Perception of Causality in Schizophrenia Spectrum Disorder

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Patients with schizophrenia spectrum disorders often maintain deviating views on cause-effect relationships, especially when positive and disorganization symptoms are manifest. Altered perceived causality is prominent in delusional ideation, in ideas of reference, and in the mentalizing ability (theory of mind [ToM]) of patients. Perceiving causal relationships may be understood either as higher order cognitive reasoning or as low-level information processing. In the present study, perception of causality was investigated as a low-level, preattentional capability similar to gestalt-like perceptual organization. Thirty-one patients (24 men and 7 women with mean age 27.7 years) and the same number of healthy control subjects matched to patients with respect to age and sex were tested. A visual paradigm was used in which 2 identical discs move, from opposite sides of a monitor, steadily toward and then past one another. Their coincidence generates an ambiguous, bistable percept (discs either "stream through" or "bounce off" one another). The bouncing perception, ie, perceived causality, is enhanced when auditory stimuli are presented at the time of coincidence. Psychopathology was measured using the Positive and Negative Syndrome Scale. It was found that positive symptoms were strongly associated with increased perceived causality and disorganization with attenuated perceived causality. Patients in general were not significantly different from controls, but symptom subgroups showed specifically altered perceived causality. Perceived causality as a basic preattentional process may contribute to higher order cognitive alterations and ToM deficiencies. It is suggested that cognitive remediation therapy should address both increased and reduced perception of causality.

Key words: cognitive remediation therapy/gestalt perception/perceived causality/psychopathology/schizophrenia/theory of mind

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Introduction

Most schizophrenia spectrum patients report that their phenomenological appraisals of self, others, and environment have undergone marked changes in the course of the disorder. Many of these changes affect agency and causality, ie, the issue of who causes actions and events and how events are causally linked. Patients' views on the relationships between causes and effects often deviate markedly from what other people think. This is especially true when positive symptoms are manifest—a person with delusional ideation, eg, believes in causal links that are deemed bizarre by external observers. In addition to paranoid and delusional ideas, a person may sense an immediate link between him- or herself and external events.[1] In such "ideas of reference," a person perceives causal relationships between him- or herself and the social environment when in fact there are none. The disorganization syndrome of schizophrenia is characterized by altered causality as well, yet in a different manner. A number of studies on perceptual organization have found that the spontaneous gestalt-like grouping and association of stimulus items are deficient in chronic patients with disorganized symptoms[2,3]; in other words, associations and, prominent among these, causal associations are reduced in disorganized cognition. Such results are consistent with the Bleulerian notion of a weakening of associations as well as the premises of gestalt psychiatry.[4] Perceiving causal relationships may be understood either as "reasoning," ie, a complex "higher" cognitive ability, or as low-level, automatic information processing resembling gestalt perception processes.[5] It has remained unclear which cognitive processes at which level of processing contribute to the altered causal inferences characterizing psychotic symptoms such as delusions and disorganized thinking.

The research on "theory of mind" (ToM) points in a similar direction[6]; ToM is defined as the ability to "mentalize," ie, to monitor and infer one's own and others' mental states. To a large extent, ToM addresses social causation. If a person's causal attributions are generally deficient or lacking, the mentalizing ability of this person will by necessity be compromised. As a consequence, social skills and interactive behavior as well as the phenomenology of patients are likely to be affected.[7] It was found that ToM was impaired particularly in

patients with positive symptoms[8] and disorganization symptoms.[9] ToM research normally used written or "comic strip" stories of social scenes. In a recent study, animated sequences displaying different types of "interactions" of geometric figures were presented on a monitor; schizophrenic patients could less well describe the inferable content of the sequences (eg, a triangle "chasing" another triangle[10]).

