



## Verbal learning and memory in schizophrenic and Parkinson's disease patients

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### Abstract

The purpose of this study was to investigate the neurofunctional substrate of verbal learning and memory impairments in schizophrenic patients. In this pilot study, our aim was to compare the memory disturbance of schizophrenic patients to the subcortico-frontal memory profile of Parkinson's disease (PD) patients. The California Verbal Learning Test, a verbal episodic memory test, was administered to 60 subjects, 20 patients with schizophrenia, 20 patients with PD and 20 healthy control subjects. All subjects were aged between 50 and 70 years and all patients were in a stable phase. Like the Parkinson patients, the schizophrenic patients showed a major deficit of retrieval characterized by deficit of recalls but contrarily to PD patients, schizophrenic patients' encoding scores were altered. These impairments in episodic memory could suggest a dysfunction of the subcortico-frontal circuits in schizophrenic patients. However, they demonstrated an additional encoding deficit associated with probable frontal in situ alteration. © 2002 Elsevier Science Ireland Ltd. All rights reserved.

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### 1. Introduction

Within the last decade, many studies have explored memory impairments in schizophrenia. These studies followed the publication of two articles suggesting that schizophrenic patients suf-

fered from important deficits of declarative long-term memory (McKenna et al., 1990; Saykin et al., 1991). These deficits appeared to be especially severe in comparison to other cognitive deficits and could not be attributed to the side effects of neuroleptic or anticholinergic medication (Saykin et al., 1994). Episodic long-term memory, the component of memory that contains personal experience memories with a spatio-temporal specification (Tulving, 1983), seems to be particularly impaired in these patients.

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In most studies, schizophrenic patients show important deficits on measures of free recall. These studies indicate that schizophrenic patients present an inability to encode information in long-term memory (Calev et al., 1983; Gold et al., 1992; Kareken et al., 1996; Brébion et al., 1997) and it is argued that the memory impairments in schizophrenic patients are mainly due to medial temporal lobe deficits (Saykin et al., 1991; Clare et al., 1993; Seltzer et al., 1997; Rushe et al., 1999). Nevertheless, the studies that compared neuropsychological testing in schizophrenic patients to patients with a temporal lobe dysfunction concluded that the temporal lobe model did not account entirely for the cognitive deficits observed in schizophrenia (Heinrichs, 1994; Duffy and O'Carroll, 1994; Gold et al., 1994). Neuropsychological studies indicate that a prefrontal contribution to memory function is in the organization of material (Gershberg and Shimamura, 1995). Several studies showed that impairment of episodic memory in schizophrenia is related to the frontally mediated central executive functions (Torres et al., 2001). In fact, both frontal and temporal deficits could be involved in the impairment of episodic memory in schizophrenia. Indeed, reduced left fronto-temporal activation in patients with schizophrenia was observed during episodic memory encoding and retrieval (Ragland et al., 2001). Hazlett et al. (2000) also suggested that impairments in function of the prefrontal and temporal cortex play a central role in the pathophysiology of learning and memory deficit in schizophrenia. Besides the involvement of frontal and temporal cortex in episodic memory, the subcortical structures such as striatum also have to be considered in both schizophrenia and PD patients. Memory deficit of non-demented Parkinson's patients has been largely qualified as a fronto-striatal dysfunction (Pillon et al., 1993, 1994, 1995; Daum et al., 1995) and the fronto-striatal structures are involved in the pathophysiology of schizophrenia (Robbins, 1990). Most investigators have found decreased metabolic rates in the basal ganglia in unmedicated patients (Buchsbaum and Hazlett, 1998). Both treated and untreated schizophrenic patients also showed decreased striatal metabolic rates during a verbal learning task (Shihabuddin et al., 1998). Paulsen

et al. (1995a) provided important support for a striato-frontal involvement in the memory deficits of the schizophrenic patients. Their results suggested that most schizophrenic patients demonstrated a pattern of learning and memory impairments that resembles the pattern seen in patients with primary subcortical (specifically striatal) pathology. This cognitive pattern was characterized by a prominent retrieval deficit associated with a mild encoding deficit. Moreover, several similarities can be observed in schizophrenia and subcortical dementias like Parkinson's disease (PD) (Pantelis et al., 1992). Dopaminergic dysfunctions play an important role in both disorders. Neuroleptics can cause extrapyramidal symptoms in schizophrenic patients, while levodopa can cause psychotic symptoms in PD patients. Furthermore, in both disorders a prefrontal dysfunction has been observed, which could be due to a disruption in the subcortical structures (Pantelis et al., 1992). As direct comparison between schizophrenic memory profile and performance of patients with specific subcortico-frontal dysfunction has not been done, we decided to compare schizophrenic patients' memory profile to the memory performance of non-demented, highly selected PD patients using the California Verbal Learning Test (CVLT), an episodic memory test which evaluates the learning, encoding and retrieval memory processes (Delis et al., 1987). A similar learning and memory pattern among the two groups would suggest an important role of basal ganglia and frontal cortex in memory deficit in schizophrenia and would suggest a 'subcortical' profile of cognitive impairments. This 'subcortical' profile can be characterized by a prominent retrieval deficit with relatively spared recognition memory in the context of greater impairment in delayed recall (Paulsen et al., 1995a). In contrast, a cortical profile of impairment could be inferred if encoding and retrieval deficits are associated with similarly altered recognition and delayed recall processes.

## 2. Methods

### 2.1. Subjects

Sixty subjects were included in this study: 20 patients who met DSM-IV (American Psychiatric

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