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Evidence for perceptual deficits in associative visual (prosop)agnosia: a single-case study

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Abstract

Associative visual agnosia is classically defined as normal visual perception stripped of its meaning [Archiv für Psychiatrie und Nervenkrankheiten 21 (1890) 22/English translation: Cognitive Neuropsychol. 5 (1988) 155]: these patients cannot access to their stored visual memories to categorize the objects nonetheless perceived correctly. However, according to an influential theory of visual agnosia [Farah, Visual Agnosia: Disorders of Object Recognition and What They Tell Us about Normal Vision, MIT Press, Cambridge, MA, 1990], visual associative agnosics necessarily present perceptual deficits that are the cause of their impairment at object recognition Here we report a detailed investigation of a patient with bilateral occipito-temporal lesions strongly impaired at object and face recognition. NS presents normal drawing copy, and normal performance at object and face matching tasks as used in classical neuropsychological tests. However, when tested with several computer tasks using carefully controlled visual stimuli and taking both his accuracy rate and response times into account, NS was found to have abnormal performances at high-level visual processing of objects and faces. Albeit presenting a different pattern of deficits than previously described in integrative agnosic patients such as HJA and LH, his deficits were characterized by an inability to integrate individual parts into a whole percept, as suggested by his failure at processing structurally impossible three-dimensional (3D) objects, an absence of face inversion effects and an advantage at detecting and matching single parts. Taken together, these observations question the idea of separate visual representations for object/face perception and object/face knowledge derived from investigations of visual associative (prosop)agnosia, and they raise some methodological issues in the analysis of single-case studies of (prosop)agnosic patients.

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1. Introduction

Visual agnosia is a deficit in object recognition confined to the visual modality, despite intact elementary visual processes such as visual acuity, visual field, visual scanning and attention, and which is not due to general problems in language, memory or deficiency in intellectual abilities. Neuropsychological investigations have described cases of visual agnosia belonging to the two broad classes of disorders introduced by Lissauer (1890), namely visual *apperceptive*, and visual *associative* agnosia. As defined in the literature, apperceptive agnosics present visual deficits, which prevent them to elaborate a correct percept of the stimulus. Associative agnosics, on the other hand, are considered as being able to construct a normal visual percept that cannot be adequately associated with visual representations of objects stored in memory. This distinction has been extended to a particular type of visual agnosia, the inability to recognize faces, or prosopagnosia (Bodamer, 1947): *apperceptive prosopagnosics* cannot elaborate a correct percept of a face, whereas *associative prosopagnosics* are unable to give any meaning to a correctly elaborated visual representation of an individual face (De Renzi, Faglioni, Grossi, & Nichelli, 1991).

Humphreys and Riddoch (1987) have elaborated upon the basic distinction of apperceptive and associative agnosia, drawing a clear boundary between perceptual and mnesic processes involved in object recognition. Referring to a classical hierarchical cognitive architecture of visual object recognition, these authors have described different forms of apperceptive and associative agnosias, depending

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on the stage at which the visual object processing is impaired.

A different view has been put forward by Farah (1990). Although this author also described a taxonomy of apperceptive agnosias and acknowledged the heterogeneity of associative agnosias,[1] a review of 99 cases of associative agnosias described over the century in the English-speaking literature led her to conclude that there was no clear evidence of the sparing of high-level visual processes in the cases of associative visual (prosop)agnosia[2] reported. She argued that all cases of associative visual agnosia present perceptual deficits that are the cause of their impairment at object recognition, and thus that perceptual and mnesic representations involved in object recognition were not clearly distinct. Farah's account is based on the observation that very few of the patients who have been described as associative agnosics have been adequately tested for perceptual abnormalities. In the rare cases where particularly demanding visual tasks have been presented to associative agnosic patients, their results have suggested some critical deficits at high-level visual processes (Farah, 1990).

