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Prism adaptation in schizophrenia ☆

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Abstract

The prism adaptation test examines procedural learning (PL) in which performance facilitation occurs with practice on tasks without the need for conscious awareness. Dynamic interactions between frontostriatal cortices, basal ganglia, and the cerebellum have been shown to play key roles in PL. Disruptions within these neural networks have also been implicated in schizophrenia, and such disruptions may manifest as impairment in prism adaptation test performance in schizophrenia patients. This study examined prism adaptation in a sample of patients diagnosed with schizophrenia ($N = 91$) and healthy normal controls ($N = 58$). Quantitative indices of performance during prism adaptation conditions with and without visual feedback were studied. Schizophrenia patients were significantly more impaired in adapting to prism distortion and demonstrated poorer quality of PL. Patients did not differ from healthy controls on after-effects when the prisms were removed, but they had significantly greater difficulties in reorientation. Deficits in prism adaptation among schizophrenia patients may be due to abnormalities in motor programming arising from the disruptions within the neural networks that subserve PL.

Keywords: Schizophrenia; Procedural learning; Prism adaptation; Neurocognition; Basal ganglia; Cerebellum

1. Introduction

Prisms displace the visual field of subjects and initially disturb the accuracy in reaching a visual target, but subjects accommodate with successive trials, a phenomenon called prism adaptation (Kitazawa, Kohno, & Uka, 1995). Upon subsequent removal of the prisms, subjects will again miss the target, but this time in the opposite direction. This latter phenomenon is known as aftereffect. Prism adaptation is a form of visuomotor procedural learning (PL) in which visual and motor systems adjust to the displaced visual fields through experience.

PL refers to the incremental acquisition of knowledge or skill through practice without conscious awareness of what is being learned (Cohen & Squire, 1980; Squire & Zola-Morgan, 1996). PL has been demonstrated in both motor and cognitive tasks (Schwartz, Rosse, Veazey, & Deutsch, 1996). The neuronal substrates involved in the PL of skilled behaviors are poorly understood. However, increasing evidence suggests that three main brain regions may play a key role in adaptation to prisms: the basal ganglia (the striatum in particular), the frontal cortex, and the cerebellum (Alexander & Crutcher, 1990; Doyon et al., 1997; Doyon et al., 2002; Grafton et al., 1992; Hikosaka et al., 1999; Jenkins, Brooks, Nixon, Frackowiak, & Passingham, 1994; Krebs, Hogan, Hening, Adamovich, & Poizner, 2001; Pascual-Leone et al., 1993). Patients with Huntington's disease, Parkinson's disease or cerebellar lesions show impairments in PL when assessed using tests of smooth pursuit, prism adaptation, and variants of the serial reaction time task (Doyon et al., 1997, 1998; Gomez-Beldarrain, Garcia-Monco, Rubio, & Pascual-Leone, 1998; Lang & Bastian, 2002; Martin, Keating, Goodkin, Bastian, & Thach, 1996; Molinari et al., 1997; Pascual-Leone et al., 1993;

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Straube, Scheuerer, & Eggert, 1997; Willingham, Koroshetz, & Peterson, 1996). Positron emission tomography (PET) studies also suggest that the early stages of learning motor skills involve distributed neural circuits that include the basal ganglia and their connections to the frontal and parietal cortices (Clower et al., 1996; Grafton et al., 1992; Grofton, Hazeltine, & Ivry, 1995; Grofton, Woods, & Tyszka, 1994).

While the pathophysiology of schizophrenia remains unknown, multiple brain areas, including regions critical to the neural circuitry subserving PL, have been identified as being abnormal (Andreasen et al., 1992, 1996, 1997; Andreasen, Paradiso, & O'Leary, 1998; Crespo-Facorro et al., 1999; Jernigan et al., 1991; O'Leary et al., 1996; Schmand, Brand, & Kuipers, 1992; Wiser et al., 1998). Studying PL in schizophrenia patients may, therefore, provide insights into the pathophysiology of schizophrenia.