The phenomenology of schizophrenia as well as the findings on ToM deficiencies indicate that notions of causality are altered in schizophrenia, with ensuing impacts on social cognition and behavior. As is the case in phenomenology, the origins of ToM deficiencies remain largely undetermined. ToM research typically addresses higher cognition, ie, mental modules that process abstract information, as for instance metarepresentational information (thinking about what others think). ToM research therefore has relied to a great extent on the verbal descriptions and interpretations that are provided by study participants.[10] This renders localization of the origin of ToM problems encountered by schizophrenic patients quite difficult. The locus of deficiencies may lie anywhere in higher cognition (eg, in linguistic skills; in inductive reasoning), but also in basic cognition (eg, memory, attention, vigilance), or even in perception (eg, preattentional binding processes[11]). To date, ToM research has not unambiguously disentangled the multiple mental processes, of varying cognitive complexity levels, on which the mentalizing ability likely rests.

Which therapeutic consequences result from these findings? If patients with schizophrenia spectrum disorders suffer (specifically) from inadequate ToM and causal attribution, such deficiencies are promising targets for cognitive remediation therapy.[12,13] The efficacy of targeted cognitive-behavioral intervention, however, can be optimal only with detailed information on the nature of these deficiencies. Even if there are multiple sources of dysfunctional cognition on causal relationships, steps toward selective indication for therapy are possible and necessary. It is thus essential to investigate the cognitive processes underlying causal attributions.

The Belgian gestalt psychologist Michotte[5] developed an empirical research program for the investigation of low-level causality perception using simple psychophysical paradigms. In Michotte's prototypical "launch experiment," some geometric object A moves toward object B which is stationary. After "collision," object A is stationary and B moves away from A evoking an immediate perception that the first motion caused the second—"A pushed B." Michotte viewed this phenomenal causality attribution as an example of a gestalt perception illusion akin to apparent motion.[14] This illusionary perception of motion has found to be altered in patients with schizophrenia (W. Tschacher, P. Dubouloz, R. Meier, and U. T. Junghan, unpublished data, 2006). Michotte's experimental setup thus has the potential to
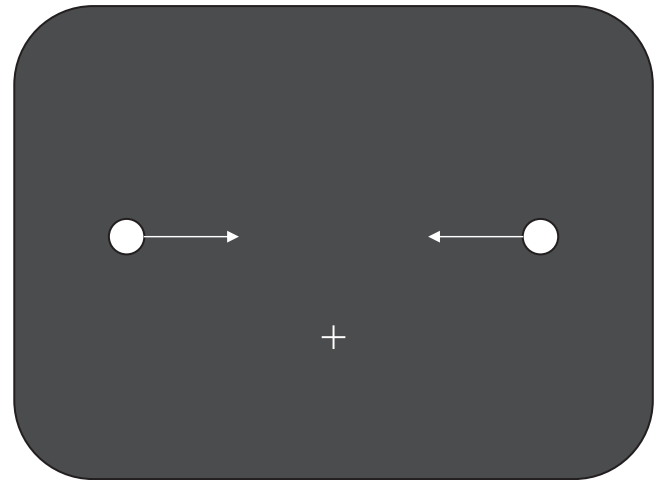
Fig. 1. Screen display (schematic) for the perceived causality paradigm. Two discs move horizontally toward each other with constant speed, coincide in the screen center, and continue moving until they are again separated by their initial distance. An auditory stimulus is presented at the time of coincidence.

clarify high-level causal attributions by investigating "early," preattentional causality perception. A variant of Michotte's experiments was used to study visual motion perception in the presence of additional, auditory stimuli.[15] In this paradigm, 2 identical discs move, from opposite sides of a monitor, steadily toward and then past one another (figure 1). Their coincidence generates an ambiguous, bistable percept: the discs appear to either "stream through" or to "bounce off" one another. The probability of a bouncing perception (ie, perception of causal interaction between the discs) is low in general but is elevated when a sound is presented near the time of collision of the discs.[16]

