

Cognitive-behavioral group therapy for obsessive–compulsive disorder: a 1-year follow-up

Braga DT, Cordioli AV, Niederauer K, Manfro GG. Cognitive-behavioral group therapy for obsessive–compulsive disorder: a 1-year follow-up.
Acta Psychiatr Scand 2005; 112: 180–186. © Blackwell Munksgaard 2005.

Objective: The aim of this study was to evaluate the results of cognitive-behavioral group therapy (CBGT) for obsessive–compulsive disorder (OCD) over a 1-year follow-up period.

Method: Forty-two OCD patients, who completed 12 sessions of CBGT, were followed for 1 year. Measures of the severity of symptoms were obtained at the end of the acute treatment and at 3, 6, and 12 months post-treatment using the Yale-Brown obsessive–compulsive scale (Y-BOCS) and the clinical global impression (CGI).

Results: The reduction in the severity of symptoms observed at the end of the treatment was maintained during 1 year ($F_{2,41} = 1.1$; $P = 0.342$). Eleven patients (35.5%) relapsed in the follow-up period. The intensity of improvement (log rank = 12.97, GL = 1, $P = 0.0003$) and full remission (log rank = 6.17; GL = 1; $P = 0.001$) were strong predictors for non-relapsing.

Conclusion: The CBGT is an effective treatment for OCD and its results are maintained for 1 year. However, further long-term randomized controlled trials are needed in order to confirm this finding.

**D. T. Braga, A. V. Cordioli,
K. Niederauer, G. G. Manfro**

Anxiety Disorders Program, Department of Psychiatry,
Hospital de Clínicas de Porto Alegre, Federal University
of Rio Grande do Sul, Porto Alegre, Brazil

Key words: obsessive–compulsive disorder;
psychotherapy; cognitive therapy; group; longitudinal
studies; recurrence

A. V. Cordioli, Department of Psychiatry, Universidade
Federal do Rio Grande do Sul. Rua: Ramiro Barcelos,
2350, Room 400N, 90035-903 Porto Alegre, RS–Brazil.
E-mail: acordioli@terra.com.br

Accepted for publication March 8, 2005

Introduction

Obsessive–compulsive disorder (OCD) is a chronic disorder with symptoms that rarely remit spontaneously (1, 2). In a naturalistic study that followed 144 OCD patients for 40 years, it was found that only 20% of these patients met criteria for remission (3). In another prospective study, the estimated probability for full remission in patients with OCD at the 2-year follow-up evaluation was only 12% (4).

Although the effectiveness of acute treatments for OCD is well established, it is still not known whether the results are maintained over time (5, 6). Eighty percent of patients relapse at 2 to 4 months after discontinuing anti-obsessional medication (7), and the maintenance of drug therapy seems to prevent relapses, especially if high doses are taken

for a long time (8). Moreover, relapses are less frequent and occur later when patients are treated with exposure and response prevention (ERP) in comparison to drug therapy (9–11).

Cognitive-behavioral group therapy (CBGT) is a cost-effective treatment alternative for OCD with further benefits (12, 13). It is not known, however, whether the response of this modality of treatment is maintained for a long period of time and which factors are associated with favorable outcomes. Several clinical features and predictors have been associated with short and long-term outcome in OCD patients treated with ERP, cognitive-behavioral therapy (CBT) and medications. Age at onset of the OCD symptoms (14, 15), severity of the symptoms at baseline (16–20), duration of the disease (17, 18, 20–22), and the intensity of overvalued ideas (23–25) were studied as predictors

of the outcome in the short-term. In the long-term, a fast response to treatment (26) and intensity of improvement (11, 19) were associated with the maintenance of therapeutic benefits. However, a high relapse rate was observed following the discontinuation of anti-obsessional drugs (7). Prevention of relapses with the continued use of anti-obsessional drugs was found in one study which suggested that patients receiving fluoxetine had numerically lower relapse rates compared with those receiving placebo, although the difference was not significant (8). In another study, O'Sullivan et al. (11) found that although clomipramine was no better than placebo in long-term outcomes, exposure therapy and compliance to the exposure therapy homework were associated with better outcomes.

