Is cognitive–behavioural therapy a worthwhile treatment for psychosis?

DOUGLAS TURKINGTON / PETER McKENNA

INTRODUCTION

Cognitive–behavioural therapy (CBT) is of proven benefit for treatment of depression and has, over the past 5 years, been increasingly advocated as a treatment modality for schizophrenia. There has been considerable enthusiasm for the use of CBT in schizophrenia from psychiatrists, psychologists, psychiatric nurses and users alike. However, this enthusiasm may have precluded dispassionate evaluation of the effectiveness of this treatment. Even though an intervention is popular and is thought to be generally a ‘good thing’, we should not then take short-cuts through the evidence. In an era of limited resources every hour spent with a patient or training staff in a new technique must be justified. Is CBT really a worthwhile and effective treatment for patients with psychosis? This issue is debated by Dr Douglas Turkington, Senior Lecturer in Psychiatry in Newcastle upon Tyne and author of one of the leading texts on CBT for schizophrenia, and Dr Peter McKenna, consultant psychiatrist with the Cambridge Psychiatric Rehabilitation Service.

FOR

Patients with schizophrenia and their carers have tended to be the passive recipients of care delivered by mental health services. This situation has largely developed because of the negative results reported with psychodynamic psychotherapy and the failure to implement family therapy interventions proven to reduce relapse. Cognitive–behavioural therapy as applied to schizophrenia has become increasingly available across the UK over the past 10 years. It has been welcomed by patients and carers alike because it brings them into the treatment team as active participants in the management of psychotic symptoms. The therapy primarily facilitates engagement and the establishment of collaborative empiricism, with reality-testing based on guided discovery rather than confrontation or collusion. This working relationship allows for the testing and working-through of hypotheses concerning causation and maintenance of distressing symptoms. Hallucinations, delusions, negative symptoms and depression have all been shown to be responsive to CBT (Sensky et al, 2000). Techniques range from more-superficial peripheral questioning of delusional content to deeper work on underlying dysfunctional beliefs about the self (e.g. ‘I am evil, deficient, damaged’ or ‘I am special, unique, different’). Homework exercises allow patients, often with the help of key-workers or carers, to begin to make sense of their distressing experiences and to see the effects of working on avoidance, rational responding or changing coping strategies. Cognitive–behavioural therapy is therefore an individualised intervention based on a case formulation which helps the patient to answer the question, ‘Why have I changed so much?’ and to begin to see the point in taking medication and attending social treatment options. Such improvements in insight and adherence have led to reductions in relapse and rehospitalisation. Carers who have worked in this way have often moved from the expression of frustration and guilt to a more hopeful and less alienated position.

Cognitive–behavioural therapy has been well tested in relation to the treatment of residual symptoms of schizophrenia and is of proven efficacy and cost-effectiveness (National Institute for Clinical Excellence, 2002). Other psychological treatments (supportive therapy and befriending) also seem to have an effect at the end of 20 sessions. However, CBT is the only psychological treatment in chronic schizophrenia with proven durability at short-term follow-up (Gould et al, 2001). It has also recently been proven in a pragmatic randomised controlled trial that the benefits of CBT translate into community settings (Turkington et al, 2002). In this study, community psychiatric nurses were trained in CBT for schizophrenia over a 10-day period and given weekly supervision. They used CBT effectively in terms of insight improvement and reduction in overall symptoms and depression. Cognitive–behavioural therapy would appear to be very acceptable to patients, with an average rate of drop-out across studies of 12–15%. The therapy would also appear to be safe, with no evidence of increasing suicidal ideation, agitation or violence in any study to date.

It is, however, certainly true that, for certain types of psychotic symptoms (e.g. command hallucinations linked to trauma, or systematised or grandiose delusions), distressing affects can emerge as the psychotic symptom is worked with. Brief CBT is often not indicated for such presentations and 20–50 sessions with a CBT expert can be indicated.

Future progress will depend on the further development of psychological models of psychotic symptom onset and maintenance and on the development of more refined treatment manuals. Cognitive–behavioural therapy would appear to have the possibility of an enhanced effect when given with cognitively sparing antipsychotic medication (Pinto et al, 1999) or when combined with cognitive remediation. It will be very interesting to note any functional imaging changes through a course of CBT when psychotic symptoms are improving. Similarly, CBT for bipolar disorder has been shown to reduce relapse and improve illness management through early-signs monitoring. Cognitive–behavioural therapy has proven efficacy,
durability and cost-effectiveness in psychosis. Why would any general psychiatrist not wish to have a member of their community team trained to deliver CBT to their patients with psychosis and their carers?

