Cognitive Rehabilitation for Schizophrenia: Is It Possible? Is It Necessary?

by Alan S. Bellack

Abstract

Limitations of available psychosocial interventions combined with the increasing evidence that schizophrenia is characterized by diverse deficits in information processing has stimulated great interest in the possibility of cognitive rehabilitation. However, the current optimism seems unjustified. The precise role of information processing in the behavioral handicaps evidenced by schizophrenic patients is not clear, and the neuropsychologic and experimental psychopathology tasks used to assess information processing generally cannot specify precisely which cognitive functions are deficient. Thus, the choice of cognitive targets for rehabilitation is arbitrary. The strategies currently employed for rehabilitation emphasize an exercise model of treatment and the use of complex mnemonics. Neither approach has been successful in rehabilitating brain-injured patients, and preliminary results with schizophrenic patients are not very promising. It is concluded that the field might be better served by focusing on environmental change and compensatory strategies until we determine how and why schizophrenic patients fail.

The articles by Brenner and colleagues (1992, this issue), Liberman and Green (1992, this issue), and Spring and Ravdin (1992, this issue) are thought provoking and timely. Interest in psychosocial interventions for schizophrenic patients reached a nadir in the 1970’s as the shortcomings of traditional group and individual psychotherapy approaches became increasingly apparent (Mosher and Keith 1980). There were scattered reports of effective behavior therapy programs (Meichenbaum and Cameron 1973; Paul and Lentz 1977), but they were generally regarded as anomalies. The pessimistic outlook has now begun to change, because a series of well-controlled studies has demonstrated that family education (Goldstein 1984), behavioral family therapy (Falloon et al. 1982), and social skills training (Morrison and Bellack 1984) programs could have a demonstrable effect in reducing relapse and symptomatology. Enthusiasm for psychosocial interventions has recently increased further with the demonstration that clozapine may allow a large number of previously intractable patients to respond to rehabilitation programs.

While available psychosocial interventions clearly have beneficial effects, they are far from a panacea. The family interventions are designed primarily to reduce relapse and symptomatology by decreasing environmental stress (e.g., expressed emotion in the home). They do not directly “treat” the patient, and they have little impact on psychosocial functioning. Social skills training is designed primarily to improve psychosocial functioning. While the technology is highly effective in teaching new skills, the impact of the training on role functioning in the community is uncertain. Consequently, it is clear that new and more effective techniques are needed.

The developments in psychosocial treatment have been paralleled by equally exciting advances in neurobiology. New imaging technologies and neuropsychological research...
methods have demonstrated that schizophrenia results from some form of brain dysfunction that is acquired or inherited or both (Weinberger 1987; Crow et al. 1989). While the precise nature and locus of this dysfunction are subject to considerable debate, there is consensus that it results in a functional disruption of information processing. Schizophrenic patients have been shown to have significant deficits in a wide range of cognitive processes, including memory, attention, reasoning ability, and language (Nuechterlein and Dawson 1984; Braff and Geyer 1990). As with the debate about the nature of the underlying brain dysfunction, there is also considerable disagreement about the core cognitive impairment that underlies the diverse information-processing deficits. Nevertheless, as indicated by Brenner and colleagues (1992, this issue), Liberman and Green (1992, this issue), and Spring and Ravdin (1992, this issue), it is now widely assumed that these specific deficits are responsible, at least in part, for the profound disruptions in social behavior and role functioning that categorize the illness. This assumption has revitalized the search for cognitive rehabilitation strategies.

Cognitive rehabilitation approaches are characterized to varying degrees by several common assumptions, which are reflected in Brenner and colleagues (1992, this issue), Liberman and Green (1992, this issue), and Spring and Ravdin (1992, this issue): (1) Cognitive impairments play a central role in the social disability and other problems schizophrenic patients experience in daily living. (2) These impairments must be rectified if we are to achieve effective rehabilitation. (3) The prognosis for cognitive rehabilitation, as reflected in preliminary studies, is quite positive. While each of these assumptions has considerable face validity and may ultimately prove to be true, current knowledge provides scant support for any of them and a more conservative view seems justified. Limitations of each of these assumptions will be considered in the following pages. The discussion will focus on social behavior and social skill, which has been the predominant concern of cognitive rehabilitation programs.

