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Research report

Agnosic vision is like peripheral vision, which is limited by crowding

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ABSTRACT

Visual agnosia is a neuropsychological impairment of visual object recognition despite near-normal acuity and visual fields. A century of research has provided only a rudimentary account of the functional damage underlying this deficit. We find that the object-recognition ability of agnosic patients viewing an object directly is like that of normally-sighted observers viewing it indirectly, with peripheral vision. Thus, agnosic vision is like peripheral vision. We obtained 14 visual-object-recognition tests that are commonly used for diagnosis of visual agnosia. Our "standard" normal observer took these tests at various eccentricities in his periphery. Analyzing the published data of 32 apperceptive agnosia patients and a group of 14 posterior cortical atrophy (PCA) patients on these tests, we find that each patient's pattern of object recognition deficits is well characterized by one number, the *equivalent eccentricity* at which our standard observer's peripheral vision is like the central vision of the agnosic patient. In other words, each agnosic patient's equivalent eccentricity is *conserved* across tests. Across patients, equivalent eccentricity ranges from 4 to 40 deg, which rates severity of the visual deficit.

In normal peripheral vision, the required size to perceive a simple image (e.g., an isolated letter) is limited by acuity, and that for a complex image (e.g., a face or a word) is limited by crowding. In *crowding*, adjacent simple objects appear unrecognizably jumbled unless their spacing exceeds the *crowding distance*, which grows linearly with eccentricity. Besides conservation of equivalent eccentricity across object-recognition tests, we also find conservation, from eccentricity to agnosia, of the relative susceptibility of recognition of ten visual tests. These findings show that agnosic vision is like eccentric vision.

Whence crowding? Peripheral vision, strabismic amblyopia, and possibly apperceptive agnosia are all limited by crowding, making it urgent to know what drives crowding. Acuity does not (Song et al., 2014), but neural density might: neurons per deg^2 in the crowding-relevant cortical area.

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1. Introduction

1.1. *Visual apperceptive agnosia*

Visual agnosia is a neuropsychological disorder characterized by the inability to recognize familiar objects. Visual agnosia patients are generally unable to recognize visually presented objects, but they can successfully name the object on the basis of tactile exploration and they correctly describe the object's function from its name. Such impairment must be distinguished from early sensory deficits (e.g., low visual acuity or contrast sensitivity), oculomotor disturbances, attentional deficits, aphasic syndromes, and mental deterioration (De Renzi, 1996; Farah, 1990). It is remarkable that these patients can recognize a tiny letter when tested for acuity, yet cannot recognize everyday objects.

