The Need to Integrate Neuropsychological and Experimental Schizophrenia Research

by Mark R. Serper and Philip D. Harvey

Although the experimental and the neuropsychological approaches to the study of schizophrenic cognition have coexisted for many years, they remain detached, with virtually independent methods and literatures. The result has been a needless duplication of work and effort. In this article we review the emergence and representative research from each approach, examine model integrative studies, and suggest ways in which the two approaches can be better integrated to generate comprehensive cognitive schizophrenia theories and research.


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and quantitative units of measurement established psychology as a laboratory science. However, the study of mentalistic processes such as attention were widely neglected during the first half of the century (Hilgard 1987). With the notable exception of Shakow and colleagues (e.g., Huston et al. 1937), schizophrenia researchers in the early part of the 20th century (e.g., Goldstein 1939; Cameron 1944) were concerned primarily with perceptual and abstraction deficits in the Gestaltist tradition, rather than with the study of schizophrenic attention and cognition (Neale and Oltmanns 1980). Observations made by both Bleuler and Kraepelin in the area of schizophrenic attentional dysfunction appeared to have their greatest influence on the experimental study of cognition and neuropsychological functioning in the second half of this century, when information processing theory became increasingly popular.

In the late 1950s, cognitive processes became an important concern in schizophrenia research, primarily because of advances in the study of animal and human cognition (the “cognitive revolution”; Hilgard 1987). The information-processing model advanced by Broadbent (1958) was perhaps the major launching point for much of the research by experimental psychopathologists in the next 35 years. Shortly after Broadbent’s seminal work, Payne et al. (1959) hypothesized that schizophrenia patients have a faulty information-processing “filtering” mechanism. Expanding on Goldstein’s (1939, 1944) work while adopting the new information-processing perspective, Weckowicz and Blewett (1959) proposed that abstraction deficits were secondary to a primary selective attention deficit. McGhie and Chapman (1961) also concluded, much as Kraepelin and Bleuler had 50 years earlier, that the primary deficit in schizophrenia was in the control and direction of attention. Since the late 1950s and early 1960s numerous aspects of schizophrenic attentional/cognitive dysfunction have been identified (for a review, see Nuechterlein and Dawson 1984).

Rise of the Neuropsychological Approach

The beginnings of modern clinical neuropsychology stem from the work of Hebb (1949), Teuber (1950), Piercy (1959), and McFie (1960), who were interested in examining the behavioral correlates of brain disorders. Dissatisfied with efficacy of traditional psychological tests in examining the behavior of brain-damaged patients, McFie (1960), among others, advocated the use of special tests to detect brain damage by assessing the deviation of the patient’s test scores from those of individuals with established brain syndromes or dysfunctions.

The view of schizophrenia as a brain disease has become widely accepted, and evidence for the neurobiological basis of the disorder has been accumulating (e.g., Nasrallah and Weinberger 1986). Paralleling the widespread appreciation of the neurobiological basis of schizophrenia, there has been an exponential increase in the extent to which the methods of clinical neuropsychology have been applied to schizophrenia patients. Consequently, schizophrenia patients have been examined with clinical neuropsychological tests measuring cognitive functions putatively associated with brain dysfunctions in a number of cortical and subcortical areas. Since many of the cognitive impairments and clinical symptoms in patients with focal brain damage resemble those of schizophrenia patients (see Davison and Bagley 1969 for a classic review and Benson and Stuss 1990 for a recent integration), the use of these tests with schizophrenia patients appears to be valid. In fact, schizophrenia patients manifest relatively consistent deviance on neuropsychological measures (Braff et al. 1991; Saykin et al. 1991), and the concept of the “neuropsychological deficit” (e.g., Bilder et al. 1985; Goldberg et al. 1989a) in schizophrenia has become a standard rubric under which cognitive dysfunction has been studied.

