HOW SPECIFIC IS THE GESTALT-INFORMED APPROACH TO SCHIZOPHRENIA?

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Specificity: Claims, Revisions, Disillusionment?

UHLHAAS & SILVERSTEIN (2003a) argue that Gestalt psychology has continuing relevance for an understanding of schizophrenia. Their line of argumentation is based on perception; on the Gestalt-like grouping of visual elements into a coherent whole. They draw on numerous empirical studies showing that patients with schizophrenia frequently display deficits in perceptual organization. The authors claim that the perceptual organization model is superior in accounting for the data than alternative cognitive theories. Such more general theories have focused on deficits of processing speed, processing capacity, or attention in patients with schizophrenia. The finding of PLACE & GILMORE (1980) of a circumscribed superiority of schizophrenia subjects in grouping tasks is proposed as a particularly strong demonstration of the validity of the perceptual organization model. In the course of the article UHLHAAS & SILVERSTEIN broaden the scope of their discussion by addressing neurobiological research and theories as well as phenomenological approaches (cf. KÖHLER's isomorphism). They conclude:"schizophrenia may be characterized by a general impairment in Gestalt processes affecting consciousness as a whole and its neural substrate" (p. 272).

PLAUM's commentary to UHLHAAS & SILVERSTEIN (2003a) addresses a number of methodological issues (e.g. the weight of PLACE & GILMORE's findings, the problem of selecting adequate control groups). As a main topic, PLAUM (2003) introduces the perennial question of the specificity of the defect(s) found in people with schizophrenia. PLAUM mentions the observation of temporal fluctuations of schizophrenia-related deficits and symptoms, which make identifying a specific deficit even more difficult.

In their response to PLAUM's comments, UHLHAAS & SILVERSTEIN (2003b) develop a position that, at least in my own reading, revises their original viewpoint to some extent. They now emphasize, for instance, that "schizophrenia patients are not deficient in all tasks assessing perceptual organization" (p. 290), that "deficits in perceptual organization fluctuate with severity of clinical signs and symptoms" (p. 291), and that "dysfunctional perceptual organization (...) may be indicative of a neurodevelopmental subtype of schizophrenia" (p. 292). In other words, not all schizophrenia patients show Gestalt deficits, and such deficits are probably not stable markers of schizophrenia.

The development of UHLHAAS & SILVERSTEIN's position becomes a little ambiguous here; initially, their article (2003a) clearly transmits the message that schizophrenia can be characterized by a deficit of perceptual, especially visual, organization. This "perceptual organization deficit hypothesis" is put forward as the best explanatory model to date. To me this sounds like a pretty specific hypothesis. Yet in their ensuing response to PLAUM, UHLHAAS & SILVERSTEIN (2003b) state that it is *un*likely that the varieties of clinical signs, perceptual and other dysfunctions of schizophrenia "represent a single unitary pathological mechanism" (p. 292).

What has happened? – *„Als Tiger abgesprungen und als Bettvorleger gelandet"*? I perceived that UHLHAAS & SILVERSTEIN started out as proponents of a specific deficit hypothesis, but by the end of the second paper have arrived at a considerably modified conclusion – namely that heterogeneity dominates in schizophrenia and „that any single current psychological or neurobiological theory (or a combination of both) is likely to underestimate the complexities of schizophrenia" (p. 295).

Perhaps one should not criticize the authors for the subtle ambiguity of their statements if the evidence they have so expertly collected is itself ambiguous. Nevertheless, a first reaction may be disillusionment: A specific Gestalt explanation of schizophrenia was promised, but a generalized "Gestalt-informed" deficit model resulted finally. Must Gestalt theory queue up together with all the other "explanations" of this enigmatic disorder – all the theories that had claimed initially to hold specific explanatory value for schizophrenia, but ultimately failed?

Isomorphism

Sharing the author's opinions about the attractiveness of Gestalt theory applied to psychopathology, I highly esteemed for UHLHAAS & SILVERSTEIN's great job in reviewing the literature and putting together pieces of evidence from cognitive research, neuroscience, and phenomenology. The body of research on perceptual grouping originating from this group is without doubt an excellent achievement.

