visual field.

The lesions in achromatopsia are usually on the inferior surface of the temporooccipital region, in the lingual and fusiform gyri. In full achromatopsia they are bilateral, and in hemiachromatopsia they are confined to the hemisphere contralateral to the color vision defect. This localization accords well with functional neuroimaging studies in which the substrates of color perception have been isolated by comparing cerebral activation patterns while subjects view colored displays to patterns resulting when gray-scale versions of the same displays are viewed (e.g., Zeki et al., 1991). Chapter 20 reviews achromatopsia in further detail, as well as distinguishing it from a number of other disorders of color cognition.

Single-cell recording has also been used to elucidate the neural systems of motion perception in the monkey. Area MT (for middle temporal) contains neurons whose response properties suggest a primary role in motion perception. Consistent with this, humans with damage in the homologous region have developed *cerebral akinotopsia* (see Zeki, 1993, for a review). By far the best-studied case is that of Zihl et al. (1983). This was case L.M., a 43-year-old woman who, following bilateral strokes in the posterior parietotemporal and occipital regions, was left with but one major impairment, namely the complete inability to perceive visual motion. Zihl et al. (1983) tested L.M.'s visual perception in a variety of simple experimental tasks and compared her performance with that of normal subjects. In her color and depth perception, object and word recognition, and a variety of other visual abilities tested by these authors, L.M. did not differ significantly from normal subjects. In addition, her ability to judge the motion of a tactile stimulus (wooden stick moved up or down her arm) and an auditory stimulus (tone-emitting loudspeaker moved through space) was also normal. In contrast, her perception of direction and speed of visual motion in horizontal and vertical directions within the picture plane and in depth was grossly impaired.

In her everyday life she was profoundly affected by her visual impairment. When she was pouring tea or coffee, the fluid appeared to be frozen, like a glacier. Without being able to perceive movement, she could not stop pouring at the right time and frequently filled the cup to overflowing. She found it difficult to follow conversations without being able to see the facial and mouth movements of each speaker, and gatherings of more than two other people left her feeling unwell and insecure. She complained that "People were suddenly here or there but I have not seen them moving." The patient could not cross the street because of her inability to judge the speed of a car. "When I see the car at first, it seems far away. But then, when I want to cross the road, suddenly the car is very near." She gradually learned to estimate the distance of moving vehicles by means of the sound as it became louder.

As with achromatopsia, the existence of akinotopsia implies a high degree of cerebral specialization, with one cortical area being necessary for motion perception and not necessary for other aspects of perception. Although L.M.'s lesions were fairly large and encompassed both parietal and temporal cortex, the critical lesion site has been inferred to be the posterior middle temporal gyrus. Functional neuroimaging studies of motion perception, comparing brain activation patterns to moving and static displays, show their maximum in this same region (Zeki et al., 1991).

An Organizing Framework for Higher-Level Visual Disorders

Vision has two main goals, the identification of stimuli and their localization. Although a bit of an oversimplification, this dichotomy has provided a useful organizing framework for the neuropsychology of high-level vision. The two goals, sometimes abbreviated as *what* and *where,* are achieved by relatively independent and anatomically separate systems, located in ventral and dorsal visual cortices, respectively, as shown in Fig. 8-2. These have been termed *the two cortical visual systems* (Ungerleider and Mishkin, 1972).

Note that the color and motion disorders discussed in the previous section fit naturally into this framework: color is an aspect of appearance that is useful for object recognition but plays little role in spatial function. The critical lesion site for achromatopsia lies on the ventral surface of the brain. Motion is, by its very nature, a spatial property—change of location over time—and is one of the most powerful cues for summoning spatial attention. The critical lesion site for akinotopsia is dorsolateral to this, in the posterior temporal lobe.

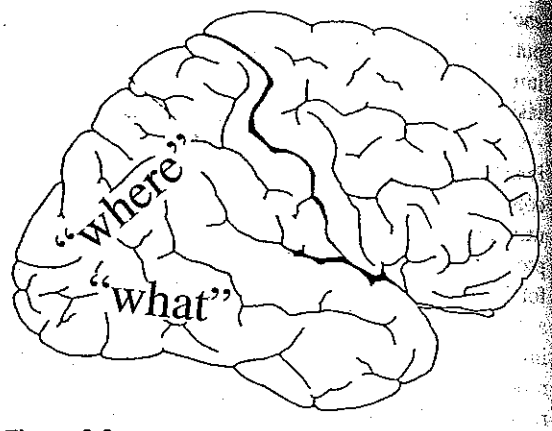

Figure 8-2
The two cortical visual systems: Dorsal visual areas are particularly important for spatial or "where" processing, and ventral visual areas are particularly important for appearance or "what" processing.

