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A Trial of Two Cognitive–Behavioural Methods of Treating Drug-Resistant Residual Psychotic Symptoms in Schizophrenic Patients: I. Outcome

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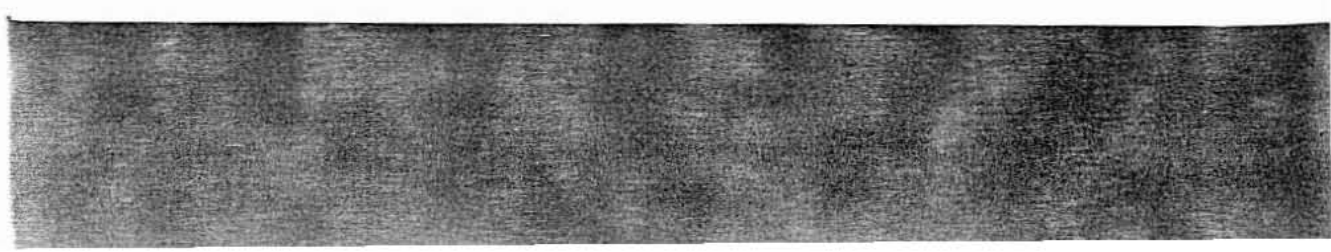
Despite neuroleptic medication, many schizophrenic patients continue to experience residual positive psychotic symptoms. These residual symptoms cause distress and disability. We report a controlled trial of two cognitive–behavioural treatments to alleviate residual hallucinations and delusions. Forty-nine patients were recruited into the trial, of whom 27 entered the trial and completed post-treatment assessment, and 23 were reassessed at six-month follow-up. Patients were randomly allocated to either coping strategy enhancement (CSE) or problem solving (PS). Half the patients were allocated to a high-expectancy positive demand condition and half to a counter-demand condition to evaluate expectation of improvement. Patients receiving either cognitive–behavioural treatment showed significant reductions in pyschotic symptoms compared with those in the waiting period, who showed no improvement. There was some evidence, although equivocal, that patients receiving CSE improved more than those receiving PS. There was no evidence that improvements generalised to negative symptoms or social functioning, nor was there evidence that expectancy of treatment benefit contributed to the treatment effect.

Despite advances in pharmacological treatments for positive schizophrenic symptoms, many sufferers of schizophrenia continue to experience residual psychotic symptoms. Although these symptoms may be less severe than during the acute episode, they do not appear to respond further to medication. For example, in a three-year follow-up study, 47% of patients continued to experience some psychotic symptoms (Harrow & Silverstein, 1977; Silverstein & Harrow, 1978). Similarly, in a seven-year follow-up study, 23% of patients were found to be experiencing florid symptoms (Curson *et al*, 1985). Similar results to these, which were demonstrated in community settings, are found in investigations of hospital populations. Curson *et al* (1988), in a survey of all patients in a London psychiatric hospital, found that nearly half were experiencing either hallucinations or delusions despite long-standing and frequently 'energetic' medication. Besides being extremely distressing in themselves and a frequent cause of anxiety and depression (Brier & Strauss, 1983; Tarrier, 1987), persistent symptoms also contribute significantly to general disabilities and handicaps (Falloon, 1986). There is also a high risk of suicide among patients experiencing persistent symptoms (Falloon & Talbot, 1981).

A number of psychological approaches to alleviating psychotic symptoms have been reported in the literature, for example operant methods such as, social reinforcement (Liberman *et al*, 1973; Bulow *et al*, 1979), time out (Davis *et al*, 1976), and punishment (Weingaertner, 1971; Turner *et al*, 1977; Fonagy & Slade, 1982); assertive training (Nydegger, 1972); exercise (Belcher, 1988); stimulus control (Slade, 1972, 1973); self-instruction (Meichenbaum & Cameron, 1973); belief modification (Watts *et al*, 1973; Milton *et al*, 1978; Hole *et al*, 1979); thought stopping (Lamontagne *et al*, 1983); control of stimulus input (Birchwood, 1986; Morley, 1987); biofeedback (Schneider & Pope, 1982); and self-control (Alford *et al*, 1982). Most of these reports, however, have been of single case studies or uncontrolled trials, so the general efficacy of these methods has still to be demonstrated. Furthermore, many of these studies, especially those consisting of contingency management techniques, have dealt with chronic institutionalised populations and it is unclear whether the symptoms themselves or merely the patient's reports of them have been reduced (for comprehensive reviews of this area see Hemsley, 1986; Heinrichs, 1988; Slade & Bentall, 1988; Tarrier, 1992*a*,*b*).

Some studies have examined whether patients try to cope with their symptoms and, if so, how effective these methods are. Here 'coping' signifies an active attempt or attempts to control, master, or overcome the symptoms or their consequences. Despite differences in methods and definitions, these studies have produced consistent results indicating that symptoms can be precipitated by environmental factors and that schizophrenic patients do use coping strategies to alleviate their symptoms (Falloon &



Talbot, 1981; Brier & Strauss, 1983; Kanas & Barr, 1984; Cohen & Berk, 1985; Tarrier, 1987; Carr, 1988). However, whether these self-initiated coping strategies are effective in alleviating psychotic symptoms is much less clear. For example, Tarrier (1987) found that patients who used coping strategies reported 24% of these strategies ineffective. It was also found that patients who reported that coping was effective were more likely to use multiple strategies. In contrast, Falloon & Talbot (1981) found that patients who were least handicapped by their symptoms used fewer coping strategies but found specific and consistently used methods to be more effective.

