Rheumatology 2003;42:435–441
doi:10.1093/rheumatology/keg144, available online at www.rheumatology.oupjournals.org

Long-term efficacy of a cognitive behavioural treatment from a randomized controlled trial for patients recently diagnosed with rheumatoid arthritis

L. Sharpe, T. Sensky[1], N. Timberlake[2], B. Ryan[2] and S. Allard[2]

Objectives. **This study examined the long-term efficacy of a cognitive behavioural intervention for patients with recent-onset, seropositive rheumatoid arthritis (RA).**
Methods. **Fifty-three consecutive patients with less than a 2-yr history of classic or definite RA were recruited into the trial. All participants received routine medical management during the study, and half were randomly allocated to receive an 8-week adjunctive psychological intervention. All assessments were conducted blind to the allocation. This paper reports intention-to-treat analyses of the 18-month follow-up.**
Results. **Consistent with short-term results, significant differences were found between the groups in depressive symptoms. The intervention group maintained improvements in joint function, although those in routine care made similar improvements over the ensuing 18 months. At follow-up, group differences emerged for disability and anxiety.**
Conclusions. **These results indicate that cognitive behavioural intervention offered as an adjunct to standard clinical management early in the course of RA is efficacious in producing improvements in both psychological and physical indices. Furthermore, improvements appear to increase 18 months after a brief, time-limited psychological treatment.**

KEY WORDS: Arthritis, Rheumatoid, Psychological intervention, Cognitive behavioural treatment, Depression, Outcome assessment.

There is evidence to suggest that cognitive behavioural psychotherapy (CBT) is an effective, adjunctive treatment in chronic rheumatoid arthritis (RA) [1]. Research has demonstrated that broadly based CBT directed at symptom management is effective in improving psychopathology, levels of pain, disability, joint function and even biological indicators of disease, such as erythrocyte sedimentation rate (ESR) [2–7]. Evidence suggests that interventions are less effective in chronic, progressive disease where symptoms are likely to deteriorate with or without psychological intervention [8, 9]. Two recent studies have demonstrated the efficacy of intervention early in the course of the illness [10, 11]. Both studies demonstrated benefits in psychological and physical domains.

Despite the large number of trials that have now been reported in the literature for patients with RA [2–11], few have reported data on longer-term outcomes. This omission is perplexing given that for the majority of patients RA is a chronic illness with symptoms that wax and wane over time accompanied by general deterioration in physical function. Of the early outcome studies, Bradley *et al.* [7] found therapeutic benefits that lasted for 6 months following the intervention. Parker *et al.* [8] reported improvements for 12 months following treatment, but only for those participants who were highly adherent to treatment strategies. If psychological interventions are to be useful in the routine management of RA, then longer-term efficacy must be demonstrated.

School of Psychology, Clinical Psychology Unit F12, University of Sydney, Australia, [1]Imperial College of Science, Technology and Medicine, London, UK and [2]West Middlesex University Hospital, Isleworth, UK.

Submitted 19 March 2001; revised version accepted 4 September 2002.

Correspondence to: L. Sharpe, Department of Psychology, Clinical Psychology Unit F 12, University of Sydney, NSW, Australia 2006. E-mail: louises@psych.usyd.edu.au

Of those studies investigating intervention early in the disease process, Parker *et al.* [10] found that benefits were maintained at 15-month follow-up in some psychological factors and level of pain. In a 6-month follow-up, Sharpe *et al.* [11] found maintenance of improvements in mood, but also found an emergence of further improvements in joint function. Considerable evidence now suggests that the first 2 yr of illness represent a particular window of opportunity in the physical treatment of RA [12–14]. The success of a psychological intervention within the crucial first 2 yr of illness is, as a result, particularly encouraging for the role of psychological factors in RA. However, the importance of examining the longer-term outcome is fundamental to the clinical utility of a treatment, particularly in the case of early intervention. The primary aim of early intervention is to intervene while patients remain relatively able bodied and have not yet developed unhelpful ways of managing the illness. The importance of early intervention lies in its ability to reduce the subsequent risk of either psychological and/or physical burden in the longer term. Since CBT is an intervention that expressly aims to help patients learn strategies that will improve their management of symptoms and develop more adaptive attitudes towards the illness, one might expect that the efficacy of the treatment should increase over time rather than decrease.