Duplication of Effort and Need for Integration of the Two Approaches

Although the goals of clinical neuropsychological studies have varied from understanding the normative standards for performance of a pathologic group on a relatively simple test, such as the pursuit rotor, to localizing a brain dysfunction on the basis of a clinical neuropsychological measure, such as the Wisconsin Card Sorting Test (WCST; Heaton 1981), the basic cognitive deficits of schizophrenia patients have not changed. Many findings generated by studies from the experimental perspective have recently been duplicated or “reinterpreted” in terms of functional brain deficits. For example, Goldberg et al. (1989b) asserted, after duplicating the already well-established finding of schizophrenia patients’ greater deficit in re-
call than in recognition in a serial learning test, that this deficit reflected a deficit in functions of the prefrontal cortex. Furthermore, the "generalized neuropsychological deficit" that has been discussed recently was reviewed in detail by Hunt and Cofer (1944) nearly 50 years ago. Consequently, many of the "new" interpretations of the results of assessment of cognition in schizophrenia patients are based on cognitive deficits that were identified in experimental cognitive studies conducted 25 or even 50 years ago. Many recent studies of the neuropsychology of schizophrenia have examined the same cognitive functions (e.g., verbal learning and memory) that have been examined extensively by investigators interested in the characterization of cognitive functions and not necessarily in brain localization.

To avoid duplication of effort and, more important, duplication of methodologic problems and conceptual dead ends, it may be productive for researchers interested in the neuropsychological implications of cognitive deficits to incorporate the experience acquired during the long history of experimental schizophrenia research dating back to Kraepelin. Of importance in the process of application of new assessment strategies to the study of cognitive impairments in schizophrenia is the long history of methodologic difficulties in schizophrenia research. Extensive reviews of methodologic concerns and suggestions for improvements from the perspective of the experimental psychopathology and information-processing approaches have been published (e.g., Chapman and Chapman 1973; Neale and Oltmanns 1980); these reviews offer valuable suggestions for new neuropsychological studies of schizophrenia.

In addition, a greater understanding of the specific psychological mechanisms underlying more global schizophrenic cognitive deficits is needed before brain function correlates can be identified (Frith and Done 1988). Many symptoms and deficits manifested by schizophrenia patients on cognitive tests have multiple potential psychological determinants (Alpert 1985) and may therefore be inappropriate for specific neuropsychological hypotheses. For example, deficits on the Continuous Performance Test (CPT; Rosvold et al. 1956) have been explained by impaired sustained attention or vigilance deficits that interfere with processing of the stimuli (Nuechterlein 1977), as well as by motor abnormalities that interfere with generation of responses to target stimuli (Walker and Green 1982). Similarly, the causes of the reaction time crossover effect have been explained by competing theories (i.e., "segmental set" [Shakow 1962] vs. "redundancy associated deficit" [Bellissimo and Steffy 1972]). Varying explanations of psychological components of cognitive deficits may in fact lead in different directions as far as neuropsychological localization of impairment. An additional practical implication of this issue has been raised by a recent discussion of "cognitive rehabilitation" strategies for schizophrenia patients (e.g., Spaulding 1992). Undeniably, a greater understanding of the components of the cognitive impairments in schizophrenia is required before cognitive rehabilitation interventions can be effective.

We now briefly address three general areas of cognitive dysfunction in schizophrenia in terms of the great possibilities for integration between perspectives. We selected these areas—learning and memory, conceptual and executive functioning, and cognitive capacity—because they are central to the current research in experimental and neuropsychological perspectives, but they are by no means exhaustive or unique in terms of the need for integration.

Learning and Memory

The Experimental Perspective.

Difficulties in learning and memory have been studied extensively by researchers from the experimental perspective. Reviewing all of these findings is beyond the scope of this article, but a pattern of consistent findings has emerged. Relative to various normal and nonschizophrenia patient control samples, schizophrenia patients have demonstrated slower acquisition of serially presented verbal material (Koh et al. 1973), reduced tendencies toward utilization of intrinsic organization in presented material (Bauman 1971), and reduced benefits from strategies designed to increase recall perform-
These findings have led to the suggestion that schizophrenia patients manifest a primary deficit in the encoding of verbal material. Schizophrenia patients have also been found to manifest a greater decrement in recall than in recognition performance on memory tests (Bauman and Murray 1968), although their recognition performance may also be impaired relative to that of normal control subjects (Calev and Monk 1982). Forgetting of successfully acquired material, however, appears to be present in only certain schizophrenia patients, typically those who have a more chronic course of illness (Calev et al. 1983).