A number of studies have also been reported that used a treatment strategy of teaching patients methods which they could use to cope with and hence reduce their symptoms (Fowler & Morley, 1989; Tarrier *et al*, 1990). The method developed by Tarrier and his colleagues (Tarrier *et al*, 1990; Tarrier 1992*b*) has been termed coping strategy enhancement (CSE). CSE attempts to identify coping strategies that may already be used by the patient and to use these as a basis from which to systematically train the patient in a battery of coping techniques. This treatment approach is based on a conceptualisation of psychotic phenomena which explains such phenomena as manifest due to a complex interaction of biological, environmental, and behavioural elements. Two factors are potentially important in increasing the probability of symptom occurrence. Firstly, the presence of environmental cues or precipitators, and secondly, the patient's cognitive, behavioural, or physiological reaction to experiencing hallucinations or delusions. The aim of CSE is to decrease symptoms by training the patient to cope with and control both the cues and reactions to symptoms.

This paper reports on a controlled trial in which CSE is compared with another cognitive-behavioural treatment, problem solving (PS), to test their efficacy in reducing residual psychotic symptoms in schizophrenic patients. Problem solving was selected as a control treatment since it is an established cognitive-behavioural treatment method (D'Zurilla & Goldfried, 1979; Hawton & Kirk, 1989) with applicability to a wide range of problems such as "dealing with handicaps resulting from . . . psychiatric illness" (Hawton & Kirk, 1989, p. 407). PS was considered suitable as it was a credible treatment which may well result in benefits to the patient but would not be expected to directly address psychotic symptoms. Furthermore, individual PS had been used as a control treatment in the trials of family management of schizophrenia (Falloon *et al*, 1984).

It was predicted that CSE should result in a direct improvement in psychotic symptoms which would have a secondary effect of decreasing general psychopathology and increasing social functioning. Problem solving would be predicted to increase functioning but not have a direct action upon positive psychotic symptoms.

Method

Referrals to the project were solicited from mental health workers, principally from injection clinics, consultant psychiatrists, and community psychiatric nurses, working within Salford District Health Authority. Patients were recruited into the study if they fulfilled the following criteria:

(a) they met DSM-III-R criteria for schizophrenia (American Psychiatric Association, 1987)
(b) they had been experiencing psychotic symptoms (i.e. hallucinations or delusions) for at least six months which did not appear to be responding further to medication
(c) there was no evidence of organic pathology which could have explained the psychopathology
(d) they were between the ages of 16 and 65
(e) they were receiving regular and stable neuroleptic medication.

Of the 75 referrals, 49 were considered suitable for the project; the others were not included because of ambiguities in diagnosis or evidence of organic pathology. A further ten patients did not complete the initial assessments: three refused; the wife of one patient refused to allow her husband to participate; two patients were incoherent, one of whom was thought to be suffering from dementia; one patient was admitted to the regional secure unit after being involved in a violent crime; another was very disturbed and was admitted to long-term care; one other was rediagnosed as hypomanic; and it was not possible to elicit psychotic symptoms from another.

Thus 39 patients were recruited into the study; however, a further 12 did not receive treatment: five refused to participate further; four patients were admitted, two to the regional secure unit, and two for severe depression; one patient moved from the area; one patient was transferred to another health authority; and one patient moved residence and was untraceable. Therefore, 27 patients entered the study, completed the treatment and were assessed at follow-up. Of these, 23 were assessed at six-month follow-up: of the four who were not, three refused and one was withdrawn from the study on the request of her consultant.

Details of the patient sample were as follows: mean (s.d.) age 42.77 (12.32) years; mean (s.d.) duration of illness 12.2 (9.21) years; mean (s.d.) number of admissions 4.06 (2.34); mean (s.d.) time since the last admission 3.7 (3.99) years. Twelve (24.5%) lived with a parent or parents, 12 (24.5%) lived with a spouse or cohabitee, 6 (12%) lived in hostel accommodation, 15 (31%) lived alone, and 4 (8%) lived with some other relative. Comparisons between patients who entered the trial and those who did not indicated that those who did enter were significantly younger

(40.92 years v. 50.78 years; $t=2.22$, $P=0.029$), but there were no other significant differences.

Once patients were recruited to the study they were randomly allocated to one of two treatments, either coping strategy enhancement (CSE) or problem solving (PS). Patients were also allocated to psychologists so that there was as equal a balance as possible between the four different therapists (NT, RB, AB, and LY) and the treatments they administered. Initially the procedure was designed so that each psychologist would treat ten patients, five with each treatment. However, patient drop-out and refusal did not allow this to be achieved exactly.

Half of each group were then allocated to a waiting period of six weeks, equivalent in time to the duration between pre- and post-treatment assessments. Patients were then assessed for the first time: those in the waiting-period group were asked to wait for a short period before receiving treatment, while the others entered treatment after receiving their pre-treatment assessment. After the six-week waiting period, this group received the pre-treatment assessment and entered treatment. Half of each treatment group was assigned to high expectancy and half to neutral expectancy. Each treatment programme lasted five weeks, and patients received the post-treatment assessment one week after the end of treatment. Follow-up assessment was six months after the finish of treatment.

Waiting period (WP)

A period of time equivalent to the duration of the treatment programme was used to assess whether patients changed spontaneously or changed because of the reassessment. This period was used as an index of baseline stability. It was not considered clinically desirable to have a no-treatment control group that would be reassessed at six-month follow-up without any clinical input, even though such a design would have been more rigorous.

Coping strategy enhancement (CSE)

The aim of the treatment was to ascertain which environmental factors were maintaining the psychotic symptoms and their emotional consequences at their present level. The programme then aimed to modify these factors and reduce symptoms and accompanying negative emotions. This analysis included the patients' endeavours to cope with their symptoms. The patient was first given this rationale for treatment: it was suggested that patient and therapist engage in a 'collaborative endeavour' to decrease the symptoms and their negative emotional consequences. If the patient did not accept that the experiences were illness-based then it was suggested that although the therapist and patient might hold different causal explanations of the patient's experiences (i.e. illness- v. reality-based) these differing explanations could be put to the test. Furthermore, both would agree that the consequential emotional distress was undesirable and should be tackled. Then a semi-structured interview was used to assess: the symptoms, their antecedents, their consequences, and any coping strategies (described in detail by Tarrier, 1992*b*).

