"CAN GESTALT PSYCHOLOGY INFORM THE SEARCH FOR THE ETIOLOGY OF SCHIZOPHRENIA?"

A Response to Ernst Plaums Commentary

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In his informative commentary, PLAUM raises a number of important issues regarding the validity and relevance of Gestalt psychology-informed models of cognitive deficits for the understanding of schizophrenia. We would like to respond briefly to the most salient points of his argument and use this opportunity to review several issues which were not addressed in our target article.

1) Deficits in perceptual organization in schizophrenia: Generalized deficit or specific cognitive dysfunction?

PLAUM provides a review of the methodological difficulties associated with research into cognitive deficits in schizophrenia patients. Specifically, he raises the question whether dysfunctions in perceptual organization in schizophrenia reflect a deficit in the organization of stimuli or are due to additional factors, such as other cognitive deficits which are present in the large majority of patients (e.g., problems in attention, memory, etc.) and/or secondary effects of medication (e.g., sedation, restlessness/akithesia), poor motivation etc. Poor performance due to either of the latter two classes of factors has been referred to as the *generalized deficit* in schizophrenia (CHAPMAN & CHAPMAN 1978). The generalized deficit hypothesis, when applied to experimental psychopathology research, assumes that schizophrenia patients will be significantly impaired on all task conditions, or if differential significance emerged, such differences would covary with the difficulty level of the condition. Accordingly, demonstration of a deficit on a given cognitive task may not be very informative for the identification of impairments in specific cognitive processes in schizophrenia. CHAPMAN and CHAPMAN (1978) suggested the solution of designing studies using two tasks matched on reliability and difficulty levels. Using this design, a performance deficit relative to a control group on one task but not the other could not be attributed to a generalized deficit. KNIGHT and SILVERSTEIN (1998, 2001), however, criticized this solution because it does not ensure that the tasks used are process-specific, as opposed to being confounded by multiple cognitive processes. In addition, they noted that the matched-tasks solution is not appropriate for many cognitive psychological paradigms, where differences in difficulty level are built into a multiple-condition task in order to assess factors that affect the integrity of a specific cognitive process (e.g., variations in exposure duration, brightness, level of target-noise similarity, etc.).

In our previous work (KNIGHT 1984, KNIGHT & SILVERSTEIN 2001) we proposed a process-oriented approach as an alternative to the matched-task solution advocated by the CHAPMANS'. The process-oriented approach advocates the use of well-established models from cognitive psychology to predict theory driven patterns of performance within and across tasks that should be found when specific stages of processing function either adequately or inadequately. Moreover, these predicted patterns are different from those predicted by the generalized deficit hypothesis or by other theories. KNIGHT (1984) delineated four ways in which predictions of the general deficit model can be refuted:

1. Disconfirmation strategy; this strategy is implemented by providing convincing evidence of patients' competence in a specific cognitive process.

2. Superiority strategy; this strategy involves the demonstration that a specific cognitive impairment can lead to an advantage in an experimental task.

3. Relative superiority strategy; the distinguishing characteristic of the relative superiority strategy is that it hypothesizes a specific reversal, compared with normal controls, in the relative performance level of at least two tasks or conditions in the experiment.

4. Multiparadigm strategy; in this strategy, cognitive theory is used to predict and test a pattern of performance across multiple tasks indicating a specific deficit that is not confounded with the obvious predictions of a general deficit model.

As we describe below, research into perceptual organization in schizophrenia provides paradigmatic examples of how the process-oriented approach can be implemented.