The Role of Cognitive Impairment in Social Disability

There is no question that schizophrenic patients have marked deficits in social skill (Bellack et al. 1990) and information processing. It is also clear that information processing plays a key role in social behavior. Cognitive demands in social encounters include face and affect recognition, recall of past interactions, decision-making and judgment in conflictual interactions, and the use of language. By implication, it seems logical to conclude that the impairments in social skill are, at least partially, a consequence of the cognitive deficits. There are ample data to support this conclusion. Cornblatt et al. (in press) have found that attentional dysfunctions in childhood predict social deficits in adults at risk for schizophrenia. Ohman and colleagues (1989) reported that deficient electrodermal orienting response was associated with poor social functioning. Similarly, Saccuzzo and Braff (1981) showed that poor premorbid schizophrenic patients displayed persistent vulnerability to visual masking stimuli compared with good premorbid patients and controls.

These data suggest that there is a robust relationship between cognitive impairment and social dysfunction. However, the data are primarily correlational and do not demonstrate a causal relationship. They may reflect a common diathesis, such as neuroleptic side effects or anergia. The results also are based on very general measures of social role functioning (e.g., premorbid social competence). Such measures are better markers of symptomatology and neurological anomalies (e.g., enlarged ventricles) than of social competence per se. In any case, little is known about precisely which information-processing deficits compromise social behavior in schizophrenia or the amount of variance accounted for by cognitive deficits.

The adequacy of social performance is influenced by a number of factors, including social skill, social perception, and motivation to interact (see table 1). In some cases, such as negative symptoms, medication side effects, and social anxiety, the role of information processing is indirect at best. In other cases, such as social perception and social problem solving, cognitive processes appear to play an important role. But even when the operation of cognitive parameters can be inferred, there are few data to document an important role for any specific cognitive process. For example, simple response skills such as greetings and social reinforcers are relatively automatic and probably require little more than procedural memory. Conversely, social problem solving undoubtedly requires higher level reasoning, episodic and semantic memory, sustained attention, and high processing capacity, but the interaction and relative contribution of these diverse elements are not known even in normal populations (Bellack et al. 1989).

Social perception also presents a difficult case for analysis. One im-
Table 1. Factors affecting social competence

<table>
<thead>
<tr>
<th>Primary influences</th>
<th>Possible consequences</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social skill</td>
<td>Gaze avoidance</td>
</tr>
<tr>
<td>Social perception</td>
<td>Failure to recognize criticism and hostility</td>
</tr>
<tr>
<td>Social problem-solving ability</td>
<td>Inability to negotiate or compromise</td>
</tr>
<tr>
<td>Affect</td>
<td>Avoidance due to social anxiety</td>
</tr>
<tr>
<td>Negative symptoms</td>
<td>Paucity of speech or gestures</td>
</tr>
<tr>
<td>Positive symptoms</td>
<td>Disruptive conversations due to derailing</td>
</tr>
<tr>
<td>Medication side effects</td>
<td>Akinesia, akathisia</td>
</tr>
<tr>
<td>Motivation</td>
<td>Loss of interest due to repeated failure</td>
</tr>
</tbody>
</table>

important type of social perception—
affect recognition—is a fundamental human capability that appears early in life and is consistent across cultures (Dimberg 1988; Ekman 1989). It may be a phylogenetically determined capacity that depends on specific brain structures (Frith and Frith 1986; Chelune 1987). As such, it might require little high-level cognitive processing. A number of studies have demonstrated that schizophrenic patients have deficits in affect recognition, particularly for negative affect displays. In two studies from my laboratory, schizophrenic patients consistently underestimated the intensity of negative affect expressed by others, although they still rated these displays as more negative than positive affects (Morrison et al. 1988; Bellack et al., in press). These findings are consistent with several different hypotheses about the underlying impairment. There could be a lesion in neural structures critical for the perception of negative affect, resulting in aprosodia. The problem could also result from reduced processing capacity. Negative affect entails a more complex array of stimuli than positive affect (Ekman et al. 1972). It is possible that schizophrenic patients cannot process all of the relevant cues because of reduced processing speed or inadequate short-term memory. Or, they may have learned to modulate stress by damping or inhibiting their perception of negative affect cues, or they may simply not know the meaning of critical cues or combinations of cues (e.g., the difference between sarcasm and facetiousness).