Patients were then taught to monitor their symptoms. They were initially taught to recognise hallucinations and the unusual or bizarre thought content of delusions. The false perceptions of the former were relatively easy to identify as they were, as a rule, discrete and intermittent. Patients experiencing delusions were taught to become aware of 'unusual thoughts' in a manner similar to the way in which depressed and anxious patients are taught to become aware of maladaptive thought patterns as a prelude to cognitive–behaviour therapy. Some patients were able to identify the abnormal thought content of delusions as such; those with little or no insight were asked to attend to emotional and behavioural reactions to specific thoughts identified by the therapist as delusional. Symptoms were targeted for treatment in agreement with the patient on the basis of either:

(a) potential ease of treatment (so that a graded approach to the teaching of coping could be taken) or
(b) when a reduction of a specific symptom was high priority (i.e. a specific symptom was causing considerable distress or was disruptive to the patient's functioning).

The following categories of coping strategies were identified as being potentially usable, alone or in combination, in the intervention:

(a) cognitive strategies: attention switching, attention narrowing, self-instruction
(b) behavioural strategies: increasing solitary activities, increasing social interactions, social disengagement, reality testing
(c) strategies to produce physiological change (such as relaxation, breathing exercises).

Once a symptom had been chosen for intervention an appropriate coping strategy was identified and broken down into component parts. The strategy was then practised under simulated conditions. If the patient experienced symptoms during the session then this situation was used for *in-vivo* practice. Homework exercises of implementing coping strategies between sessions were set, and reviewed in detail at the beginning of each session. If reasonable progress was being made, or if no progress was made on that particular symptom after two to three sessions, then the next symptom was targeted, and so on. Throughout the sessions, progress was reviewed and patients were encouraged to generalise coping skills to other symptoms and situations. During the final sessions, potential ways of resolving difficulties that might occur in the future were rehearsed.

A more detailed account of this treatment method can be found in Tarrier (1992*b*).

Problem solving (PS)

The aim of this treatment was to improve the patient's cognitive functioning by teaching a cognitive plan for problem solving and by encouraging its application. This approach was derived from the original description of problem solving by D'Zurilla & Goldfried (1971).

(a) The patient was initially given the rationale that one of the consequences of schizophrenia was the impairment

of the ability to resolve daily and practical problems due to an inability to think in an organised manner. The aim of the treatment was to improve the individual's ability to approach problems in a systematic way and to implement and evaluate an action plan to resolve their problems.

(b) The patient was invited to list or record situations which were currently causing them difficulty or dissatisfaction. These would be available for detailed analysis later in the programme.

(c) The therapist then explained the notion of the 'stages of problem solving' which were: identify and clearly specify the problem, generate a range of alternative solutions to solve the problem, evaluate the potential utility and outcome for each alternative, choose the most appropriate alternative, implement the chosen problem-solving strategy, evaluate the outcome; if the outcome is not as desired, select and implement an alternative solution for solving the problem, and give self-reinforcement for the use of the problem-solving strategy.

(d) The problem-solving strategy was then applied to an abstract situation such as a simple game like draughts or noughts and crosses. The aim of the game was specified and all potential moves were described and their consequences evaluated: on this basis the best move was selected and implemented. This procedure was carried out aloud for each player and each move. Initially the therapist performed the problem solving and then the patient was encouraged to increase his/her participation.

(e) The problem-solving strategy was then applied to standard 'real life' situations, such as 'getting a job', 'finding somewhere to live', 'making friends'.

(f) Lastly, problems within the patient's own life were targeted for the implementation of problem solving. Difficulties that the patient had experienced which had been previously generated (in Stage b) were now approached, and a detailed analysis of each problem was performed; initially implementation of alternatives was discussed and performed in imagination. Finally, the patients set themselves behavioural targets for implementation, the results of which were carefully monitored. The patient then received feedback from the therapist and was encouraged to provide her/himself with positive self-reinforcement for attempting the exercise. During this procedure the therapist attempted to shape the patient's problem-solving skills towards the desired capability.

Expectancy

In an attempt to examine certain non-specific effects of treatment, expectations of treatment success were manipulated in some subjects. A previous study by one of the authors had found that expectancy effects explained some, but not all, of the improvement in the treatment of generalised anxiety (Tarrier & Main, 1986). The possibility of improvement being the result of a non-specific effect such as the patient's expectation of improvement was examined by this manipulation. During the sessions it was continually emphasised to patients who were allocated to the high-expectancy condition that the treatment approach should have a beneficial and accumulative effect and that improvement would be immediate and proportional to the amount of practice and homework performed. No such explanation was provided for the neutral expectancy group, who were told that benefit would occur but they should not expect it before the post-treatment assessment. This positive demand and counter-demand manipulation attempts to control for any improvement resulting from the patients' expectations of improvement produced solely because they are entering treatment (Steinmark & Borkovec, 1974).

Assessment

Assessment was carried out at: pre-waiting period (where appropriate), pre-treatment, post-treatment, and at six-month follow-up. Assessments were performed by one of the team (SH) who was independent of treatment delivery but not blind to group allocation. The following assessments were used.

Each individual psychotic symptom elicited by the Present State Examination (PSE; Wing *et al*, 1974) was rated on the seven-point Brief Psychiatric Rating Scale (BPRS; Lukoff *et al*, 1986) for hallucinations or unusual thought content (the rating scale was changed slightly from a 1–7 scale to 0–6; where 0 = absent, 6 = extremely severe). For each patient, two scores were produced: (a) the number of symptoms present (number of symptoms) and (b) the sum of the BPRS scores for all psychotic symptoms (total symptom severity).

