

PROSOPAGNOSIA AND ALEXIA WITHOUT OBJECT AGNOSIA

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ABSTRACT

Following a trauma causing bilateral posterior brain damage, a patient complained of dyslexia and prosopagnosia, but not object agnosia. On testing she showed intact recognition of object drawings, even when it was assessed with perceptually demanding tasks such as Ghent's overlapping figures and Street completion test. This pattern of deficit is inconsistent with Farah's (1990) prediction that the simultaneous occurrence of alexia and prosopagnosia is invariably associated with object agnosia.

The patient's reading performance had the features typically found in letter-by-letter readers. On face tests, she showed a discrepancy between the impairment exhibited in familiarity recognition and famous face naming and the correct (though slow) performance in matching the names of famous persons with their photographs. This apparent contradiction was clarified by showing that the patient had maintained the ability to generate the mental images of famous faces in response to the presentation of their names. We assume that face recognition units were intact, but partially disconnected from the output of perceptual processing.

Key words: alexia, object agnosia, prosopagnosia

INTRODUCTION

Ever since the early descriptions of visual recognition disorders, it has been recognized that they can be prevalent or even restricted to a specific class of stimuli, leaving other visuo-perceptual domains unimpaired. For instance, alexia and prosopagnosia are seldom associated and both have been reported without object agnosia. To account for these findings it has been hypothesized that discrete brain structures and mechanisms are specialized for the processing of particular types of stimuli. However, it is not clear whether the specificity concerns perception or stored knowledge. Farah (1990) favoured the former view, arguing that not only apperceptive agnosia but also associative agnosia is contingent upon defective perceptual processing, which would affect "high-level (i.e., relatively late, abstract) perceptual representation of shape". She further speculated that the processing of visual stimuli is mainly mediated, depending on their nature, by two discrete perceptual abilities, involving either the analysis of their constituent parts or their apprehension as a whole. Words and faces are examples of stimuli that undergo different processing modes. The encoding of words requires that they be broken down into the letters composing them, while faces are encoded as a global gestalt. Either mechanism can be independently

disrupted and alexia and prosopagnosia can, therefore, occur independently from one another. In Farah's view, objects are not represented in one encoding mode, but share both, thus allowing a certain degree of compensation, when just one of the two is deranged. Object agnosia will, consequently, result either from the severe impairment of one encoding mode (in which case it will be associated with alexia or prosopagnosia), or from the disruption of both (in which case all three of the agnosic symptoms will be presented). Clinical support for this theory was found in a review of the literature, which showed a spectrum of patterns of association and dissociation among the three forms of recognition deficit. There were, however, two notable exceptions: object agnosia never occurred in isolation and alexia and prosopagnosia were never associated in the absence of object agnosia. These are the findings predicted by the two representational capacity model and Farah explicitly stated that the it would be falsified by the report of a case of object agnosia without alexia or prosopagnosia or a case of alexia and prosopagnosia without object agnosia.

A case showing the former dissociation was published by Rumiati, Humphreys, Riddoch et al. (1994) and a brief report of a patient with the latter type of dissociation was presented by Buxbaum, Closer and Coslett (1996) at Tennet VII. The patient described here showed alexia and prosopagnosia, but no deficit of object recognition. She was given a wide spectrum of tests, which provided a means to localize the functional damage underlying the impairment of face recognition at the level of the defective activation of the face representation store.

CASE REPORT

Anna (a fictitious name) was a 43-year-old, right-handed woman, when in July 1993, she was involved in a car accident resulting in thoracic and cranial trauma. She was comatous on admission to the intensive care unit of a near-by hospital. At this time there were pulmonary complications and she was operated upon for fractures of the humerus and femur. She progressively regained consciousness over 20 days and was eventually able to live an autonomous life and to return to work. She teaches mathematics at the university and is married with no children.

When we first saw her in June 1994, neurological examination revealed only a mild motor impairment of the left hand. However, she complained of having difficulties in reading and recognising familiar faces. She reported that, although possible, reading was so slow and wearing that she was forced to give it up, which made lecture preparation difficult. Familiar people identification was impaired in real life, especially when they were met out of the usual context. She also failed to recognise persons first met after the disease (e.g., she never recognised any of us until we introduced ourselves).

MRI, carried out in June 1994, showed bilateral areas of hypodensity, which were more marked in the right hemisphere, where a large lesion involved the infra and the supracalcarine areas 18 and 19, the posterior cingulate gyrus (areas 23 and 31) and the mesial part of the superior parietal lobe (areas 5 and 7). A separate lesion encroached upon the white matter of the motor and premotor area and the lateral and mesial part of the superior parietal lobe. In the left hemisphere the lesion involved the lateral part of area 19, bordering on area 37. Figure 1 shows the mapping of the lesions onto the template set developed by Damasio and Damasio (1989).

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