Experimental researchers have suggested that these learning difficulties indicate that schizophrenia patients manifest difficulties in the information-processing operations that are required to actively process novel material for storage (Neale and Oltmanns 1980). The methodologic refinements that have been advanced include careful separation of the component processes of verbal learning and memory. This has been done to identify the specific components of learning and memory deficits and to identify specific deficit processes that have been examined with experimental manipulation (e.g., Harvey 1987; Koh 1978).

The Neuropsychological Perspective. Neuropsychological researchers have provided a number of interpretations regarding learning and memory deficits in schizophrenia. Goldberg et al. (1989b), following Hecaen and Albert (1978), suggested that schizophrenia patients' differential deficit in recall as compared with recognition performance may be mediated by deficits in prefrontal cortical functions (i.e., forgetting to remember). Saykin et al. (1991) suggested that schizophrenia patients' deficits in the acquisition and retention of verbal material may be due to abnormalities of the medial temporal lobe, whereas Tamlyn et al. (1992) suggested that the pattern of memory impairment demonstrated by schizophrenia patients is similar to that seen in classic amnestic syndromes.

Integration. In terms of integration of the two perspectives, researchers from the experimental perspective, focusing exclusively on the functional components of cognitive deficits, have identified several psychologically valid components of learning and memory. The neuropsychological studies have focused either on cortical localization of functions or on the similarity between cognitive deficits in schizophrenia and those in other brain diseases with well-understood pathology, but these studies have selected aspects of cognition that are very broad and that have multiple discrete subcomponents. Some studies from the neuropsychological perspective have not benefited from the methodologic and conceptual advances made by researchers from the experimental psychology perspective. For example, in Goldberg et al.'s (1989b) study, the recall and recognition tasks were certainly very different in their psychometric characteristics. In Saykin et al.'s (1991) study, memory measures were grouped for statistical analysis on the basis of similarity of stimuli (e.g., verbal vs. spatial) without attempt to separate the tasks into measures of psychologically valid components. An integrated approach would be to attempt to identify the functional cortical correlates of processes such as encoding, categorization, retention, and retrieval so that the conceptual sophistication of the experimental perspective could be combined with the potentially useful goal of identifying the regional brain dysfunctions associated with learning and memory impairments in patients with schizophrenia. This is a complex task, but one in which much progress has been made with integrative approaches in the study of the effects of other brain diseases, such as epilepsy (e.g., Milner et al. 1991).

Conceptual and Executive Functioning

Impairment in abstraction ability has long been recognized as a major component of schizophrenia (Goldstein 1944). This area has received increased attention in recent years owing to advances in neuroimaging techniques that have shown that deficits on tests of conceptual functions using the WCST are associated with specific patterns of regional brain dysfunction (i.e., the dorsolateral prefrontal cortex; Weinberger et al. 1986). Schizophrenia patients manifest poor functioning on a variety of measures of executive and conceptual functioning, such as the WCST, the Tower of Hanoi, and the Halstead Category Test (Halstead 1947) (Goldberg et al. 1989a).