In their theoretical considerations I especially liked that they address (and advance) KÖHLER's concept of isomorphism. The big challenge of today's neurocognitive science is to come to grips with how mind and brain can interact (if they are in fact separable entities) or, alternatively, how mind and brain can be identical (if some monist approach is chosen). In either case, isomorphism (or some concept with congruent meaning) must be at the very heart of the discussion.

My own understanding is that the concept of isomorphism should be freed from its historical, quite concretist, connotations by showing the deep *structural* analogies present in emergent phenomena (or "Prozessgestalten", TSCHACHER 1997). The appropriate instrument for that purpose may be non-linear systems theory such as synergetics (HAKEN 2000). The structure of Gestalts emerging from complex dynamical systems of different domains can thus be described using a unitary methodological framework. UHLHAAS & SILVERSTEIN speak of an "empirical verification" of isomorphism (p. 261) brought about by synchronization of neurons in the gamma band. Even if the expression "verification" may be a bit tough in such a deli-

cate philosophical context, I readily agree with UHLHAAS & SILVERSTEIN that isomorphism may be expected to describe the commonalities of Gestalt processes in different substrates and at different levels of cognitive processing. My additional suggestion here is that an integrative background theory (such as non-linear systems theory) should be very advantageous in this respect because it would provide clearer concepts and mathematical tools for modelling.

A core concept of systems theory, known as an "attractor", concerns the attribute of stability of emerging patterns. From the point of view of systems theory, all perceptual Gestalts are attractors. Moreover, applications have been reported for emergent structures in other fields; e.g. such attractors were described in movement coordination (MECHSNER et al. 2001), cognitive coordination (KELSO 2003), in psychological Bartlett scenarios (KRUSE & STADLER 1990), and in systems of interacting individuals (KRIZ 1997). In addition, non-linear systems theory has its roots in the natural sciences and is used to model self-organization phenomena in complex physical, chemical, and biological systems. It was thus a straightforward development to adopt this approach in neurocognitive science (HAKEN 1996; VARELA et al. 1991; TSCHACHER & DAUWALDER 1999, 2003). Further important elements in this systems-theoretical approach are the control parameters; these parameters denote the environmental forcing of complex systems, they capture the valent context of Gestalt attractors.

In other words, the systems approach is promising, in my view, *because* of its generalized stance. Only with the aid of a *generalized* theoretical background may we arrive at an alternative understanding of *specificity* when we employ this theory to understand a manifestly heterogeneous disorder such as schizophrenia. "Specific" may not mean that the key to an understanding of schizophrenia is confined to perception, attention, executive function, or any other single cognitive faculty. Instead, a specific dysfunction may dwell inside an entirely general mechanism.

In this vein we may discriminate the following broad categories of neurocognitive specificity (happily ignoring the bulk of viral, genetic, social, psychoanalytic and further specific hypotheses of schizophrenia research):

- faculty specificity (e.g. a deficit of perception; a localized impairment in the brain);
- function specificity (e.g. a circumscribed deficit of a process not confined to a single cognitive faculty; a circumscribed deficit of neuronal assemblies not localized in a single part of the brain);
- stage specificity (specific dysfunctions of schizophrenia are nonstationary, i.e. they vary with time or with the stage in the evolution of the disorder);
- no specificity.

As mentioned, the position of UHLHAAS & SILVERSTEIN contains some opacity where it concerns the topic of specificity. They argue for several categories of specificity: (i) based on the perceptual grouping database they propose deficits in perceptual organization as a specific finding (faculty specificity); (ii) later in their

discussion they address a potential dysfunction of a general cortical processing algorithm (function specificity); and finally, (iii) in response to PLAUM, UHLHAAS & SILVERSTEIN state that there is probably no single cognitive dysfunction (no specificity).

Yet, the "no specificity" result would be particularly uninteresting, especially if we choose to approach schizophrenia in a Gestalt-inspired fashion. It would mean that all the single findings concerning schizophrenia would not result in an integrated "holistic" picture of this disorder, but add up to little more than a summative assembly of elements – in this case, a sum of isolated findings. This would entail a less than elegant research program. Therefore, let us first explore other notions of specificity, starting with function specificity. We refrain from discussing faculty specificity here because the state of schizophrenia research indicates that deficits are not confined to a single faculty of information processing. Schizophrenia is obviously not a perception problem alone, nor is it restricted to memory, or attention, or executive functions.