Damage further along the dorsal and ventral visual streams is responsible for a variety of neurobehavioral syndromes. The disorders of spatial perception and attention discussed in Chaps. 11, 14, and 15 result from damage to posterior parietal cortex, part of the dorsal *where* route, whereas the disorders of object and face recognition discussed in Chaps. 9 and 10 result from damage to inferior temporal cortex, part of the ventral *what* route.

VISUAL MENTAL IMAGERY

The most obvious function of the cortical visual system is the analysis of retinal inputs. Yet under some circumstances it is also used in thinking, as when we generate a visual image from memory. Brain damage can affect the process of generating a visual mental image in two ways: by impairing the visual representations themselves or by impairing the process of activating those representations in the absence of a stimulus.

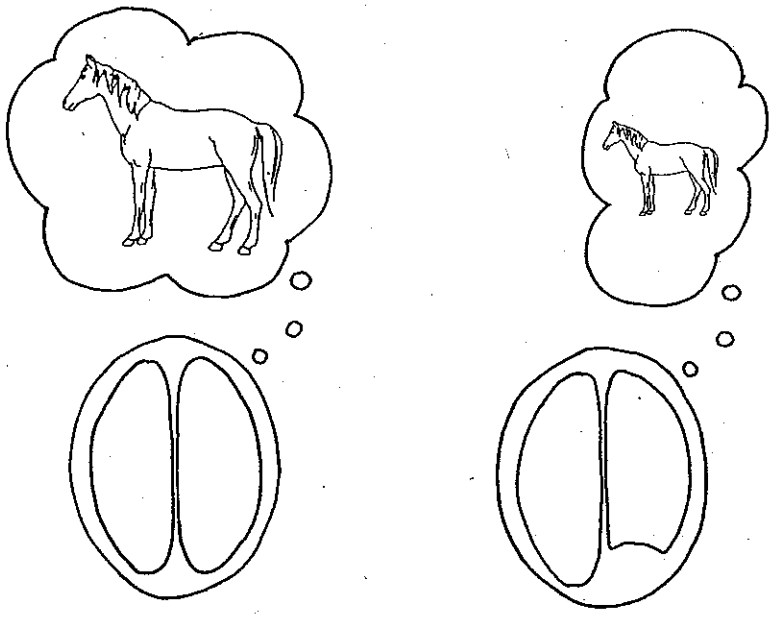

Figure 8-3
Depiction of the effects of unilateral occipital lobectomy on the visual angle of the mind's eye. (From Farah MJ, in Gazzaniga MS (ed): The Cognitive Neurosciences. *Cambridge, MA: MIT Press, 1996, with permission.)*

Disorders of Image Representation

If imagery and perception are both impaired after brain damage, this suggests that the functional locus of damage is the representations of visual appearance used by both. There are many reports of parallel impairments of imagery and perception, and these have attracted interest for what they can tell us about mental image representation. Specifically, they imply that mental imagery shares representations with the cortical visual system.

A clear-cut example of parallel imagery-perception impairment comes from a study comparing visual and "imaginal" fields. We were able to test an epileptic woman before and after a right occipital lobectomy. If mental imagery consists of activating representations in the occipital lobe, then it should be impossible to form images in regions of the visual field that are blind due to occipital lobe destruction. This predicts that after surgery, she should have both a narrower visual field and a narrower imaginal field. By asking her to report the distance of imagined objects such as a horse, breadbox, or ruler when they are visualized as close as possible without "overflowing" her imaginal field, we could compute the visual angle of that field. We found that the size of her biggest possible image was reduced after surgery, as represented in Fig. 8-3. Furthermore, by measuring maximal image size in the vertical and horizontal dimensions separately, we found that only the horizontal dimension of her imaginal field was significantly reduced. These results provide strong evidence for the use of occipital visual representations during imagery.

Other parallels have been noted as well—for example, between disorders of color perception and disorders of color imagery, between left visual neglect and

inattention to the left sides of mental images, and between the ability to recognize objects from their visual appearance and the ability to imagine their appearance. A fuller discussion of these findings, as well as cases of visual impairment in which imagery ability is not affected, may be found in Farah (2000).

