A CASE OF PROSOPAGNOSIA FOLLOWING MODERATE CLOSED HEAD INJURY WITH LEFT HEMISPHERE FOCAL LESION

Audrey J. Mattson[1], **Harvey S. Levin**[2] **and Jordan Grafman**[3]

([1]Neuropsychology Service, Western State Hospital, Steilacoom, Washington; [2]Departments of Physical Medicine and Rehabilitation, Psychiatry and Behavioral Sciences, and Neurosurgery, Baylor College of Medicine, Houston, Texas; [3]Cognitive Neuroscience Section, Medical Neurology Branch, National Institutes of Health, Bethesda, Maryland)

Abstract

This study describes a left handed woman with prosopagnosia following traumatic brain injury with a focal lesion confined to the left-posterior hemisphere. Few cases of prosopagnosia following unilateral left hemisphere lesions have previously been reported in the literature.

Corrected visual acuity was 20/70 (binocular), color vision was intact on screening, and shape detection was borderline. Impairments in higher order visual perception were evident to varying degrees on nonfacial tasks. Matching of unfamiliar faces was very slow but accurate. A marked impairment in the ability to recognize familiar faces and learn new face-name associations was evident on experimental tasks relative to the performance of healthy control subjects. In contrast, identification of characteristics of faces (gender, age) and identification and matching of facial expressions were relatively preserved. We discuss the cognitive processing stages that appear to be disrupted using Bruce and Young's (1986) model of facial recognition and perception as a framework.

Introduction

Prosopagnosia, an inability to recognize familiar faces, is usually the result of vascular insult or cerebral tumor, but cases have been reported in patients following head trauma and infectious disease (Levin and Peters, 1976; Levine and Calvino, 1989; De Renzi, Faglioni, Grossi et al., 1991). Prosopagnosia cannot be attributed to aphasia, global cognitive impairment or pervasive visual perceptual disturbance because the inability to recognize familiar faces is more severe than would be expected on the basis of the visual perceptual or cognitive deficits alone (Damasio, Damasio and Van Hoesen, 1982). However, patients often exhibit visually related dysfunction, especially visual field defects and central achromatopsia, alexia, and visual object agnosia (Damasio et al., 1982). Clinical case reports and analysis of post-mortem data indicate that prosopagnosia usually occurs in patients with bilateral damage to inferior occipital and temporal association cortices (Damasio et al., 1982; Damasio and Damasio, 1986; Benton,1990; and Boeri and Salmaggi, 1994). The presence of bilateral lesions is illustrated in De Renzi and Di Pellegrino's (1998) case of prosopagnosia following severe closed head injury in which magnetic resonance imaging (MRI) one year postinjury showed right hemisphere lesions in visual

areas 18 and 19, the posterior cingulate gyrus (areas 23 and 31), the mesial part of the superior parietal lobe (areas 5 and 7), and the white matter of the motor and premotor area and the lateral and mesial part of superior parietal lobe. A left hemisphere lesion involving the lateral part of area 19 was also present. A few cases of prosopagnosia with unilateral posterior, right hemisphere lesions (De Renzi, 1986; Benton, 1990; De Renzi, Perani, Carlesimo et al., 1994; Takahashi, Kawamura, Hirayama et al., 1995) have raised the question of whether bilateral lesions are necessary to disrupt recognition of familiar faces. Investigations of face perception in brain damaged patients (Bruyer, 1986; Carlesimo and Caltagirone, 1995) and in normal subjects using functional imaging techniques (Mishkin, Ungerleider and Macko, 1983; Haxby, Grady, Horwitz et al., 1991; Grady, Maison, Horwitz, et al., 1994; McIntosh, Grady, Ungerleider et al., 1994; Sergent and MacDonald, 1992) also indicate that face perception is primarily a function of posterior brain regions, particularly right occipital-temporal association areas.

Traditionally two forms of visual agnosia, as well as prosopagnosia, have been described in the medical and neuropsychological literature, one characterized by marked visual perceptual impairment and one related to a mnestic disturbance (Damasio et al., 1982; De Renzi, Faglioni, Grossi et al., 1991). Several cognitive models of facial recognition have been developed recently which are based on neuropsychological and experimental studies (Rhodes, 1985; Young, Newcombe, De Haan et al., 1993; Wilson, Clare, Young et al., 1997). These models conceptualize face processing and recognition as made up of various processing modules, or components, which can be differentially disrupted depending on the site of lesion. Rhodes' (1985) model assumes that face perception is a complex and dynamic process (i.e., the component processes occur simultaneously with "bottom up" and "top down" feedback loops), and indicates that while face processing is primarily carried out within the right hemisphere, the left hemisphere is involved to varying degrees at different stages. Rhodes' (1985) model and neuropsychological findings in brain injured subjects (Carlesimo and Caltagirone, 1995) indicate that the left hemisphere role is limited primarily to recall of proper names and possibly other semantic information about the individuals being perceived. Bruce and Young's (1986) cognitive model of face recognition contains separate and distinct functional pathways for the processing of facial characteristics including expressions, the directed visual analysis required to match unfamiliar faces, and the recognition of familiar faces. Consistent with Bruce and Young's model, De Renzi and Di Pellegrino's case of posttraumatic prosopagnosia exhibited intact recognition of recurring unfamiliar faces and random designs, accurate matching of facial emotional expressions with their corresponding verbal label, and preserved object recognition. Young and coworkers (Young, Newcombe, De Haan et al., 1993) also provided partial support for this model by identifying patients with unilateral left or right posterior hemispheric lesions who exhibited difficulty identifying facial expressions, but whose ability to recognize familiar faces and match unfamiliar faces was intact. Although Young et al. provided evidence that the processes involved in the recognition of facial expression are distinct from the processes involved in face recognition and face matching, they