Previous attempts to evaluate PL in schizophrenia have used a variety of tasks such as rotor pursuit, Tower of Hanoi and serial reaction time (Green, Kern, William, McGurk, & Kee, 1997), but have produced equivocal findings. In general, some studies have reported intact (Clare, McKenna, Mortimer, & Baddeley, 1993; Goldberg, Saint-Cyr, & Weinberger, 1990; Granholm, Bartzokis, Asarnow, & Marder, 1993), and others impaired PL (Goldberg et al., 1990; Scherer, Stip, Paquet, & Bedard, 2003; Schwartz et al., 1996) in schizophrenia patients. Varying task complexity and cognitive processes involved in PL may have led to these differences in results. The prism adaptation test (PAT), on the other hand, provides a unique way to assess motor PL while eliminating confounds of speed (affected in rotor pursuit and serial reaction time tasks) and attention (affected in Tower of Hanoi).

This study is the first to our knowledge to explore PL in schizophrenia using the PAT. The objective of our study is to test whether the acquisition of prism adaptation is impaired in schizophrenia patients. Since schizophrenia patients have abnormalities in frontostriatal circuits (Manoach et al., 2000; Pantelis et al., 1997; Raemaekers et al., 2002) that mediate PL, we predict that patients would demonstrate slow learning during PAT compared with healthy normal controls.

2. Method

2.1. Participants

Ninety-one patients diagnosed with DSM-IV schizophrenia ($N = 76$) or schizophreniform disorder ($N = 15$) and 58 healthy volunteers were recruited through the Iowa Mental Health Clinical Research Center. After a complete description of the study, written informed consent was obtained. All subjects underwent an extensive evaluation, including phenomenological assessment, a neurological examination, a neuropsychological battery, and a magnetic resonance imaging scan of the brain. A semi-structured interview, the Comprehensive Assessment of Symptoms and History (CASH) (Andreasen, Flaum, & Arndt, 1992a), which includes the Scale for the Assessment of Negative Symptoms (SANS) (Andreasen, 1983) and the Scale for the Assessment of Positive Symptoms (SAPS) (Andreasen, 1984), was completed for all patients. All participants were right-handed. Patients with a history of head trauma or neurological disorders were excluded.

Healthy volunteers were recruited from the community through newspaper advertisements. They were initially screened by telephone, and further evaluated using an abbreviated version of the CASH to exclude subjects with a current or past history of medical, neurological or psychiatric illnesses.

Sociodemographic and clinical characteristics of the sample are summarized in Table 1. Patients were significantly younger. There was a significantly greater proportion of males in the patient group compared to controls. Patients also had less education and lower Full Scale IQ, although parental socioeconomic status was comparable between patient and control groups.

Table 1
Sample demographics and clinical characteristics

Variables	Controls ($N = 58$)	Patients ($N = 91$)	T (p)
Sociodemographics			
Age (years)	30.76 (9.61)	25.36 (9.85)	3.29 (<.001)
Gender (%)	41.38 (M); 58.62(F)	69.23 (M); 30.77(F)	$X^2 = 4.19$ (.04)
Education	14.76 (1.87)	12.56 (2.70)	5.42 (<.0001)
Parent's SES[a]	2.95 (0.35)	2.87 (0.75)	0.76 (.45)
Clinical characteristics			
FSIQ	109.35 (12.09)	91.25 (11.54)	8.87 (<.0001)
Age of onset (years)	NA	20.65 (7.26)	
Duration of Illness (weeks)	NA	107.85 (200.28)	
Negative symptoms	NA	11.37 (3.64)	
Psychotic symptoms	NA	6.99 (2.31)	
Disorganized symptoms	NA	5.00 (3.06)	

[a] Hollingshead–Redich Scale ranging from 1 (Professional with top rank social prestige) to 5 (Unskilled Worker). Hollingshead & Redich (1958).