Disorders of Image Generation

In the absence of visual perceptual disorder, visual imagery may be impaired because of damage affecting image generation ability. Patients with an image generation deficit are disproportionately impaired at answering questions such as "Which is bigger, a grapefruit or a cantaloupe?" or "Does a kangaroo have a short or a long tail?" compared to questions that do not evoke imagery such as "Do kangaroos wash their food before eating it?" Other indications are that their drawing from memory is sketchy despite good copying ability, and their ability to report the color of objects from memory depends upon the availability of verbal associations (e.g., the colors of the sky, lemons, and fire engines can be retrieved without imagery, but the color of a coke can, a mailbox, or a peanut cannot). Some typical cases include those of Farah and coworkers (1988), Goldenberg (1992), Grossi and colleagues (1986), and Riddoch (1990). The critical lesion site in these cases appears to be left temporooccipital cortex (see Farah, 1995, for a review of lesion and neuroimaging data).

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Chapter 9

VISUAL OBJECT AGNOSIA

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The term *visual object agnosia* refers to the impairment of object recognition in the presence of relatively intact elementary visual perception, memory, and general intellectual function. This chapter reviews the different subtypes of agnosia, their major clinical features and associated neuropathology, and their implications for cognitive neuroscience theories of visual object recognition.

The study of agnosia has a long history of controversy, with some authors doubting that the condition even exists. For example, Bay[1] suggested that the appearance of disproportionate difficulty with visual object recognition could invariably be explained by synergistic interactions between mild perceptual impairments on the one hand and mild general intellectual impairments on the other. The rarity of visual object agnosia has contributed to the slowness with which this issue has been resolved, but several decades of careful case studies have now shown, to most people's satisfaction, that agnosic patients may be no more impaired in their elementary visual capabilities and their general intellectual functioning than many patients who are not agnosic. Therefore, most current research on agnosia focuses on a new set of questions. Are there different types of visual object agnosia, corresponding to different underlying impairments? At what level of visual and/or mnestic processing do these impairments occur? What can agnosia tell us about normal object recognition? What brain regions are critically involved in visual object recognition?

APPERCEPTIVE AGNOSIA

Lissauer[2] reasoned that visual object recognition could be disrupted in two different ways: by impairing visual perception, in which case patients would be unable to recognize objects because they could not see them properly, and by impairing the process of associating a percept with its meaning, in which case patients would be unable to recognize objects because they could not use the percept to access their knowledge of the object. He termed the first kind of agnosia *apperceptive agnosia* and the second kind *associative agnosia*. This terminology is still used today to distinguish agnosic patients who have frank perceptual impairments from those who do not, although the implicit assumption that the latter have an impairment in "association" is now questioned.

Behavior and Anatomy

One might wonder whether apperceptive agnosics should be considered agnosics at all, given that the definition of agnosia cited at the beginning of this article excludes patients whose problems are caused by elementary visual impairments. The difference between apperceptive agnosics and patients who fall outside of the exclusionary criteria for agnosia is that the former have relatively good acuity, brightness discrimination, color vision, and other so-called elementary visual capabilities. Despite these capabilities, their perception of shape is markedly abnormal. For example, in the classic case of Benson and Greenberg,[3] pictures, letters, and even simple geometric shapes could not be recognized. Figure 9-1 shows the attempts of their patient to copy a column of simple shapes. Recognition of real objects may be somewhat better than recognition of geometric shapes, although this appears to be due to the availability of additional cues such as size and surface properties such as color, texture, and specularity rather than object shape. Facilitation of shape perception by motion of the stimulus has been noted in several cases of apperceptive agnosia. In most cases of apperceptive