In the present study, this phenomenon was used to investigate basic causality perception in schizophrenia spectrum disorder. Causality perception was viewed as a neurocognitive binding process, by which the brain—prior to conscious processing—integrates multiple sensory inputs in order to create perceptual configurations.[17] Specifically, it was intended to assess the relationship between schizophrenia and causality perception by comparing patients with age- and sex-matched controls and evaluating the influence of symptoms on perception of causal interactions. The primary hypothesis specified that perception of causality depends on the dimensions of psychopathology in the schizophrenia spectrum patients. On the basis of previous findings[11] and the ToM literature,[6] it was further expected that the various symptom dimensions show differential associations with causality perception; positive symptoms should enhance perceived causality, whereas disorganization symptoms should be associated with reduced perceived causality. The second hypothesis targeted causality perception as a trait marker of schizophrenia spectrum disorder. Differences between

Table 1. Characteristics of the Healthy Control Group and Schizophrenic Patient Group

Variable	Healthy Controls (n = 31)		Schizophrenic Patients (n = 31)	
	M	SD	M	SD
Age (y)	27.4	7.9	27.7	7.3
Male-to-female ratio	24/7		24/7	
Education	4[a]	2–6[b]	3[a]	1–5[b]
Age at first hospitalization			23.9	5.0
Number of hospitalizations			2.8	3.7
GAF at entry			41.7	12.8
CPE (mg/d)			270	222
PANSS positive			2.0	0.9
PANSS negative			1.9	0.9
PANSS excitement			1.5	0.4
PANSS depression			1.9	0.6
PANSS cognitive			1.6	0.7

Note: PANSS, Positive and Negative Syndrome Scale; CPE, chlorpromazine equivalents; GAF, Global Assessment of Functioning Scale. The following difference was statistically significant: education, schizophrenic patients < healthy controls (Kruskal-Wallis test, $P < .01$).
[a]Median.
[b]Range.

patients and controls and potential specific covariates of causality perception were investigated.

Materials and Methods

Participants

The study sample consisted of 31 patients (24 [77%] men and 7 [23%] women with mean age 27.7 years [SD 7.3]) and the same number of healthy control subjects (male-to-female ratio 24:7; mean age 27.4 years, SD 7.9; table 1). Patients were recruited from units of the University Hospital of Psychiatry in Bern, Switzerland. Thirteen inpatients had been admitted to a community-based acute unit (Soteria Bern), and 18 patients were undergoing psychiatric outpatient treatment in 2 day hospitals. All patients had been diagnosed as suffering from schizophrenia spectrum disorder according to the International Classification of Diseases, ICD-10 (F20 schizophrenia, 25; F21 schizotypal disorder, 1; F23 acute psychotic disorder, 2; F25 schizoaffective disorder, 3). The mean chlorpromazine equivalent prescribed on the day of testing was 270 mg (SD 222 mg). The entire patient sample was receiving atypical neuroleptics, with the exception of 1 individual who was being treated with haloperidol decanoate. Patients participated in standardized clinical interviews (Positive and Negative Syndrome Scale [PANSS][18]), dur-

ing which their individual psychopathological states were assessed. Trained staff psychologists, who were not associated with the project and were naive to its hypotheses, acted as interviewers. The model developed by Lindenmayer et al[19] was utilized to group PANSS psychopathology into the 5 factors of positive, negative, excitement, depression, and cognitive. The positive factor includes symptoms characteristic of florid psychosis, such as, delusions, hallucinatory behavior, and unusual thought content; the negative factor describes deficit symptoms of schizophrenia commonly manifested as emotional and social withdrawal; the excitement factor consists of items such as excitement, hostility, tension, and impulsivity; the depression factor consists of the PANSS items of depression, anxiety, guilt feelings, somatic concern, and preoccupation; and finally, the cognitive factor incorporates signs of cognitive disorganization, such as conceptual disorganization, disorientation, and difficulty in abstract thinking. The mean symptom burden of patients included in the study was moderate to low. As a further measure, the Global Assessment of Functioning Scale (GAF, published in *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*) was implemented to assess the psychological and social levels of functioning in patients upon entering their present course of treatment. The number of previous hospitalizations and the patients' ages at the time of their first psychiatric hospitalization were recorded on the basis of case histories. The highest degree of education achieved was operationalized using a 6-point ordinal scale (ranging from 1, high school not completed, to 6, university degree; adapted to the Swiss schooling system). In summary, this sample of young adults may be characterized as follows (see table 1): low to medium symptomatology on average, all participants except 1 had completed high school or obtained a higher education degree, and the patient group had achieved a lower degree of education than the control group. All participants agreed to take part in the study on the basis of prior written informed consent. The study had been previously evaluated and approved by the Canton of Bern Ethics Committee.