The Experimental Perspective. A few studies have examined conceptual and executive functioning from a uniquely experimental psychological perspective. These studies have identified deficits in cognitive functions that have been suggested to underlie symptoms...
such as delusions, hallucinations, and formal thought disorder. Experimental psychology research in the area of “reality monitoring” (Johnson and Raye 1981) has led several investigators to hypothesize that schizophrenia patients may be deficient in their ability to discern whether an event is self-generated (e.g., a thought) or a product of the outside world. Studies of hallucinations have found evidence that schizophrenia patients are relatively poor at identifying their own previously recorded thoughts (Heilbrun 1980) and that they demonstrate a bias toward attributing experiences to an external source in conditions of uncertainty (Bentall and Slade 1985). Frith and Done (1989) demonstrated that patients who had delusions of alien control had difficulty in monitoring ongoing cognitive functions. Harvey (1985) found that thought-disordered schizophrenia patients, compared with non-thought-disordered schizophrenia patients, had a bias toward attributing self-generated information to external sources.

The Neuropsychological Perspective. Since the earliest conceptualization of the disorder, investigators such as Kraepelin, Alzheimer, and Bleuler theorized that schizophrenia was the result of frontal lobe dysfunction (Robbins 1990). Given the primacy of deficits in executive functions in both patients with known frontal lobe disease and schizophrenia patients, many neuropsychological researchers have attempted to behaviorally validate the notion that schizophrenia is a disorder of the frontal lobes. Although schizophrenic deficits on the WCST have been widely publicized in recent years as a reflection of neuropsychological frontal dysfunction, many other tests that are sensitive to frontal lobe or executive dysfunctions have been applied to schizophrenia patients (e.g., Bilder et al. 1985; Goldberg et al. 1989a). Imaging studies with executive functioning tasks have demonstrated promise in correlating deficient test performance with regional dysfunction (e.g., Weinberger et al. 1986; Rubin et al. 1991).

Integration. In terms of the integration of experimental and neuropsychological perspectives, an attempt can be made to help determine whether the brain functioning deficits underlying reality monitoring are similar to those associated with deficits in similar appearing executive functions. Similarly, in studies of executive dysfunction in schizophrenia, attempts to identify the specific psychological mechanisms of such phenomena as perseveration (a hallmark of frontal cortical dysfunction in brain-injured patients [Milner 1964; Stuss and Benson 1984]) are needed to determine the cognitive mechanisms underlying these phenomena.

It should be noted that David (1992) cautions against overgeneralizing about the relationship between frontal lobe neuropathology and psychiatric illness (particularly schizophrenia) and warns schizophrenia researchers of the risks of becoming “frontal lobologists.” A greater understanding of the cognitive mechanisms behind perseveration and “concreteness,” as well as an understanding of brain mechanisms implicated by deficits in reality monitoring, is needed to advance the field beyond the circular explanation that frontal lobe-related deficits are caused by frontal lobe pathology (David 1992).

Cognitive Capacity

The cognitive capacity approach is based on the study of the complex cognitive operations that constitute the normal ability to perceive, attend to, select, and retain information present in the environment. While much of the study of information processing is based on research in experimental psychology that is at present only marginally relevant to the understanding of impaired cognition, several concepts that have been introduced in this area have received substantial attention in the applied literature. Most prominent of these is the concept of information-processing capacity. Simply stated, capacity models focus on the notion that all aspects of cognitive function can be characterized in terms of the performance demands that they make on an individual (Wickins 1984). Within this conceptualization, cognitive processes can be divided into “controlled” versus “automatic” operations. This concept, introduced by Schneider and Shiffrin (1977) and applied to the schizophrenia literature by Neale and Oltmanns (1980) and Callaway and Naghdi (1982), divides cognitive functions into those that require cognitive resources and effort (controlled) and those that do not (automatic). Processes that are automatic can be performed concurrently with other processes (i.e., in a dual-task situation) with no functional decline in either task (commonplace examples are driving while talking or integrating auditory and visual information presented on television). Although this division makes intuitive sense, schizophrenia patients often complain that they
experience difficulties with tasks related to this concept (see McGhie and Chapman 1961 for a classic example), there are some problems with the data supporting this division and its validity in schizophrenia populations (see Serper et al. 1990 for a discussion). In addition, theorists working from an information-processing approach have not directly addressed issues of neuropsychological localization in substantial detail, leading to dualistic interpretations in which cognitive processes are approached as if they were somehow independent of the functioning of the brain.