Function Specificity

Cognitive coordination, binding, and Gestalt formation: the common demominator of these concepts is that they suggest an integration of single elements into a resulting whole or Gestalt, thereby employing some mechanism of complexity reduction. The tradition of Gestalt psychology has shown that such processes are encountered ubiquitously; they are not confined to perception. In fact, such a theory "(...) cuts across all the traditional distinctions (...)" (PHILLIPS & SILVERSTEIN, 2003). Yet perceptual demonstrations may be most convincing because of the phenomenological ease of access.

Since Gestalts are concepualized as attractors (i.e. resultant subspaces in cognition), then from a complex systems perspective, *stability* is a core property of them. A number of neuropsychological tasks seem especially appropriate to study the dimension of stability and destabilization. One of these consists of presenting bistable or multistable stimuli generated in the context of an *apparent motion paradigm*. One reason that I hold apparent motion to be an appropriate candidate is because Wertheimer's original phi-phenomenon is a major, striking example of the emergence of Gestalt qualities, ranking next to the Gestalt laws of perceptual grouping. Another reason for selecting apparent motion is that its attractor-like dynamics are well investigated (KRUSE et al. 1996) and neuronal signs of binding (gamma enhancement) have been reported to occur at the time of apparent motion flips (BASAR-EROGLU et al. 1996).

In a recent, as yet unpublished study (cf. TSCHACHER & SCHULER 2004) the stability of apparent motion illusions were measured in a circular apparent motion display (CAM) and in a stroboscopic apparent motion (SAM) paradigm. Almost all subjects instantaneously perceive one of the apparent motion illusions (for instance, clockwise CAM), and the illusion almost always flips spontaneously into a qualitatively different illusion (for instance, counterclockwise CAM) after a certain period of time. Stability was operationalized by the mean duration of each CAM or SAM perception. A sample of schizophrenia patients treated in a psychiatric hospital was

compared to a non-patient control group matched with respect to age and sex. In this investigation, no significant group differences for stability were found in the CAM and SAM paradigms.

A more sophisticated way to operationalize the stability of Gestalts can be achieved by measuring *hysteresis*. Hysteresis, or the tendency for a percept to remain unchanged across trials, is a phenomenon which characterizes bistable attractor displays (e.g. SAM) where in general one of two motion Gestalts can be perceived (in this case, either horizontal or vertical apparent motion). The probability of either Gestalt perception depends on a control parameter (in this case, horizontal distance between the stimuli). First, the control parameter is gradually increased from minimum to maximum values to elicit a Gestalt flip (control parameter value = CP1). Then in a second run, the direction of parameter variation is reversed (decrease from maximum to minimum). In this case the Gestalt flip may be perceived at a different parameter value (control parameter value = CP2). The reason for asymmetry lies with the delay of the flip events due to the stability of the Gestalt established before. The difference –CPdiff = CP1-CP2 – is is a measure of hysteresis in the paradigm; a CPdiff different from zero is an indicator of attractor dynamics. The magnitude of CPdiff may be considered a direct measure of the stability of the apparent motion Gestalts.

We measured CPdiff in a stroboscopic Gestalt paradigm. CPdiff very significantly deviated from zero in both the patient and the control groups, indicating stable attractors. No statistical difference, however, was observed between the group averages of hysteresis.

Stability was put forward as one essential property of Gestalts. In the case of perceptual grouping, the dependent variables are usually reaction times or error rates in degraded or noisy stimuli. One may assume that these variables assess the salience / *Prägnanz* of the corresponding Gestalts, thus their stabilities. Yet stability per se may not represent the *specific*, critical parameter of cognitive Gestalt formation in schizophrenia. It seems likely that specificity rests with a further aspect of Gestalt formation. A candidate aspect is the relationship between the control parameters and any emergent Gestalt. Thus, the decisive distinction between functional and dysfunctional Gestalt formation may not be the stability of Gestalts (or the "ease" of Gestalt emergence) per se, but instead the contextual adequacy of a Gestalt given a certain control parameter. This (lack of) adequacy corresponds to the functional disconnection of motivational and cognitive brain regions mentioned by UHLHAAS & SILVERSTEIN (2003b, p. 294).

