Backward Masking Performance in Hypothetically Psychosis-Prone Subjects

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Backward masking performance was evaluated in three groups defined by scores on scales of schizotypic signs. Normal control subjects (n = 12) and subjects scoring high on the Perceptual Aberration-Magical Ideation scales (n = 10) were predicted to show greater interference from a cognitive mask compared to a pattern mask, while Physical Anhedonics (n = 12) were predicted not to show this differential response. The predicted differences were not found. Possible reasons for the lack of a significant group X mask type interaction and the theoretical significance of this finding are discussed.

During the past decade, much attention has been focused on the hypothesis of a dual-syndrome construct of schizophrenia, most well known as the postive-negative symptom distinction hypothesized by Crow (eg., Crow et al., 1982). The positive-negative terminology can be thought of as the presence of deviant functioning vs. an absence or deficit of normal functioning. In this sense, "positive" symptoms are defined as hallucinations, delusions, bizarre behavior, and formal thought disorder; whereas "negative" symptoms are defined as alogia (poverty of speech production and content), avolition, affective flattening, social withdrawal, and anhedonia (or the lack of a subjective capacity for pleasureful experience) (Andreasen, 1982).

The inference that there might be distinct etiological processes underlying these phenotypic symptom categories was originally founded on the clinical evidence among schizophrenic patients of an association between poor vs. good premorbid histories and the subsequent development of corresponding symptom patterns and outcomes. The use of the good vs. poor premorbid heuristic for determining clinical prognosis was fairly well-accepted by the time Crow proposed a connection with newer neurophysiological data that seemed to be consistent with this diagnostic schema. Thus, these data provided additional support for a dual syndrome construct of schizophrenia.

With the advent of neuroleptic treatment, we now had research that indicated a better response of positive vs. negative symptoms to neuroleptics (e.g., Johnstone et al., 1978). We also had more sophisticated technologies that allowed for the detection of structural abnormalities in the brain, which appeared to have specificity to negative-symptom schizophrenics (e.g., Andreasen et al., 1982). Finally, neuropsychological testing revealed an association between generalized cognitive impairment and negative symptoms (Andreasen, 1982; Opler et al., 1984). Together, these data seemed to imply a mediation of distinct organic processes, in the expression of corresponding behavioral syndromes: For postive-symptom schizophrenics, an underlying neurochemical imbalance seemed to be implicated by the responsiveness of psychotic symptoms to dopamine-reducing neuroleptics; whereas for negative-symptom schizophrenics, the focus was on structural abnormalities in the brain, that were presumed to account for observed cognitive deficits.

This new support for the conceptualization of schizophrenia as a heterogenous disorder led researchers to devise experiments that could help us to understand the exact nature of the deficits that might exist behind varying symptom pictures. To this end, researchers looked to cognitive psychology, which by this time also had newer technologies, and had developed increasingly more sophisticated experimental paradigms that allowed for the isolation of very specific information-processing functions in normal subjects. It made sense that the comparison of performances obtained by normals and by schizophrenics on specialized cognitive tasks, might serve to clarify the nature of the cognitive deficit presumed to be underlying the negative symptom syndrome.

For example, a visual-processing task called backward-masking has been useful in differentiating poor pre-morbids from other schizophrenic and control subjects by their performance deficit on this task. Yet the reason for this deficit remains unclear. Early researchers had hypothesized a deficit in early visual processing called the iconic memory store. However, subsequent researchers were able to demonstrate the apparent adequacy of this function among schizophrenics, by obtaining estimates of iconic capacity and duration equivalent to that of normal controls (Knight et al., 1978).

A more recently revised construct (Phillips, 1974) of early visual information processing provides a possible clue to the reason for such contradictory findings. New evidence suggests that the initial stage of visual input previously referred to as "iconic memory" can actually be divided into two distinct stages with different durations and processing functions.

Stage 1, which Phillips (1974) calls the sensory store, dacays rapidly within the first 100 ms. after exposure to the visual stimulus. In normal subjects, it is hypothesized that during this stage, perceptual analysis of a visual stimulus begins with a passive, automatic, global structuring or organizing of the visual field in which meaningful elements are distinguished from non-meaningful elements and a "decision" is made accordingly, whether or not to allocate additional processing.