A series of studies by our group (SILVERSTEIN, KNIGHT et al. 1996a, Study 1, SILVERSTEIN et al. 1996b, UHLHAAS, SILVERSTEIN et al. 2003a, SILVERSTEIN, UHLHAAS et al. in press) and those of others (FERMAN, PRIMEAU et al. 1999; ORLOWSKI, KIETZMAN et al. 1985; PLACE & GILMORE 1980; PETERS, NUNN et al. 2002; WELLS & LEVENTHAL 1984) has found reliable evidence for absolute and relative performance advantages for schizophrenia patients in tasks assessing perceptual organization. In addition, three studies (ORLOWSKI, KIETZMAN et al. 1985; RABINOWICZ, OPLER et al. 1996; WELLS & LEVENTHAL 1984) replicated the findings by PLACE and GILMORE (1980) who reported superior performance of schizophrenia patients at counting the number of lines in tachistoscopically presented arrays. Performance advantages have also been observed in schizotypal subjects (GOODARZI, WYKES et al. 2002; UHLHAAS, SILVERSTEIN et al. in press). Similarly, schizophrenia patients are not deficient in all tasks of perceptual organization. Perceptual organization in schizophrenia is intact for stimuli with strong configural properties, such as symmetry (KNIGHT, MANOCH et al. 2001). Preservation of perceptual organization in schizophrenia patients for stimuli with strong configural properties has been interpreted as indicating that stimulus assembly processes relying on prespecified feature hierarchies (i.e., those patterns for which the visual system is "hard-wired" to respond) are intact. In contrast, the evidence for performance abnormalities reviewed above suggests that schizophrenia patients' performance deficiencies involve dynamic organizational processes, which create novel groupings

that can be specified only after the input is known (see PHILLIPS & SILVERSTEIN 2003, and WATT and PHILLIPS 2000, for explication of the distinction between these two mechanisms involved in perceptual organization). The converging evidence for deficits in dynamic organization in schizophrenia demonstrated using the process-oriented approach (see KNIGHT & SILVERSTEIN 1998, 2001 for reviews) strongly suggests that these findings of perceptual organization deficits in schizophrenia are not the result of a generalized deficit.

2) Temporal characteristics of dysfunctions in perceptual organization in schizophrenia

Another important issue is whether dysfunctions in perceptual organization represent a stable, trait-like characteristic (stable vulnerability marker) or whether such cognitive deficits are more strongly related to the clinical state of the patient (episode indicator). The presence of deficits in perceptual organization in non-psychotic schizotypal subjects (UHLHAAS et al. in press) and in chronic and acute schizophrenia patients argues against the interpretation of dysfunctional perceptual organization as an episode indicator. Nevertheless, in a recent study (UHLHAAS 2003b) a close link was demonstrated between improvement in scores on the Cognitive/Disorganized factor of the Positive and Negative Syndrome Scale (PANSS) and normalization of perceptual organization ability across approximately three weeks of acute inpatient treatment. This, and other findings of relationships between increased clinical disorganization and impaired perceptual organization in schizophrenia, suggest that impaired perceptual grouping may be a mediating vulnerability marker (NUECHTERLEIN & DAWSON 1984) for the illness. This refers to a finding that while present across the schizophrenia spectrum, deficits in perceptual organization fluctuate with severity of clinical signs and symptoms.

3) Specificity of dysfunctions in perceptual organization and the heterogeneity of schizophrenia

PLAUM rightly points out that the search for cognitive dysfunctions specific to schizophrenia is a difficulty task. The recent history of experimental psychopathology provides many examples to support this view. Our research has so far indicated that deficits in perceptual organization represent a relatively specific cognitive dysfunction which is not present in other psychotic disorders. For example, SILVERSTEIN et al. (1996a, Study1) compared good premborbid and poor premorbid schizophrenics to patients with psychotic disorders other than schizophrenia (mainly schizoaffective and bipolar disorder with psychotic features). In this study, dysfunctional perceptual organization was found only among poor premorbid schizophrenia patients. Other studies by our group (see KNIGHT 1992 for a review) have confirmed that dysfunctions in perceptual organization are not found in other psychotic disorders. In addition, dysfunctional perceptual organization in schizophrenia is associated with a number of clinical characteristics. These include poor premorbid social history, poor outcome,

and elevated nailfold plexus visibility, a putative biological marker for schizophrenia (for a review see KNIGHT & SILVERSTEIN 1998). Together, these features suggest that dysfunctional perceptual organization in schizophrenia spectrum disorders may be indicative of a neurodevelopmental subtype of schizophrenia (JONES, GUTH, LEWIS & MURRAY 1994). This is supported by the co-occurence of impairments in perceptual organization and certain neurodevelopmental disorders, such as autism (HAPPE 1999) and Williams syndrome (PANI, MERVIS & ROBINSON 1999).