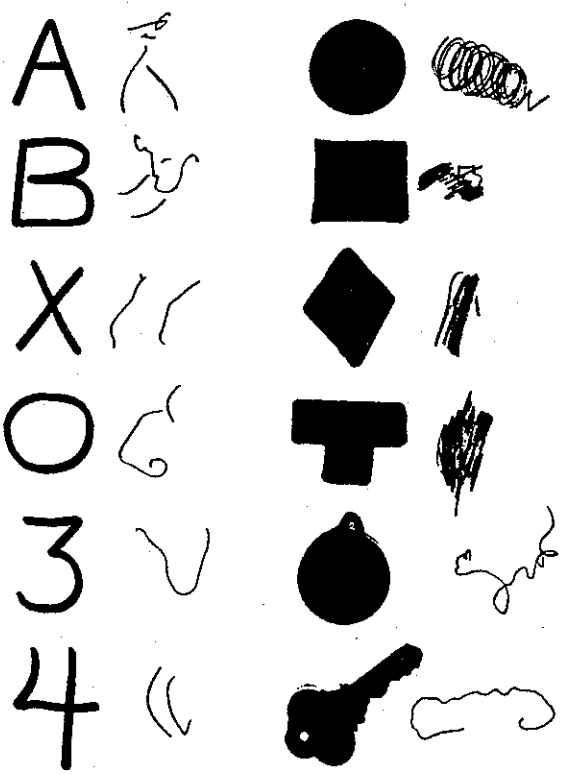

Figure 9-1
The attempts of an apperceptive agnosic patient to copy simple shapes. (From Benson and Greenberg,[3] with permission.)

agnosia, the brain damage is diffuse, often caused by carbon monoxide poisoning. For a review of other cases of apperceptive visual agnosia, see Ref. 4.

Interpretation of Apperceptive Agnosia

One way of interpreting apperceptive agnosia is in terms of a disorder of grouping processes that normally operate over the array of local features representing contour, color, depth, and so on.[4] Outside of their field defects, apperceptive agnosics have surprisingly good perception of local visual properties. They fail when they must extract more global structure from the image. Motion is helpful because it provides another cue to global structure in the form of correlated local motions. The perception of form from motion may also have different neural substrates from the perception of form from static contour,[5] and may therefore be spared in apperceptive agnosia.

Relation to Other Disorders

Some authors have used the term *apperceptive agnosia* for other, quite different types of visual disorders, including two forms of simultanagnosia and an impairment in recognizing objects from unusual views or under unusual lighting conditions. *Simultanagnosia* is a term used to describe an impairment in perception of multielement or multipart visual displays. When shown a complex picture with multiple objects or people, simulanagnostics typically describe them in a piecemeal manner, sometimes omitting much of the material entirely and therefore failing to interpret the overall nature of the scene being depicted.

Dorsal simultanagnosia is a component of Balint's syndrome in which an attentional limitation prevents perception of more than one object at a time.[4,6–8] Occasionally attention may be captured by just one part of an object, leading to misidentification of the object and the appearance of perception confined to relatively local image features. The similarity of dorsal simultanagnosia to apperceptive agnosia is limited, however. Once they can attend to an object, dorsal simultanagnosics recognize it quickly and accurately, and even their "local" errors encompass much more global shape information than is available to apperceptive agnosics. Their lesions are typically in the posterior parietal cortex bilaterally.

Despite some surface similarity to apperceptive agnosia and dorsal simultanagnosia, *ventral simultanagnosia* represents yet another disorder.[4,9] Ventral simultanagnosics can recognize whole objects, but are limited in how many objects can be recognized in a given period of time. Their descriptions of complex scenes are slow and piecemeal, but unlike apperceptive agnosics their recognition of single shapes is not obviously impaired. The impairment of ventral simultanagnosics is most apparent when reading, because the individual letters of words are recognized in an abnormally slow and generally serial manner (letter-by-letter reading, see Chap. 20). Unlike the case with dorsal simultanagnosics, their detection of multiple stimuli appears normal; the bottleneck is in recognition per se.

Unlike apperceptive agnosics, they perceive individual shapes reasonably well. Their lesions are typically in the left inferior temporooccipital cortex.

Some patients have roughly normal perception and recognition of objects except when viewed from unusual perspectives or under unusual lighting. Their impairment has also been grouped with apperceptive agnosia by some, but for clarity's sake can also be called *perceptual categorization deficit* because they cannot categorize together the full range of images cast by an object under different viewing conditions. This disorder does not have great localizing value, although the lesions are generally in the right hemisphere and frequently include the inferior parietal lobe.[4,10]