Since these two theoretical proposals have been formulated, a number of acquired cases of visual agnosias for objects (e.g. Behrmann & Kimchi, 2003; Behrmann, Moscovitch, & Winocur, 1994; Behrmann, Winocur, & Moscovitch, 1992; Humphreys & Rumiati, 1998; Moscovitch, Winocur, & Behrmann, 1997; Turnbull & Laws, 2000) and for faces (e.g. De Renzi & di Pellegrino, 1998; De Renzi et al., 1991; Henke, Schweinberger, Grigo, Klos, & Sommer, 1998; McNeil & Warrington, 1991; Schweich & Bruyer, 1993) have been reported, that may be classified as the 'associative' type. However, these studies investigated a number of different theoretical questions related to object and face recognition, without explicitly testing the hypothesis of the necessary visual impairments in the 'associative' cases that they described.

Here we report the case study of NS, a case of acquired visual agnosia for objects and faces with intact knowledge of object function as well as object recognition through tactile and auditory modalities. NS' case is particularly interesting because he has no low-level visual deficits, and his performances at copying and matching objects appears remarkably normal. Furthermore, tested with a classical face recognition battery (Bruyer & Schweich, 1991), he was found to be normal at all tasks of perceptual processing of faces but dramatically impaired at recognition processes. In this report, we investigate NS' high-level visual processes in detail, in order to test the claim that a patient defined as a visual associative (prosop)agnosia, should necessarily show some visual impairments that are the cause of his/her deficit. Our investigation was motivated by recent studies of prosopagnosic patients and normal subjects, which have raised a number of issues regarding the conclusions that are usually drawn from analyses of patterns of errors alone, using classical object and face recognition tests. For instance, recent evidence indicates that normal subjects can perform reasonably well at the Benton facial matching test as well as at the face recognition test of Warrington (1984)—two standards in the literature—when they have to rely exclusively on external features, and the time to perform the test is not taken into account (Duchaine & Weidenfeld, 2003; see also Davidoff & Landis, 1990; Sergent & Signoret, 1992). Claims for normal abilities in object recognition have also been made in several cases in which the quality of the stimuli set used was poor, allowing the patient to attend to a single salient feature of the object or its background to perform the task (e.g. McNeil & Warrington, 1993). In addition to a lack of control in the use of stimuli, most studies report performance score only, without any precise information about the time taken by the patients to perform the object matching tasks, for instance. However, recent studies have shown that measuring response times was critical in revealing abnormal performances at object perception tasks in cases of visual agnosia and prosopagnosia (Gauthier, Behrmann, & Tarr, 1999).

Given these concerns, we tested our patient extensively with different visual tasks performed on computer, measuring accuracy rates and RTs. We compared the performance and RTs of our patient to normal subjects matched for age and level of education. Doing these tests, we were also particularly aware to the fact that apparently slight abnormal performances in tests of visual perception may arise of difficulties in accessing stored visual representations or to damage to these representations themselves. Accordingly, in addition to the use of RTs measures, a distinctive characteristic of the present study was the use of novel objects in several visual tasks. This strategy allowed us to cancel any support for matching tasks that could be extracted from prior knowledge of the stimuli for control subjects, and would thus make them perform relatively better than NS.

2. Case study

2.1. NS: clinical history

NS (born 1951) is a right-handed man who was 40 years old when he was hit by a car while cycling, and remained unconscious for 23 days. He was first evaluated in September 1991, a few days after recovery. The neuropsychological examinations revealed sensory transcortical aphasia, a severe dyslexia and dysgraphia, signs of apraxia, anterograde amnesia, anosognosia, a bilateral superior quadranopsia and a severe visual agnosia for objects, faces, colors and places. Over the next 2 years, NS underwent cognitive rehabilitation,

[1] Although Farah (1990, 1991) makes a clear distinction between visual associative agnosia, on the one hand, and on the other hand, the cases of visual modality-specific anomia ("optic aphasia") and the loss of general semantic knowledge (not confined to the visual modality). See Section 4.

[2] Since Farah's view concerns both visual agnosia for objects and prosopagnosia, and our patient suffers from clear deficit at both object and face recognition, we will use the term visual agnosia in the larger sense, i.e. including prosopagnosia.

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