4) Phenomenology and disintegration of Gestalt processes

PLAUM agrees with our emphasis on the phenomenology of visual perception in schizophrenia as an important source of data. He cites in this context a number of clinical vignettes which suggest that a description of cognitive deficits in schizophrenia in terms of a 'disintegration of Gestalt processes' may not adequately capture the clinical picture. For example, PLAUM refers to a patient who during the GOLDSTEIN-SCHEERER Object Sorting Test proposed that the common factor among three objects was that all of them were 'yellow' although one object, a black and white dog, obviously did not fit into this category. Upon questioning, the patient suggested that this toy dog had been manufactured in Japan and since all Japanese people are 'yellow', the same quality could be assigned to the toy dog. While it is unknown at this point whether overinclusive thinking is related to abnormal Gestalt processing in schizophrenia, several theorists have proposed that this is the case. For example, MATUSSEK (1987) suggested that delusions become possible when the loosening of the visual context allows for the occurrence of highly infrequent, idiosyncratic, and personalized combinations of object meanings and qualities. An example of this comes from one of MATTUSEK's patients, who stated: "Out of these connections came the absolute awareness that my ability to see connections had been multiplied many times over" (MATUSSEK 1987, p.96). Similarly, CARR and WALE (1986) proposed, within the framework of cognitive psychology, that disorganization leads to efforts at reconstitution involving abnormal integration of sensory input, leading to delusional beliefs. They describe such symptoms as "…instances of ideational organization, creations of higher cortical processes by which disorganized inputs are ordered or structured (according) to ideational schemata" (p.150, parentheses added).

5) Heterogeneity of Gestalt processes and cognitive deficits in schizophrenia

Deficits in perceptual organization and the associated, wider dysfunctions in language and thought in schizophrenia, and the clinical symptoms of the disorder are unlikely to represent a single unitary pathological mechanism. We agree with the position of PLAUM who rightly raises this important issue and suggests, that in addition to dysfunctions in elementary sensory processes, higher, complex cognitive functions are impaired in schizophrenia which may not be due a single dysfunction.

Our brief discussion of this issue below covers three main points: 1) heterogeneity exists in schizophrenia, including in the area of cognitive impairment; 2) in some

cases, what is initially seen as multiple deficits can be understood to be the result of a common underlying impairment; and 3) even after reconceptualizing certain cognitive deficits and symptoms as manifestations of core dysfunctions, heterogeneity can be seen to exist.

The existence of heterogeneity in schizophrenia was demonstrated long ago and continues to be a focus of research. Factors such as premorbid history, treatment response, symptomatology, cognitive deficits, and neurobiological structural and functional abnormalities are known to be heterogeneous in schizophrenia. HEINRICHS (2001) provides a comprehensive discussion of the heterogeneity of cognitive deficits and associated neurobiological findings in schizophrenia. This review makes it clear that there is no single finding that is characteristic of all patients, and that, typically, any finding considered to be significant can be expected to be found in only about 30–40% of patients.

Recently, efforts have been made to move away from the reporting of multiple performance deficits among schizophrenia patients towards attempts to understand classes of dysfunctions as reflecting impairments in one or more basic mechanisms (CARR & WALE 1986; COHEN & SERVAN-SCHREIBER 1992). For example, we (PHILLIPS & SILVERSTEIN 2003; SILVERSTEIN, BAKSHI et al. 1998; SILVERSTEIN, KOVACS et al. 2000; SILVERSTEIN & SCHENKEL 1997) have proposed that dysfunctions in perceptual organization in schizophrenia represent a wider deficit in the cognitive coordination of contextually related information. Although a detailed description of this theory is beyond the scope of this response, a brief summary of its main tenets can be seen to be relevant to the present discussion. These are: 1) data from experimental psychology, neurobiology, and computational neuroscience suggest the existence of a cortical algorithm involving contextual coordination that operates across cortical regions; 2) this coordinating operation serves to increase the salience of stimuli that are predictably related to the context in which they occur; 3) the similarity in contextual coordinating processes that occurs across cortical regions is due to the presence of a single cytoarchitecture throughout the cortex. This cytoarchitecture and its accompanying neurobiology allows for the formation of dynamic long-range interactions between pyramidal cells that are mediated by NMDA receptors, whose combination of voltage and ligand gated properties provide the basis for modulatory effects on processing; following #s 1, 2, and 3, a widespread dysfunction in cytoarchitecture and/or NMDA receptor functioning would be expected to cause impairments in contextual coordination in multiple functions from perception to thought and language, to behavior; 5) evidence for multiple forms of contextual coordination deficits have been found in schizophrenia and these are significantly correlated; 6) evidence for cytoarchitectural abnormalities and for NMDA receptor hypofunction have also been found in schizophrenia; and 7) recent ERP data indicate that reduced gamma phase synchrony, thought to reflect contextual coordination, has been related to abnormal Gestalt perception in schizophrenia (SPENCER, NESTOR et al. 2003).