Aims of the study

To investigate whether the reduction of the severity of OCD symptoms obtained with 12 sessions of CBGT would be kept for a 1-year period. It was also evaluated whether the clinical features most consistently demonstrated in the literature such as the age of OCD onset, severity of the symptoms, intensity of overvalued ideas at baseline, intensity of symptoms reduction, comorbidities and full remission at the end of treatment could be associated with relapses and better outcomes.

Material and Methods

Subjects

Patients from the Anxiety Disorders Program of the Hospital de Clínicas de Porto Alegre (HCPA) who were diagnosed with OCD according to the DSM-IV were selected to participate in CBGT. To be included in the study, patients should be aged between 18 and 65 years; have scores ≥ 16 on the Yale-Brown obsessive–compulsive scale (Y-BOCS); and, if in use of anti-obsessive drugs, they had to have been taking adequate and stable doses of these medications for at least 3 months. Sixty-five patients were initially evaluated and 18 were excluded for different reasons: depression with suicidal risk ($n=2$), OCD initiated after traumatic brain injury ($n=1$), severe social phobia ($n=2$), mental retardation ($n=1$), severe anorexia nervosa ($n=1$), severe personality disorders ($n=2$), or a score < 16 on the Y-BOCS ($n=3$). In addition, six patients, although suitable, refused treatment. The Ethics Committee of the HCPA approved the research and all participants

signed the written informed consent before entering the study (12).

Forty-four patients completed the CBGT based on a manual, from October 2000 through October 2001. The patients were treated in one of six groups (with a mean of seven patients per group) offering 12-sessions of treatment over 3 months provided by two therapists, both with an experience of at least 5 years in CBT.

Of the 44 patients that completed the 12 weekly protocols, 42 were evaluated during the follow-up period. Two patients were excluded: one was lost during follow-up and the other refused follow-up evaluation.

Methods

The patients were initially evaluated through a clinical interview by an experienced psychiatrist and the Mini International Neuropsychiatric Interview (MINI) (27, 28) was used to confirm the OCD diagnosis and to assess the presence of comorbidities. The ICD-10 Research Criteria was used for excluding severe personality disorders. The overvalued ideas scale (OIS) (23) was used at baseline in order to evaluate the intensity and fixidity of beliefs subjacent to OCD symptoms. Ten analog subscales with 10 points each compose this scale. The Y-BOCS (29) and the clinical global impression (CGI) (30) were used for assessing the severity of OCD symptoms at the end of CBGT and at 3, 6 and 12 month post-treatment. The Y-BOCS is comprised of 10 items with scores from 0 to 4, with five questions for obsessions and five for compulsions, and a maximum of 40 points. The CGI range is 1–7, comprising minimal to very severe OCD symptoms. In the follow-up period, the patients were evaluated by two independent evaluators, who did not participate in the CBGT sessions.

The group of patients that improved with CBGT (reduction $\geq 35\%$ on the Y-BOCS) was evaluated for maintenance of therapeutic response and relapse at 3, 6 and 12 months after the end of the treatment. It was considered a case of maintenance of therapeutic response when the patient had no changes on the Y-BOCS or on the CGI, and a case of relapse when the patient that had improved with the therapy had an increase $\geq 35\%$ on the Y-BOCS and CGI > 2 during the follow-up period. Additional treatment was offered to the patients that relapsed.

Full remission was considered to have been achieved when the patient presented a score < 8 on the Y-BOCS (4) and CGI < 2 ; and partial remission when there was a reduction $\geq 35\%$ on the Y-BOCS, but the total score on this scale was ≥ 8 and CGI = 2.

