



Visual agnosia

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The “father” of agnosia: Lissauer

Lissauer was the first scientist to provide a detailed account of a patient with visual agnosia [1]. He presented his clinical observations at a meeting of neurologists in 1888 in the context of a theoretic framework. He suggested that, from early stages of visual processing, the processing of color, form, and motion might be affected separately as a result of brain damage (as indeed subsequent work has proved; see later discussion of “Deficits in peripheral visual processing”). At the level of form perception, he proposed that visual recognition required processing through two distinct stages: the first (apperception) he described as “the stage of conscious awareness of a sensory impression”; the second (or associative stage) was believed to result from the simultaneous activation of many concepts related to the object (ie, the activation of associated memories). Taking as an example a violin, he stressed the wealth of sensory associations (image, sound, name, tactile experience from handling it, typical visual context, and so forth). The specific nature of memories and associations for any object varies across individuals; nonetheless, most people have a rich concept based on such distributed associations. An “associative agnosia” results if perceptual information for a violin, despite being derived normally, fails to activate the stored associations. Lissauer was skeptical, however, that a purely associative agnosia could exist without some concomitant impairment in apperception. He also suggested that apperceptive deficits could exist at different levels of processing, although in all such cases, the deficit should not be attributable to impaired peripheral visual processing (which may result in

This work was supported by grants from the MRC, Wellcome Foundation, and the Stroke Association.

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decreased acuity, reduction, or both in visual fields). Lissauer's account of agnosia continues to be used as a general framework, and we review evidence on agnosia in the light of Lissauer's work. In particular, we evaluate each of his proposals, dealing first with whether or not agnosic deficits are attributable to low level deficits in basic perception and whether or not disturbances to different properties of images can be established (eg, impaired color or motion vision without impairments in form vision; see following discussion). Subsequently, we discuss whether or not contrasting forms of apperceptive agnosia exist (see later discussion of "Apperceptive disorders") and whether or not associative agnosia can occur without concomitant problems in form processing (with apperceptive agnosia; see later discussion of "Associative disorders"). These arguments are reviewed in light of cases of visual agnosia reported since Lissauer's influential paper in 1888.

Deficits in peripheral visual processing

Color and motion perception

Distinct areas of the brain are specific for the processing of color, form, and motion, as indicated by independent neurologic deficits. Achromatopsia refers to the syndrome in which a patient loses the ability to see colors after cortical damage. The loss may be partial or complete and it may or may not be accompanied by other visual defects. Typically, the patient reports seeing the world in black and white and shades of gray [2] but has no difficulty in the recognition of objects (provided recognition does not depend on color). Relatively few cases have been reported with selective deficits of motion perception. The most detailed study has been that of patient LM, who suffered a superior sagittal sinus thrombosis at age 43 [3]. An MRI showed severe bilateral damage to the middle temporal gyrus and the adjacent part of the occipital gyri with subcortical damage affecting lateral, occipital, and occipitoparietal white matter [4]. LM was profoundly disabled in everyday life and found moving stimuli to be highly disturbing and unpleasant. She could not perceive actual movement, so that when objects did move (eg, cars or people), they seemed to jump from one position to the next. She found that understanding language was problematic (people's lips seemed to hop up and down), as was meal preparation (she had difficulty in pouring and measuring liquids because they appeared frozen like a glacier) [5]. LM had no visual field defect, her acuity was unimpaired, and she performed normally on tests of visual identification and recognition of objects, faces, and places.

Form perception: a problem of low-level vision?

Efron's patient, Mr. S., was able to detect differences in luminance, wavelength, and area and could respond to small movements of objects before him [6]. Critically, however, he was unable to distinguish between two objects of

the same luminance, wavelength, and area when the only difference between them was shape. Over the years, there has been some controversy as to whether or not deficits in form perception, such as that shown by Mr. S., may be classified as deficits of low-level visual processing or whether or not they form part of the agnosia continuum. In the 1950s, Bay argued that the sensitivity in visual testing was lacking in such patients. He used more detailed examination techniques than were generally applied clinically, including tests for local adaption and for sensation time, Bay defined sensation time as “the minimal time required for the appearing of a sensation” (p. 516). Stimuli were presented tachistoscopically in different parts of the visual field. The time taken to detect the stimuli was raised in areas of decreased function. Adaption occurred if a constant stimulus was applied to a fixed area of the visual field. Over time, the perception of this stimulus slowly faded. The fading time was dependent on the object’s size and its positioning in the visual field. Fading occurred more rapidly in impaired areas of the visual field and demonstrated disorders of visual functioning that had not been apparent on standard perimetric testing in a variety of brain-damaged patients, including a patient with visual agnosia [7]. Bay found that the retina in patients with apparently normal visual fields showed abnormal fatigue. The abnormality was greatest in the central retinal region but affected the periphery more (visual stimuli would tend to drop out of awareness). Bay argued that the visual field essentially was contracted and therefore would not allow the simultaneous perception of a whole visual stimulus in circumstances in which the visual angle subtended by the stimulus was greater than the remaining functional central area. As a result, it would not be possible to gain a holistic view of the stimulus, but details of it may be seen in succession. These effects must be contrasted with those occurring in cases of simultanagnosia. Here also identification is limited to one object at a time; however, now what defines the ‘object’ is critical. Luria [69] described a patient who was able to report the star of David when shown the stimulus in a single color; however, the patient only reported the presence of a triangle when the two triangles making up the star of David were shown in different colors. Identification here is not determined by the spatial extent of the stimulus but by how the parts group together (when in the same color) or segment apart when in different colors. Bay’s [7] attribution of impaired visual recognition to disturbed sensory processes resulted in more stringent assessments of many of the subsequently reported cases of visual agnosia. For example, a detailed investigation of visual abilities of a patient with visual agnosia, HJA [8], showed no tunnel vision [9]; however, he was impaired in detecting local elements embedded in more global geometric forms. Thus, whereas he performed at normal levels in detecting a line target embedded in distractor lines of a different orientation, he was unable to perform a similar task when the target was a square made of four parallel lines (Fig. 1). Similarly, HJA was abnormally slow in responding to local elements in compound global letters [10], although he could respond to the global form itself (Fig. 2).