The aim of the present study was to examine the long-term efficacy of an adjunctive CBT programme for patients with early RA. The present study investigated whether the short-term psychological and physical benefits following a cognitive behavioural intervention (post-treatment and 6-month follow-up) were maintained or improved 18 months following treatment. It was hypothesized that improvements in depression and joint function following CBT would be maintained. It was also hypothesized that improvements which failed to reach significance following treatment, such as changes in anxiety, coping strategies and disability, would emerge as significant over the follow-up period.

Patients and methods

Patients

The sample was recruited from consecutive patients attending the rheumatology clinics of three local hospitals in or near London between July 1994 and July 1996. Participants were adults between the ages of 18 and 75 yr. All participants were diagnosed by a consultant rheumatologist as having 'definite or classic' RA, according to the American Rheumatism Association criteria [15] and were seropositive for RA. Participants were excluded if they had a history of psychotic illness, current alcohol or drug abuse or poor English language skills, insufficient to complete the assessment or treatment. Power calculations were based on the effect sizes from Bradley *et al.*'s study [2]. Effect sizes (ES) of 0.88 for tender joints and 0.44 for pain, indicated that for repeated measures ANOVAs using one-tailed tests of significance, a combined sample of 40 patients should be sufficient to show changes of 5% significance with a power of at least 80%. For depression (ES = 0.27), a combined sample size of 50 was required to show the same power.

A high recruitment rate of 89% was achieved in the trial. Sixty-three participants were identified, of whom 56 agreed to take part, although three participants later had their diagnoses changed and their data were subsequently removed from the analyses. This left 53 participants in the trial, 27 allocated to the CBT group and 26 to standard care alone. Four participants in each group dropped out during the intervention stage, leaving 23 and 22 participants at post-treatment and 6-month follow-up. Only one further participant was lost in the ensuing 12-month period from the standard care group. See Fig. 1 for the flow of participants through the trial.

Prior to intervention, the mean age for participants was 55.1 yr (S.D. = 14.1), and they had been diagnosed with RA an average of 12.6 months earlier (S.D. = 8.2). Most of the patients in the sample were female (70%), and 67% were married or living in a *de facto* relationship. Twenty-four per cent of participants had completed an 'A' level or equivalent as their highest educational

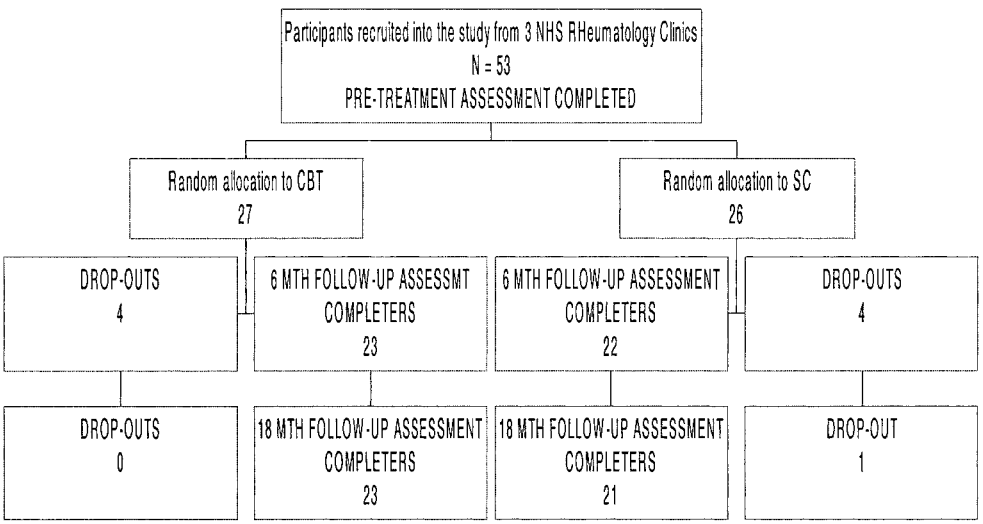

FIG. 1. Progression of participants through the trial. SC, standard care.

attainment, with 14% having completed a tertiary degree. The remainder had left school prior to completing 'A' levels. Only 34% of the patients were in paid employment: 22% working full-time and 12% part-time.