Statistical analysis

The maintenance of therapeutic gains was assessed by comparing the Y-BOCS and the CGI scores at four different moments: at the end of the treatment, and then at 3, 6, and 12 months after it, using repeated measures MANOVA. These tests were complemented by presentation of within-group effect sizes:

$$\text{Cohen's } d: ES = M_1 - M_2 / \sqrt{SD_1^2 + SD_2^2 - 2 \times r \times SD_1 \times SD_2}$$

The estimated distribution of relapses over time was calculated using a Kaplan–Meier curve. The log-rank test was used to observe the influence on the relapse rate of the following dichotomized factors: intensity of improvement, full remission, age at onset of the symptoms, symptom severity and intensity of overvalued ideas prior to the beginning of the treatment. The Fisher's exact test was used to evaluate the association between comorbidities and the relapse rates. The statistical analysis was carried out using the SPSS software, Version 10.0. The results were considered significant when $P < 0.05$.

Results

The sample included 42 subjects: 26 (61.9%) women and 16 (38.1%) men. The mean age was 36.8 years (± 13.2), the mean duration of the disease was 21.5 years (± 11.6) and the mean age of the patients at OCD onset was 14.8 years (± 6.9).

The majority of our sample (81%) had at least one comorbidity, 50% of our patients had two comorbidities and 31% had three psychiatric disorders diagnosed in Axis I. Twenty-six patients (61.9%) had comorbidity with anxiety disorders (panic disorder, agoraphobia, social anxiety disorder, post-traumatic stress disorder and generalized anxiety disorder); 22 (52.4%) had comorbidity with a depressive disorder (major depression and dysthymia); 15 (35.7%) had comorbidity with depressive and anxiety disorders, and seven (16.7%) presented other comorbidities such as eating disorders (anorexia nervosa and bulimia nervosa), impulse control disorders and tic disorders. Only eight patients (19%) did not present any comorbidity.

Seventeen (40.5%) patients were on pharmacological treatment for a long time (mean 12 years). Clomipramine was used in four patients (75–250 mg/day, mean = 175 mg/day), fluoxetine was used in nine patients (20–80 mg/day, mean =

45 mg/day), sertraline in one patient (100 mg/day) and paroxetine in one patient (60 mg/day). The combination of clomipramine (75 mg/day) and paroxetine (20 mg/day) was used in one patient and the other one was on clomipramine (75 mg/day) and fluoxetine (60 mg/day). After 1 year, 15 patients (35.7%) were still on pharmacological treatment. Six patients discontinued the use of medication and the others maintained the same dose of medication during the follow-up period. Of the patients that were not initially on medication, four started using one of the above-mentioned medications during the 1-year period (paroxetine 20 mg/day in one patient and the others started on fluoxetine 20–40 mg/day). Of the patients that relapsed, six sought additional CBT treatment.

Maintenance of therapeutic gains

The mean Y-BOCS score before CBGT was 25.6 (± 5.29). After the acute treatment the mean score decreased to 13.2 (± 7.42), and 31 (73%) patients were considered to have improved. This number increased to 32 (76%) 1 year after the treatment. The mean reduction in the scale rating scores was 48.4% at the end of the treatment and 54.5% 12 months later. However, 26.2% of our patients did not improve with the acute treatment or during the follow-up period.

Repeated measures MANOVA showed no significant difference on the Y-BOCS ($F_{3,123} = 1.44$; $P = 0.244$) or on the CGI ($F_{3,123} = 0.257$; $P = 0.799$) scores at 3, 6 and 12 months. The reduction in Y-BOCS scores and in CGI scores observed at the end of the treatment was maintained in the follow-up period as well as the calculated effect sizes (Table 1).

Full remission

At the end of the CBGT, nine (21%) patients showed full remission (Y-BOCS ≤ 8 and CGI < 2). During the follow-up period 16 (38%) patients met these criteria for full remission (Table 2).