Stage 2, which Phillips calls "short-term visual memory" (STVM), decays more slowly and is efficient up to the first 600 ms. following stimulus exposure. During this stage, visual processing procedes to an active, more detailed analysis of meaningful elements in the visual stimulus field, and it appears to provide a brief working span in which the consolidation of meaning for subsequent retention takes place (Potter, 1975, 1976).

The distinction between these stages was critical for Ray Knight's (1985) re-interpretation of the backward masking performance deficit among poor pre-morbid schizophrenics. Briefly, this task requires the subject to correctly identify a target stimulus, which is followed, after a short delay, by a second stimulus, called a "mask". The type of mask typically used consists of a visually patterned, but cognitively meaningless stimulus, hence the term "pattern mask". Poor pre-morbid schizophrenics have shown increasing performance decrements on this task when a pattern mask is presented at delays of greater than 100 ms. (Sacuzzo & Braff, 1981; Sacuzzo et al., 1974), which locates the performance deficit within Stage 2 processing.

Given the timing of the poor premorbids' susceptibility to interference of the mask, Knight thought that it seemed reasonable to assume that the hypothesized perceptual deficit might somehow be associated with their short-term visual memory.

Despite this assumption, Knight speculated that a deficit in either of the two stages of early visual processing could account for the vulnerability of poor premorbids to the interference of a pattern mask at longer delays: A Stage 1 deficit could mean that poor premorbid schizophrenics were possibly deficient in recognizing the non-meaning of the pattern stimulus, and subsequently were allocating unnecessary additional processing to it during Stage 2. That is, the poor pre-morbids' treatment of the pattern masking stimulus as though it had cognitive meaning in Stage 1, would interfere with their consolidation of the target stimulus in Stage 2, by requiring the processing of new meaningful information.

A stage 2 deficit was also a likely candidate, but it was less clear what this might be. Knight hypothesized that poor premorbids might possibly have an unstable STVM that was more easily disrupted than that of normal subjects.

To test these hypotheses, Knight et al. (1985) devised a backward masking experiment which incorporated the Stage 1/Stage 2 visual processing paradigm, by varying the content of the masking stimulus in terms of cognitive meaningfulness. An interaction effect of group by masking condition was predicted as follows: A pattern mask would not be expected to interfere with processing of the target by normal and good premorbid control subjects, because they would automatically recognize it as conceptually meaningless, and no further effort would be "wasted" in processing the mask. Only a cognitive mask would be expected to cause interference with processing of the target for these subjects, because it would be recognized as such, requiring additional processing that would result in an overload, and consequently, fewer accurate target identifications.

By contrast, the performance of poor-premorbid subjects would be expected to be equivalent across both pattern and cognitive masking conditions. A third, noise-mask condition would not be expected to interfere significantly with any subject's performance, thereby serving as a demonstration of baseline competency for all subject groups. Their data did produce this expected pattern of results.

That is, sensitivity to the pattern and cognitive masking conditions was equivalent only for the poor premorbid schizophrenics, suggesting perhaps that they were unable to differentiate a cognitively meaningful from a cognitively non-meaningful stimulus. Unfortunately, these data do not exclusively confirm a Stage 1 perceptual organization deficit while ruling out the possibility of a Stage 2 consolidation deficit. Still, Knight felt the argument was more compelling for the former interpretation, as other notable investigations had found what appeared to be a similar deficit among poor premorbid schizophrenics (Place & Gilmore, 1980; Cox & Levanthal, 1978).

The current study seeks to further test Knight's hypothesis by attempting to reproduce the same pattern of results among hypothetically schizotypic college students. Meehl (1962) proposed an early diathesis/stress model which hypothesized that a genetic factor called schizotaxia predisposed some individuals to develop a personality organization called schizotypy. Some, but not all of these individuals could be expected to eventually decompensate into schizophrenia. Thus, according to Meehl, these individuals represent the early part of the schizophrenia spectrum, and as such, are identifiable by a series of stable personality characteristics, or "signs".

Susceptibility to backward masking has long been thought to be a possible trait marker of schizophrenia, since a backward masking performance deficit has been exhibited across the entire schizophrenia spectrum--by both active and remitted schizophrenics, as well as by schizotypes (e.g., Balough & Merritt, 1985; Braff & Sacuzzo, 1981,1982; Knight et al., 1985; Knight, Scherer, & Shapiro, 1977; Merritt & Balough, 1984,1986; Sacuzzo & Braff, 1981; Schwartz, Winstead, & Adinoff, 1983; Steronko &Woods, 1978). However, earlier studies with schizotypes had not differentiated between subtypes of schizotypy. We wanted to go one additional step to test the hypothesis that a perceptual deficit, such as the one suggested by Knight, might function as a trait marker for a particular subtype of schizophrenia.