The Experimental Perspective. A widely accepted theory accounting for the pervasive cognitive deficits exhibited by schizophrenia patients is that these individuals have an abnormal reduction in the availability of processing resources compared with normal persons (Knight and Russell 1978; Gjerde 1983; Nuechterlein and Dawson 1984). The dual-task paradigm is a widely accepted experimental procedure for measuring resource limitations (Norman and Bobrow 1975). In the dual-task procedure, subjects are asked to perform two tasks simultaneously. Deficits in overall performance on both tasks during dual-task conditions, compared with performance during single-task conditions, and impairments in secondary task performance as a function of increasing processing load on the primary task are taken to reflect processing limitations (Gophor and Donchin 1986). In a recent study (Serper et al. 1990), we employed the dual-task procedure with schizophrenia patients to test their processing capacity. We found that unmedicated schizophrenia patients, relative to medicated schizophrenia patients and normal control subjects, were impaired in improving their information-processing performance under dual-task conditions, even with extended practice. Only normals and medicated schizophrenia patients were able to develop automated responses under dual-task conditions. Similarly, Granholm et al. (1991), also using the dual-task paradigm, found that schizophrenia patients had greater difficulty than normal subjects in developing automatic detection responses. In this study, as in Serper et al. (1990), schizophrenia patients had deficiencies in the use of controlled processing resources. These deficiencies were alleviated once the patients were able to develop automated responses. These two studies are consistent with the theories and findings of Callaway and Naghdi (1982), who found that schizophrenia patients had difficulty with tasks requiring effortful processing while demonstrating relatively normal automatic processing performance. Many other studies have found that schizophrenia patients manifest performance deficits when experimenters manipulate the processing demands; tasks used in such studies include the backward masking task (e.g., Saccuzzo et al. 1974), versions of the CPT (e.g., Nuechterlein et al. 1986), and the span of apprehension task (Asarnow et al. 1991).

The Neuropsychological Perspective. In contrast to the cognitive processing theories, no research to date has examined the neuropsychological aspects of attentional capacity theory. It is essential to determine the neuropsychological implications of resource limitations and automation difficulties in schizophrenia, both for brain functioning and for future rehabilitation efforts.

A consistent effort has been made to examine the attentional processes in schizophrenia through the study of nervous system activity by peripheral measures (e.g., Bernstein et al. 1981; Zahn et al. 1981; Bernstein 1987) and by central nervous system measures such as the P300 component of the evoked potential (e.g., Duncan 1988). The mechanisms underlying autonomic and central nervous system abnormalities in schizophrenia focus on cognitive and physiologic regulation of information intake. For example, the orienting response (OR) was regarded by Luria (1973) as the most basic attentional process and was thought to be involved with the onset of preattentional processes (Sokolow 1963). Recent theories consider OR to be associated with the onset of resource-limited controlled information processing (Ohman 1979). Gruzelier and Venables (1973, 1975) hypothesized that schizophrenia was bimodally distributed between patients who were OR nonresponders and patients who were OR hyperresponders. They suggested that OR nonresponding was associated with amygdaloid dysfunction and lack of OR habituation was associated with hippocampal dysfunction. Studies of autonomic nervous system activity have been related to neuropsychological functioning as well as to symptomatology and outcome. Katasonis and Iacono (1992), measuring electrodermal activity, found that schizophrenia hyporesponders manifested deficits on neuropsychological tasks that are hypothesized to be associated with temporal-limbic system dysfunctions. Dawson et al. (1992) found that heightened electrodermic ac-
tivity was associated with increased psychopathology and predictive of poor short-term prognosis. Once a greater understanding of the mechanisms underlying psychophysiological dysfunctions and their relationship to clinical and neuropsychological variables is achieved, this area has great potential for becoming a truly integrated area of schizophrenia research.