Each psychotic symptom identified on the PSE was rated at post-treatment and follow-up on an eight-point change score (where 0 = completely remitted, 4 = no change, 7 = markedly worse) (Tress *et al*, 1987).

The Psychiatric Assessment Scale (PAS; Krawiecka *et al*, 1977) was used to measure anxiety, depression, delusions, hallucinations, incoherence and irrelevance of speech, poverty of speech, flat or incongruous affect, and psychomotor retardation, on a five-point scale (where 0 = absent, 4 = severe). A composite score for negative symptoms was made by adding the latter four subscales together.

The social functioning of the patient was assessed by means of the Social Functioning Scale (SFS; Birchwood *et al*, 1990). The total score of the SFS was used.

The subjective benefit of the treatment as perceived by the patient was assessed at post-treatment and follow-up on a seven-point scale (1 = resulted in extreme exacerbation, 4 = no change, 7 = resulted in extreme benefit).

The coping skills and problem-solving capabilities of the patient were also assessed, but these results are reported elsewhere (Tarrier *et al*, 1993).

Statistical analysis was performed by use of the StatView 512 + statistics package.

Results

The means and standard deviations for the total symptom score and the number of symptoms present are given in Table 1. It should be noted that where a repeated-measures ANOVA has been used the analysis only accepts patients who have data for all the repeated assessments. Hence, where three levels of within-subject comparisons, including the follow-up assessment, are

Table 1
Mean (s.d.) scores on assessment

	Pre-treatment	Post-treatment	Follow-up
Total symptom severity scores			
CSE[1]	20.8 (11.55) ($n=15$)	9.8 (10.88) ($n=15$)	7.58 (8.36) ($n=12$)
PS	10.17 (8.78) ($n=12$)	8.0 (7.97) ($n=12$)	5.55 (6.15) ($n=11$)
WP	14.54 (10.12) ($n=14$)	14.5 (13.58) ($n=14$)	
Number of symptoms scores			
CSE	5 (2.42) ($n=15$)	2.83 (2.36) ($n=15$)	2.08 (1.94) ($n=12$)
PS	2.8 (1.7) ($n=12$)	2.46 (2.2) ($n=12$)	1.82 (1.96) ($n=11$)
WP	3.54 (2.3) ($n=14$)	3.77 (2.59) ($n=14$)	

1. CSE = Coping strategy enhancement group, PS = Problem-solving group, WP = Waiting-period group.

completed, then the four patients who were not assessed at follow-up are excluded from the analysis.

Patients entering the WP group did not differ significantly at the initial assessment from those directly entering the two treatment groups.

Analyses of change over the waiting period indicated that there were no significant changes in any assessment measures. The PSE scores of the WP group changed little – the WP mean of 3.7 (s.d. 1.03) approximates closely to a score of 4 indicating no change, whereas the PS group's mean of 2.54 (s.d. 1.69) indicates a minimal to moderate improvement, and the CSE group's mean of 1.49 (s.d. 1.27) suggests a moderate to marked improvement.

A repeated-measures ANOVA on all patients who received treatment indicated a significant decrease in the total symptom severity score (pre-treatment mean = 16.07, s.d. = 11.55; post-treatment mean = 9.0, s.d. = 9.56; $F=13.07$, $P=0.0013$) and in the number of symptoms score (pre-treatment mean = 4.04, s.d. = 2.36; post-treatment mean = 2.78, s.d. = 2.56; $F=9.499$, $P=0.0048$).

There were no significant changes in social functioning in any of the groups or between any of the assessments.

To test whether either treatment was superior, repeated-measures ANOVAs which compared two levels of between subjects (groups: CSE, PS) and three levels of within subjects (assessments: pre, post, follow-up) were carried out. Number of symptoms showed a non-significant groups effect, a significant difference across assessments ($F=11.999$, $P=0.0001$), and an interaction effect which approached significance ($F=2.21$, $P=0.066$). The total symptom severity showed a non-significant groups effect and a significant difference across assessments ($F=14.18$, $P=0.0001$) and an interaction effect ($F=4.92$, $P=0.02$).

These results indicate that there were significant decreases on all measures over treatment and follow-up periods. The significant interaction effect for the total symptom severity and, to a lesser extent, for the number of symptoms indicates that the CSE group showed more change during treatment (see Table 1). Although there were no significant differences between the CSE and PS groups at pre-treatment, reference to Table 1 indicates a non-significant difference in scores at this assessment.

To investigate the effect of pre-treatment scores on treatment outcome a multiple regression was carried out using group membership and pre-treatment scores to predict the change scores over treatment. For the total symptom severity score this analysis indicated that both group membership ($F=11.38$, $P=0.0025$) and pre-treatment scores ($F=60.97$, $P=0.0001$) significantly contributed to change scores. For the number of symptoms score this analysis indicated that pre-treatment scores ($F=9.999$, $P=0.0042$) significantly contributed to change scores, while group membership showed only a trend towards significance ($F=3.09$, $P=0.09$).

Correlations between pre-treatment scores and change over treatment indicate that these measures were highly correlated for total symptom severity ($r=0.872$) and moderately so for number of symptoms ($r=0.497$). These positive correlations indicate that patients with higher pre-treatment scores improved more over treatment.

A one-way ANOVA on the PSE change score comparing CSE and PS groups showed a trend towards significance at post-treatment (means: CSE = 1.49, PS = 2.54; $F=3.39$, $P=0.077$) which was non-significant at follow-up ($F=1.6$, $P=0.22$). The CSE group showed a greater improvement at post-treatment.

Repeated-measures ANOVAs with two levels between subjects (CSE, PS) and three levels within subjects (pre, post, follow-up) indicated that significant changes over assessment (within subject) were shown only by a decrease in the anxiety ($F=3.47$, $P=0.04$) and delusions ($F=7.5$, $P=0.0017$) subscales. No other effects were found to be significant.

