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Patients with schizophrenia show deficits on spatial frequency doubling

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ABSTRACT

There are pieces of evidence indicating that visual deficits in patients with schizophrenia can be attributed to a deficiency in the magnocellular portion of the early visual system. The main objective of this study was to investigate the neurological dysfunction of the magnocellular pathway in patients with schizophrenia using the frequency doubling technology perimetry (FDT). The FDT has been developed based on particular neural magnocellular characteristics and can examine the magnocellular dysfunction hypothesis in schizophrenia. Twenty patients with schizophrenia (12 males and 8 females) and 20 normal subjects (10 males and 10 females) participated in this study. The spatial frequency doubling task was presented via the Humphrey perimetry instrument in order to examine the magnocellular pathway of the participants. Patients with schizophrenia showed less visual field sensitivity than normal controls and their standardized age cohort in both eyes ($p < 0.001$). The results indicated impaired visual field sensitivity deficits in patients with schizophrenia that can be attributed to a deficit in the magnocellular neural pathways. This Magnocellular pathway defect may provide a physiological base to explain some of the deficits caused by schizophrenia such as cognitive deficits.

1. Introduction

Visual processing deficits in schizophrenia have long been considered by researchers. Earlier research had shown that patients with schizophrenia have deficits in visual integration and normal backward masking (Saccuzzo & Braff, 1981). More recent research indicated reduced contrast sensitivity (Butler & Javitt, 2005), reduced visual ERP amplitudes (ERPs), increased thresholds for evaluation of visual stimuli (Butler et al., 2005; Slaghuis, 1998), reduced neurophysiologic response to a single stimulus (Butler et al., 2001), deficits in motion processing (e.g. Chen et al., 2004), and deficits in spatial–temporal integration (Makarem et al., 2010) in patients with schizophrenia. The relation between these deficits is not well understood.

Some researchers have proposed that a portion of these visual processing deficits might be attributed to a potential malfunction of the magnocellular pathway in patients with schizophrenia (e.g., Kim et al., 2006; Martinez et al., 2008; Schechter et al., 2003).

The visual pathway consists of three main streams: Magnocellular, Parvocellular, and Koniocellular. The Magnocellular stream (M cells) is responsible for transferring low spatial frequency and high temporal frequency information, i.e. it is responsible for vision in low light and seeing moving objects. The Parvocellular pathway (P cells) is responsible for transferring high spatial frequency and low temporal frequency information, i.e. it is responsible for detailed vision and seeing static objects. The Koniocellular pathway (K cells) is responsible for transferring short or blue wavelength (Mashayekhy et al., 2008). It is expected that magnocellular deficits emerge at low spatial frequencies (e.g. 1.5 c/deg) and high temporal frequencies (Skottun & Skoyles, 2007a).

Research have shown that the magnocellular pathway transfers information related to movement, stereopsis, spatial localization, depth perception, hyperacuity, figural grouping, illusory border perception, and figure/ground separation (Livingstone, 1987; as cited in Patel, 2004).

A magnocellular pathway dysfunction has been reported by several investigators in patients with schizophrenia using different techniques. Martinez et al. (2008) reported a reduced activity to low spatial frequencies (but not high spatial frequencies) in several areas of the parietal and temporal lobes using functional magnetic resonance imaging. Kim et al. (2006) showed that deficits in motion processing in schizophrenia are significantly associated with reduced activity of the magnocellular vision systems using steady state Visual Evoked Potentials (ssVEP). Schechter et al. (2003) showed a significant functional impairment in schizophrenic patients' magnocellular pathways using a backward masking task. It has also been observed that patients with schizophrenia have reduced electrophysiological activity for magnocellular oriented stimuli (Butler et al., 2001).

Another effective and reliable way to separate magnocellular activities in psychosomatic tests is the measurement of contrast sensitivity (Skottun & Skoyles, 2007a). Studies on injuries to different layers of Lateral Geniculate Nucleus (LGN) of monkeys found

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that reduction in contrast sensitivity following injury in magnocellular layers is limited to cases where stimuli are of low spatial frequency or high temporal frequency (Merigan, Byrne, & Maunsell, 1991a; Merigan, Katz, & Maunsell, 1991b; Merigan & Maunsell, 1990, 1993; Schiller, Logothetis, & Charles, 1990a, 1990b; as cited in Skottun & Skoyles, 2007a). Psychosomatic studies on humans are consistent with these findings (Legge, 1978, Tolhurst, 1975; as cited in Skottun & Skoyles, 2007a).

Several studies have reported significant differences between patients with schizophrenia and the control groups in different spatial contrast sensitivity tasks indicative of magnocellular inefficiency (e.g., Butler et al., 2005; Keri et al., 2002; O'Donnel et al., 2006; Revheim et al., 2006; Schwartz, McGinn, & Winstead, 1987; Slaghuis, 1998, 2004; Slaghuis & Bishop, 2001; Slaghuis & Thompson, 2003).

Some studies investigated spatial and temporal frequencies via a combined method. In a study by Keri et al. (2005), a spatial origin task was used. The results of this study showed that, like their biological relatives, patients with schizophrenia – under both medication and non-medication – have more definitive deficits in situations of low contrast and doubled frequency compared to consistent lighting. These findings support the magnocellular deficits in schizophrenia, because low contrast and doubled frequency are likely indicators of magnocellular inefficiency. Chen et al. (2004) showed varying degrees of reduced sensitivity for part of subjects with schizophrenia. In contrast, Keri et al. (2000), Gutherie, McDowell, and Hammond (2006) and Delord et al. (2006), found no deficits related to magnocellular malfunction in patients with schizophrenia.