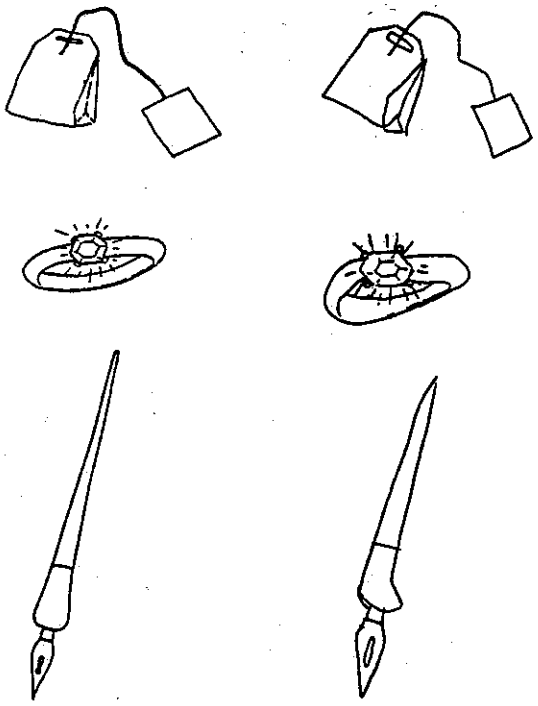

Figure 9-2
The copies of an associative agnosic patient with prosopagnosia and object agnosia. The patient did not recognize any of the original drawings. (From Farah et al.,[31] with permission.)

ASSOCIATIVE AGNOSIA

Behavior and Anatomy

In associative agnosia, visual perception is much better than in apperceptive agnosia. Compare, for example, the copies made by the associative agnosics shown in Figs. 9-2 and 9-3 with the copies shown in Fig. 9-1. Nevertheless, object recognition is impaired. Associative agnosic patients may be able to recognize an object by its feel in their hand or from a spoken definition, demonstrating that they have intact general knowledge of the object in addition to being able to see it well enough to copy it, but they cannot recognize the same

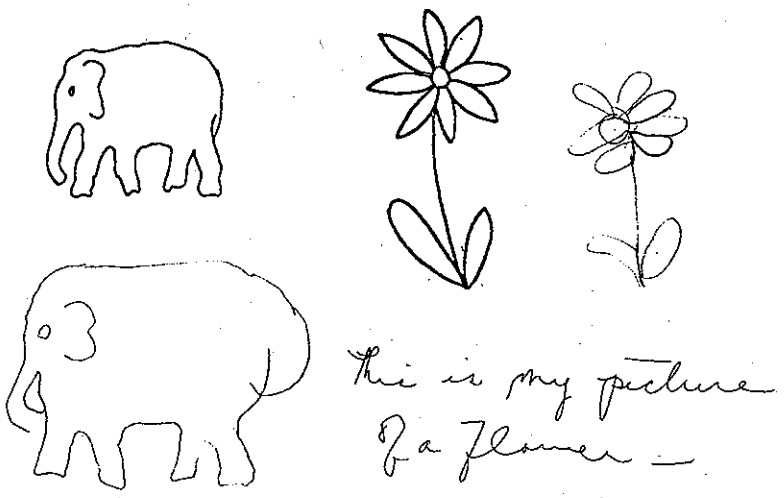

Figure 9-3
The copies of associative visual agnosic patients with alexia and object agnosia. The patients did not recognize the original drawings. Also shown is a sample of a patient's writing to dictation. After a delay, her own handwriting could not be read. (From Feinberg et al.,[16] with permission.)

object by sight alone. The impairment is not simply a naming deficit for visual stimuli; associative agnosics cannot indicate their recognition of objects by nonverbal means, as by pantomiming the use of an object or by grouping together dissimilar-looking objects from the same semantic category[11–16] (see Ref. 4 for a review of representative cases).

The scope of the recognition impairment varies from case to case of associative agnosia. Some patients encounter difficulty mainly with face recognition (see Chap. 10), while others demonstrate better face recognition than object recognition. Printed-word recognition is similarly impaired in some cases but not others. The selectivity of these impairments suggests that there is more than one system involved in visual recognition. According to one analysis,[17] there are two underlying forms of visual representation, one of which is required for face recognition, used for object recognition but not for word recognition, and the other of which is required for word recognition, used for object recognition and not required for face recognition. Indeed, if one regards associative agnosia as a single undifferentiated category, it is difficult to make any generalizations about the brain regions responsible for visual object recognition. Although the intrahemispheric location of damage is generally occipitotemporal, involving both gray and white matter, cases of associative agnosia have been reported following unilateral right-hemispheric lesions,[18] unilateral left-hemispheric lesions,[15,16,19,20] and bilateral lesions.[21–23] However, if one considers impairments in face and word recognition as markers for different underlying forms of visual recognition disorder, then a pattern emerges in the neuropathology.