Procedure

Participants had been recruited through their routine out-patient appointments. They were instructed to complete a set of questionnaires and had medical assessments (i.e. blood tests and joint assessment) prior to intervention, following treatment and at 6- and 18-month follow-up. The present data include the results of the pre-treatment, 6- and 18-month follow-up assessments. Patients were allocated to treatment groups by simple randomization that was determined according to a standard table of random numbers. The set of random numbers was related to a list of consecutive numbers (1–75) and was concealed. The research nurse, who administered the assessments, gave each participant a consecutive number from 1–56. Participants were allocated to a condition, according to the predetermined set of random numbers, on the basis of their subject number. Participants who dropped out of the study or were later excluded were not replaced. The assessment nurse remained blind to the allocation throughout the intervention study and follow-up period. In our earlier study, the research nurse had failed to guess the allocation at a rate better than chance [11].

Outcome measures

Hospital Anxiety and Depression Scales (HADS). The HADS [16], a 14-item questionnaire, excludes somatic items of anxiety and depression and was developed to assess anxiety and depression in patients with physical health problems.

Coping Strategy Questionnaire (CSQ). The CSQ [17] is a widely used and well validated measure of coping strategies. It includes seven subscales, of which two involve maladaptive strategies. A total score of active coping is calculated by subtracting the passive scale scores from the sum of the active scale.

Self-monitored level of subjective pain. Participants were asked to record pain severity on an 11-point rating scale three times daily for 1 week. Each day was divided into three periods for which the participant monitored the number of hours of pain in addition to the severity of pain symptoms upon wakening, at 4 o'clock in the afternoon and upon going to bed. A pain index score was calculated as a product of severity and duration (h) of pain for each full-day period [18].

Stanford Health Assessment Questionnaire (HAQ). The HAQ [19] assesses the level of disability associated with RA. It is a brief self-report questionnaire that is a good predictor of disease course and is sensitive to change in early RA [20, 21].

Ritchie Articular Index (RAI). The RAI [22] is a widely used measure of joint function. It was administered by a trained rheumatology nurse, blind to the allocation of participants to intervention.

Disease measures. Blood tests were conducted to provide measures of ESR and C-reactive protein (CRP). ESR was measured using Westergren's method.

Psychological intervention

Two psychologists conducted the psychological treatment from a detailed treatment manual developed for the project. The programme involved eight individual sessions, each lasting approximately 1 h, over consecutive weeks. The cognitive and behavioural intervention was developed from standard pain management approaches [23] and self-help educational material [24].

The aims of the intervention were (i) to help patients learn strategies that would be helpful in coping with the symptoms of RA and (ii) to foster an adaptive attitude towards the illness. The intervention included the following strategies: education about the illness, applied relaxation training, goal setting with an emphasis on developing an appropriate balance between rest and exercise, attention diversion training, cognitive restructuring to help foster helpful attitudes towards illness, assertiveness training and developing plans for managing flare-ups and high-risk situations. Further details of the intervention have been published elsewhere [11, 25].

Analysis

Repeated measures 2×3 multivariate analyses of variance were conducted using SPSS 9.0 for Windows, to assess treatment effects from pre-treatment and 6- and 18-month follow-up. The analyses conducted used an intention-to-treat analysis, using the last-observation-carried-forward method. A level of significance of $P < 0.05$ (one-tailed tests) was accepted as the level of significance for the trial. Results of changes between pre- and post-treatment and 6-month follow-up have been presented elsewhere [11]. Means of pre-treatment and 6- and 18-month follow-up are presented in Table 1.

In addition to the parametric analyses, it was also important to determine whether significant changes are of clinical significance. For this reason, scores on the HADS anxiety and depression scales were re-analysed using the cut-off scores conventionally taken to indicate a possible (>7) or probable (>10) clinical problem [16].

The results on the HAQ were also re-analysed according to the degree of improvement shown by patients over time between pre-treatment and 18-month follow-up. The following categories were determined: (i) worse—a deterioration in function of

TABLE 1. Means (and standard deviations) for treatment group by occasion for pre- to post-treatment comparisons (Pre and Post, respectively) and 18-month follow-up (F/up)