Materials and Procedures

The causal perception paradigm was presented on a 17-in computer screen at a viewing distance of 50 cm (the display is shown schematically in figure 1). Subjects were instructed to maintain fixation on a cross 2.5 cm (visual angle, 2.9°) below the center of the display. All tests were carried out on working days between 10:00 AM and noon at the testing facility of the hospital's research department. For each participant, 10 preparatory runs (for instructional purposes) preceded 50 evaluated runs. During instructional runs, the participants were informed about the bistable character of the stimulus, ie, that 2 alternative events could be seen, either bouncing or streaming of the discs. All data derived from the evaluated runs were used

for ensuing statistical analysis. Each run lasted approximately 2.5 s, with an interval of random duration (range, 1–3 s) between runs. Two white discs (diameter 0.5 cm, visual angle 0.6°) appeared on both sides above the fixation cross against a black background. Discs were initially separated by 12 cm (13.7°). Immediately after onset, the discs moved horizontally toward each other with a constant speed of 10 cm/s (11.4°/s), coincided in the screen center, and continued moving until they were again separated by 12 cm; discs then disappeared. At around the time of coincidence, a click sound of 40-ms duration was presented from 2 speakers next to the monitor; the click sound "clickup.wav" integrated in the computer operating system Microsoft Windows 98 was used. The exact timing of the sound defined the following 5 conditions: condition 1 (2), 150 ms (75 ms) before coincidence; condition 3, simultaneous with coincidence; condition 4 (5), 75 ms (150 ms) after coincidence. Each condition occurred with equal probability in random sequence throughout the 60 runs. After each run, an identical written text was shown that contained the instruction to press the left (right, middle) button of the computer mouse when a bouncing (streaming; unclear) perception had resulted in the run just observed.

For each subject, the probability of "bounce" responses was computed across all evaluated runs for each of the 5 conditions. To assess the global probability of causal perceptions, the probabilities of bounce responses were pooled across all conditions. This variable was labeled "perceived causality."

Statistical Treatments

The following statistical procedures were performed to assess the hypotheses of this study. First, multiple regression analysis was applied to estimate the relationships between perceived causality and psychopathology as assessed using the PANSS. This analysis was based exclusively on the patient group. In the regression analysis, perceived causality was treated as a dependent measure, and the 5 PANSS psychopathology factors were treated as predictors. In addition to whole-model regression analysis, backward stepwise regression was applied to determine the most parsimonious model by which perceived

causality could be predicted. Second, group differences between patients and controls regarding perceived causality were tested by a multivariate analysis of variance (MANOVA). In this MANOVA, the effects of the timing of the auditory signal (expressed in conditions 1 through 5) on the probabilities of bounce perceptions as well as the interaction group × condition were analyzed. Additionally, associations of patients' descriptive characteristics with perceived causality were explored.

Results

Association of Perceived Causality With Psychopathology

The schizophrenic patients group showed a highly significant and strong association between perceived causality and psychopathology (table 2). Forty-six percent of total variance of perceived causality was explained by differences in psychopathology. A stepwise regression analysis suggested that perceived causality is increased in the presence of positive symptoms (standardized beta = .79) and attenuated by cognitive symptoms (standardized beta = −.51). A post hoc analysis of this finding revealed that cognitive symptoms alone were not significantly related to perceived causality, whereas the difference of positive and cognitive symptoms was a potent predictor of perceived causality in this sample of schizophrenic spectrum patients (R^2 = .39; adjusted R^2 = .37; P = .0002), as was the ratio of positive symptoms and cognitive symptoms (R^2 = .35; adjusted R^2 = .33; P = .0005). The regressive relationship of the predictor "positive minus cognitive" with perceived causality is shown in figure 2.