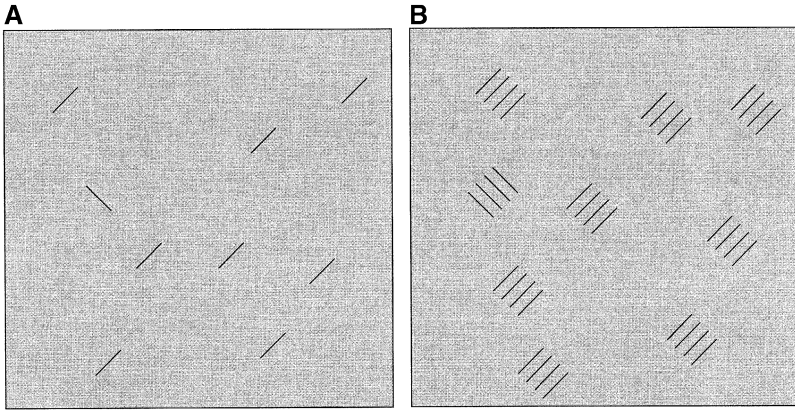


Fig. 1. Examples of the displays used by Alkhateeb et al [9]. In (A) the elements consist of single lines; in (B) they consist of groups of lines. (Adapted from Alkhateeb W, Bromley JM, Humphreys GW, Javadnia A, Riddoch MJ, Ruddock, H. Abnormal responses to multi-element spatial stimuli in a subject with visual form agnosia. *Clinical vision Science* 1992; 7:163–73; with permission.)

Alkhateeb et al [9] argue that this pattern of performance in more complex search tasks mirrors HJA's ability to identify correctly local features of visual scenes, while being unable to link them into a coherent percept (see later discussion of "Perceptual integration of form information"). Others also explored whether or not impaired low level vision (such as the thresholds for detecting and discriminating gratings, color, correlated movement, reflectance, shape from motion or luminance contrast, relative position, texture, and orientation) in separate studies in groups of patients with localized

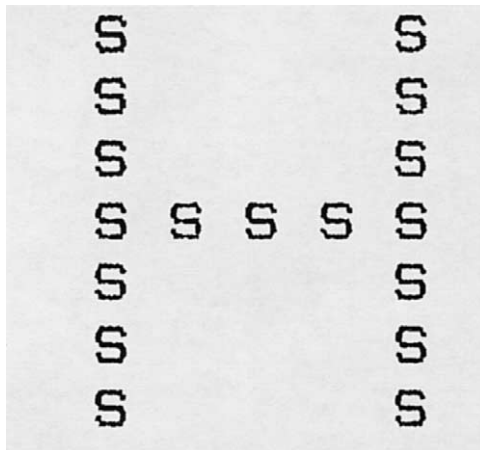


Fig. 2. A Navon-type stimulus consisting of a global shape (the letter H) made up of smaller letters (S).

brain damage can account for recognition disorders. The accumulated data show that whereas there may be elevated thresholds, there is no correlation between the pattern or the severity of impairments and the presence or absence of visual agnosia (for a discussion of these issues, see Cowey [11]).

