

Court (1967) criticizes Karras (1967) on several points regarding his test of the hypothesis that the psychological deficit of schizophrenics implicates the data-processing system of the central nervous system. Each of his eight criticisms is reviewed below.

(1) Court believes that the degree of task complexity used by Karras in his one- and two-choice reaction time tasks was insufficient. His claim is based on the finding by Harwood & Naylor (1963) that the predicted divergence (i.e., the appearance of psychological deficit) in schizophrenics occurred only after four or five items were presented to them. When it is recalled that Karras found that chronics were slower than controls on both tasks, it is apparent that the number of stimuli he used was sufficient to demonstrate psychological deficit.

(2) Court states that Yates (1966a), a proponent of abnormally slow data-processing in schizophrenics, distinguishes between the performance of paranoid vs non-paranoid schizophrenics, not between chronics and others, as does Karras. However, apparently not even Yates adheres so rigidly to this requirement. For example, Yates (1966b) cites Shakow (1962) in support of his view; yet Shakow's work focuses on the dichotomy of chronic schizophrenics vs others. Yates (1966b) also cites the findings and formulations of Pishkin, Smith, & Leibowitz (1962) for support; yet their study used undifferentiated chronic schizophrenics and normals. Finally, nowhere in his review article on psychological deficit does Yates (1966b) stipulate that his theory applies only to a paranoid-non-paranoid dichotomy.

(3) Court states that because psychiatric patients in general process data more slowly, the control group used by Karras, which consisted of acute nonschizophrenic patients, was inappropriate. The point is that so long as chronic patients are slower on both tasks than others, regardless of who they are, the data-processing hypothesis necessarily predicts a steeper slope for the chronics. This predicted outcome, and the reason for it, are explicitly stated by Karras on p. 75.

(4) Court says that because the S made a jump response the slowness of the chronics may be due to motor retardation. This explanation becomes a possibility only when Karras' finding that there was no difference in slope between the two diagnostic groups is taken in conjunction with his experimental design. The design held motor retardation constant for both tasks by holding constant the distance the S jumped for either task. Under such a condition motor retardation is an additive constant and a hypothesis based on such a view would predict parallel slopes for the groups rather than the nonparallel slopes of the data-processing formulation. Court's suggestion that King's (1954) technique, viz, measuring lift reaction, is a solution, is open to his own criticism, for in making a lift reaction the S performs a motor response.

(5) Court regrets that Karras used a median based on only nine responses. Presumably a median based on more responses would reduce error and, if so, Karras might have found that the flatter slope of the chronics was significantly different from that of the controls, an outcome still contrary to the data-processing view. Also, Court should have noted that King, whose data he uses in point (7), used only 10 trials to obtain scores on his two-choice reaction time task.

(6) Court states that perhaps significant differences failed to appear because of the possibly large within-group variances for age, education, length of hospitalization, and the number of acutes on medication. Court also states that Karras dismissed the effects of these patient variables without giving scores to support such a conclusion. Although Karras did not present scores, he did state that within each group age, education, and length of hospitalization were not significantly correlated with reaction time (the age and education ranges of both groups were virtually identical). More explicitly, the largest correlation was .13 for the chronics between complex reaction time and education; no partial correlations were performed.

The conclusion that medication was irrelevant for the control group was based on a reported preliminary analysis of variance of scores between controls on drugs and those not on drugs, which showed them to be highly similar (p. 76). More explicitly, no main effect or the interaction was significant at $p < .20$.

Court's suggestion that severity of illness might be an important variable seems a reasonable one now that it has been established that chronics in general do not process visual data more slowly.

(7) Court states that King's study already demonstrated what Karras found. He also says that King concluded that at these levels of complexity "schizophrenics do not decline in performance more rapidly than normals." Actually King's data are opposite to Karras', for the chronics' slope is much steeper than the normals or subacutes he also tested. King concluded that the *ratio* between the simple and complex reaction time tasks does not differ among chronics, normals, and subacutes. If this is true (no test of the significance of the interaction is reported), then the slopes are *not* parallel. For example, the difference between the simple and complex jump was .27 sec for chronics, but around .13 sec for normals or subacutes. The ratios for the three groups range from .76 to .79. Analogous findings are reported for the lift reaction.

It is not difficult to understand why King's data differ from Karras' because his experimental design differs in several important ways. Another sense modality was used. Subjects in King's study responded to auditory stimuli, while Karras used visual stimuli. It is in the former modality, though, that schizophrenics most often have been found to have difficulty.

Two other procedures in King's study, however,

make it difficult to believe that his study was a fair test of the data-processing hypothesis. First, the test trials of the reaction time task were separated from the practice trials by 20 min, during which time two other motor tasks were introduced, which may have induced proactive inhibition. Second, prior to the two-choice reaction time task, the S performed the one-choice task by responding to each of the auditory stimuli for five consecutive trials before switching to the other stimulus for five trials. The alternation occurred twice before the S performed the complex task. Such a procedure offers another source of proactive inhibition, for it requires the S to overcome the earlier tendency to respond to predictable alternating sequences of stimuli when he has to perform the random two-choice task. The chronic schizophrenic should have greater difficulty than others in overcoming these two kinds of interfering sets operating simultaneously, as expected from the Zahn, Rosenthal, & Shakow (1961) study which tested this possibility directly. Karras had task order reversed for one-half of each of his groups as a check on this possibility.

One last comment on Court's use of King's data: Court notes the similarity of the scores reported by King and Karras; this comparison is irrelevant because King's data are in raw scores while Karras' are in logs. (In a footnote Karras reported an analysis of

variance with raw scores which failed to find a steeper slope for chronics.)

(8) Court notes that Karras was shy a reference, for which I apologize.

In conclusion, Karras' study showed that the psychological deficit of chronic schizophrenics cannot always be explained by assuming that they process data more slowly than others.

References

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Note

1. Now at Hillside Hospital, Glen Oaks, N. Y.