We tested the relationship of control parameter values and Gestalt formation in an apparent causality paradigm introduced by MICHOTTE (1954). Two disks approaching one another on a computer screen are either perceived as "bouncing" or "streaming" (passing by each other) at the moment of visual overlap. This paradigm can thus be considered a bistable Gestalt system in the domain of causality attribution. An acoustic click stimulus with varying temporal latency relative to "collision" time of the disks has been applied as a control parameter (LEWKOWICZ 2000). This stimulus controls the emergent causality Gestalt because if the click is sounded before (after) visual overlap, the probability of attributed "bouncing" ("streaming")

is increased. This paradigm therefore pertains to non-perceptual Gestalt formation and, additionally, operationalizes the relationship between the control parameters and an emerging Gestalt. Since acoustic control parameters are combined with an optical paradigm, the MICHOTTE display is a method to explore intersensory binding. It was found in our recent study that this relationship was less pronounced in the patient group than in controls.

To conclude, we found few differences in apparent motion paradigms that might help distinguish schizophrenia patients' cognition from control subjects. A possible exception is the latter finding from MICHOTTE's paradigm, but the significance of this is yet unclear. To date the evidence in favor of function specificity is equivocal, but I agree with UHLHAAS & SILVERSTEIN that Gestalt-related function specificity remains a constructive challenge for schizophrenia research.

Stage Specificity

PLAUM (2003, p. 283) drew our attention to the temporal and even oscillating character of symptoms and dysfunctions in schizophrenia. The process character of schizophrenia has been a focal topic in our own group's research (KUPPER & TSCHACHER 2002; TSCHACHER & KUPPER 2002). Extensive longitudinal monitoring of symptoms shows that even those symptom domains that are frequently viewed as enduring (such as negative symptoms of schizophrenia) fluctuate considerably and on short (e.g. daily) time scales. This is compatible with the dynamical disease approach (TSCHACHER et al. 1997). Further clues come from Gestalt psychiatry; CONRAD's (1958) stage model of schizophrenia pointed out the clear qualitative differences between the *Trema* stage (destabilization of Gestalts) and the *Apophänie* stage (hyperstabilization of delusional Gestalts). Such observations make stage specificity a plausible assumption – function-specific deficits are still assumed to exist, but we would not expect to discover them cross-sectionally with any reliability because these specific deficits are variables depending on time.

UHLHAAS & SILVERSTEIN (2003a) review several studies that show an association of perceptual grouping with the "cognitive disorganization" factor of the PANSS ("Positive and Negative Symptom Scales", a structured psychopathology interview) but no association with other symptom domains. Contradicting results have been obtained, however, that point to a linkage between clinical signs and Gestalt formation in schizophrenia.

Our own findings on bistable Gestalts suggested that specific associations between psychopathological dimensions (measured by the PANSS) and apparent motion perception existed in a sample of schizophrenia patients. Multiple regression analyses revealed strong associations between symptomatology and various Gestalt paradigms. Positive symptoms, for instance, were linked with Gestalt instability, negative symptoms with perseveration of Gestalts both in SAM and CAM (cf. above). We also tested the paradigm of motion-induced blindness (BONNEH et al. 2001), where spontaneous figure-ground reversals induce subjective blindness impressions. This paradigm showed an association between the PANSS "excitement" as well as "positive symptoms" factors and the frequency of experienced blindness phenom-

ena. Apparent causality likewise depended on symptoms; attribution of causality (the "bouncing" perception) was attenuated in the presence of positive symptoms, whereas it was negatively linked with "cognitive disorganization".

In short, the bulk of our recent investigations support stage specificity of Gestalt deficits in schizophrenia.

Conclusions

UHLHAAS & SILVERSTEIN's proposal of a Gestalt-informed approach in schizophrenia research is highly appealing. The authors have convincingly shown that their approach can expand our understanding of this disease.

