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, Balteau 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 agnostic. 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 mnemonic 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 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, pictures, letters, and even simple geometric shapes could not be recognized. Figure 6-1 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 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 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.

**Figure 6-1**
The attempts of an apperceptive agnostic patient to copy simple shapes. (From Benson and Greenberg, with permission.)

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. Outside of their field defects, apperceptiveagnosias 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 structure from motion may also have different neural substrates from static contour, 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, simultanagnosics 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. 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 apperceptiveagnosias. Their lesions are typically in the posterior parietal cortex bilaterally.

Despite some surface similarity to apperceptive agnosia and dorsal simultanagnosia,
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. 6-2 and 6-3 with the copies shown in Fig. 6-1. Nevertheless, object recognition is impaired. Associative agnostic 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 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 (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. 7), 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, 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, unilateral left-hemispheric lesions, and bilateral lesions. 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. 7). 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. When recognition of faces, objects, and words is impaired, the lesions are generally bilateral.

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 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 and may therefore be more suited to explaining optic aphasia.
of agnosia limited to impaired naming of vis-
ul (see Chap. 23). Associative agnosia has
been explained as a disconnection between
representations and medial temporal mem-
centers.23 However, this would account for
modality-specific impairment in new learning,
the inability to access old knowledge through

The inadequacies of the disconnection ac-
cepts lead us to consider theories of associ-a-
sia in which some component of perception
for memory has been damaged. Perhaps the
widely accepted account of associative agno-
sia that stored visual memory representations
have been damaged. According to this type of ac-
cept, stimuli can be processed perceptually up
to some end-state visual representation, which
would normally be matched against stored visual
representations. In associative agnosia, the stored
representations are no longer available and recog-
nition 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 data-
based might be searched in a present-day com-
puter. 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 as-
sociative 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 accom-
plished suggests that perception is not normal in
associative agnosia and suggests yet a third expla-
nation of associative agnosia. Typically, these pa-
ients are described as copying drawings "slav-
ishly"25 and "line by line."26 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 agnostic 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 represen-
tations are compared, then the absence of such a
case suggests that a different type of computational
architecture may underlie object recognition. Par-
allel distributed processing (PDP) systems exem-
plify an alternative architecture in which the per-
ceptual and memory representations cannot be
dissociated (see Chap. 4; see also Ref. 4, Chap. 2,
for a discussion of PDP models and 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 al-
tered 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 insepara-
able, 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 dis-
tinct disorders have been labeled associative
agnosia by different authors. Visual modality-
specific naming disorders exist and are usually
termed optic aphasia, but they may on occasion be
called associative visual agnosia. Impairments of
semantic memory (see Chap. 23) will affect ob-
ject-recognition ability (as well as entirely nonvisual
abilities such as verbally defining spoken words)
and perhaps for this reason have sometimes been
called associative visual agnosia.

REFERENCES

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