In the 1980s Campion and coworkers argued that the visual recognition problem in patients with agnosia resulting from a particular etiology, carbon monoxide poisoning, is the result of multiple small scotoma (resulting from the multiple lesions caused by the poisoning). They proposed that the behavioral consequences of such lesions would be that patient viewed the world through a peppery mask [12,13]. Given that we adapt over natural “holes” because of the blind spot, however, and that any masking effect may be reduced by movement, then the import of this argument is not clear. An alternative account is that the problem results from impaired grouping processes [14,15] (see later discussion). Recently, Vecera and Gilds [16] attempted to distinguish between these possibilities in a series of experiments with normal subjects. The basic paradigm required subjects to respond to a spatially cued target. There were two conditions to the experiment: either subjects viewed the displays (two rectangular bars) through a peppery mask (masking condition), or the displays were degraded in one of two ways (grouping condition). In the grouping condition, either the midsections of the rectangles were removed (cues such as co-linearity and closure remained present, allowing grouping of the parts into an object) or the corners were removed (making it more difficult to group the parts into objects). The displays were aligned either horizontally one above and one below the fixation cross, or they were aligned vertically, again one on each side of the fixation cross. A spatial cue appeared at one end of one of the bars. This was either valid (75% of trials) or an invalid (25% of trials). After the cue, the two bars were shown empty for 200 ms. On valid trials, the target then appeared in the cued location. On invalid trials there were three possible locations for the target: (1) in the same bar as the cue but at the opposite end; (2) in the other bar in the same relative spatial location as the cue; or (3) in the other bar and at the opposite end to the relative spatial location of the cue (for examples of the stimuli, see Fig. 3). In the masking condition, RTs were slowed but the pattern of results was similar to that obtained from unmasked displays (ie, there was a valid cuing effect with responses to targets in the cued rectangle faster than those to target in the uncued rectangle). In the grouping conditions, there was no effect of degrading when only the corners of the rectangles were present, suggesting that grouping processes nullify this form of degradation. There was an effect of degrading, however, when only the midsections of the rectangles were present (ie, in the circumstances when grouping could not occur). Vecera and Gilds [16] argue that their results support the grouping-deficit account of visual agnosia (see later discussion of “Feature integration”).

Warrington [17] also argued the poor recognition in cases such as those of Efron [6] are conceptualized best as a “partial or incomplete visual field

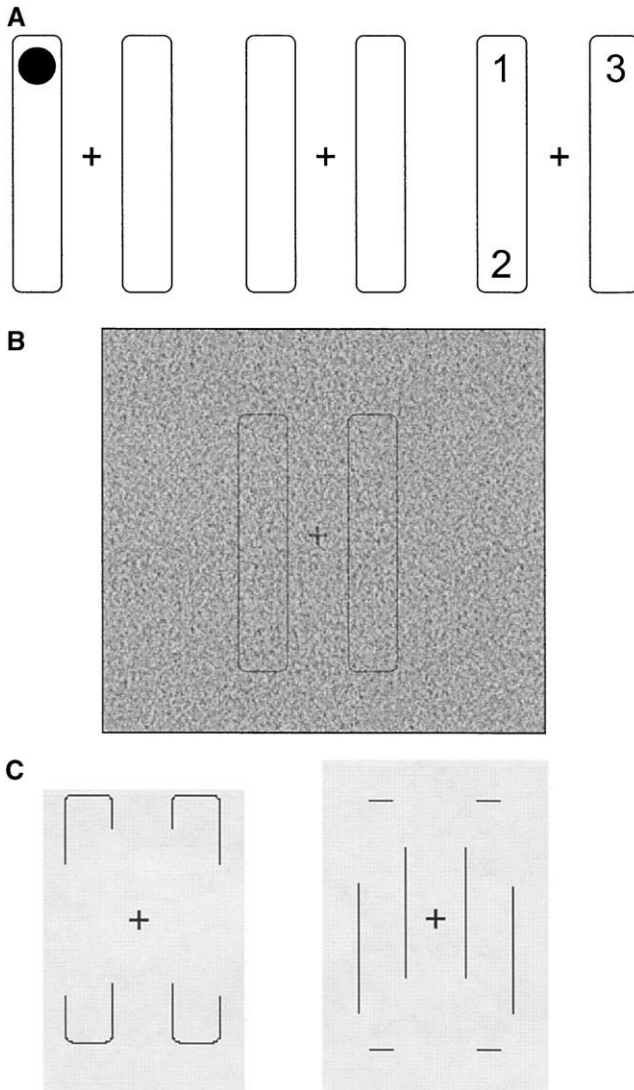


Fig. 3. Stimuli similar to those used by Vecera and Gilds [16]: (A) illustrates the sequence of events: a cue first appears at the end of one of the rectangles, there is an interstimulus interval, and the target then appears in one of three possible locations; (B) illustrates the masked displays; and (C) shows the stimuli with corners or mid sections only. (From Vecera SP, Gilds KS. What processing is impaired in apperceptive agnosia? Evidence from normal subjects. *Journal of Cognitive Neuroscience* 1998;19: 568–80; with permission.)

defect.” She describes them as “pseudo-agnosic” syndromes and argues that adequate visual functioning could not be established. More recently, Warrington and Rudge argued that adequate visual functioning reflects the ability to detect differences between nonrepresentational visual patterns; in

particular, they propose that the term “apperceptive agnosia” be reserved for disorders reflecting damage to “those sub-components of object recognition that implicate the ability to construct, by recourse to stored representations, an object specific structural description” [18]. Tests used to assess whether or not stored representations can be accessed include identifying overlapping, familiar objects [19,20], incomplete outline drawings of objects [21], or unconventional view object photographs [22]. In all these instances, patients with posterior right hemisphere damage seem to be selectively impaired. One difficulty with this account is that, although right hemisphere patients show impaired performance on a variety of perceptual tasks, they typically do not have any difficulty recognizing undegraded objects in conventional views; such a difficulty would seem to be central to the concept of visual agnosia.