Measure	Adjunctive CBT			Routine clinical management		
	Pre	Post	F/up	Pre	Post	F/up
HAD depression[a]	5.1 (3.9)	4.3 (2.8)	4.6 (3.1)	5.3 (3.2)	6.3 (4.3)	6.7 (4.3)
Coping Strategies Questionnaire (CSQ)	54.7 (28.0)	70.6 (26.3)	57.6 (31.82)	42.5 (21.4)	48.5 (31.7)	45.7 (25.4)
HAD anxiety[a]	8.6 (5.4)	7.4 (4.7)	7.1 (4.7)	7.2 (4.6)	7.5 (5.6)	8.2 (4.6)
Pain	37.6 (34.3)	27.0 (26.4)	26.7 (24.5)	46.7 (41.9)	45.0 (47.8)	44.2 (52.2)
Health Assessment Questionnaire (HAQ)[a]	0.73 (0.6)	0.67 (0.5)	0.65 (0.6)	0.67 (0.6)	0.75 (0.5)	0.85 (0.7)
Ritchie Articular Index	15.7 (11.6)	13.3 (10.7)	12.0 (9.8)	13.2 (9.1)	14.4 (11.1)	11.8 (9.1)
ESR	23.7 (14.1)	18.4 (11.1)	25.5 (21.4)	30.3 (20.4)	26.8 (19.1)	30.6 (24.7)
CRP	18.2 (16.1)	15.2 (15.0)	15.1 (17.3)	25.8 (36.4)	27.2 (34.2)	20.3 (30.7)

[a]Group $\times$ time differences significant at $P < 0.05$.

greater than 25%; (ii) unchanged—a score within 25% of the original score in either direction; (iii) (somewhat) improved—an improvement in HAQ scores of between 25 and 49%; and (iv) much improved—an improvement of 50% or greater on original HAQ scores. This method was used to calculate clinically significant changes in an earlier publication and ensures that measurement error can not be interpreted as responsible for observed changes [11]. χ^2-test was used to determine differences in rates of improvement between the two groups.

Results

Demographic and outcome variables at baseline were not significantly different between the groups except for CRP [$t = 4.98$; $P = 0.03$]. There were no other statistically significant differences between groups and no trends towards difference were observed for any of the other variables ($P > 0.1$). CRP was not correlated with any variables at baseline or outcome, except a weak correlation with ESR, and therefore has not been controlled for in the present analysis. More patients in the CBT group scored in the clinically anxious range on the HADS than in the standard care group. However, the groups did not differ significantly in their mean anxiety scores. All data were normally distributed with the exception of CRP and depression. Following data transformations, both of these variables met criteria for normal distribution allowing parametric statistics to be used. The variable of pain had outlying scores which were replaced with a score one above the rest of the data set for analysis [26].

Treatment effects—statistical changes

Between the initial, 6- and 18-month follow-up assessments, the CBT group became less depressed, while the opposite was true of the standard group (group × time interaction: $F(2, 51) = 5.357$; $P = 0.012$). This represented a large effect size of 0.5 for treatment-related changes in depression. There were no significant changes in disability between pre-treatment and 6-month follow-up, but there was an interaction between treatment group and time at 18-month follow-up, indicating that the two groups had different outcomes with regard to disability (group × time interaction: $F(2, 51) = 3.730$; $P = 0.030$). This indicated a large effect size of 0.45 for treatment-related changes in disability. Similarly, changes in anxiety had failed to reach significance at the 6-month follow-up, but a treatment effect was observed on anxiety at the 18-month follow-up (group × time interaction: $F(2, 51) = 4.378$; $P = 0.02$). The effect size for treatment-related changes in anxiety was also large (ES = 0.44). There were no significant differences between groups for pain level (group × time interaction: $F(2, 51) = 2.577$; $P = 0.115$) or coping strategies (group × time interaction: $F(2, 51) = 0.067$; $P = 0.40$).

No significant differences were observed for either ESR level (group × time interaction: $F(2, 51) = 1.274$; $P = 0.264$) or CRP level (group × time interaction: $F(2, 51) = 0.90$; $P > 0.40$). Over the three assessments, both the CBT group and the standard group demonstrated comparable changes in RAI level (main effect for time: $F(2, 51) = 5.181$; $P = 0.01$) and there was no difference between the groups at follow-up (group × time interaction $F(2, 51) = 0.385$; $P > 0.4$)

Treatment effects—clinical significance

In the 6-month follow-up [11], the base rate of 'possible' depression had more than halved in the CBT group from 17% at pre-treatment to 4% at 6-month follow-up. In contrast, the proportion of cases in the 'possible' depression range doubled from 14 to 31% [11]. At 18-month follow-up, the proportion of the sample in the standard care group with possible depression remained relatively stable at 33%, with 19% of the standard care group scoring above the cut-off point for probable depression. In the CBT group, only 13% scored as possible cases of depression with only one participant (4%) remaining in the probable range. All participants who scored in the clinical range for depression over the course of the trial had been in the clinical range at pre-treatment. There were no new cases of clinical disorder, according to the HADS, in the treatment group (see Fig. 2).