Change scores for the eight PAS subscales and the composite negative symptom scales were calculated for change over treatment. A one-way ANOVA was used to compare the CSE and PS groups. The CSE group showed a significantly greater improvement on the delusions scale (CSE mean = 1.53, s.d. = 1.69; PS mean = 0.17, s.d. = 0.94; $F=6.3$, $P=0.019$) and a trend towards a greater improvement on the anxiety scale (CSE mean = 1.13, s.d. = 1.69; PS mean = 0, s.d. = 1.13; $F=3.98$, $P=0.057$).

The distinction between statistical significance and clinical significance of improvement in the evaluation of psychotherapies has been increasingly emphasised (eg. Kazdin & Wilson, 1978; Jacobson *et al*, 1984), but there has been little consensus on the criteria for evaluating clinical significance. For example, it has been defined as: a large proportion of clients improving (Hugdahl & Ost, 1981); a change which is large in magnitude (Barlow, 1981); an improvement in the clients' everyday functioning (Kazdin & Wilson, 1978); a reduction in symptoms of 50% or more (Jansson & Ost, 1982); and an elimination of the presenting problem (Kazdin & Wilson, 1978). Moreover, most of the psychotherapy evaluation literature involves clients who can aspire to functioning within normal limits or to having their problems eliminated. This is unlikely to be true of patients suffering from chronic schizophrenia, hence there are few guidelines as to what constitutes clinical improvement in this group. Clinically significant change implies a change of practical

importance such as a reduction in both psychotic symptoms and increase in functioning. We have defined this, somewhat arbitrarily, as a reduction in symptoms of 50% or more (total symptom severity score) accompanied by an increase in social functioning of at least one standard deviation (15 points on the SFS). No patients achieved this. Two patients (one from each group) achieved a decrease in symptoms of 50% and an increase in social functioning of at least 12 points at post-treatment, which was maintained at follow-up. No other patients achieved a change in social functioning of any magnitude either at post-treatment or follow-up. At post-treatment 9 of 15 (60%) of the CSE group and 3 of 12 (25%) of the PS group had shown a decrease in symptoms of 50% or greater. A comparison of these group differences was almost significant ($\chi^2 = 3.307$, $P = 0.0653$). At follow-up, 5 of 12 (42%) of the CSE group and 4 of 11 (36%) of the PS group showed a 50% or greater improvement from pre-treatment level, but this difference between the two treatment groups was not significant. It is perhaps difficult to argue that clinically significant results were achieved, however, since levels of functioning were not improved. Large reductions in symptoms were achieved in a sizeable group of patients, especially those who received CSE.

One-way ANOVAs were completed between the perceived benefit scores of the CSE and PS groups at post-treatment and follow-up. Neither of these comparisons was significant. Both groups demonstrated mean scores close to 6, indicating that patients estimated they received moderate benefit from both treatments.

To test whether treatment gains were maintained at follow-up, repeated-measures ANOVA were completed comparing two-levels between-subject (CSE, PS) and two-levels within-subject (in the first analysis pre-treatment and follow-up assessment and in the second post-treatment and follow-up assessment) comparisons. For total symptom severity in the first analysis, the group comparison was non-significant, while the effect across assessments ($F = 20.18$, $P = 0.0002$) and the interaction ($F = 5.06$, $P = 0.035$) were significant. This significant group × assessment effect indicated that the CSE group showed greater improvements (see Table 1). In the second analysis the between-group comparison and interaction were non-significant while the effect across assessments ($F = 5.6$, $P = 0.028$) was significant. For the number of symptom scores the first analysis between-groups comparison was non-significant ($F = 2.01$, $P = 0.17$), the effect across assessments was significant ($F = 17.84$, $P = 0.0004$), and the interaction was almost significant ($F = 3.52$, $P = 0.075$). In the second analysis, the between-group comparison and interaction were non-significant while the effect across assessments ($F = 9.43$, $P = 0.006$) was again significant.

These results indicate a significant improvement at follow-up from both pre- and post-treatment levels. There is some indication that CSE is superior to PS in the change from pre-treatment to follow-up. The difference between the two groups appears to be over the treatment period, and there were no differences from post-treatment to follow-up.

To test whether the expectation of improvement affects improvement, comparisons were made between the high-expectancy (HEX) and neutral-expectancy (NEX) conditions.

Repeated-measures ANOVAs compared two levels (HEX, NEX) between subject and three levels within subject (pre, post and follow-up assessment). For the total symptom severity score the group effect ($F = 6.56$, $P = 0.018$) and the effect across assessments ($F = 11.5$, $P = 0.001$) were significant. The interaction was not significant (means: HEX pre = 17.9, post = 12.3, follow-up = 10.3; NEX pre = 10.5, post = 4.6, follow-up = 2.8). For the number of symptoms score the group ($F = 6.696$, $P = 0.017$) and the effect across assessments ($F = 11.1$, $P = 0.0001$) were significant. The interaction was not significant (means: HEX pre = 4.3, post = 3.7, follow-up = 3.1; NEX pre = 3.1, post = 1.7, follow-up = 0.9).

Changes in PSE score and the subjective benefit score, indicated no significant differences between HEX and NEX groups.

References to these results and the means indicated that the HEX group had higher scores throughout treatment but there was no differential effect of expectancy on improvement over treatment. It can be concluded that the manipulation of expectancy did not contribute to treatment effects.