Relapses

We studied the relapse rates in the subsample of 31 patients that had improved with therapy. Three months after finishing CBGT, 3 (9.7%) patients relapsed, four patients relapsed at 6 months (12.9%), and at 12 months four (12.9%) other patients relapsed. Therefore, in the follow-up period 11 patients (35.5%) relapsed. The estimated relapse distribution over 1 year was illustrated using the Kaplan–Meier survival curve (Fig. 1).

Table 1. Y-BOCS and CGI scores at the end of treatment and at 3, 6, and 12 months of the follow-up period [mean, SD and effect size (ES)] ($n= 42$)

Scale	Baseline (mean \pm SD)	Post-treatment (mean \pm SD), ES	3 months (mean \pm SD), ES	6 months (mean \pm SD), ES	12 months (mean \pm SD), ES
Y-BOCS	25.6 \pm 5.3	13.2 \pm 7.4, 1.54	11.8 \pm 8.8, 1.55	12.9 \pm 9.3, 1.3	11.6 \pm 9.7, 1.46
Obsessions	12.8 \pm 2.9	6.7 \pm 3.9	5.7 \pm 4.7	6.4 \pm 5.0	5.5 \pm 5.2
Compulsions	12.5 \pm 3.0	6.5 \pm 4.0	6.0 \pm 4.5	6.5 \pm 5.0	6.0 \pm 5.0
CGI	4.6 \pm 0.9	2.3 \pm 1.2, 2.01	2.1 \pm 1.3, 2.00	2.2 \pm 1.2, 2.20	2.2 \pm 1.2, 2.04

Analysis of variance for repeated measures (MANOVA), comparing the end of the treatment with 3, 6, and 12 months post-treatment (Y-BOCS: $F_{3,123} = 1.44$; $P = 0.244$; CGI: $F_{3,123} = 0.257$; $P = 0.799$).

Table 2. Number of patients with full remission, partial remission and those who had not improved at the end of treatment, and at 3, 6 and 12 months into the follow-up ($n= 42$)

	End of treatment [n (%)]	3 months [n (%)]	6 months [n (%)]	12 months [n (%)]
Not improved	11 (26)	7 (17)	13 (31)	10 (24)
Full remission	9 (21)	14 (33)	13 (31)	16 (38)
Partial remission	22 (52)	21 (50)	16 (38)	16 (38)

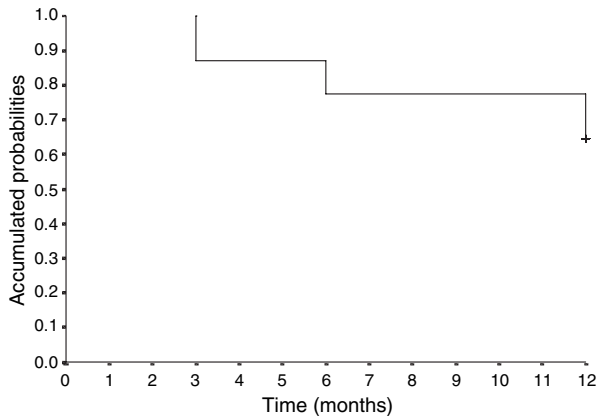


Fig. 1. Accumulated probabilities of patients relapsing over 1 year (Kaplan–Meier survival curve) ($n= 31$).

Predictors of relapses

For analysis of the influence of different factors on relapse rates, the group of patients that improved with treatment ($n= 31$) was dichotomized, using the mean values of the following parameters as criteria: intensity of improvement (reduction in Y-BOCS $\geq 54.5\%$ = more responsive OCD; reduction in Y-BOCS $< 54.5\%$ = less responsive OCD); intensity of overvalued ideas (IS ≥ 59 = more severe overvalued ideas; IS < 59 = less severe overvalued ideas); symptom severity (Y-BOCS ≥ 25 = more severe OCD, Y-BOCS < 25 = less severe OCD), full remission (Y-BOCS < 8 = presence of full remission, Y-BOCS ≥ 8 = absence of full remission) and age at OCD onset (≤ 18 years = early onset; > 18 = late onset). Patients that presented a reduction in Y-BOCS ≥ 54.5 with CBGT relapsed