Until recently, the literature on information processing in schizophrenia and on social competence in schizophrenia have followed divergent paths. It has been reliably demonstrated that schizophrenic patients have diverse information-processing deficits, but the functional significance of those deficits in relation to the demands imposed by daily life has not been studied. For example, what does a 100-ms deficit in ability to avoid the effects of a stimulus mask (viz. backward masking protocols) tell us about a patient's ability to process the rapid flow of interpersonal cues in a conversation? How do perseverative errors on the Wisconsin Card Sorting Test (WCST; Heaton 1981) relate to problem solving in the natural environment? How does decreased ability to sustain attention to degraded computer images relate to one's ability to sustain attention in a heated conversation? Schizophrenic patients might exhibit statistically significant deficits on these tests compared with nonpatients, but that does not demonstrate thereby that the deficits are of significant magnitude or relevance to impede social performance. Writing from a different perspective, Ekman (1989) argues that it might not be necessary for people to perceive the full panoply of facial, vocal, and gestural affect cues to make accurate judgments about a partner's mood state. If valid, this hypothesis raises questions about the functional significance of the affect recognition deficits found in schizophrenia. Patients might miss subtle differences in mood but still be able to detect the most salient affect states.

It should be noted that schizophrenic patients are not decorticated. They can learn and perform the cognitively complex tasks of reading and arithmetic. They can find their way around the hospital and neighborhood; they remember names, faces, and events from one day to the next; their conversations sometimes are sprinkled with logical errors, but they are substantially understandable and they can detect when other people are being illogical (Harlow and Miller 1980). They can solve many problems in daily living, including how to borrow or steal cigarettes and money for alcohol and street drugs, and they know how to lie to avoid criticism (Bellack et al., in press). They can even remember which neuropsychologic tests they dislike from one testing session to the next and exhibit selective noncompliance. They may not be able to perform consistently the social and instrumental tasks needed to sustain themselves in the community, but their information-processing system is substantially more intact than impaired.

In light of this confusing picture, it is not clear which cognitive process or processes should be targeted for rehabilitation. Should we focus on response skills, social perception, or
Is Cognitive Rehabilitation a Viable Goal?

The Focus of Rehabilitation Programs. The previous section suggested that cognitive rehabilitation programs might be premature because we do not know the relationship between specific social disabilities and specific cognitive impairments. Given current knowledge about information processing and the functioning of the brain, it is not at all clear that the rehabilitation strategies now being investigated would be viable even if we knew what to target.

The creative program developed by Brenner and colleagues (1990) is substantially responsible for rekindling interest in cognitive rehabilitation and is representative of the general approach. It emphasizes two generic rehabilitation strategies: strengthening or restoration of function by practice, and the use of mnemonics to guide behavior. Patients are first given training on information-processing tasks that parallel the neuropsychologic tests and experimental paradigms used to evaluate cognitive functioning. Training then targets successively more complex aspects of social perception and social skill, culminating in a social problem-solving module similar to that developed by Liberman and Wallace (Wallace et al. 1985; Liberman et al. 1986). The problem-solving component entails use of a complex mnemonic to guide behavior. I have previously discussed the limitations of this strategy (Bel-lack et al. 1989). Most notably, it is based on a model of problem solving that is not well suited to the interpersonal problems confronting people (including patients) in daily life, and there is little evidence that it can be employed effectively by schizophrenic patients. The focus of the present discussion is the more direct training in cognitive operations, which presents the critical question for the current debate: Can fundamental information-processing systems be enhanced with training?