Research conducted to date has investigated the magnocellular deficits in schizophrenia and other disorders using different tasks. Each of these tasks considers a specific deficit in schizophrenia (e.g. backward masking, motion processing, contrast sensitivity, etc.) or measures low spatial frequencies or high temporal frequencies which are features of the magnocellular pathway. There are many disputes about the reliability and validity of these instruments. Although the results of these studies, are consistent with the magnocellular system malfunction in schizophrenia disorder, some other studies tend to disprove them (e.g. Barch et al., 2003; Braus et al., 2002; Delord et al., 2006; Gutherie, McDowell, & Hammond, 2006; Selemon & Begovic, 2007; Skottun & Skoyles, 2007a, 2007b; Slaghuis & Bishop, 2001). There are substantial controversies over research methods and it seems that a measurement instrument is required that specifically focuses on features unique to the magnocellular pathway and which evaluates its function validly and reliably.

Kelly (1966; as cited in Patel, 2004) introduced a phenomenon named the doubled frequency illusion. In this frequency-doubling task the stimuli include alternating black and white stripes with low spatial frequency (0.25 c/deg). The place of these white and black stripes changes alternatively with high temporal frequency (25 Hz) creating a flickering appearance. In this situation, the number of black and white stripes appears to be doubled.

In the 1990s, this phenomenon was linked to a subset of type M (Magnocellular) cells that could selectively become damaged in glaucoma. Since then many researchers have studied this phenomenon (Maddess & Henry, 1992; Quigley et al., 1987; Johnson, 1994; Johnson & Samuel, 1997; Maddess et al., 1999; Maddess & Severt, 1999; Kalaboukhova & Lindblom, 2003, all cited in Patel, 2004). It is believed that the perception of this low spatial frequency sinusoidal grating with high flickering temporal frequency occurs due to the non-linear magnocellular mechanisms (Johnson & Demirel, 1997).

The Humphrey Matrix is the latest perimetry generation of frequency doubling technology (FDT) which was developed in 2003 with seven functional tests for the eye care professions (Patel, 2004). It is considered as a detailed, fully equipped and reliable visual field testing instrument that evaluates the magnocellular pathway directly taking into account its special features. Doubled frequency perimetry provides a contrast sensitivity test for detecting magnocellular pathway deficit (Anderson & Johnson, 2003). Many studies have shown that doubled frequency perimetry can detect impairments in the visual field that are overlooked by other methods (Dublin, 2003). The Humphrey Matrix has high differential ability in detecting early functional damage in patients at risk (Spry et al., 2005; as cited in Zeppieri & Johnson, 2008). Various studies have provided promising results using the Humphrey Matrix which is the result of its precision, accuracy, sensitivity, specificity, and reliability (Johnson et al., 1999; Turpine et al., 2003; Arts et al., 2005; Anderson & Johnson, 2005; all cited in Zeppieri & Johnson, 2008).

The FDT visual field instrument selectively examines the magnocellular visual pathway (Patel, 2004), but has not previously been used to investigate visual function in schizophrenia. This study utilizes doubling frequency technology to test the magnocellular deficit hypothesis in patients with schizophrenia.

2. Materials and methods

2.1. Design

The research method for this study was causal–comparative. The independent variable was schizophrenia (belonging to the group) and the dependent variable was Magnocellular deficit measured through evaluation of visual field sensitivity by means of Humphrey perimetry frequency doubling technology.

2.2. Subjects

The experimental group included all non-hospitalized patients with schizophrenia who referred to Hafez Hospital, Shiraz. The control group included eye clinic staff and patients' companions. The sample included 20 patients (12 males and 8 females) and 20 healthy persons (10 males and 10 females) as control group. Inclusion criteria for the experimental group (selected via convenience sampling method) restricted to patients in the 18–50 years old age range, with normal vision (20/20 as tested by Snellen chart), and with a diagnosis of acute schizophrenia, as diagnosed by a psychiatrist based on clinical interview. The patients were selected based on DSM-IV-TR diagnostic criteria. The exclusion criteria included physical and mental illnesses apart from the main diagnosis (such as drug abuse, mental retardation or having a severe emotional disturbance). The control group participants were selected via convenience sampling method. The exclusion criteria for this group included any psychiatric diagnosis or history of psychiatric diagnosis, neurological illness, head injury, accident or medical eye disorders. A review of these variables was accompanied by participants' or their companions' reports.

Patients, who referred to Department of Psychiatry in Hafez Hospital and had schizophrenia diagnosis, were referred to the researcher by the psychiatrist. After interviewing each patient and ensuring that they met the criteria of schizophrenia and other inclusion criteria, the researcher explained the perimetry test and its duration and the purpose of research. The patients were told that the test was performed in the eye ward of Shiraz Motahari Clinic. The patients who agreed with all of these conditions signed "the informed consent form to participate in research project" that included the name of the research project, and the related school and executives, confidentiality issues, benefits, the right to reject or cancel, and response to all questions. Any expenses including patients' travelling and testing costs were incurred by the researcher. Both of the patient's eyes were tested using the