The data supporting the above statements have been used to support a global theory of cognitive dysfunction in schizophrenia. However, it has also been noted that not all schizophrenia patients are characterized by these deficits, and that some patients may

have different degrees of impairment in different domains of contextual coordination. We have argued elsewhere (PHILLIPS & SILVERSTEIN 2003) that this may reflect differences in types of developmental trajectories of cortical pruning differences in which several of the 5 subunits of the NMDA receptor are affected (WATANABE et al. 1992), and other factors in the development of schizophrenia. Thus, it is to be expected that there will be heterogeneity even in what we have viewed as an example of a dysfunction in a core computation algorithm. In addition, it is likely that other aspects of schizophrenia will affect other aspects of the CNS, including specific regions such as the frontal and temporal lobes and basal ganglia, which will cause deficits unrelated to those of cognitive coordination (although these may interact, see below). Clarifying the heterogeneity in cognitive and neurobiological abnormalities in schizophrenia, and how these interact, represents a formidable task for future research.

Affective disturbances and Gestalt processes in schizophrenia

PLAUM is correct in suggesting that the large majority of cognitive models in schizophrenia neglect the potential role of affective factors in producing cognitive deficits. This is somewhat surprising since as far back as 1919, the relationship between motivational and attentional deficits in schizophrenia was noted by KRAEPELIN, who wrote that the lack of motivation observed in many patients "is without doubt clearly related to the disorder of attention which we very frequently find conspicuously developed in our patients. It is quite common for them to lose both the inclination and ability on their own initiative to keep their attention fixed for any length of time" (1919, p. 5–6).

Recent attempts to model the relationships between motivation and cognition (BROWN & PLUCK 2000; GROSSBERG 2000) view affect (including motivation) and attention as having such strong reciprocal interactions that studying any one component in isolation will produce a distorted picture. Both views also highlight relationships between limbic and cortical regions. For example, the emotional centers of the brain, such as the amygdala, interact with sensory and prefrontal cortices to generate affective states, to attend to salient stimuli and events, to link affective information to sensory information so that stimuli and situations can acquire reinforcing properties, and to generate adaptive responses involving stimuli and events (GROSSBERG 2000). This suggests that some form of disconnection between the neurobiological substrates of motivation and attention is occurring in schizophrenia. SVENSSON (2000) proposed that this involves a functional disconnect between the subcortical, mesolimbic dopamine system (involved in reward and motivational functions) and the mesocortical dopamine system (involved in attention and other aspects of cognition), and more specifically, a reduced ability of the prefrontal cortex to exert a selective controlling influence over the phasic activity of ventral tegmental (VTA) (subcortical) dopamine neurons. In general, positive affect alters a wide range of cognitive processes via moderately increasing DA activity (ASHBY et al. 1999), and different affective states can either facilitate or reduce prefrontal functions (GRAY et al. 2002). Therefore, we agree that PLAUM that comprehensive theories of cognitive performance in schizophrenia will have to include considerations of affective factors.

