

Differential Impact of Parvocellular and Magnocellular Pathways on Visual Impairment in Apperceptive Agnosia?

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Abstract

The term “visual form agnosia” describes a disorder characterized by problems recognizing objects, poor copying, and distinguishing between simple geometric shapes despite normal intellectual abilities. Visual agnosia has been interpreted as a disorder of the magnocellular visual system, caused by an inability to separate figure from ground by sampling information from extended regions of space and to integrate it with fine-grain local information. However, this interpretation has hardly been tested with neuropsychological or functional brain imaging methods, mainly because the magnocellular and parvocellular structures are highly interconnected in the visual system.

We studied a patient (AM) who had suffered a sudden heart arrest, causing hypoxic brain damage. He was/is severely agnosic, as apparent in both the Birmingham Object Recognition Battery and the Visual Object and Space Battery. First- and especially second-order motion perception was also impaired, but AM experienced no problems in grasping and navigating through space. The patient revealed a normal P100 in visual evoked potentials both with colored and fine-grained achromatic checkerboards. But the amplitude of the P100 was clearly decreased if a coarse achromatic checkerboard was presented.

The physiological and neuropsychological findings indicate that AM experienced problems integrating information over extended regions of space and in detecting second-order motion. This may be interpreted as a disorder of the magnocellular system, with intact parvocellular system and therefore preserved ability to detect both local features and colors.

Introduction

Benson and Greenberg (1969) described a syndrome which they named “visual form agnosia.” The case they described suffered from no serious intellectual impairment, but from object recognition problems and poor copying abilities. As shown by Efron (1969) the patient was unable to distinguish between simple geometric shapes. Several other patients with similar impairments have been described (Adler, 1944, 1950; Abadi *et al.*, 1981; Campion and Latto, 1985). Extensive testing of these patients showed multiple dissociations between “figure-ground separation” (Kartsounis and Warrington, 1991), “shape discrimination (Davidoff and Warrington, 1993), and “luminance and color discrimination” (Heywood *et al.*, 1994). These dissociations have been taken as an argument for segregated visual pathways in the human brain, which can be lesioned focally and therefore

produce different classes of symptoms. A subgroup of these patients cannot distinguish between different shapes, sizes, and shadings of grey. One popular explanation for these symptoms proposes a selective damage of the magnocellular visual pathway (Milner *et al.*, 1991; Davidoff and Warrington, 1993).

The term “magnocellular” refers to a subdivision of the visual system originating in the alpha ganglion cells of the retina, projecting to layers 4B and 4C of the striate cortex (V1), and thence to the “thick stripes” of the adjacent area V2 (Livingstone and Hubel, 1988). The magnocellular pathway is supposed to predominantly analyse depth and motion cues, while it is “color blind.” On the other hand, because of the larger receptive fields of its neurons, it has also been related to the perception of size and length differences