Whilst acknowledging these arguments, the weight of opinion is that deficits of form processing can occur and that these are independent of early visual processes that may be reflected in poor visual acuity, reduced visual fields or impairments in color, or movement or depth perception. In addition, some studies report measures of contrast sensitivity, visual evoked responses, eye movements, and detection of targets defined by luminance, which can be preserved even though problems in form perception are demonstrated [23].

Apperceptive disorders

Lissauer suggested two ways to assess the integrity of form perception: (1) shape discrimination and (2) shape copying. He reported the results of these tests with his patient, GL. With shape discrimination, GL initially was asked to detect obvious differences between simple shapes and then minimal differences between complex shapes. He performed well with simple tests (eg, he was asked to discriminate between shapes similar to the letter 3; one of the shapes had an additional curl at the top), but performance slowed with more complex tests (eg, he was asked to distinguish between two boxes, each of which contained 12 equally distributed crosses; in one box, the center was empty, whereas the other was filled with a cross). On the basis of these observations, Lissauer argued that perceptual abilities might lie on a continuum, with the implication that a simple shape discrimination test is not sufficient to determine whether or not a perceptual deficit is present. GL's differing performance on the discrimination tests suggests that one may distinguish his processing of form (which the authors imply is intact based on his performance in the easy discrimination test) from his ability to integrate local and global aspects of form (which may be impaired, given his performance on the difficult discrimination test).

Lissauer's patient was able to copy; however, again it is clear that Lissauer did not regard copying ability as definitive proof of intact perception. He indicates that the procedure used by the patient was very slow with many pauses. He also noted that GL found it easier to copy line drawings than real

objects, because it was difficult to perceive the outline contour when shadows were present. Recently, the differences between drawing real objects and copying line drawings have been explored empirically in a patient with visual agnosia [24]. The patient, HJA, was asked to copy line drawings or to draw objects, but in conditions of good lighting, without shadow. There were several conditions: either HJA's head was fixed or he was allowed free movement. Also, objects were positioned within or outside reaching range in order to vary the potential contribution of stereoscopic depth (stereo cues being stronger within reachable space [25]) (see Fig. 4 for examples of HJA's performance). This enabled judgment of the role of such cues in HJA's object identification. All the drawings subsequently were presented to a group of control subjects who were asked to name the depicted objects. The results showed no reliable difference in the identification of line drawings versus real objects when the objects were close, even when head movements were prevented. When HJA's head was fixed, however, and objects were viewed from a distance, few of his drawings could be identified. In this condition, he typically failed to depict all the parts of the object successfully. The data indicate that stereo depth cues may play an important part in such patients perceiving objects, with perception abnormally impaired when stereo depth cues are reduced, by preventing motion and stereo disparities (with a fixed head position and more distant stimuli). In line with Lissauer's original observations, these data also suggest that the ability to copy items varies as a function of test conditions and that apparently good copying cannot necessarily be taken as evidence of intact perception. Given this proviso, we review the cases of several patients in the literature, taking their ability to discriminate shapes and to copy drawings as a starting point. We show that some patients are unable to perform even these simple tasks (presumably because of impaired form perception) and that, whereas others succeed, performance is far from normal. In these instances, we propose that the deficit is described best as a perceptual deficit specifically affecting processes of visual integration.

Impaired form perception

Many patients have been described with a profound deficit in form perception who are unable to either discriminate between simple shapes or to copy drawings successfully [6,12,26–28]. Interestingly, all these cases suffered carbon monoxide poisoning. Color, brightness, and movement discrimination typically were preserved, whereas the ability to perceive figural properties, such as size, orientation, and shape, was lost [6,28]. For instance, Efron's patient, Mr. S., was unable to discriminate between a square and a rectangle that were matched for total flux. Task difficulty was manipulated by varying the ratio of length to the width of the oblong. Mr. S. was barely above chance, except on the easiest discriminations. In a more extensive report of patient DF, Milner and colleagues [28] indicate that she performed at chance on a similar test to that described by Efron [6]; she also was unable