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AGAINST

In order to protect patients from false hope and doctors from self-deception, a cumbersome but foolproof methodology has evolved for evaluating new treatments in medicine. This is the double-blind, placebo-controlled trial, and its rules also apply to trials of psychological treatments. Although double-blind assessments may be difficult to achieve, no one would argue that blind evaluation can be dispensed with. Psychological interventions are also susceptible to an exact equivalent of the placebo effect in drug trials, the Hawthorne effect, a distortion of research results caused by the response of subjects to the special attention they receive from researchers.

From the outset, CBT for schizophrenia has been associated with claims made on the basis of evidence that is less rigorous than in the rest of medicine. For example, one of most widely quoted trials in support of its effectiveness is that of Kuipers et al. (1997). Yet this employed neither a control intervention nor blind evaluations. It found a significant effect of cognitive therapy on overall scores on the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962) but not on delusions, hallucinations or any other measure of symptoms or functioning.

Of the 13 trials of CBT in schizophrenia included in a Cochrane meta-analysis (Cormac et al., 2002), only four used a control intervention and were carried out under blind conditions. These are shown in Table 1, together with a more recent study by Lewis et al. (2002). The two largest studies (Sensky et al., 2000; Lewis et al., 2002) showed no significant advantage for CBT over the control intervention. Tarrier et al. (1999) found a non-significant difference in favour of CBT on a score based on delusions and hallucinations, and no difference for negative symptoms. One of two small studies (Turkington & Kingdon, 2000) found a significant benefit for the CBT group, but the other (Haddock et al., 1999) had findings in the opposite direction. The pooled effect size for these studies is close to zero.

Table 1: Effect sizes for improvement with cognitive–behavioural therapy (CBT) in studies using blind evaluation and a control intervention

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample size</th>
<th>Finding</th>
<th>Effect size</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tarrier et al. (1999)</td>
<td>23 CBT, 21 SC</td>
<td>NS</td>
<td>-0.47</td>
<td>-1.07 to -0.13</td>
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<tr>
<td>Positive symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Tarrier et al. (1999)</td>
<td>23 CBT, 21 SC</td>
<td>NS</td>
<td>-0.07</td>
<td>-0.66 to 0.52</td>
</tr>
<tr>
<td>Negative symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Haddock et al. (1999)</td>
<td>8 CBT, 10 SC</td>
<td>NS</td>
<td>+0.57</td>
<td>-0.38 to 1.52</td>
</tr>
<tr>
<td>Sensky et al. (2000)</td>
<td>46 CBT,</td>
<td>NS</td>
<td>-0.08</td>
<td>-0.50 to 0.33</td>
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<td></td>
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<tr>
<td>Turkington et al. (2000)</td>
<td>10 CBT, 5 SC</td>
<td>CBT &gt; SC</td>
<td>-1.14</td>
<td>-2.28 to 0.01</td>
</tr>
<tr>
<td>Lewis et al. (2002)</td>
<td>78 CBT, 71 SC</td>
<td>NS</td>
<td>+0.10</td>
<td>-0.22 to 0.42</td>
</tr>
<tr>
<td>Overall effect</td>
<td></td>
<td></td>
<td>-0.028</td>
<td>-0.25 to 0.19</td>
</tr>
</tbody>
</table>

1. Negative value favours CBT.
2. Effect size calculation based on standard deviations in Cormac et al. (2002).
3. For this calculation the average of the two effect sizes in Tarrier et al. (1999) was used.

CCT, supportive counselling; NS, no significant difference.

If CBT were a drug, these studies would have been sufficient to consign it to history. As yet, however, this has not happened and advocates of the treatment continue to plead their case. Thus, despite finding no advantage over befriending at the end of their 9-month study period, Sensky et al. (2000) made the extraordinary statement that ‘[c]ognitive–behavioural therapy is effective in treating negative as well as positive symptoms in schizophrenic patients resistant to standard antipsychotic drugs’. Lewis et al. (2002) felt able to conclude that CBT speeded remission from acute symptoms in early schizophrenia, even though a significant difference between the two treatments was found only for auditory hallucinations, and not for delusions, positive symptoms or total symptom scores.

Finally, on the basis of a significant improvement over befriending that appeared 9 months after the end of treatment, Sensky et al. (2000) have also argued that CBT leads to sustained clinical improvement in schizophrenia. However, this finding has to be balanced against the results of the Cochrane meta-analysis (Cormac et al., 2002), which found no convincing evidence of an effect in the longer term.

REFERENCES AND FURTHER READING