Self-report measures have been developed by the Chapmans and their associates for many of the schizotypic signs described by Meehl. Propper et al. (1987) found that the Chapman schizotypy measures seem to cluster into groups comparable to the poor premorbid/good premorbid distinction found in schizophrenic patients. The Physical Anhedonia Scale is used to assess personality characteristics that may be thought of as part of the "negative" schizophrenic syndrome, and the Perceptual Aberration and Magical Ideation Scales assess personality traits that may be thought of as part of the "positive" schizophrenic syndrome.

Thus, it was predicted that subjects meeting the criteria for Physical Anhedonia would perform similarly to poor premorbid schizophrenics, and that subjects meeting the criteria for Perceptual Aberration and Magical Ideation would perform similarly to good premorbid schizophrenics on a backward masking task patterned after Knight et al.

Method

Subjects. Experimental subjects were college students selected on the basis of their scores on three measures of schizotypy: Anhedonic subjects (n=12) scored at least two standard deviations above the mean on the Physical Anhedonia Scale (Chapman, Chapman, & Raulin, 1976); Per/mag subjects (n=10) scored at least two standard deviations above the mean on either the Perceptual Aberration Scale (Chapman, Chapman, & Raulin 1978) or the Magical Ideatuion Scale (Eckblad & Chapman, 1983); as these two scales are routinely combined or interchanged for research purposes due to a high interscale correlation. Control subjects (n=12) scored no more than .5 standard deviations above the mean on all three of the schizotypy scales.

Design and Procedure. A 2 X 3 X 3 design (sex by group by mask type) was used. The within-subjects variable of mask type had three levels: a noise mask (dot or line matrices), a pattern mask (jigsaw puzzle pieces), and a cognitive mask (recognizable objects). All masking stimuli varied within the same size range. After being tested for visual acuity, the subject was seated in front of a three-field tachistoscope. The subject's task was to identify single target letters (A,B,C,D,E,G,H,K,P,R). At each presentation, the target letters were followed by one of the three types of masking stimuli described above. The length of time that the target letter appeared on the screen was set at the subject's critical stimulus duration (CSD), which was determined during a practice period before the experiment. Prior determination of CSD served as an important control for individual differences in perceptual thresholds that might confound performance. The interval between onset of the target and onset of the mask, or stimulus onset asynchrony (SOA) was set at 50 ms. (Although we had originally planned to fix our SOA at an interval greater than 100 ms., piloting proved it necessary to shorten this interval in order to produce sufficient masking effects). Individual variations in CSD were then factored into this fixed interval. All masking stimuli were presented for 150 ms. The three masking conditions were counterbalanced within subjects. Three accuracy scores were computed for each subject--one for each of the three masking conditions. All subjects were tested by a researcher who was blind to the subject's schizotypy scores.

Results

No significant differences between groups were found on critical stimulus duration times. The results of the performance on the masking task are summarized in Figure 1. Mean correct target identifications are broken down here as a function of group and mask-type. A significant main effect of mask type was found, $F(2,56) = 117.07$, $p < .001$. However, only the noise vs. pattern mask comparison reached significance ($F(1,28) = 197,79$, $p < .05$). The comparison of pattern vs. cognitive mask effects was non-significant. Therefore we were unable to draw conclusions about the main hypothesis of interest with the planned comparison of pattern vs. cognitive mask effects across subject groups.

In essence, we are left with a traditional backward masking experimental design, and so must restrict our focus to the absence of a significant difference in pattern mask effects between groups.

Although the sample size was small, there was still reasonable power to detect group differences if they existed, and our sample size was comparable to that of an earlier study in which significant differences were obtained for schizotypes vs. controls in pattern mask vulnerability (Balogh & Merritt, 1985).

Of interest is a significant main effect of sex ($F(1,28) = 4.45$, $p < .05$) and a significant sex by group interaction ($F(2,28) = 3.55$, $p < .05$), neither of which had been predicted. That is, females did better than males in overall performance and although post-hoc comparisons within groups were all non-significant, examination of cell means indicates that sex differences are evident within both controls and anhedonic schizotypes.