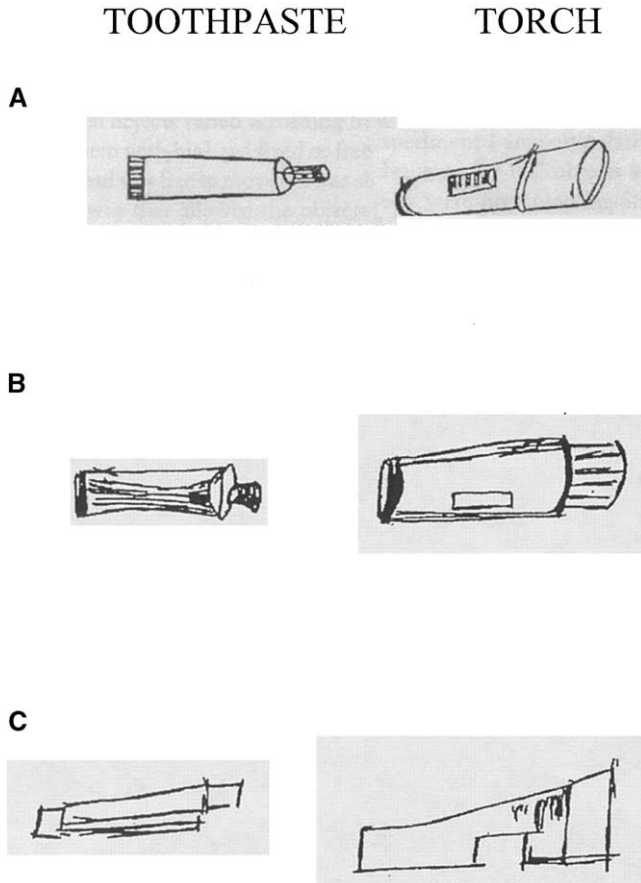


Fig. 4. Examples of HJA's copies of (A) copies of line drawings; (B) real objects presented at a distance with the head not fixed; (C) copies of real objects (distant condition, head fixed); (From Chainey H, Humphreys GW. The real-object advantage in agnosia: evidence for a role of surface and depth information in object recognition. *CognitiveNeuropsychology* 2001;18:175–91; with permission.)

to segment figure from ground (ie, detecting an O, X, or + shape against a ground consisting of a noise mask); she was at chance at determining whether or not abstract geometric shapes were symmetric or not, and so forth. Although she was unable to discriminate between simple shapes, she was able to use shape information in order to orient correctly her hand to match the orientation of a slot and to appropriately scale her grasp to objects of different sizes [28,29]. These data suggest dissociation between the visual information used for recognition and perceptual judgments and the visual information used for action.

A recent case who does not seem to have as extreme an impairment in form perception as that shown in Mr. S. [6] and DF [28] is SMK. He suffered

anoxia, which resulted in impairment in visual recognition that, although relatively severe (he was reported as able to name only one of the first six of the Oldfield pictures and three of twelve silhouette drawings of common objects), was not total (unlike DF, he could identify capital letters; also, DF failed to recognize any line drawings) [30]. SMK also seemed to have adequate elementary visual function: acuity was normal, visual fields were full, he had normal stereopsis, and his ability to discriminate between surface appearances of lightness, color, and shading was normal [30]. DF, in contrast, had severe visual deficits: her visual fields could not reliably be measured; brightness judgments were inaccurate; there was impaired detection of low spatial frequencies, although high spatial frequencies could be detected normally; depth perception and movement perception were also impaired. Although performance was poor, SMK was not at chance either on the Efron test (assessing the ability to discriminate between squares and rectangles), scoring 60% and 70% correctly for difficult and easy discriminations, respectively, or on a similar test using squares made from interrupted lines, scoring 58% and 80% correctly for difficult and easy discriminations, respectively. Although SMK found it difficult to make judgments on the basis of shape, he performed well on tests of figure–ground segmentation (for instance, detecting an X in the presence of background noise, detecting the number of items when geometric shapes are arranged concentrically or when they overlap, and so forth). From this, Davidoff and Warrington [30] argued that the process of figure–ground perception is separate from processes involved in form perception, with only the latter impaired in SMK. It is clearly the case that simple figure–ground discrimination can be dissociable from form perception, because figures can be distinguished from ground on the basis of image properties other than form, including color [31,32], depth [33–37], and motion [3]. In these cases figure–ground segmentation is based on shape perception derived from cues other than luminance-based contours. These other cues (color, motion, depth) may result from processing within separate modules to those involved in the processes in deriving shape from luminance contours. A more critical issue for understanding the relations between form perception and figure–ground coding is whether or not the two dissociate, even when form perception may be important for figure–ground coding. Here the results are less clear. Peterson and colleagues observed that the identification of occluded forms biases figure–ground organization. They propose that objects are recognized first on the basis of image features and occluded contours are computed afterwards (ie, a top-down model of figure–ground organization) [38]. Giersch et al [39], alternatively, show that impaired figure–ground perception can be related to processes involved in form coding. Their patient, HJA, was poor at discriminating occluded and occluding figures, but primarily when parts of the occluded figure were close and so grouped, despite the presence of the occluder. Under these circumstances, HJA sometimes ascribed the occluded edge to the front shape, consistent with a problem in figure–ground assign-

ment. This problem in figure–ground assignment reflects a more general problem of linking parts to a whole in this patient, with the parts-to-whole problem exacerbated under conditions of figure–ground ambiguity (Fig. 5).

Feature integration

Lissauer's patient, GL, did not perform well when form perception was tested stringently (eg, discriminating between complex shapes with minimal perceptual differences; see previous discussion); he also performed poorly on a test of figure–ground segmentation. Lissauer placed two books in front of him, which had identical ornamental borders on their covers. Lissauer marked one aspect of the ornamental detail on one of the books and asked GL to find it on the other. This he was unable to do, presumably because the assignment of the ornamental figure against the ground of the book was difficult.