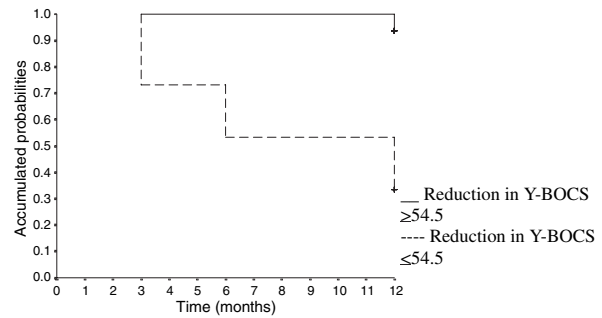


Fig. 2. Reduction in the severity of symptoms at the end of treatment and probability of relapses over a 1-year period (Kaplan–Meier survival curve).

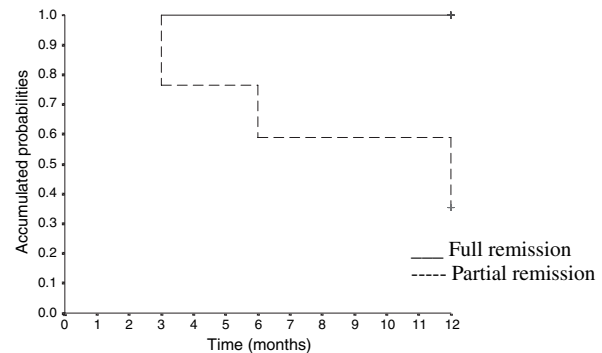


Fig. 3. Full remission at the end of treatment and probability of relapses over a 1-year period (Kaplan–Meier survival curve).

less than those presenting a lower reduction (log rank = 12.97, GL = 1, $P = 0.0003$) (Fig. 2). A significant difference was observed in the estimated rates of relapse over the period of 1 year for the patients that presented full remission at the end of treatment (Y-BOCS < 8) when compared with the other patients (log rank = 6.17; GL = 1; $P = 0.001$) (Fig. 3).

No significant difference was observed in the estimated relapse rates between the more severe patients at baseline, i.e. Y-BOCS scale ≥ 25 before CBGT compared with less severe ones (log rank = 0.1; GL = 1; $P = 0.75$). Also, no significant difference was found in patients that had more intense overvalued ideas (average of IS ≥ 59) compared

with those with less intense overvalued ideas (log rank = 0.18; GL = 1; $P = 0.67$), and in patients that had an early onset of OCD (age ≤ 18 years) compared with those with late onset (log rank = 1.54, GL = 1, $P = 0.21$). Moreover, no significant difference was observed in the estimated relapse rates between OCD patients with depressive disorders (Fisher's exact test; $P = 0.999$), anxiety disorders (Fisher's exact test; $P = 0.452$) or without comorbidity (Fisher's exact test; $P = 0.999$).

Discussion

For 1 year, the present study followed 42 patients that attended 12 weekly manualized CBGT sessions for 3 months. At the end of the acute treatment, 31 (73%) patients were considered to have improved, and 1 year later the number went up to 32 (76%). Of the patients that remitted, 20 (64.5%) maintained their gains over the 1-year post-treatment follow-up, and 11 (35.5%) relapsed. We found a 38% probability of achieving full remission and a 38% probability of achieving, at least, partial remission over the same period.

These data suggest that the effectiveness of CBGT was consolidated after 1 year and this modality of treatment can be carried out with OCD patients at any stage of their treatment once it can provide a reliable and lasting improvement (31). Our results are in agreement with the results of Barrett et al. (32), which found that cognitive-behavioral family group therapy was effective for child OCD and the results were also maintained at 3 and 6 months after the end of therapy. These results demonstrate the efficacy and durability of CBGT in treating both adult and child OCD patients.