The nature of visual agnosia is debated, and patients within this gross category are diverse. For a recent review, see Behrmann and Nishimura (2010). Neuropsychological studies of brain-damaged patients have found selective deficits for words (pure alexia), objects (pure visual object agnosia), and faces (prosopagnosia) (Farah, 2004). The inhomogeneity of the visual agnosic population reported in the literature may reflect the various neural sites of the lesion and the varying degree of neural damage (Adler, 1944; Behrmann & Nishimura, 2010; Benson & Greenberg, 1969; Campion & Latto, 1985; Milner et al., 1991; Vecera & Behrmann, 1997). The classical description (Lissauer, 1890/1988) distinguishes "apperceptive" agnosia, which is a perceptual processing deficit, from "associative" agnosia, which is a deficit either in semantic knowledge of visual objects or in accessing that knowledge. Associative agnosia patients have trouble recognizing a variety of visually presented objects, despite their intact visual perception, which is usually demonstrated by having the patients copy objects that they cannot recognize. Patients that show highly selective impairment of one object category (i.e., pure alexia, pure visual object agnosia, and prosopagnosia) are classically said to have an associative deficit (Farah, 2004). We will consider the debate about category-specific deficits in visual agnosia in the Discussion section. Putting associative deficits aside, here we focus on apperceptive agnosia. Loosely, "apperceptive" refers to sensation without perception, marked by detection without recognition. The apperceptive deficit comprises a broad range of symptoms. Several authors have proposed a detailed taxonomy of visual apperceptive agnosia, differentiating it into: shape/form agnosia (Efron, 1968; Milner et al., 1991; Riddoch, Johnston, Bracewell, Boutsen, & Humphreys, 2008), integrative agnosia (Riddoch & Humphreys, 1987), transformational agnosia (Humphreys & Riddoch, 1987; Warrington, 1985), and perceptual categorization deficit (Farah, 2004). The deficits range from severe—in patients who cannot even discriminate simple geometric shapes (shape agnosia)—to mild—in patients who seem unimpaired in their daily lives, but who fail, at the clinic, to recognize familiar objects in photographs taken from unusual perspectives (transformational agnosia). Between these two extremes, there is a wide intermediate range of deficit that is sometimes called "integrative agnosia" (Riddoch & Humphreys, 1987). Here, we apply the term *apperceptive agnosia* to this broad category of patients with an intermediate degree of deficit. These patients with visual agnosia are profoundly impaired in object recognition, face recognition, word recognition, and reading. They may show signs of achromatopsia and topographical agnosia as well. They do recognize an isolated letter. They typically perform better with real objects than with drawings and photographs, but only if the objects are presented in isolation or in motion. This syndrome is usually associated with either bilateral occipito-temporal lesions or unilateral right occipito-temporal lesion sparing striate cortex and parietal areas (Humphreys, 1999).

Popularized by Oliver Sacks (1998) in "*The man who mistook his wife for a hat*," apperceptive agnosia has long attracted keen interest for the investigation of integration in object recognition and how we produce a single coherent percept (Behrmann & Kimchi, 2003; Lissauer, 1890; Riddoch & Humphreys, 1987). Despite severely impaired visual recognition of the object, these patients can verbally describe what they perceive, though their descriptions are often piecemeal. When presented with a drawing of a paintbrush, HJA (one of the most famous and well-studied cases of visual agnosia) said, "it appears to be two things close together; a longish wooden stick and a shorter, darker object, though this can't be right or you would have told me." (Riddoch & Humphreys, 1987, p. 60).

Despite detailed descriptions of individual patients spanning the whole range of symptoms associated with this syndrome, there is still no comprehensive account. According to Riddoch and Humphreys (1987), apperceptive agnosia is an "integration deficit": The patients can process local visual elements but cannot integrate them into a whole. However, contrary to this generalization, some apperceptive agnosia patients perform better with silhouettes than with drawings (Humphreys, Riddoch, & Quinlan, 1985; Lê et al., 2002), which presumably requires some integration (Humphreys, 1999). On the other hand, these patients are still impaired in recognizing a single part of a complex object. For instance, they are slower than normally-sighted observers in processing a "local" letter embedded in "global" letter (Behrmann & Kimchi, 2003; Navon, 1977). The interplay between impairments of recognition of single parts and complex objects remains mysterious.

Apperceptive agnosia severely impairs vision yet spares acuity and visual fields. Patients with visual agnosia can recognize small simple shapes (e.g., a letter) when presented in isolation. Most visual impairments (e.g., macular degeneration or anisometropic amblyopia) restrict visual field or acuity, and are well characterized by those restrictions. However, there are several conditions, like apperceptive agnosia, that impair central vision while sparing acuity and fields. We focus on perceptual deficits (hence apperceptive agnosia), putting aside high-level attentional deficits such as neglect and simultanagnosia (and associative agnosias). The perceptual deficits of central vision that spare acuity include: apperceptive agnosia, achromatopsia (color agnosia), akinetopsia (motion blindness), dysmetropsia (failure of size constancy), transformational agnosia (inability to recognize objects seen from an unusual perspective), and depth perception deficits. Among them, only apperceptive agnosia specifically impairs recognition of complex shapes.