**Model Integrative Theories and Studies**

The theories that attempt to specify discrete psychological processes that arise from brain dysfunctions provide unique and testable models for understanding schizophrenic brain dysfunction and their contribution to psychotic symptomatology. In generating a comprehensive model of schizophrenia, Frith and Done (1988) pointed out the need for understanding symptoms in terms of psychological processes that can then be related to the functions of specific brain systems. The following are examples of the integration of experimental psychology and neuropsychology studies that benefit from the unique aspects of each perspective.

An example of an integrationist approach in the area of learning and memory comes from a recent study conducted by Gold et al. (1992). These authors examined memory performance in schizophrenia and found deficits in recall, recognition, semantic encoding, and frequency estimation. They interpreted these deficits in the context of recent neuropsychological findings. Their study employed the methodologic refinements that have been developed in the experimental literature, including the use of word lists from category norms and the matching of stimuli on category salience and word frequency. Gold et al. discussed their results with experimentally derived implications (e.g., the importance of effortful and automatic processing deficits in schizophrenia) as well as neuropsychological significance (e.g., temporal and frontal lobe contributions to learning and memory dysfunction in schizophrenia patients) in mind.

In an innovative report, McGrath (1991) integrated data from neuropsychology and aphasiology and combined these findings into a cognitive theory of executive functioning deficits and thought disorder in schizophrenia. Results from linguistic studies analyzing thought-disordered patients' failures in communication (e.g., Rochester and Martin 1979) were linked to findings of deficits in frontal executive functions to construct an integrative conceptual model. McGrath suggested that deficits in the functioning of the prefrontal cortex are the substrate for deficits in reality monitoring, which then lead to communications failure through loss of the ability to monitor the contents of consciousness. In constructing a plausible neuropsychological model, McGrath expanded on the interpretation of data presented in studies (e.g., Harvey and Serper 1990) that did not consider the neuropsychological implications of reality monitoring failures.

An example of successful integration of neuropsychological tasks with clearly identifiable underlying cognitive processes in the area of attentional allocation and selective attention comes from the laboratory of Posner and colleagues. Posner and colleagues found that schizophrenia patients had difficulty in disengaging their visual search operations in their right visual field, leaving these patients with a decreased ability to extract relevant information from visual arrays (Posner et al. 1988; Posner and Early 1990). The interpretation of the results of these studies was taken as support for the involvement of the inferior parietal cortex in an attentional network responsible for attention to both spatial and verbal material. This study is important because of its contribution to network models of neurocognition (e.g., Weinberger et al. 1992) and because of its move away from a strict regionalizing perspective.

**Conclusions**

The recent past has seen an unparalleled advance in knowledge regarding the cognitive functions of schizophrenia patients. At the root of this explosion of interest and information has been the focus on cognitive deficits as mediators of certain psychotic symptoms, as behavioral reflections of core neurological deficits, as markers of vulnerability, as predictors of outcome, and as etiological determinants of schizophrenia. Advances in understanding schizophrenia will accelerate if neuropsychologically based schizophrenia researchers take advantage of the knowledge base that has accrued from investigations of schizophrenic cognition that were not explicitly neuropsychological in nature. It should be noted that in clinical neuropsychology there have been recent strong statements regarding the need to focus on meaningful cognitive concepts and their interrelationship rather than on attempts to specify a multiplicity of specific brain region deficits, each with unique behavioral
correlates (Shallice 1988; see Cohen and Servan-Schreiber 1992 for a similar discussion on cognitive measures from the experimental psychology perspective).

Research data and methodologic advances from neuropsychological studies oriented toward brain localization and dysfunction must be integrated with experimental psychology studies focusing on deficits in cognitive function to advance our knowledge regarding the characteristics and determinants of cognitive deficits in schizophrenia.

References


Duncan, C.C. Event-related brain potentials: A window on information processing in schizophrenia.


Saykin, A.J.; Gur, R.C.; Gur, R.E.; Mozley, R.D.; Mozley, L.H.; Resnick, S.M.; Kester, D.B.; and Stafiniak, P. Neuropsychological function in schizophrenia: Selective impairment in memory and learn-


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