We observed an increase in the number of patients achieving full remission during the follow-up period. This fact is in agreement with the results of De Haan et al. (19), which suggest that some patients had a late response to treatment. This finding is relevant for clinical practice, as it indicates that some patients need a longer treatment to show a reduction in symptom severity. The probability that our findings were related to late response to pharmacological treatment is unlikely, given the history of chronicity and the non-response to pharmacotherapy of our sample, yet, however, some patients may require longer periods to respond to CBGT.

Our data also suggest that the response from OCD patients to CBGT is quite diverse and it is still a challenge: some patients met criteria for full remission in a short-term at the end of the treatment and did not relapse over a year; but

the majority of the patients that had partial improvement relapsed. In addition, the intensity of the symptoms did not change in a small number of patients. As a matter of fact, 26.2% of our patients did not improve with the acute treatment or during the follow-up period, and the reasons for this non-response remain unclear. The heterogeneity of OCD as a disorder, considering etiology, clinical manifestations, treatment response and outcome may account for the differences found in this study. It is also possible that some non-specific factors like motivation, quality of therapeutic relationship, and the group effect may have influenced our results, yet they were not evaluated in this study.

This study also intended to identify factors that could be associated with relapses. The age at OCD onset (early or late), the comorbidities, the severity of the symptoms and the intensity of overvalued ideas at baseline were not associated with relapse. On the contrary, the intensity of symptom reduction at the end of the CBGT was found to be a strong predictor for non-relapsing. No patients with full symptom remission at the end of the treatment relapsed during the follow-up period. This finding suggests that the group of patients with a robust and fast response to CBGT tends to maintain their gains over time, which is in agreement with the literature (19). This finding is also of clinical relevance as full remission at the end of the treatment should be one of the main goals of any treatment modality. Our data also suggest that the failure in achieving full remission is associated with an increased risk of relapse and poor prognosis, as described in pediatric OCD (33) and other disorders such as depression (34, 35) and panic disorder (36). The addition of CBT should, therefore, be emphasized in any step of OCD treatment in order to achieve remission, once many studies have also demonstrated that CBT is very effective for OCD patients who do not have full response to medication (37, 38).

Our results are limited by the small sample size, absence of a control group and the influence of the use of medication and additional CBT in the follow-up period. Although outcomes cannot be attributed unequivocally to the CBGT, it is highly unlikely that our results are due simply to non-specific factors or maturation, given the chronicity of our sample. It is also unlikely that our results are due to pharmacological treatment because $< 50\%$ of our sample were on medications and the ones that used them, were using for a long time (mean 12 years). Also, the use of independent evaluators to assess the outcome reduces the likelihood that these results are driven by clinician expectation.

Some studies have shown that there are many similarities between clinical practice and randomized controlled trials and have suggested that CBT can be effective in routine OCD clinical practice (39), although additional controlled studies should be performed in order to evaluate the use of CBGT for OCD over the time.

Although the effectiveness of CBGT is documented in the literature and it appears to be a cost-effective treatment for OCD, application of this treatment is relatively limited, likely because of the lack of professionals trained in this modality. Although this study suggests that CBGT can be considered an effective treatment for OCD, and that its results are maintained for 1 year, long-term randomized controlled trials are necessary to evaluate whether the relapse rate with CBGT is smaller than it would be expected.

Acknowledgements

This work was supported by a grant from Coordenação de Aperfeiçoamento de Ensino Superior (CAPES), Ministry of Education, Brazil.