Discussion

The persistence of residual psychotic symptoms in schizophrenic patients is an enormous clinical problem. Although there have been numerous case studies and some uncontrolled studies, to the best of our knowledge this is the first controlled group-treatment evaluation of psychological management of this problem. The results are encouraging, although not unequivocal; they indicate that patients do not change on any of the assessment measures over the waiting period, as predicted. Although the waiting period is only six weeks it is equivalent in time to the treatment period and is indicative of a stable baseline. Patients who received the cognitive–behavioural treatments showed a significant improvement on symptom-related assessments. There were also significant changes over treatment on the anxiety and delusions subscales of the PAS, but not on those for depression, hallucinations, or negative symptoms. Nor were there any significant changes on the measure of social functioning.

A number of points of interest are raised by these results. Firstly, the cognitive–behavioural treatments showed clear improvements on measures relating to the target symptoms but did not generalise to wider areas of functioning including negative symptoms. Secondly, improvements appeared to occur with delusions but not with hallucinations as measured on the PAS. The target symptom ratings measured by the seven-point BPRS scales may be more sensitive to change than the PAS scales which may need a more marked improvement to reach significance. Thirdly, improvements were found in anxiety but not

in depression. Possibly this is because anxiety may be more closely related to the presence of psychotic symptoms than depression and hence would decrease as symptoms decrease.

Comparisons to determine the superiority of one of the two treatments, CSE or PS, were equivocal in their results. There is some evidence that CSE is superior in producing symptom reduction. Significant interactions between treatment groups and assessments in the ANOVAs suggest a greater change resulting from CSE (see Table 1). A significantly greater change score on the delusions subscale of the PAS also supports this. Furthermore, a greater percentage (60%) of patients who received CSE demonstrated a 50% or greater improvement in their symptoms compared with those who received PS (25%). There is, however, an indication that a difference in scores at pre-treatment contributed, at least in part, to the greater change over treatment in the CSE group. There is a suggestion that this effect was greater in the change in the number of symptoms rather than the total symptom score. Unfortunately the random allocation of patients to treatment groups resulted in pre-treatment differences: although these were non-significant they have contributed to some difficulty in clearly interpreting these results.

A number of further issues are raised by these results. Firstly, if both treatments have some beneficial effects would they be more effective in combination? This question is impossible to answer at present but deserves further investigation. The second point is whether a greater treatment effect could have been produced and maintained by an extended treatment programme. Treatment lasted for ten sessions which were spread over five weeks. We feel that it would be unwise to directly transfer the parameters of a research protocol to a service setting, and in a clinical service the use of booster sessions or the extension of treatment over an extended time period to meet the needs of the individual patient may well be advisable. The results suggest that treatment benefits are maintained at six months, although it was noticeable that the percentage of patients who improved by at least 50% was reduced somewhat at follow-up in the CSE group (60% to 42%).

The next question is why do the treatments work? CSE has demonstrated significant decreases in psychotic symptoms and related anxiety. But there were no 'ripple out' improvements in mood, negative symptoms, or social functioning, as had been predicted. This failure to generalise supports Hall's (1989) view that the relationship between change in symptomatic behaviour and adaptive or social functioning is low. Furthermore, problem solving, which was predicted to increase social functioning and not psychotic symptoms, had the reverse effect. In an accompanying paper (Tarrier *et al*, 1993) we have examined the treatment-specific changes that occur in the patients' coping and problem-solving skills. These results indicate that patients taught coping strategies significantly increase their coping skills, whereas patients taught problem solving significantly increase their problem-solving ability but show a reduction in coping skills. There is evidence, therefore, for treatment-specific skill improvements. Evidence for specific treatment effects is therefore equivocal in the problem-solving group. Either problem solving results in some kind of cognitive change that inhibits delusions or hallucinations (e.g. it is in itself an attention-switching process) or a number of non-specific effects are operating. However, a pure expectation of improvement due to receiving treatment does not appear to be one of these.

Two clinical anecdotes may serve to raise some of the difficulties in both measuring and maintaining clinical improvements. A 52-year-old man with a 16-year history of schizophrenia suffered continuous and severe delusions and hallucinations. Over the years the professional staff, and to an extent his local community, had ignored his illness-related conversations. However, the intervention which targeted his psychopathology and encouraged him to monitor his experiences also increased the importance of his psychopathology to him and the frequency with which he spoke of his symptoms. This was reflected in his post-treatment assessment scores. After the treatment programme finished he appeared to become frustrated and angry that his symptoms were no longer a source of attention and he later refused the follow-up assessment. The second case was a 62-year-old woman with a 30-year history of schizophrenia who reduced her social activities because of a delusional interpretation of events that had occurred at her church social group. She was encouraged to test the reality of her delusional interpretation against the therapist's interpretation of events. This was successful and she agreed that her thoughts had been related to the illness and returned to the social group meetings. Some weeks later she was still attending the groups but she had changed her attribution of events back to a delusional interpretation. However, she was no longer distressed by her delusional thoughts since she reasoned that because her friends were not upset by what had happened (she thought that her friends heard her obscene thoughts broadcast out loud) neither should she be upset.

As is typical with this patient group there is a considerable drop-out and refusal rate. Of the 48 suitable patients (the patient who may have suffered from dementia has been excluded from these figures) only 27 (56%) continued to post-treatment and 23 (48%) to six-month follow-up. Hence it was possible to keep less than 50% of the patients in treatment for any period of time. Two important factors must be balanced against this difficulty. Firstly, this problem of engagement and drop-out is not uncommon in the treatment of psychiatric and psychological disorders generally (Emmelkamp & Foa, 1983) and has frequently been reported with psychosocial interventions with schizophrenic patients (Smith & Birchwood, 1990; Tarrier, 1991). A high rate of refusal and early drop-out is, therefore, to be expected with this highly disturbed patient group. Secondly, the patients were not achieving further sustained therapeutic advances despite optimum pharmacological treatment. Therefore, any benefit derived from psychological intervention is of clear importance. In support of this point it should be noted that at post-treatment 60% of those patients who received CSE and 25% of those who received PS improved by at least 50%. Furthermore, two patients in each group were completely symptom free at post-treatment, that is, 19% of the patient sample were in complete remission. At six-month follow-up, 42% of those who had received CSE and 36% of those who received PS showed at least 50% improvement, and five patients (22% of the follow-up sample) were in complete remission. Even if this figure is considered as a percentage of all suitable patients whether they received treatment or not, then 10% achieved total remission, not an insignificant number.