From a scientific perspective, one cannot prove the null hypothesis; thus, the conservative answer to that question is maybe. But, there are two fundamental problems with existing programs that limit the likelihood of success. As has been indicated, the information-processing demands of social behavior have not been determined. Similarly, the mechanisms underlying neuropsychologic test performance are far from clear. For example, schizophrenic patients exhibit marked deficits on the WCST (Goldberg et al. 1987). The WCST purportedly measures reasoning ability and has been linked to frontal lobe dysfunction, but it also taps short- and long-term memory, distractibility, sustained attention, and learning ability (Heaton 1981; Goldberg and Seidman, in press). Backward masking is another reliable marker of cognitive deficit in schizophrenia (Saccuzzo and Braff 1986). It has been variously thought to tap diverse cognitive processes, including iconic representation, short-term memory, processing speed, information transfer, and cognitive capacity (Schuck and Lee 1989). Most other neuropsychologic tests and experimental psychopathology tasks also tap multiple cognitive domains. In the next decade, these measures may play a seminal role in unraveling the mystery of schizophrenia, and they may also be useful as dependent measures for evaluating the outcome of cognitive rehabilitation programs. However, they do not have enough specificity to serve as the basis of these programs (Wilson 1989).

In light of these limitations, the choice of which cognitive process to target in existing programs is relatively arbitrary. Moreover, as now formulated, the rehabilitation approach serves to reify hypothetical constructs, such as "processing capacity," by using the same measures for training and evaluation of outcome. Patients are taught to perform better on tests or computer games that purportedly tap underlying cognitive processes, and improved test performance is interpreted to reflect improvement in the underlying cognitive parameter. In no case is there an independent assessment of the cognitive dimension or of the impact of improved test performance on other aspects of functioning. Without such convergent evidence, it is difficult to conclude that improved performance reflects anything other than practice effects on parallel tasks.
Rehabilitation Strategies. The discussion thus far has addressed primarily the focus of current rehabilitation efforts. More fundamental questions are raised about the conceptual underpinnings of these approaches. There is an implication that brain function can be improved by exercise in a manner analogous to rehabilitation of muscles after a bone is broken. This conception is not consistent with current knowledge about the brain, and it has not proved useful in the rehabilitation of brain-injured patients (Schacter and Glisky 1986). There is a good deal of cerebral plasticity early in life, but there is scant evidence for tissue regeneration or transfer of functions across neural structures in adulthood (Schacter and Glisky 1986; O'Connor and Cermak 1987). Recent traumatic brain damage in adults is often associated with partial recovery, but the evidence suggests that the neurologic impairment associated with schizophrenia develops or is acquired early in life (Weinberger 1987; Cornblatt et al., in press).

Practice on cognitive tasks can improve performance on that specific task, but there is little evidence for the generalizability of such training. For example, chess masters can very quickly recognize the configuration of the entire chessboard because of their experience with characteristic relationships between pieces. But they do not have greater recall or more rapid visual recognition capacity when the pieces are arranged in random groupings rather than in typical game configurations (Squire 1987). Another example of this phenomenon comes from a widely cited case study in the neuropsychologic literature (Schacter and Glisky 1986). A college student was able to increase his digit span to 80 digits by use of a special mnemonic strategy, but his span for letters remained at 7. The student, like the chess masters, improved his performance on one task by extensive practice and use of a very specific strategy, but he did not enhance the underlying cognitive capacity. Moreover, the strategy did not spontaneously generalize to other tasks, even when they were structurally very similar. These anecdotal observations are consistent with the results of controlled studies on patients with brain injury (O'Connor and Cermak 1987; Benedict 1989) and schizophrenia (Bentall et al. 1987). In general, there is minimal empirical or conceptual support for the exercise model of cognitive rehabilitation or for the restoration of lost functions.