For anxiety, the rate of clinically significant levels of anxiety was higher at pre-treatment for the CBT group (see Fig. 3), with 57% scoring above the cut-off point for possible anxiety, almost all of whom met criteria for probable cases of clinical anxiety (52%). The proportion of participants scoring above the cut-off for possible anxiety at 6-month (52%) and 18-month follow-up (50%) remained constant in the CBT group. However, the proportion scoring in the probable range for clinical disorder reduced from 52% at pre-treatment, to 23% at 6-month follow-up and 26% at 18-month follow-up. In the control group, only 32% scored above the possible anxiety cut-off score at pre-treatment, with only 9% scoring in the probable range. At 6-month follow-up, there was an increase in the standard care group with 50% meeting criteria for a possible anxiety problem, of whom 31% met criteria for probable anxiety. Similarly, at 18-month follow-up, 50% of the standard care group still fell within the clinical

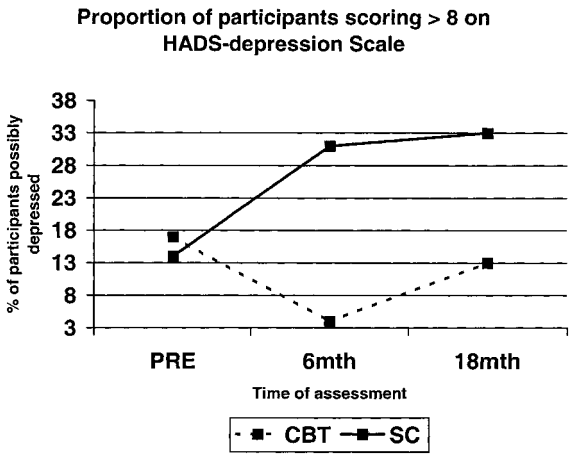

FIG. 2. Proportion of participants across treatment and at 18-month follow-up for CBT and standard care scoring as possible cases of depression (>8 on HADS depression scale). CBT, cognitive behavioural therapy with standard care; SC, standard care only.

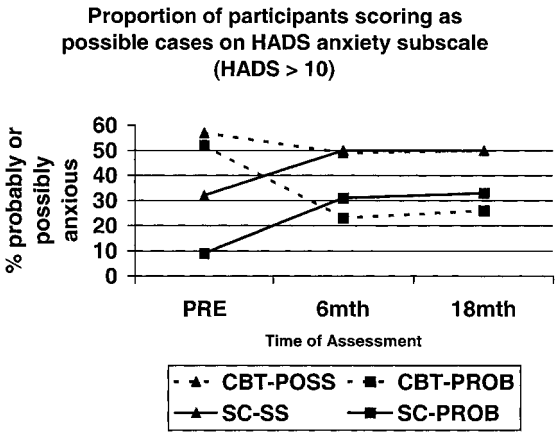

FIG. 3. Proportion of participants across treatment and at 18-month follow-up for CBT and standard care (SC) scoring as possible cases of anxiety (>8 on HADS anxiety scale). POSS, possible clinical anxiety (HAD-A = 8–9); PROB, probable clinical anxiety (HAD-A >10).

range (possibly anxious), with 33% scoring in the range of probable anxiety. These results indicate that while the proportion of those in the CBT group scoring either in the possible and/or probable group fell only slightly across time, the proportion in the control group increased. Importantly, for those in the CBT group initially scoring as probable cases, only half exceeded the criteria for probable anxiety at each follow-up period, indicating a reduction in anxiety symptoms likely to be clinically important.

For disability, analyses indicated that there were significant differences between the groups ($\chi^2 = 14.882$; $P < 0.001$). Examination of the resulting proportions in each category (Fig. 4) indicate that whereas 52% of the standard care group had become worse over time, that was true for only 13% of the CBT group. In the standard care group, 19% had remained the same, 19% were somewhat improved and 10% were much improved. For the CBT group, on the other hand, 52% were unchanged, only 4% were somewhat improved and nearly one-third (30%) were much improved. These results clearly indicate that CBT is helpful in protecting patients from continued deterioration in their level of function over time.