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References

ALFORD, G. S., FLEECE, L. & ROTHBLUM, E. (1982) Hallucinatory–delusional verbalisations: modification in a chronic schizophrenic by self-control and cognitive restructuring. *Behaviour Modification*, **6**, 421–435.

BARLOW, D. H. (1981) On the relation of clinical research to clinical practice: current issues, new directions. *Journal of Consulting and Clinical Psychology*, **49**, 147–155.

BELCHER, T. L. (1988) Behavioural reduction of overt hallucinatory behaviour in chronic schizophrenics. *Journal of Behaviour Therapy Experimental Psychiatry*, **19**, 69–71.

BIRCHWOOD, M. (1986) Control of auditory hallucinations through occlusion of monoaural auditory input. *British Journal of Psychiatry*, **149**, 104–107.

——, SMITH, J., COCHRANE, R., *et al* (1990) The social functioning scale: the development and validation of a scale of social adjustment for use in family intervention programmes with schizophrenic patients. *British Journal of Psychiatry*, **157**, 853–859.

BRIER, A. & STRAUSS, H. S. (1983) Self-control in psychotic disorders. *Archives of General Psychiatry*, **40**, 1141–1145.

BULOW, H., OEI, T. P. S. & PINKNEY, B. (1979) Effects of contingent social reinforcement with delusional chronic schizophrenic men. *Psychological Reports*, **44**, 659–666.

CARR, V. (1988) Patients techniques for coping with schizophrenia: an exploratory study. *British Journal of Medical Psychology*, **61**, 339–352.

COHEN, C. I. & BERK, B. S. (1985) Personal coping styles of schizophrenic out-patients. *Hospital and Community Psychiatry*, **36**, 407–410.

CURSON, D. A., BARNES, T. R. E., BAMBER, R. W., *et al* (1985) Long term depot maintenance of chronic schizophrenic outpatients. *British Journal of Psychiatry*, **146**, 464–480.

——, PATEL, M., LIDDLE, P. F., *et al* (1988) Psychiatric morbidity of a long stay hospital population with chronic schizophrenia and implications for future community care. *British Medical Journal*, **297**, 819–822.

DAVIS, J. R., WALLACE, C. J., LIBERMAN, R. P., *et al* (1976) The use of brief isolation to suppress delusional and hallucinatory speech. *Journal of Behaviour Therapy and Experimental Psychiatry*, **7**, 269–275.

D'ZURILLA, T. J. & GOLDFRIED, M. (1971) Problem solving and behaviour modification. *Journal of Abnormal Psychology*, **78**, 107–126.

EMMELKAMP, P. M. G. & FOA, E. B. (1983) Failures as a challenge. In *Failures in Behaviour Therapy* (eds E. B. Foa & P. M. G. Emmelkamp). New York: Wiley.

FALLOON, I. R. H. (1986) Cognitive and behavioural interventions in the self-control of schizophrenia. In *Psychosocial Treatment of Schizophrenia* (eds J. Strauss, W. Boker & H. D. Brenner), pp 180–190. Bern: Hans Huber.

—— & TALBOT, R. E. (1981) Persistent auditory hallucinations: coping mechanisms and implications for management. *Psychological Medicine*, **11**, 329–339.

——, BOYD, J. L. & MCGILL, C. (1984) *Family Care of Schizophrenia*. New York: Guilford.

FONAGY, P. & SLADE, P. (1982) Punishment vs negative reinforcement in the aversive conditioning of auditory hallucinations. *Behaviour Research and Therapy*, **20**, 483–492.

FOWLER, D. & MORLEY, S. (1989) The cognitive–behavioural treatment of hallucinations and delusions: a preliminary study. *Behavioural Psychotherapy*, **17**, 267–282.

HALL, J. (1989) Chronic psychiatric handicaps. In *Cognitive Behaviour Therapy for Psychiatric Problems* (eds K. Hawton, P. M. Salkovskis, J. Kirk, *et al*), pp. 315–338. Oxford: Oxford Medical Publications.

HARROW, M. & SILVERSTEIN, M. L. (1977) Psychotic symptoms in schizophrenia after the acute phase. *Schizophrenia Bulletin*, **3**, 608–616.

HAWTON, K. & KIRK, J. (1989) Problem solving. In *Cognitive Behaviour Therapy for Psychiatric Problems* (eds K. Hawton, P. Salkovskis, J. Kirk, *et al*), pp. 406–426. Oxford: Oxford Medical Publications.

HEINRICHS, D. W. (1988) The treatment of delusions in schizophrenic patients. In *Delusional Beliefs* (eds T. F. Oltmanns & B. A. Maher), pp. 275–294. New York: Wiley.

HEMSLEY, D. (1986) Psychological treatment of schizophrenia. In *A Handbook of Clinical Psychology* (eds S. Lindsay & G. Powell). London: Gower.

HOLE, R. W., RUSH, A. J. & BECK, A. T. (1979) A cognitive investigation of schizophrenic delusions. *Psychiatry*, **42**, 312–319.

HUGDAHL, K. & OST, L. (1981) On the difference between statistical and clinical significance. *Behavioral Assessment*, **3**, 289–295.

JACOBSON, N. S., FOLLETTE, W. C. & REVENSTORF, D. (1984) Psychotherapy outcome research: methods for reporting variability and evaluating clinical significance. *Behavior Therapy*, **15**, 336–350.