References

1. LENSI P, CASSANO GB, CORREDDU G, RAVAGLI S, KUNOVAK JL, AKISKAL HS. Obsessive-compulsive disorder: familial-developmental history, symptomatology, comorbidity and course with special reference to gender-related differences. *Br J Psychiatry* 1996;**169**:101-107.
2. RASMUSSEN SA, EISEN JL. The epidemiology and differential diagnosis of obsessive-compulsive disorder. *J Clin Psychiatry* 1994;**55**:5-14.
3. SKOOG G, SKOOG I. A 40-year follow-up of patients with obsessive-compulsive disorder. *Arch Gen Psychiatry* 1999;**56**:121-127.
4. EISEN JL, GOODMAN WK, KELLER MB et al. Patterns of remission and relapse in obsessive-compulsive disorder: a 2-year prospective study. *J Clin Psychiatry* 1999;**60**:346-351.
5. PICCINELLI M, PINI S, BELLANTUONO C, WILKINSON G. Efficacy of drug treatment in obsessive-compulsive disorder. A meta-analytic review. *Br J Psychiatry* 1995;**166**:424-443.
6. VAN BALKOM AJLM, VAN OPPEN P, VERMEULEN AWA, VAN DYCK R, NAUTA MCE, VORST HCM. A meta-analysis on the treatment of obsessive-compulsive disorder: a comparison of antidepressants, behavior and cognitive therapy. *Clin Psychol Rev* 1994;**14**:359-381.
7. PATO MT, ZOHAR KADOUCHE R, ZOHAR J, MURPHY DL. Return of symptoms after discontinuation of clomipramine in patients with obsessive-compulsive disorder. *Am J Psychiatry* 1988;**145**:1521-1525.
8. ROMANO S, GOODMAN W, TAMURA R, GONZALES J. Long-term treatment of obsessive-compulsive disorder after an acute response: a comparison of fluoxetine versus placebo. *J Clin Psychopharmacol* 2001;**21**:46-52.
9. FOA EB, GRAYSON JB, STEKETEE GS, DOPPELT HG, TURNER RM, LATIMER PR. Success and failure in the behavioral treatment of obsessive-compulsives. *J Consult Clin Psychol* 1983;**51**:287-297.