Many patients have been reported with intact shape perception (as assessed using the Efron shape-matching test) [2,6,8,40,41] but who are

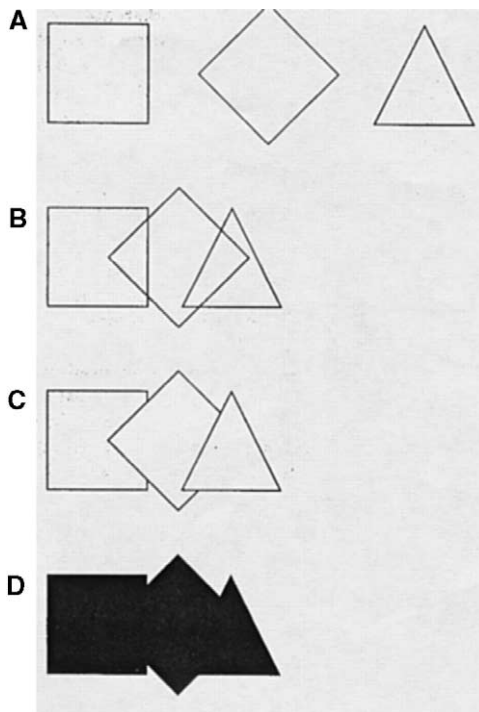


Fig. 5. Examples of the stimuli used by Giersch et al [39]: (A) non-overlapping, (B) overlapping non-occluding, (C) overlapping, occluding, and (D) overlapping silhouettes. (From Giersch A, Humphreys GW, Boucart M, Koviáks I. The computation of occluded contours in visual agnosia: evidence of early computation prior to shape binding and figure-ground coding. *Cognitive Neuropsychology* 2000;17:731–59; with permission.)

impaired when more stringent tests are used. One of these, HJA [8], was previously discussed, and his case is discussed in more detail here because it reflects a particular problem in coding complex shapes. Like Lissauer's patient, GL, HJA was able to produce accurate drawings of objects he could not recognize, although, like GL, he was very slow. He also performed the Efron [6] shape test well and could make orientation and size-matching judgments at a normal level [42]. He showed flat search functions (ie, no effect of the number of distractors) on visual search tasks in which the target differed from the distractors in terms of a single feature (eg, line orientation) [14,43]. These data suggest that HJA is able to process simple form information in a spatially parallel manner. On other tests, however, he did not do as well. For instance, he was at chance on an object decision task in which non-objects were constructed using parts of real objects, although in an alternative version of the task in which silhouettes rather than line drawings were used, he performed within normal limits. The internal detail in the line drawings seemed to disrupt rather than enhance his performance (non-brain damaged subjects find line drawings easier to discriminate than silhouettes [44]). Furthermore, as indicated in the previous section on "Impaired form perception," whereas HJA was able to identify the individual items in overlapping figures, he was slow relative to control performance [8]. In addition, we also have shown that HJA's visual recognition is compromised if time restraints are imposed. With unlimited exposure, he was able to name 80% of a set of line drawings, selected on the basis of previous successful performance; however, at 100 ms, he was only able to name 15% of them. These findings suggest that HJA's perceptual abilities were impaired (1) under speeded conditions and (2) when there were multiple segmentation cues. Riddoch and Humphreys [10] characterized HJA's deficit as a failure to integrate local visual elements into perceptual wholes. This is not to say that HJA is insensitive to global shape information. As noted, using Navon-type stimuli in which large "global" letters are made up of smaller "local" letters [45] (see Fig. 2), HJA responds faster to the global than to the local letter (as with control subjects). Unlike normal subjects, however, the identity of the local letter had no effect on his responses to the global letter. His responses to the local letter when presented in isolation were also within the normal range, but he was slow when he had to identify the local letter in the context of the global letter. This suggests that there is an abnormal effect of global context that hinders his accurate perception of local detail. Consistent with this, HJA was impaired at grouping homogeneous distractors in visual search tasks in order to detect targets efficiently [15]. Normally, a homogeneous context facilitates search by helping distractors form a separate group from the target. The detrimental effect of the distractor context, for HJA, is indicative of interference from more global groups on the coding of a target part.

To account for this pattern of performance, Riddoch and Humphreys proposed that HJA had impaired perceptual integration of shape elements

into coherent wholes. It is not that HJA is insensitive to perceptual wholes, but more that holistic contexts disrupt his coding and integration of parts. This problem of part integration is particularly telling when there are local edges within the wholes (eg, internal lines in line drawings), which prompt parts to be segmented (in line drawings rather than silhouettes) (Fig. 6).

We have provided substantial detail about the case of HJA on the grounds that the studies of his case provide perhaps the most thorough investigation to date of perceptual organization in agnosia. Other patients have been reported showing some similarities to HJA in the tasks they can and cannot do. One example is Annalisa, a 21-year-old girl who suffered a traffic accident resulting in damage to the right temporo-occipital region. After this, Annalisa had a severe impairment in object recognition [41]. Like HJA, she performed the Efron shape perception test well; she also performed within the control range in a (possibly) more stringent test of shape perception devised by Talland [46] (here the stimuli consist of vertically oriented oblongs that end at their upper side with an arch of graded sharpness; all possible combinations of pairings of the stimuli are presented and the patient must decide which one of a pair has the sharper point). She scored at ceiling on a same–different match task with complex figures that were distinguishable only by minor differences in their external configuration