Discussion

This study did not corroborate the results of Knight et al. in that no group by masking condition interaction was found. One difference between this study and the Knight et. al study was that we used a fixed stimulus onset asynchrony (SOA) interval, whereas Knight et. al included SOA as another variable in the design. The group differences in their study were more pronounced at certain SOA's. Also, they used pictures as targets, and all stimuli had the three-dimensional quality of color photographs, whereas our stimuli were two-dimensional, black and white graphics. Furthermore, our procedure required subjects to identify each target immediately after presentation, whereas the procedure Knight employed involved the presentation of an entire series of targets, after which subjects were then required to pick out the targets they had seen from a sample pool of stimuli. Their task therefore may have involved more active visual encoding and memorization processes than ours did.

However, more recent findings by Silverstein et al. (1989), suggest another possible interpretation. They found that the hypothesized perceptual organization deficits in the early stage of visual processing for anhedonics were not there, in spite of the fact that previous studies had found deficits in several aspects of visual processing among anhedonics that were similar to those found in poor premorbid schizophrenics. They suggested that these early-stage deficits may not be the markers of risk previously hypothesized, and perhaps are more "state" than "trait".

Alternatively, the deficits may be present among true schizotypes, but there may be a problem in equating anhedonia with schizotypy. The question is whether anhedonia is specific to schizophrenia. In fact, it is likely that subject samples screened for anhedonia are quite heterogenous. Current longitudinal data on the Chapman scales indicate that the scales do not identify a predisposition to schizophrenia per se, but rather, a more general psychosis-proneness (Chapman & Chapman, 1985).

Taken together with the mixed findings of perceptual deficits among schizotypes, these data raise the possibility that anhedonic schizotypy samples are differentially composed of people who have and don't have a perceptual deficit, and those who will or won't later decompensate, along with "nuisance" subjects who are at risk for other disorders. Perhaps it would be worthwhile to gather longitudinal data to see whether schizotypes who do appear to have this perceptual deficit, and are anhedonic, later decompensate to negative symptom schizophrenia.

In this sense, it is possible that anhedonia may not qualify as one of the schizotypal signs that Meehl hypothesized. According to the modifying influence model of Pogue-Geile and Harrow (1985, as cited by Silverstein et al. 1989), negative symptoms result from an interaction between an independent trait and a more specific schizophrenic diathesis. In this view, the independent trait does not directly increase risk for developing schizophrenia, but does influence the development of the schizophrenic phenotype. In the case of anhedonia, it is possible that anhedonia does not in itself increase the risk for schizophrenia, but can shape the clinical manifestation of schizophrenia toward negative symptoms and a poorer prognosis. In this scenario, the cognitive deficit remains viable as part of a diathesis specific to schizophrenia, whereas anhedonia essentially takes on the role of stressor.

With regard to the sex differences, our small sample sizes do not allow us to rule out the possibilty that these are merely statistical artifacts. However, recent data confirm that the Physical Anhedonia scale identifies subjects with different symptom clusters in males and females (Edell & Joy, 1989). Also, the Chapmans were well aware of sex differences when they developed different norms for males and females on their screening scales. Clinical studies have suggested that the experience itself of schizophrenia may be different for men and for women (eg. Allan & Hafner, 1989). Furthermore, epidemiological studies have found sex differences in diagnostic prevalence, age at onset, pre-morbid competence, and chronicity (Seeman, 1985).

Although our data may not provide conclusive evidence, the abundance of data on generalized sex differences in schizophrenia certainly leaves ample room for speculation that backward masking and other cognitive studies of schizophrenia may have ignored an important variable.

Finally, in the past several years, there has been increasing disillusionment with regard to the usefulness of the positive/negative distinction. Despite the clear heuristic appeal, many researchers now agree that subtyping along dichotomous dimensions may not reflect the true complexity of the clinical and neurophysiological spectrum of schizophrenia. Newer research directly contradicts the assumption of two independent syndromes, and suggests that the heuristic elements that comprise the positive/negative distiction are better thought of as interrelated dimensions.

The high-risk paradigm of schizophrenia remains a consistently intriguing area of study, perhaps for its very complexity. Nevertheless, its validation continues to present a formidable challenge to researchers.

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