When face recognition alone is impaired or when face and object recognition are impaired but reading is spared, the lesions are generally either on the right or bilateral. De Renzi has proposed that the degree of right-hemispheric specialization for face recognition may normally cover a wide range, such that most cases of prosopagnosia become manifest only after bilateral lesions, but in some cases a unilateral lesion will suffice (see Chap. 10). When reading alone is impaired or when reading and object recognition are impaired but face recognition is spared, the lesions are generally on the left. In a series of patients studied by us and additional cases of agnosia sparing face recognition culled from the literature, the maximum overlap in lesion locus was in the left inferior medial region involving parahippocampal, fusiform, and lingual gyri.[16] When recognition of faces, objects, and words is impaired, the lesions are generally bilateral.

The hypothesis of two underlying systems explains the pairwise dissociations among three different stimulus categories—words, objects, and faces—in a parsimonious way, with only two systems. In addition, it reveals a systematicity in lesion sites not previously apparent. Nevertheless, the hypothesis has been questioned following more recent reports of patients with patterns of spared and impaired recognition abilities that are inconsistent with an impairment in one of just two underlying systems. One patient with impaired face and word recognition but relatively less impaired object recognition has been reported.[24] The presence of a degree of object agnosia precludes strong inferences, however. Another patient with an isolated object recognition impairment has also been reported.[25] In this case, however, the object recognition impairment was evident to a degree on purely verbal tasks, limiting its relevance to visual agnosia.

Functional neuroimaging of normal subjects has largely supported the idea of a bilateral- or right-lateralized system for face recognition and a left-lateralized system for word recognition, with object recognition using both,[26] but has also raised the possibility of additional specialization within those systems, for example, specialization for orthography per se.[27]

Interpreting Associative Agnosia

Is associative agnosia a problem with perception, memory, or both? Associative agnosia has been explained in three different ways that suggest different answers to this question. The simplest way to explain agnosia is by a disconnection between visual representations and other brain centers responsible for language or memory. For example, Geschwind[28] proposed that associative agnosia is a visual–verbal disconnection. This hypothesis accounts well for agnosics' impaired naming of visual stimuli, but it cannot account for their inability to convey recognition nonverbally. Associative agnosia has also been explained as a disconnection between visual representations and medial temporal memory centers.[23] However, this would account for a

modality-specific impairment in new learning, not the inability to access old knowledge through vision.

The inadequacies of the disconnection accounts lead us to consider theories of associative agnosia in which some component of perception and/or memory has been damaged. Perhaps the most widely accepted account of associative agnosia is that stored visual memory representations have been damaged. According to this type of account, stimuli can be processed perceptually up to some end-state visual representation, which would then be matched against stored visual representations. In associative agnosia, the stored representations are no longer available and recognition therefore fails. Note that an assumption of this account is that two identical tokens of the object representation normally exist, one derived from the stimulus and one stored in memory, and that these are compared in the same way as a database might be searched in a present-day computer. This account is not directly disconfirmed by any of the available evidence. However, there are some reasons to question it and to suspect that subtle impairments in perception may underlie associative agnosia.

Although the good copies and successful matching performance of associative agnosics might seem to exonerate perception, a closer look at the manner in which these tasks are accomplished suggests that perception is not normal in associative agnosia and suggests yet a third explanation of associative agnosia. Typically, these patients are described as copying drawings "slavishly"[29] and "line by line."[30] In matching tasks, they rely on slow, sequential feature-by-feature checking. It therefore may be premature to rule out faulty perception as the cause of associative agnosia.