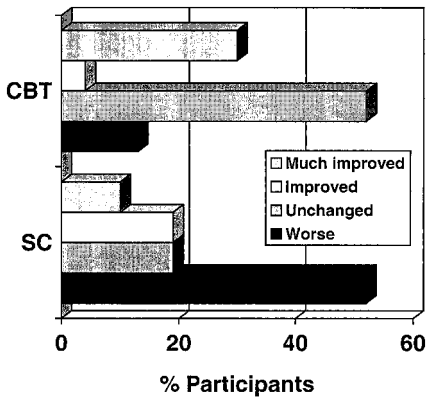

FIG. 4. Proportion of participants from standard care (SC) and CBT groups showing different changes in disability between pre-treatment and 18-month follow-up. Worse—more than 25% deterioration in disability; unchanged—follow-up disability score within 25% of pre-treatment disability; improved—25–50% improvement in disability; much improved—more than 50% improvement in disability.

Discussion

This study provides evidence of the longer-term efficacy of CBT as an adjunct to medical care in early RA. Strengths of this study include a high recruitment rate, relatively low attrition rate and participants recruited from three different hospital clinics, and therefore likely to be representative of patients with early RA attending rheumatology clinics. Levels of disability and disease parameters overall indicate that this was a relatively high functioning group receiving a good standard of medical care. Changes over time in the whole sample are likely to reflect genuine changes in the disease course. The intervention itself was manual-based and administered by two therapists of varying levels of experience. The results should therefore be generalizable to routine clinical practice.

Despite this careful attention to the study design and methodology, it also had limitations which must be considered when interpreting the results. The sample size, though sufficient to detect large and clinically significant effects, may have obscured other effects that were small to moderate. Nonetheless, large and clinically meaningful changes identified in this trial are those of most interest in developing services for patient care. Although the groups were not ideally matched, statistically significant differences between groups were observed for CRP favouring the CBT group. Scores on CRP did not correlate with the outcomes that changed significantly and therefore are unlikely to affect results. There was also a higher proportion of clinically anxious participants in the CBT group, despite the fact that the groups were not significantly different in their mean anxiety scores. The conventional HADS cut-off scores for possible and probable anxiety have been well validated and widely used, but they were not validated in the present study and may not have been optimal, giving rise to the apparent discrepancies just described. Nevertheless, they are more useful in demonstrating the clinical importance of symptoms than the mean scores. Many of the participants had above average scores on anxiety that, however, failed to score above the cut-off for probable or possible anxiety. Over time, these participants in the routine management condition developed clinically significant levels of anxiety when untreated. Given the initial differences in the rates of clinical disorders for anxiety, these results should be interpreted with caution.

Another limitation is the lack of a control group for therapist attention. Given the time and resources available for the study, and the projected recruitment rate, introducing a third arm into the trial would have resulted in inadequate statistical power. There are, however, two important reasons why support was unlikely to produce

the clinically significant and durable changes reported here. First, previous studies using attention control groups with patients with RA have failed to find extensive benefits of support-based interventions [2]. Second, in a recent prospective study of early RA, pain, attitudes and coping skills in addition to mood have been found consistently to predict depression over time [27]. CBT works specifically through changing patient's attitudes towards illness, their coping strategies and the way in which they manage pain. Hence, changes in these factors which have been shown to predict deterioration in mood are likely candidates for the mechanism of treatment change. These factors are not targeted in supportive interventions.

This study reports the 18-month follow-up of a single-blind, randomized, controlled trial of CBT for patients recently diagnosed with RA. The results partially supported the initial hypotheses. That is, for depression and joint function, gains made over treatment and 6-month follow-up [11] were maintained 12 months later, although for joint function the control group had made comparable improvements in that time. In the 12 months to the final assessment, the groups diverged significantly in anxiety and disability, with the CBT group showing significantly more favourable outcomes. However, no differences were observed for coping strategies, pain level or indices of disease activity.

For anxiety and depression, those patients who did not receive the psychological intervention showed a tendency to deteriorate over time. However, CBT appeared to prevent this deterioration over time, and led to small but consistent improvements. Indeed, despite the relatively small changes in mean scores, the effect sizes were large for treatment-related differences for both depression and anxiety. Moreover, changes in anxiety and depression appeared to represent clinically meaningful changes. Patients who entered the trial who were already in the clinical range (probable or possible) for depression or anxiety largely remained in the clinical range following treatment. However, there was a shift in the patients who had CBT in category from probable to possible range in half the participants for both mood states. Importantly, in the standard care group, the rate of likely clinical disorder actually increased substantially over time. The results for anxiety should be interpreted cautiously given the initial differences in the rates of likely clinical disorder between the CBT and standard care group. However, the results for depression are extremely robust. Indeed, not one participant in the CBT group who scored in the normal range on depression prior to treatment subsequently scored in the clinical range. This was not the case for those in the standard care group, many of whom developed clinically significant depressive symptomatology in the course of the trial. These results strongly suggest that CBT prevented the development of clinically significant levels of depression for a number of participants.