Conclusion

The critique that has been presented would have little currency if the data provided strong support for the clinical utility of existing programs. Unfortunately, that is not the case. Brenner and colleagues (1990) have been able to produce modest improvements in performance on a few measures, but their program has not yielded significant gains on most of the tasks employed in training or on more applied measures. The evidence in support of the Liberman and Wallace (Wallace et al., in press) program is also modest at best. I am aware of no data that document that patients can learn the problem-solving strategy or apply it in the community, or that the gains in community functioning associated with the overall program are tied to improved problem-solving skill. In a seminal study, Meichenbaum and Cameron (1973) reported that schizophrenic patients could be taught to use a self-verbalization strategy to decrease distractibility. This study has often been touted as a model for cognitive therapy with schizophrenic patients, but it has not been replicated. Patients can be taught to use a self-instructional strategy in training situations and when prompted, but they exhibit minimal generalization (Bentall et al. 1987).

Is cognitive rehabilitation for schizophrenic patients an achievable goal? If by rehabilitation one means restoration of function or modification of fundamental cognitive processes, or both, the answer for the immediate future is probably not. If on the other hand, rehabilitation is viewed from the perspective of alleviation of handicaps and improved functional capabilities, the answer is an emphatic yes! The literature on rehabilitation of brain-injured patients is replete with successful interventions. However, improved performance characteristically involves modification of the environment and the use of compensatory strategies rather than repair or replacement of damaged tissue (Schacter and Glisky 1986; O'Connor and Cermak 1987).

This issue is clearly underscored by Wilson (1989, p. 127), discussing cognitive rehabilitation of neurologic patients.

Given the major learning and generalization difficulties experienced by our patients, perhaps we should avoid creating a stage which requires transferring skills in a limited clinical context to situations that occur in the “real world.” Would it not be better to start on the real life skills themselves? This is not to say that computers should be abandoned for assessment and research purposes, nor is it the case that training programs cannot be devised which avoid the difficulties described above. It is nevertheless difficult to find evidence supporting the efficacy of exercise, practice, or stimulation, when these are the only strategies involved in a rehabili-
tation programme. This holds true for computer training packages as well as for cognitive rehabilitation programmes in general.

The task for schizophrenia researchers is also to develop real world training programs. For the most part, the emphasis should be on modification of the environment and on compensatory strategies that place limited demands on those aspects of the information-processing system that are compromised. Family interventions that decrease stress are an example of the former. Overlearning of social skills that deflect criticism and hostility is an elementary example of the latter. However, as previously indicated, current approaches are limited by lack of knowledge about the precise factors that impede the social and role performance of schizophrenic patients as well as the abilities they retain despite the illness. With the exception of the generic assumption that social performance is vulnerable to heightened stress, we know very little about why schizophrenic patients can perform adequately on some occasions and in some situations but not in others.

A parallel can be drawn from the literature on amnesia. Until recently it was assumed that anterograde amnesia was associated with gross inability to store new information. However, it has now been demonstrated that amnesic patients often develop implicit memories for recent events (Squire 1987). They can exhibit procedural memory or the effects of priming even when they are unable to recall stimuli or events (Schacter and Glisky 1986). The problem appears to be in retrieval, not in encoding or memory per se. This finding has led to the development of new interventions that circumvent the retrieval process. This is not to say that the recall deficits exhibited by schizophrenic patients have a similar basis. Rather, it suggests that better understanding of their strengths and handicaps is required.

I am optimistic that we will be able to develop new and effective interventions in the next decade. I also believe that efforts to understand the underlying cognitive deficits are critical, both for explaining the disorder and for leading to the development of new remediation techniques. However, I think the current efforts to develop cognitive rehabilitation programs are premature and are unlikely to be successful. They may have heuristic value, but they run the risk of promising too much and leading to disappointment among patients and their families.

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The booklet describes "The World of the Schizophrenic Patient" through the use of analogy. It briefly describes what is known about causes—the influence of genetics, environment, and biochemistry. It also discusses common treatment techniques. The booklet closes with a discussion of the prospects for understanding schizophrenia in the coming decade and the outlook for individuals who are now victims of this severe and often chronic mental disorder.

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