Recent studies of the visual capabilities of associative agnosic patients confirm that there are subtle visual perceptual impairments present in all cases studied.[4] If the possibility of impaired recognition with intact perception is consistent with the use of a computational architecture in which separate perceptual and memory representations are compared, then the absence of such a case suggests that a different type of computational architecture may underlie object recognition. Parallel distributed processing (PDP) systems exemplify an alternative architecture in which the perceptual and memory representations cannot be dissociated (see Chap. 7; see also Refs. 4 and 5 for discussions of computational approaches to agnosia). In a PDP system, the memory of the stimulus would consist of a pattern of connection strengths among a number of neuronlike units. The "perceptual" representation resulting from the presentation of a stimulus will depend upon the pattern of connection strengths among the units directly or indirectly activated by the stimulus. Thus, if memory is altered by damaging the network, perception will be altered as well. On this account, associative agnosia is not a result of an impairment to perception *or* to memory; rather, the two are in principle inseparable, and the impairment is better described as a loss of high-level visual perceptual representations that are shaped by, and embody the memory of, visual experience. It will thus be of great interest to see whether future studies of associative agnosics will ever document a case of impaired recognition with intact perception.

Relation to Other Disorders

As with apperceptive agnosia, a number of distinct disorders have been labeled associative agnosia by different authors. Visual modality–specific naming disorders exist and are usually termed *optic aphasia* (see Chap. 10), but they may on occasion be called *associative visual agnosia. Impairments of semantic memory* (see Chap. 27) will affect object-recognition ability (as well as entirely nonvisual abilities such as verbally defining spoken words) and perhaps for this reason have also sometimes been called *associative visual agnosia.*

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Chapter 10

PROSOPAGNOSIA

Martha J. Farah

Visual object agnosia, discussed in the previous chapter, does not always affect the recognition of all types of stimuli equally. Quite often, the recognition of faces seems disproportionately or even exclusively impaired, a condition known as *prosopagnosia*. Prosopagnosia can be so severe that the patient cannot recognize close friends, family members, or even his or her own face in a photograph. Yet nonfacial knowledge of people is preserved, and prosopagnosics typically resort to recognizing individuals by their voices or even by nonfacial visual cues such as clothing. Of course, such strategies have only very limited effectiveness. Prosopagnosia is therefore a serious problem for patients and is usually discovered because of the patient's complaint rather than by testing or examination.

The most straightforward explanation of prosopagnosia is that a specialized brain system for recognizing faces has been damaged. In recent years, much of the research on prosopagnosia has been aimed at testing this explanation against various alternative explanations. The reason that so much attention has been paid to this issue is that it bears directly on a larger controversy in cognitive science concerning the unity versus modularity of cognitive processes (e.g., Fodor, 1982). Does the brain support intelligent behavior with a relatively small set of general-purpose information processing mechanisms, or has it evolved to carry out its many functions by the use of dedicated, special-purpose mechanisms?

THE FUNCTIONAL DEFICIT IN PROSOPAGNOSIA: FACE-SPECIFIC?

The most straightforward interpretation of prosopagnosia is consistent with anatomically separate recognition systems for faces and objects. More precisely, prosopagnosia suggests that there is some system that is necessary for face recognition and either unnecessary or less important for object recognition. An alternative interpretation is that faces and all other types of objects are recognized using a single recognition system and that faces are simply the most difficult type of object for the recognition system. Prosopagnosia can then be explained as a mild form of agnosia, in which the impairment is detectable only on the most taxing form of recognition task.

The first researchers to address this issue directly were McNeil and Warrington (1993). They studied case W.J., a middle-aged professional man who became prosopagnosic following a series of strokes. After becoming prosopagnosic, W.J. made a career change and went into sheep farming. He eventually came to recognize many of his sheep, although he remained unable to recognize most humans. The authors noted the potential implications of such a dissociation for the question of whether human face recognition is "special" and designed an ingenious experiment exploiting W.J.'s new-found career. They assembled three groups of photographs—human faces, sheep faces of the same breed kept by W.J., and sheep faces of a different breed—and attempted to teach subjects names for each face. Normal subjects performed at intermediate levels between ceiling and floor in all conditions. They performed better with the human faces than with sheep faces, even those who, like W.J., worked with sheep. In contrast, W.J. performed poorly with the human faces and performed normally with the sheep faces.

The issue of whether prosopagnosia is selective for faces relative to common objects was addressed by my colleagues and myself with patient L.H., a well-educated professional man who has been prosopagnosic since an automobile accident in college (Farah et al., 1995). We employed a recognition memory paradigm in which L.H. and control subjects first studied a set of photographs of faces and nonface objects,

such as forks, chairs, and eyeglasses. Subjects were then given a larger set of photographs, and asked to make "old"/"new" judgments on them. Whereas normal subjects performed equally well with the faces and nonface objects, L.H. showed a significant performance disparity, performing worse with faces than with objects. In a second experiment, we used a similar method to contrast L.H. and normal subjects' recognition performance with 40 faces and 40 eyeglass frames and again found that L.H. was disproportionately impaired at face recognition. This, as well as the results of testing W.J. with human and sheep faces, implies that prosopagnosia is not a problem with recognizing specific exemplars from any visually homogeneous category but is specific to faces.