JANSSON, L. & OST, L. (1982) Behavioural treatments for agoraphobics: an evaluation review. *Clinical Psychology Review*, **2**, 311–336.

KANAS, N. & BARR, M. A. (1984) Self-control of psychotic productions in schizophrenia. *Archives of General Psychiatry*, **41**, 919–920.

KAZDIN, A. E. & WILSON, G. T. (1978) *Evaluation of Behaviour Therapy: Issues, Evidence and Research Strategies*. Cambridge: Ballinger.

KRAWIECKA, M., GOLDBERG, D. & VAUGHAN, M. (1977) A standardised psychiatric assessment scale for rating chronic psychotic patients. *Acta Psychiatrica Scandinavica*, **55**, 299–308.

LAMONTAGNE, Y., AUDET, N. & ELIE, R. (1983) Thought stopping for delusions and hallucinations: a pilot study. *Behavioural Psychotherapy*, **11**, 177–184.

LIBERMAN, R. P., TEIGAN, J., PATTERSON, R., *et al* (1973) Reducing delusional speech in chronic paranoid schizophrenics. *Journal of Applied Behaviour Analysis*, **6**, 57–64.

LUKOFF, D., NUECHTERLEIN, K. H. & VENTURA, J. (1986) Manual for expanded Brief Psychiatric Rating Scale (BPRS). *Schizophrenia Bulletin*, **12**, 594–602.

MEICHENBAUM, D. & CAMERON, R. (1973) Training schizophrenics to talk to themselves: a means of developing attentional control. *Behavior Therapy*, **4**, 525–534.

MILTON, F., PATWA, V. K. & HAFNER, J. (1978) Confrontation vs belief modification in persistently deluded patients. *British Journal of Medical Psychology*, **51**, 127–130.

MORLEY, S. (1987) Modification of auditory hallucinations: experimental studies of headphones and earplugs. *Behavioural Psychotherapy*, **15**, 240–251.

NYDEGGER, R. V. (1972) The elimination of hallucinatory and delusional behaviours by verbal conditioning and assertive training: a case study. *Journal of Behaviour Therapy and Experimental Psychiatry*, **3**, 225–227.

SCHNEIDER, S. J. & POPE, A. T. (1982) Neuroleptic-like electroencephalographic changes in schizophrenics through biofeedback. *Biofeedback and Self-Regulation*, **7**, 479–490.

SLADE, P. D. (1972) The effects of systematic desensitisation on auditory hallucinations. *Behaviour Research and Therapy*, **10**, 85–91.

—— (1973) The psychological investigation and treatment of auditory hallucinations: a second case report. *British Journal of Medical Psychology*, **46**, 293–296.

——& BENTALL, R. P. (1988) *Sensory Deception: Towards Scientific Analysis of Hallucinations*. London: Croom Helm.

SILVERSTEIN, M. L. & HARROW, M. (1978) First rank symptoms in the post acute schizophrenic: a follow-up study. *American Journal of Psychiatry*, **135**, 1481–1486.

SMITH, J. & BIRCHWOOD, M. (1990) Relatives and patients as partners in the management of schizophrenia: the development of a service model. *British Journal of Psychiatry*, **156**, 654–660.

STEINMARK, S. W. & BORKOVEC, T. D. (1974) Active and placebo treatment effects on moderate insomniac under counter demand and positive demand instruction. *Journal of Abnormal Psychology*, **83**, 157–163.

TARRIER, N. (1987) An investigation of residual psychotic symptoms in discharged schizophrenic patients. *British Journal of Clinical Psychology*, **26**, 141–143.

—— (1991) Some aspects of family interventions in schizophrenia. I: Adherence with family intervention programmes. *British Journal of Psychiatry*, **159**, 475–480.

—— (1992*a*) Psychological treatment of schizophrenic symptoms. In *Schizophrenia: An Overview and Practical Handbook* (ed. D. Kavanagh). London: Chapman & Hall.

—— (1992*b*) Management and modification of residual psychotic symptoms. In *Innovations in the Psychological Management of Schizophrenia* (eds M. Birchwood & N. Tarrier). Chichester: Wiley.

—— & MAIN, C. (1986) Applied Relaxation Training (ART) with patients suffering from generalised anxiety and panic attacks: the efficacy of a learnt coping strategy on subjective reports. *British Journal of Psychiatry*, **149**, 330–336.

——, HARWOOD, S., YUSOPOFF, L., *et al* (1990) Coping Strategy Enhancement (CSE): a method of treating residual schizophrenic symptoms. *Behavioural Psychotherapy*, **18**, 283–293.

——, SHARPE, L., BECKETT, R., *et al* (1993) A trial of two cognitive-behavioural methods of treating drug-resistant residual psychotic symptoms in schizophrenic patients: II. Treatment specific changes in coping and problem solving skills. *Social Psychiatry and Psychiatric Epidemiology* (in press).

TRESS, K. H., BELLENIS, C., BROWNLOW, J. M., *et al* (1987) The Present State Examination Change rating scale. *British Journal of Psychiatry*, **150**, 201–207.

TURNER, S., HERSON, M. & BELLACK, A. (1977) Effects of social disruption, stimulus interference and aversive conditioning on auditory hallucinations. *Behaviour Modification*, **1**, 249–258.

WATTS, F. N., POWELL, G. E. & AUSTIN, S. V. (1973) The modification of abnormal beliefs. *British Journal of Medical Psychology*, **46**, 359–363.

WEINGAERTNER, A. H. (1971) Self-administered aversive stimulation with hallucinating hospitalised schizophenics. *Journal of Consulting and Clinical Psychology*, **36**, 422–429.

WING, J. K., COOPER, J. E. & SARTORIUS, N. (1974) *Measurement and Classification of Psychiatric Symptoms: An Instruction Manual for the PSE and Catego Programme*. Cambridge, Cambridge University Press.

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