Differences Between Patient Group and Healthy Control Group

The test for overall group differences regarding perceived causality and the effect of the timing conditions are given in table 3. High Cronbach α values were found over all conditions (entire set, α = .95). In MANOVA, a tendency for lower perceived causality in schizophrenic patients was found ($F_{1,60}$ = 3.12, $P < .1$). There was a highly significant effect for conditions ($F_{4,57}$ = 10.06, $P < .0001$) showing that perceived causality depended on the timing of the auditory stimulus. The group × conditions interaction,

Table 2. Summary of Whole-Model Tests and Stepwise Regression Analyses for Psychopathology Factors Predicting Perceived Causality

Multiple Regression Whole Model		Stepwise Regression (backward)		
R^2 (adjusted R^2)	F	R^2 (adjusted R^2)	F	PANSS Predictors (standardized beta)
.46 (.36)	4.34**	.41 (.37)	9.84***	Positive (.79)**** Cognitive (−.51)** Excitement/negative/depression (NS)

Note: PANSS, Positive and Negative Syndrome Scale; n = 31.
**$P < .01$; ***$P < .001$; ****$P < .0001$; NS, not significant.
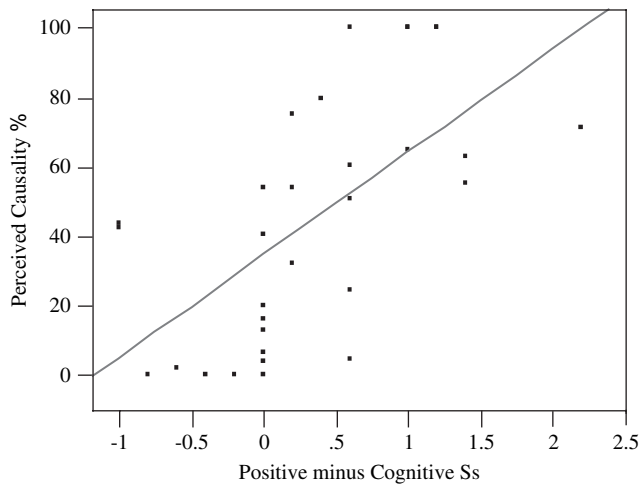
Fig. 2. Linear regression of the predictor "positive minus cognitive symptoms" on perceived causality (probability of bounce perceptions in percent).

however, was insignificant—the impact of timing conditions did not distinguish patients from controls.

Associations of Patient Characteristics and Perceived Causality

No significant correlations between perceived causality and patient characteristics were found for age, education, neuroleptic dosage, and the number of previous psychiatric hospitalizations (table 4). Lower GAF at entry tended to be correlated with increased perception of causality. Also, younger age at first hospitalization was associated with enhanced perception of causality. Sex was related to the perception of causality in all participants; more causality was perceived by male participants in the control group ($F_{1,29} = 9.8$, $P < .01$) but not the schizophrenic patient group ($F_{1,29} = 0.54$, not significant).

Discussion

This study focused on the causality perception of schizophrenia spectrum patients because causal inferences and causal attributions are essential constituents of higher cognitive processes, such as ToM and social cognition. The paradigm applied here defined "perceived causality" as a basic preattentional cognitive function akin to gestalt-like neurocognitive binding processes. This function was found to be closely related to psychopathology, especially positive symptoms and the cognitive (disorganization) factor. Positive symptoms were associated with increased perceived causality, whereas disorganization was associated with attenuated perceived causality. Group differences yielded no clear evidence of perceived causality as a trait marker of schizophrenia; only a statistical trend pointed toward decreased causal binding in patients. The manner in which the timing of the auditory stimulus shaped causal perception (ie, the group × conditions interaction) appeared unaffected in the patient group. A methodological caveat, however, may be adequate here. The very high consistencies (Cronbach α) over the entire set of timing conditions suggest that the conditions may have been distinguished insufficiently. It may thus be advisable to consider using auditory stimuli with higher salience and/or timing steps larger than 75 ms.