These results suggest that for people with early RA who have already developed clinically important symptoms of anxiety or depression, an eight-session CBT approach may be insufficient to ameliorate these symptoms completely, but may be important in preventing the development of clinically significant levels of distress. These results would suggest that offering intervention prior to the development of clinical depression or anxiety is likely to make a clinically important contribution to preventing the development of secondary psychological problems for patients with RA.

For joint function, the CBT group maintained the observed improvements reported at 6-month follow-up, although the standard care group made additional improvements over the ensuing 12 months. At 18-month follow-up, there was no difference between the groups. This is likely to reflect the fact that RA is known to be at its most aggressive within the first 2 yr of illness and therefore one would expect a reduction in the acute inflammation after that time. However, earlier changes in joint function are likely to have a long-term impact on disability [12, 13], which is consistent with the current findings. As with the psychological factors measured, disability showed small, but reliable changes over the 18 months. For the CBT group the initially low levels of disability slightly improved, whereas the standard care group had become slightly more disabled over time. This is consistent with expected increased disability as the disease progresses. Among those who did not receive the psychological intervention, over half showed worsening disability scores at follow-up, compared with 13% of those in the CBT group (Fig. 4). These results again suggest that CBT can play a preventative role in minimizing the expected deterioration in disability over the longer term course of illness.

Despite the positive changes found in the present study, an eight-session individual intervention is expensive and present data do not allow assessment of cost-effectiveness. However, there are reasons to think that intervening early may have benefits in terms of cost to health services. The present data would suggest that without intervention a high proportion of newly diagnosed patients are at risk of developing a depressive or anxiety disorder (or both). Typically once a clinical disorder has presented, psychological interventions require longer than eight sessions. The burden of untreated depression or anxiety is considerable, not only on the patient but also on his/her use of resources. Therefore, once a clinical disorder develops, the cost to health services for treatment is likely to be substantially higher than the intervention provided in the present trial. Moreover, if the effects of intervention on disability are maintained in the long term, the costs of the intervention are very likely to be offset by a reduction over the course of the disease in required health service resources. This, coupled with the benefits to participants in their quality of life, argues for increased availability of appropriate resources to manage the psychological needs of patients with RA.

Our results indicate that an intervention aimed at training patients in self-management skills, which they can subsequently use to cope with challenges imposed upon them by their illness, is likely to improve not only

psychological adjustment, but also long-term physical function. It is particularly important that the benefits of CBT for mood and disability have emerged despite initially low levels of disability and mood disturbance. The trial did not target individuals who showed mood disturbance at baseline, or who were identified as particularly vulnerable, but included a group of patients representative of those with early RA. These results suggest that there would be direct clinical benefits to patients in offering routine access to psychological services early in the course of illness. Clearly, further follow-up is required to determine whether the improvements demonstrated can influence the longer-term course of this chronic illness.

Acknowledgements

This study was supported by a grant from North Thames Regional Research and Development Programme. We are grateful to S. Davidson for her assistance in entering the data for this study. We also thank D. Holmes, West Middlesex University Hospital, R. Hughes and M. Carr, St. Peter's Hospital, Chertsey and the rheumatology staff at Charing Cross Hospital for their help in recruiting patients into the study. We would like to thank two anonymous reviewers for their helpful comments on an earlier version of this manuscript.