Another source of evidence for the independence of face and object recognition comes from patients who show the opposite dissociation—namely, more difficulty with object recognition than with face recognition (Feinberg et al., 1994; Moscovitch et al., 1997). The existence of such cases also supports the interpretation that prosopagnosia is not simply a mild general visual agnosia, because such an interpretation is inconsistent with the possibility of relatively preserved face recognition with object agnosia.

A different kind of alternative interpretation of prosopagnosia does not deny that visual recognition involves some specialized subsystems that are necessary for face recognition. However, according to this alternative, the nature of the specialization is subtly different from that discussed so far. Gauthier and collaborators have proposed that we have a recognition system that is specialized for objects that require expertise to discriminate from one another and which share an overall configuration. Faces fall into this category, but other objects can as well. These include birds or dogs for expert bird watchers and dog show judges (Tanaka and Taylor, 1991) and a set of artificial creatures devised by Gauthier and Tarr (1997) called "greebles." Greeble recognition has been shown to have many similarities to face recognition, and recent efforts to teach a prosopagnosic to recognize Greebles were unsuccessful, adding further support to Gauthier's hypothesis. Of course, it is possible to view such demonstrations as evidence that people occasionally recruit their specialized face recognition system for use with other stimuli.

ANATOMIC BASES OF FACE RECOGNITION

If we are interested in knowing precisely where, in the human brain, face recognition is carried out, individual cases are rarely very informative. L.H. sustained head injuries followed by surgery and W.J. suffered at least three strokes, resulting in widely distributed damage in both cases. Surveys of the lesions in larger groups of prosopagnosics are more helpful for localization, as the regions of overlap among different patients can be identified. Damasio et al. (1982) conducted a survey of the literature for autopsied cases of prosopagnosia, and studied three of their own patients, concluding that the critical lesion site is in ventral occipitotemporal cortex bilaterally. De Renzi and colleagues (1994) reviewed much of the same case material, along with more recent cases and data from living patients whose brain damage was mapped using both structural magnetic resonance imaging (MRI) and positron emission tomography (PET). Their findings supported the ventral localization of face recognition but called for a revision of the idea that bilateral lesions are necessary. Some patients became prosopagnosic after unilateral right hemisphere damage. The possibility of hidden left hemisphere dysfunction in these cases was reduced by the finding of normal metabolic activity in the left hemisphere by PET scan. De Renzi et al. conclude that there is a spectrum of hemispheric specialization for face recognition in normal right-handed adults. Although the right hemisphere may be relatively better at face recognition than the left, most people have a degree of face recognition ability in both hemispheres. Nevertheless, in a minority of cases, face recognition is so focally represented in the right hemisphere that a unilateral lesion will lead to prosopagnosia. The lesion sites associated with prosopagnosia are, as a group, clearly different from the lesions associated with object agnosia in the absence of prosopagnosia. The latter syndrome is almost invariably the result of a unilateral left hemisphere lesion, although confined to roughly the same intrahemispheric region (Feinberg et al., 1994).

Converging evidence about the localization of face recognition in the human brain comes from functional neuroimaging of normal individuals. The

most relevant experimental design for comparison with prosopagnosics' lesions is one in which brain activity while viewing faces is contrasted with brain activity while viewing nonface objects. Kanwisher and coworkers (1996) used functional MRI to compare regional brain activity while subjects viewed photographs of faces and of objects. An objects-minus-faces subtraction revealed areas more responsive to objects than faces and the reverse subtraction revealed an area more responsive to faces than objects. Both types of stimuli activated inferior temporooccipital regions, with face-specific activation confined to part of the right fusiform gyrus. A follow-up study identified the same fusiform face area and systematically verified its specificity for faces by comparing its response to faces and to scrambled faces, houses, and hands (Kanwisher et al., 1997). Similar conclusions were reached by McCarthy and coworkers (1997), who found right fusiform activation unique to passive viewing of faces relative to objects or scrambled objects, and left fusiform activation unique to flowers relative to scrambled objects.

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