These results of the present investigation allow 3 conclusions. First, fundamental symptom domains of schizophrenia spectrum disorder are associated with perceived causality in a meaningful way. A possible interpretation of results is that altered causality perception may contribute to the development of positive and cognitive symptoms in patients. Second, findings suggest that patients may be compromised at the fundamental stage of perceptual organization. This can be inferred from the fact that perceived causality studied with the present paradigm is a preattentional function that occurs independent of conscious, linguistic, or metacognitive processing; for exactly this reason, the paradigm has been used previously in research with infants younger that 1 year.[16] Third, indications for perceived causality as a trait marker of schizophrenia were weak. This may have been due to the fact that the present sample represented the broader scope of schizophrenia spectrum patients. It included a subsample of acute patients with elevated measures of perceived causality as well. Results are thus in line with previous findings of excessive perceptual organization, specifically in prodromal and initial stages of schizophrenia.[4,11,20] Findings are also consistent with studies reporting perceptual organization deficits particularly in chronic patients, yet not in other symptom subgroups.[3]

Disturbances of perceptual organization may well be a key cognitive symptom in many schizophrenia spectrum

Table 3. Perceived Causality in Schizophrenic Patient Group and Healthy Control Group

Group	Conditions: Timing of Auditory Stimuli					
	Across Conditions	−150 ms	−70 ms	0	+70 ms	+150 ms
Schizophrenic patients	44.5	46.3	37.2	38.6	48.8	51.5
Healthy controls	58.9	59.9	47.7	47.1	66.4	73.2

Note: Numbers are probabilities of bounce events (in %), indicating perceived causality.
"−150 ms," click stimulus presented 150 ms before time of coincidence of discs.

Table 4. Pearson Correlations of Perceived Causality With Patient Characteristics

	Age	Education[a]	CPE	GAF at Entry	Number of Hospitalizations	Age of First Hospitalization
Perceived causality	−.27	−.15	−.01	−.33[†]	−.16	−.38*

Note: $n = 31$. CPE, chlorpromazine equivalents; GAF, Global Assessment of Functioning Scale.
[a]Spearman correlations.
*$P < .05$; [†]$P < .1$.

patients. Therefore, perceptual organization can instigate novel approaches for the psychological treatment of cognitive dysfunction in schizophrenia—cognitive remediation therapy. Disturbances of perceptual organization differ from other cognitive deficits found in schizophrenia in that they are not strictly performance deficits, such as reduced attention, reduced memory abilities, or reduced planning skills. Rather, deregulated perceptual organization can take both the form of a "gestalt deficit," ie, under-responsiveness to holistic stimulus features, and the form of a "gestalt excess," ie, overresponsiveness. Because disturbances of perceptual organization are likely to be markedly different in subgroups of patients, a selective indication approach to cognitive remediation is appropriate, eg, remediation for gestalt excesses and remediation for gestalt deficits. Gestalt excess may be at the basis of the well-described cognitive symptom of "jumping to conclusions" in schizophrenia.[21,22] Gestalt excess may be responsive to both more cognitive, informative, and practice-oriented remediative procedures. Cognitive procedures can inform patients about their possible tendencies to make premature judgments and practice modified responses to their perceptions. Presumably, the secondary cognitive and emotional processing of gestalt excess perceptions, in the form of fear, paranoid thinking, or avoidance, may be more suitable targets for intervention than the gestalt excess per se. Therapy for gestalt deficits could be accomplished through practice-oriented interventions. In applying pattern recognition exercises (eg, using abstract visual patterns and materials from social perception), the subgroup of schizophrenic patients with a gestalt deficit must generally be supported in expecting, and searching, causal structures and meaning.

It is unclear so far which of these low-level cognitive processes and deficits can be modified by cognitive remediation therapy. The complexity of the issue underlines that therapy process research is required as an extension to efficacy trials in cognitive remediation approaches.

Acknowledgments

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