UHLHAAS & SILVERSTEIN have integrated quantitative findings from both sides of the mind-brain divide into their model. It is hardly surprising that conflicting evidence must trickle into this grand scheme as soon as it is put to the test; the authors have integrated contradicting findings in their revised position. Nevertheless, I am personally confident that the Gestalt approach will provide specific answers, assumedly of the "stage specificity" type, and will not share the "no specificity" destiny. In my view, schizophrenia is not a "sum of findings" sort of entity.

Gestalt theory will likely become more powerful with a broader, generalized theoretical platform that can be developed by adopting a systems-theoretical underpinning for Gestalt theory as proposed by synergetics. This platform will entail, among other benefits, a more concise definition of Gestalt formation and of cognitive coordination. Yet only encompassing future operationalizations and empirical testing will ascertain the feasibility of this "broad approach".

There is a second, additional, way by which the Gestalt discussion can be reinforced in psychopathology research. The tradition of Gestalt psychology has created a wealth of attractive paradigms that can be exploited. A beginning renaissance of Gestalt paradigms can currently be observed in neurocognitive science; making an effort to expand it to schizophrenia research would certainly be helpful. While perceptual grouping is an important subdomain, it does not represent all brands of Gestalt formation that occur at various levels of cognition. It is therefore quite promising to explore further options, such as sensorimotor Gestalts in the HAKEN-KELSO-BUNZ model (HAKEN et al. 1985, MECHSNER & PRINZ 2003), "negative" emergent phenomena and motion-induced blindness, and intersensory binding (DENNETT & KINSBOURNE 1992), to mention just a few. My personal top candidate is "apparent intentionality", the interplay between cognitive control parameters and ensuing Gestalt formation at multiple stages of cognition (TSCHACHER et al. 2003), and the attached favorite hypothesis for the "key of schizophrenia" is that a specific dysfunction may be detected in exploring this interplay.

Summary

This commentary on UHLHAAS & SILVERSTEIN's Gestalt theoretical approach focuses on the problem of discovering specific dysfunctions in schizophrenia. Several categories of specificity are defined – deficits found in schizophrenia may be specific with respect to: a cognitive faculty or module (such as perception); a distributed neurocognitive function (e.g. binding); or a neurocognitive function depending on the temporal stage of the illness. It is argued that findings from various Gestalt psychological paradigms, including my own studies using apparent motion tasks, tend to support the latter notion of "stage specificity". Additionally, it is suggested that the Gestalt-informed approach to schizophrenia may profit from a broader nonlinear systems embedding (provided by synergetics and self-organization theory). Schizophrenia research may advance particularly by applying a variety of Gestalt paradigms not necessarily confined to perceptual grouping.

Zusammenfassung

Dieser Kommentar zum gestalttheoretischen Ansatz von UHLHAAS & SILVERSTEIN bezieht sich auf das Problem, schizophreniespezifische Defizite aufzudecken. Verschiedene Kategorien von Spezifität werden definiert. Die aufgefundenen Defizite können in verschiedener Hinsicht spezifisch sein: sie können sich auf ein einzelnes kognitives Modul, wie etwa Wahrnehmung, beziehen; sie können spezifisch für eine ansonsten verteilte neurokognitive Funktion sein (z. B. binding); sie können spezifisch für eine neurokognitive Funktion unter Berücksichtigung des Krankheitsstadiums sein. Es wird argumentiert, daß die Befunde aus verschiedenen gestaltpsychologischen Paradigmen, einschließlich eigener Studien aus dem Bereich der Scheinbewegung, für die letztgenannte „Stadiumsspezifität" sprechen. Zusätzlich wird vorgeschlagen, daß der Gestaltansatz in der Schizophrenieforschung von der Einbeziehung in eine breitere Theorie (nichtlineare Systeme, Synergetik, Selbstorganisationstheorie) profitieren könnte. Die Schizophrenieforschung würde zudem durch die Nutzung der Vielzahl verfügbarer Gestaltparadigmen, nicht notwenigerweise nur aus dem Bereich der Wahrnehmung, vorangetrieben werden.

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