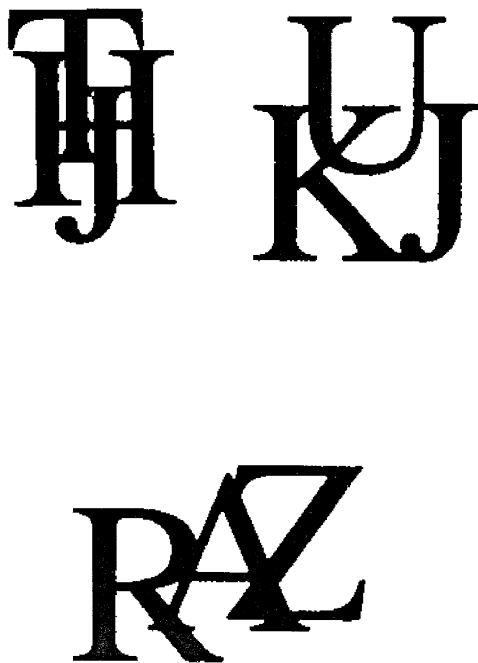


Fig. 6. Examples of overlapping letters as used by Riddoch and Humphreys [8].

(eg, hand postures) or internal structure (eg, figures from Ray's Progressive Matrices). Against her good performance on tests of form perception, Annalisa was impaired on tests of figure-ground segmentation: these included detecting a fragmented letter superimposed on a noisy background [47] and a test based on that described by Ghent [48], in which displays of three to five overlapping drawings of either meaningful or meaningless items (in separate conditions) were presented with 10 single drawings of items from the appropriate condition. The task was to choose which of the single items were present in the target array.

Similar effects have been reported recently in two further cases (SZ and AP) [49]. SZ had bilateral lesions implicating the posterior watershed distribution with sparing of the primary visual cortex, and AP had deficits affecting bilateral middle and inferior temporo-occipital cortices again sparing the primary visual cortices. Both patients performed reasonably well when asked to match a target item (a geometric shape) to one of three items (80% and 100% correct for SZ and AP, respectively). Performance dropped, however, when a similar task was performed with items consisting of partially overlapping geometric shapes (31% and 70% correct for SZ and AP, respectively). Likewise, SB is reported as being able to match identical shapes, but was at chance when asked to segment overlapping figures [50] (also see Kartsounis and Warrington [41]). Butter and Trobe [51] reported another patient, SM, who, like HJA, was not only impaired at overlapping forms but was also worse with line drawings than with silhouettes. Indeed, when presented with drawings of single objects, SM sometimes identified them as two separate items, indicating segmentation of the parts and poor coding of the whole. We suggest that normally an integrated representation of the parts is linked to more holistic descriptions based on low spatial frequency components, so that we perceive wholes that are articulated and identifiable. This linkage process is impaired in integrative agnosia. Integrative agnosia represents a form of apperceptive agnosia in which the coding of single shapes is relatively unimpaired, with the deficits revealed under conditions that stress visual segmentation and grouping.

Associative disorders

In contrast to the above cases, in which forms of perceptual deficit are apparent, many patients have been reported with impaired recognition of visually presented objects but who perform well on tests of perception [52,53]; thus, it seems that Lissauer was not correct in his assumption that some form of perceptual deficit underlies the recognition problem in all patients with visual agnosia. We suggest that there exist relatively pure forms of associative agnosia, without a concomitant apperceptive deficit. Riddoch and Humphreys [53,54] distinguished two forms of stored knowledge serving object recognition: stored structural descriptions and stored semantic knowledge. Structural descriptions are stored representations of

the structural properties of objects, used to match on-line descriptions encoded by the visual system. Semantic knowledge specifies information about object function and interobject associations that forms the basis of our full concept of an object. For objects to be recognized, with functional and associative knowledge about them retrieved, stored structural and stored semantic knowledge needs to be accessed. Accordingly, the association stage can be separated into two substages, each concerned with accessing a particular type of stored knowledge. Object decision tests have been used to assess the ability to access stored knowledge of object shape. With “perceptually good” nonobjects constructed from the parts of real objects, object decisions are contingent on access to stored structural descriptions and cannot rely on judgments from on-line perceptual representations alone. In 1987, we [53] demonstrated that patients could perform difficult object decisions (with nonobjects drawn from parts of two different objects from the same category) when their ability to access “semantic” (associative and functional) knowledge was impaired. The single case who showed this, JB, was relatively poor at making judgments about which two of three objects may be associated or used together, when they all came from the same category (eg, a hammer, a nail, and a screw). This was not because of a failure to understand the test, because he performed perfectly when presented with words rather than objects. The problem was in gaining access to this knowledge from vision. Given JB’s good object decision performance, we proposed that there was a deficit in visual access to stored semantic knowledge but crucially this took place after intact access to stored structural knowledge about objects. That is, there was an associative deficit after intact perception allowed contact to perceptual knowledge for particular stimuli. Since this original case, this pattern of deficit has been reported on several occasions [52,55,56]. The pattern confirms the distinction between structural and semantic knowledge and between apperceptive and associative agnosia. In some of the other cases, the problem is not one of access to semantic knowledge per se, but more a disorder of semantic knowledge itself. For example, the patient of Sheridan and Humphreys [55] was impaired at asking questions about the functional properties of objects from their names and when the objects were presented visually, so the problem generalized across modalities. This may be expected from a disorder of semantic knowledge, rather than a disorder of access to semantic knowledge from a specific modality (eg, spoken words vs pictures).