Some reasons why this is relevant to the present discussion about Gestalt processing include: 1) several studies indicate that some cases of perceptual organization dysfunction in schizophrenia reflect reduced top-down feedback to basic perceptual processes and/or that increasing top-down feedback can normalize task performance; 2) this suggests the possible role of the prefrontal cortex in mediating perceptual organization ability (especially in cases where expectations and strategic factors affect what is grouped with what); 3) it has already been demonstrated that spatial working memory (SWM) task performance in schizophrenia (which is typically abnormal) can be improved by increasing affective input during engagement in the task. This has been accomplished in various ways, including changing the stimuli whose location is to be remembered from dots to facts or other personal meaningful material, providing verbal praise during task performance, and/or socializing with the patient for a few minutes before he/she begins the task (PARK et al. in press); 4) the prefrontal cortex is thought to be involved in SWM task performance, and, as noted above, its activity can be normalized under conditions of positive affect; 5) this raises the possibility that increasing affective input to some optimal level during performance of perceptual organization tasks could normalize performance among schizophrenia patients, secondary to enhancing prefrontal cortical activity; and 6) while the specific role of affective factors in Gestalt perception is not yet known, the evidence cited above suggests that this issue is worthy of further investigation. New insights into this issue could help clarify the mechanisms involved in interactions between higher and lower forms of cognition, factors involved in deficient task performance among schizophrenia patients (including how test performance is affected by its social and affective context), and possible premorbid, developmental, affective precursors of cognitive deficits in schizophrenia.

Outlook and prospects for Gestalt theory informed models of cognitive dysfunctions in schizophrenia

Schizophrenia represents one of the most puzzling disorders of the human mind. Despite a hundred years of research, the causes and mechanisms of the disorder remain largely unknown. The inherent problems of research into the psychology of schizophrenia led Karl JASPERS (1959), for example, to conclude that any attempt to solve the enigma of schizophrenia was doomed to failure. Although we do not share this pessimistic outlook, we acknowledge that any single current psychological or neurobiological theory (or a combination of both) is likely to underestimate the complexities of schizophrenia. Yet, we believe that Gestalt theory contains valuable concepts and insights which will advance our understanding of schizophrenia.

Progress will, in part, depend on further developments in cognitive neuroscience to identify the neural and cognitive underpinnings of Gestalt mechanisms at various levels of the cognitive system. Recent research has clarified distinct cognitive and neurobiological mechanisms involved in normal perceptual organization. These provide clues to the pathophysiology of schizophrenia. The continued interest in cognitive neuroscience among psychologists working within the Gestalt tradition makes us hopeful that a Gestalt-theory informed approach of schizophrenia will continue to contribute to our understanding of this complex disorder.

Summary

Dysfunctions in Gestalt perception represent a specific deficit in the grouping of stimulus elements. Specifically, performance advantages of schizophrenia patients on measures of Gestalt perception disprove the prediction derived from the generalized deficit model (Chapman & Chapman, 1978). The explanation of cognitive deficits in schizophrenia in terms of a loss of the Gestalt structure may not be sufficient, however. Phenomenological data indicate that delusional perception, for example, may involve an abnormal organization of sensory information. In addition, dysfunctions in Gestalt perception may be relevant for the understanding of deficits in basic sensory processes as well as in higher cognitive functions, such as thinking and language, as well as for the explanation of affective disturbances. The heterogeneity of cognitive and affective disturbances in schizophrenia can be accomodated by recent models of the pathophysiology of the disorder.

Zusammenfassung

Die gestörte Gestaltwahrnehmung bei Schizophrenen ist das Resultat eines spezifischen Defizits in der Gruppierung von Stimuluselementen. Insbesondere die besseren Testleistungen von Patienten widerlegen die Hypothesen des ‚General Defizit Models' (CHAPMAN & CHAPMAN 1978). Jedoch mag eine Charakterisierung der Schizophrenie im Sinne eines Zerfalls der Gestaltstruktur unzureichend sein. Die Phänomenologie der Schizophrenie deutet darauf hin, daß insbesondere Wahnvorstellungen auf einer abnormen Gestaltbildung basieren könnten. Ferner handelt es sich bei Defiziten der Gestaltwahrnehmung bei Schizophrenen um eine Beeinträchtigung, die frühe sensorische Verarbeitungsprozesse als auch Defizite in komplexeren, kognitiven Prozessen, wie z. B. im Denken und in der Sprache, sowie affektive Störungen umfaßt. Die Heterogenität kognitiver und affektiver Störungen bei Schizophrenen läßt sich aufgrund neuerer Modelle zur Pathophysiolgie der Schizophrenie erklären.

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