References

1. Young LD. Psychological factors in rheumatoid arthritis. J Consult Clin Psychol 1992;60:619–27.
2. Bradley LA, Young LD, Agudelo CA, Anderson KO, McDaniel LK. Effects of cognitive behavioural therapy on pain behavior of rheumatoid arthritis patients. Scand J Behav Ther 1985;14:51–64.
3. Applebaum KA, Blanchard EB, Hickling EJ, Alfonso M. Cognitive behavioural treatment of a veteran population with moderate to severe rheumatoid arthritis. Behav Ther 1988;19:489–502.
4. Achterberg J, McGraw P, Lawlis GF. Rheumatoid arthritis: a study of relaxation and temperature biofeedback training as an adjunctive therapy. Biofeedback Self Regul 1981;6:207–23.
5. O'Leary A, Shoor S, Lorig K, Holman HR. A cognitive behavioural treatment for rheumatoid arthritis. Health Psychol 1988;7:527–44.
6. Mitchell KR. Peripheral temperature autoregulation and its effect on the symptoms of rheumatoid arthritis. Scand J Behav Ther 1986;15:55–64.
7. Bradley LA, Young LD, Anderson KO *et al.* Effects of psychological therapy on pain behavior of rheumatoid arthritis patients: Treatment outcome and six month follow up. Arthritis Rheum 1987;30:1105–14.
8. Parker JC, Frank R, Beck N *et al.* Pain management in rheumatoid arthritis. Arthritis Rheum 1988;31:593–601.
9. Kraaimaat FW, Brons MR, Geenen R, Biljsma JWJ. The effect of cognitive behaviour therapy in patients with rheumatoid arthritis. Behav Res Ther 1995;33:487–95.
10. Parker JC, Smarr KL, Buckelew SP *et al.* Effects of stress management on clinical outcomes in rheumatoid arthritis. Arthritis Rheum 1995;38:1807–18.
11. Sharpe L, Sensky T, Timberlake N, Ryan B, Brewin CR, Allard S. The efficacy of a cognitive-behaviour therapy programme as a treatment for recently diagnosed rheumatoid arthritis: Preventing psychological and physical morbidity. Pain 2001;89:275–83.
12. Emery P. Therapeutic approaches for early rheumatoid arthritis: How early? Br J Rheumatol 1995;34:87–90.
13. Fex E, Jonsson K, Jonsson U, Eberhardt K. Development of radiographic damage during the first 5–6 years of rheumatoid arthritis. A prospective follow-up of a Swedish cohort. Br J Rheumatol 1996;35:1106–15.
14. Devlin J, Gough A, Huissoon A *et al.* The acute phase and function in early rheumatoid arthritis: C-reactive protein levels correlate with functional outcome. J Rheumatol 1997;24:9–13.
15. Arnett FC, Edworthy SM, Bloch DA *et al.* The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. Arthritis Rheum31:315–24.
16. Zigmond AS, Snaith RP. The Hospital Anxiety and Depression Scale. Acta Psychiatr Scand 1983;67:361–70.
17. Rosensteil AK, Keefe RJ. The use of coping strategies in chronic low back pain patients: relationship to patient characteristics and current adjustment. Pain 1983;17:33–44.
18. Spence SH, Sharpe L, Newton-John T, Champion D. Effect of EMG biofeedback compared to applied relaxation training with chronic, upper extremity cumulative trauma disorders. Pain 1995;63:199–206.
19. Fries JF, Spitz P, Kraines RJ, Holman HR. Measurement of patient outcome in rheumatoid arthritis. Arthritis Rheum 1980;23:137–45.
20. Sherrer YS, Bloch DA, Mitchell DM, Young DY, Fries JF. The development of disability in rheumatoid arthritis. Arthritis Rheum 1986;29:494–500.
21. Van der Heide A, Jacobs JWG, Haanen HCM, Bijlsma JWJ. Is it possible to predict the first year of pain and disability for patients with rheumatoid arthritis? J Rheumatol 1995;22:1466–70.
22. Ritchie DM, Boyle JA, McInnes JM *et al.* Clinical studies with an articular index for the assessment of joint tenderness in patients with rheumatoid arthritis. Q J Med 1968;127:393–8.
23. Spence SH. Long-term outcome of cognitive-behaviour therapy in the treatment of chronic, occupational pain of the upper limbs: a two year follow-up. Behav Res Ther 1991;29:503–9.
24. Lorig K, Fries JF. The arthritis helpbook. Boston: Addison-Wesseley, 1986.
25. Sharpe L, Sensky T, Timberlake N, Allard S, Brewin CR. The role of cognitive behavioural therapy in facilitating adaptation to illness in rheumatoid arthritis: A case series. Behav Cogn Psychother 2002;29:303–9.
26. Tabachnik B, Fiddel L. Using multivariate statistics, 3rd edn. New York: Harper & Collins, 1996.
27. Sharpe L, Sensky T, Allard S. The course of depression in recent onset rheumatoid arthritis: The predictive role of disability, illness perceptions, pain and coping. J Psychosom Res 2002;51:713–9.