One other interesting aspect of JB’s deficit is that his problem in object identification was worse for some categories of objects than others. For instance, he was worse at naming living things than at naming nonliving things (tools, household items, and so forth). This apparent “category-specific” deficit has been reported in substantial numbers of patients and was first investigated experimentally by Warrington and Shallice (see references 57–60 for recent reviews). Warrington and Shallice [57] proposed that a deficit for living things could be linked with impairment to stored knowledge

about the perceptual properties of objects, because they assumed that perceptual knowledge was key to identifying living things. In contrast to this, nonliving things may be distinguished more on the basis of their functional properties (what they do or how they are used) and, thus, nonliving things would not be as dependent on access to detailed perceptual knowledge. Thus, damage to stored knowledge about the functional properties of objects would disturb nonliving things more than living things (for evidence, see Warrington and McCarthy [61,62]). Other investigators argued that such category specific deficits can reflect the categoric nature of our semantic representations, which may be organized modularly to separate animals from plant life and “other things.” At present, there is a good deal of study targeted at distinguishing these and other relevant accounts [60]. For our purposes, the important lesson is that disorders of semantic knowledge can be described as associative agnosias, and they can arise in patients with no apparent perceptual problems. A case such as JB’s is particularly interesting, however, because his problem is not in semantic knowledge itself, but in accessing this knowledge from vision. How could a deficit in visual access also generate an apparent category-specific deficit? Humphreys, Riddoch, and Quinlan [63] demonstrated that category effects are found not only in patients but also in normal participants (albeit on measures of reaction time rather than error). They argued that the effects occurred because many living things have a similar perceptual structure, and this created increased competition for recognition and naming. In a patient such as JB, competition at a semantic level may be exacerbated if the lesion adds “noise” to activation that is transmitted from the structural description system. Living things are more difficult to identify as a consequence (for a simulation, see Humphreys et al [64]).

The notion that one cause of category-specific deficits for living things arises in visual access to semantic knowledge also is supported by patients with relatively milder deficits than JB, who present with a naming rather than a recognition impairment for living things. SRB and DM suffered damage to the left medial and inferior occipito-temporal regions and they demonstrated a particular impairment in naming animate items [65,66]. Naming inanimate items was relatively preserved (71% versus 95% correct for SRB, and 46% versus 75% correct for DM for animate versus inanimate line drawings, respectively). In both cases there was no effect of name frequency, and the patients provided specific semantic information about the items they were unable to name (eg, for “kangaroo”—it lives in Australia, it carries its young in a pouch, it hops). Both patients performed within the normal range on standardized tests of semantics; however, on other tests, which stress stored knowledge of the visual features of items, performance was not as good. Thus, when drawing from memory, their drawings of animate items received lower ratings from control subjects than their drawings of inanimate items (control subjects were presented with all the drawings and asked to rate how good a representation of a target object each one was; good

representations received higher scores). Both patients were impaired somewhat, particularly when they were required to make within-category discriminations of real living objects from nonobjects created by combining the parts of living things [67]. In addition, when the patients were required to produce a name in response to definition stressing either the perceptual properties of objects (eg, the name for an orange or cone-shaped vegetable) or functional and associative properties (eg, the name of the root vegetable that is said to help you see in the dark), the patients did well with the functional and associative definitions (92% and 89% correct for SRB and DM, respectively) but more poorly on the perceptual definitions (51% and 42% correct for SRB and DM, respectively (the control mean was 93.4% and 74% correct for inanimate and animate items, respectively). The data from SRB and DM suggest that they have a mild disorder of stored perceptual knowledge about objects, along with intact semantic knowledge of the functional characteristics of objects. Furthermore, this can produce an apparent naming problem for living things, wherein patients sometimes seem to be within a tip-of-the-tongue state. Humphreys, Riddoch, and Price [68] argue that bottom-up activation of semantic knowledge from vision may be insufficient to invoke a name and that object naming requires recurrent activation of stored perceptual knowledge to differentiate activation of a target object from activation of other similar representations. Subtle impairments of stored perceptual knowledge in cases such as SRB and DM produce naming deficits because recurrent activation operates less well. This may be particularly important for the naming of living things, given the perceptual similarity between their category members.

Summary

As was originally proposed by Lissauer, visual recognition may break down either at an apperceptive or at an associative level. At an apperceptive level, finer grain distinctions may be made; the authors distinguished here between disorders of shape recognition and perceptual integration. It is not the case, however, that all patients with visual recognition deficits have impaired perception: poor recognition and naming may also result from damage to stored perceptual knowledge (eg, structural descriptions), from problems in accessing semantic knowledge, from perceptual knowledge, or from impairments to semantic knowledge itself. These represent different types of associative deficit. In some cases, mild damage to stored perceptual knowledge also generates problems that are more severe on naming than on recognition and more severe for some categories of objects than others.

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