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Neuropsychology

Visual agnosia and focal brain injury

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INFO ARTICLE

Article history:
Received 27 February 2017
Received in revised form
11 July 2017
Accepted 17 July 2017
Available online 24 August 2017

Keywords:
Visual agnosia
Prosopagnosia
Alexia
Topographagnosia
Orientation agnosia

ABSTRACT

Visual agnosia encompasses all disorders of visual recognition within a selective visual modality not due to an impairment of elementary visual processing or other cognitive deficit. Based on a sequential dichotomy between the perceptual and memory systems, two different categories of visual object agnosia are usually considered: 'apperceptive agnosia' and 'associative agnosia'. Impaired visual recognition within a single category of stimuli is also reported in: (i) visual object agnosia of the ventral pathway, such as prosopagnosia (for faces), pure alexia (for words), or topographagnosia (for landmarks); (ii) visual spatial agnosia of the dorsal pathway, such as cerebral akinetopsia (for movement), or orientation agnosia (for the placement of objects in space). Focal brain injuries provide a unique opportunity to better understand regional brain function, particularly with the use of effective statistical approaches such as voxel-based lesion–symptom mapping (VLSM). The aim of the present work was twofold: (i) to review the various agnosia categories according to the traditional visual dual-pathway model; and (ii) to better assess the anatomical network underlying visual recognition through lesion-mapping studies correlating neuroanatomical and clinical outcomes.

1. Introduction

The cognitive consequences of focal brain injury create a window into greater understanding of the visual recognition network. Despite the strengths of functional neuroimaging, including the study of normal brains to reveal distinguishable activations that correlate with selective cognitive process, detailed patient-based research is the only method for revealing causal relationships among brain systems.

Visual agnosia encompasses all disorders of visual object recognition confined to a selective perceptual (visual) modality not due to an impairment of elementary visual processing or some other cognitive deficit (such as language or memory). Visual agnosia usually refers to visual *object* agnosia. However, this report considers the entire spectrum of visual agnosia disorders, including visual *spatial* agnosia.

There is no unique taxonomy for visual object agnosia [1]. Based on a sequential dichotomy between perceptual and memory systems, the most accepted proposition distinguishes 'apperceptive agnosia' and 'associative agnosia' [2]. Patients with apperceptive agnosia fail to recognize a visual stimulus because of an impairment in perceptual processing, excluding elementary visual deficits (such as a

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http://dx.doi.org/10.1016/j.neurol.2017.07.009
0035-3787/

visual field deficit), whereas those with associative agnosia fail to associate the correct result of their visual analysis with their memory stores of the functional and semantic properties of the stimulus. However, this distinction between the two broad categories has been refined and extended ever since to reflect more elaborate perceptual models, such as the computational approach to vision [3] and the hierarchical model of object recognition [4].

Within the so-called apperceptive domain, three kinds of object agnosia are described: visual form agnosia; integrative agnosia; and transformational agnosia. Within the so-called associative domain, two sorts are described—multimodal associative agnosia (not confined to the visual modality), and semantic agnosia (a deficit of conceptual knowledge)—although both extend beyond the scope of visual agnosia *stricto sensu*. Patients with visual form agnosia are unable to recognize, match, copy or discriminate simple visual stimuli, such as a square from a circle. They perceive the traits and lines of objects, but are impaired at discerning distances, lengths and orientations. Only a few cases have been reported, with most having suffered from carbon monoxide poisoning [5–9], one from mercury poisoning [10], another from oligodendroglioma [11] and a further one from stroke [12]. Patients who have integrative agnosia are able to proceed with the individual elements of each form, but are unable to put those elements together into a perceptual whole. Object recognition is sometimes preserved through a feature-by-feature identification strategy. Two such patients have been extensively tested, one after a stroke [13], the other following meningoencephalitis [14]. The apperceptive nature of integrative agnosia has been described, and suggests that this kind of agnosia could be a specific type [15]. Finally, patients with transformational agnosia are unable to create a viewpoint-independent representation of an object, leading to difficulties in identifying objects in unusual perspectives, but not when seen in canonical views [16]. This disorder is also known as 'perceptual categorization deficit' [1].

Most patients with visual—whether object and spatial—agnosia have extensive lesions involving both hemispheres. However, studies of deficits resulting from more focal brain injuries have allowed descriptions of selective agnosia categories, such as prosopagnosia, pure alexia and topographagnosia (see Martinaud [17] for a review). In addition, the lesion-mapping approach offers a better understanding of regional brain function, beginning with a description of the traditional visual dual-pathway model [9]. Methodological improvements using statistics, such as voxel-based lesion–symptom mapping (VLSM) [18], have further enhanced our capacity to identify critical anatomical sites [19]. Thus, the aim of the present work was to review the various agnosia categories and to better assess the anatomical network underlying visual recognition, based on lesion-mapping studies correlating neuroanatomical and clinical outcomes.

2. Visual dual-pathway model

Two distinct visual pathways, the 'what' and 'where', were first reported in macaque monkey brains [20], then adjusted with some modifications in humans to include a dissociation between perception and action [21]: (i) the ventral pathway, involving the occipitotemporal cortex, allows identification of visual stimuli and their semantic attributes; and (ii) the dorsal pathway, involving the occipitoparietal cortex, allows visual control of actions, spatial localization of visual stimuli, and identification of their spatial attributes, such as orientation, depth and movement.

This model of two visual systems was inspired by the observation of double dissociations in patients with visual form agnosia due to a lesion in the ventral pathway and patients with optic ataxia due to a lesion in the dorsal pathway [22]. Two patients, R.V. and D.F., were reported as classic illustrations of this dissociation [23]. R.V., a 55-year-old woman, suffered from optic ataxia after several strokes involving bilateral lesions of the occipitoparietal region [23]. Patients with optic ataxia are unable to reach out or grasp objects, although they have no difficulty in recognizing or describing those same objects. D.F., a 34-year-old woman, developed visual form agnosia due to lesions in the ventrolateral occipital region following carbon monoxide poisoning [9]. In a well-known experiment using a slot that can be placed in many different orientations and a hand-held card, D.F. showed great difficulty in indicating the orientation of the slot, yet performed as well as normal subjects in reaching out and inserting the card into the slot [24]. More experiments with patients suffering from optic ataxia (for example, A.T.) [25,26] and those with visual form agnosia (such as J.S.) [12] have since been reported, thereby supporting this double dissociation, as has the recent study of a severe developmental impairment affecting the perception of visual objects while sparing motion processing [27].

To provide a more accurate account of reported patients with visual agnosia, different subclasses have been proposed according to the damaged pathway. A lesion in the ventral pathway could lead to a deficit of visual object identification, depending on the category of visual stimuli (face or word), whereas a lesion in the dorsal pathway could lead to a deficit linked to visuospatial attributes (movement or orientation) of the visual object. What follows here is a brief description of the main agnosia categories and their neural correlates according to lesion-mapping studies.

3. Visual object agnosia of the ventral pathway

3.1. *Cerebral achromatopsia*

In this syndrome, the patient is unable to perceive colors, which is different from color agnosia, an impaired knowledge of colors but with no difficulties in perceptual tests [28], and color anomia, an impaired ability to name colors despite no difficulties in perception or knowledge of colors [29]. The first review of 14 patients with perturbed perception of colors emphasized the bilateral involvement of the fusiform gyrus, the lingual gyrus, or both, close to the striate cortex [30]. The rarity of cerebral achromatopsia may be due to the fact that large lesions destroy the striate cortex, leading to homonymous hemianopia or cortical blindness. The most recent review of 92 cases of cerebral achromatopsia underlined the