10. FOA EB, KOZAK MJ, STEKETEE GS, MCCARTHY PR. Treatment of depressive and obsessive-compulsive symptoms in OCD by imipramine and behavior therapy. *Br J Clin Psychol* 1992;**31**:279-292.
11. O'SULLIVAN G, NOSHIRVANI H, MARKS I, MONTEIRO W, LEILLIOT P. Six year follow-up after exposure and clomipramine therapy for obsessive-compulsive disorder. *J Clin Psychiatry* 1991;**52**:150-155.
12. CORDIOLI AV, HELDT E, BOCHI DB et al. Cognitive-behavioral group therapy in obsessive-compulsive disorder: a randomized clinical trial. *Psychother Psychosom* 2003;**72**:211-216.
13. VAN NOPPEN BL, PATO MT, MARSLAND R, RASMUSSEN SA. A time-limited behavioral group for treatment of obsessive-compulsive disorder. *J Psychother Pract Res* 1998;**7**:272-280.
14. ACKERMAN DL, GREENLAND S, BYSTRISKY A, MORGENSTERN H, KATZ RJ. Predictors of treatment response in obsessive-compulsive disorder: multivariate analyses from a multicenter trial of clomipramine. *J Clin Psychopharmacol* 1994;**14**:247-254.
15. MCLEAN PD, WHITTAL ML, THORDARSON DS et al. Cognitive versus behavior therapy in the group treatment of obsessive-compulsive disorder. *J Consult Clin Psychol* 2001;**69**:205-214.
16. BASOGLU M, LAX T, KASVISKIS Y, MARKS IM. Predictors of improvement in obsessive-compulsive disorder. *J Anxiety Disord* 1987;**2**:299-317.
17. GER P, KEIJERS J, CEES AL, HOOGDUIN L, CAS P, SCHAAP DR. Predictors of treatment outcome in the behavioural treatment of obsessive-compulsive disorder. *Br J Psychiatry* 1994;**165**:781-786.
18. STEKETEE G, EISEN J, DYCK I, WARSHAW M, RASMUSSEN S. Predictors of course in obsessive-compulsive disorder. *Psychiatry Res* 1999;**89**:229-238.
19. DE HAAN E, VAN OPPEN P, VAN BALKOM AJ, SPINHOVEN P, HOOGDUIN L, VAN DYCK R. Prediction of outcome and early vs. late improvement in OCD patients treated with cognitive behaviour therapy and pharmacotherapy. *Acta Psychiatr Scand* 1997;**96**:354-361.
20. DEARAÚJO LA, ITO LM, MARKS IM. Early compliance and other factors predicting outcome of exposure for obsessive-compulsive disorder. *Br J Psychiatry* 1996;**169**:747-752.
21. MAWSON D, MARKS IM, RAMM L. Clomipramine and exposure for chronic obsessive-compulsive rituals: III. Two-year follow-up and further findings. *Br J Psychiatry* 1982;**140**:11-18.
22. FOA EB, GOLDSTEIN A. Continuous exposure and complete response prevention in the treatment of obsessive-compulsive neurosis. *Behav Res Ther* 1978;**9**:821-829.
23. NEZIROGLU FA, STEVENS KP, YARYURA-TOBIAS JA. Overvalued ideas and their impact on treatment outcome. *Rev Bras Psiquiatr* 1999;**21**:209-216.
24. SALKOVSKIS PM. Obsessional-compulsive problems: a cognitive-behavioural analysis. *Behav Res Ther* 1985;**23**:571-583.
25. ITO LI, DEARAÚJO LA, HEMSLEY DR, MARKS IM. Beliefs and resistance in obsessive-compulsive disorder: observations from a controlled study. *J Anxiety Disord* 1995;**9**:269-282.
26. MARKS IM, HODGSON R, RACHMAN S. Treatment of chronic obsessive-compulsive neurosis by in-vivo exposure: a two-year follow-up and issues in treatment. *Br J Psychiatry* 1975;**127**:349-364.
27. SHEEHAN DV, LECRUBIER Y, SHEEHAN KH et al. The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic

- psychiatric interview for DSM-IV and ICD-10. *J Clin Psychiatry* 1998;**59**:22–33.
28. AMORIN P. Mini-International Neuropsychiatric Interview (M.I.N.I.): validation of a short structured diagnostic psychiatric interview. *Rev Bras Psiquiatr* 2000;**22**:106–115.
 29. GOODMAN WK, PRICE LH, RASMUSSEN AS et al. The Yale-Brown Obsessive-Compulsive Scale: development, use, and reliability. *Arch Gen Psychiatry* 1989;**46**:1006–1016.
 30. GUY W. ECDEU assessment manual for psychopharmacology. Washington, DC: U.S. Department of Health, Education, and Welfare, 1976;218–222.
 31. MARKS IM. The maturing of therapy: some brief psychotherapies help anxiety/depressive disorders but mechanisms of action are unclear. *Br J Psychiatry* 2002;**180**:200–204.
 32. BARRETT P, HEALY-FARRELL L, MARCH JS. Cognitive behavior family treatment of childhood obsessive-compulsive disorder: a controlled trial. *J Am Acad Child Adolesc Psychiatry* 2004;**43**:46–62.
 33. STEWART SE, GELLER DA, JENIKE M et al. Long-term outcome of pediatric obsessive-compulsive disorder: a meta-analysis and quantitative review of the literature. *Acta Psychiatr Scand* 2004;**110**:4–13.
 34. KENNEDY S. Full remission: a return to normal functioning. *J Psychiatry Neurosci* 2002;**27**:233–234.
 35. MCINTYRE R, KENNEDY S, BAGBY M, BAKISH D. Assessing full remission. *J Psychiatry Neurosci* 2002;**27**:235–239.
 36. FAVA GA, ZIELEZNY M, SAVRON G, GRANDI S. Long term effects of behavioral treatment for panic disorder with agoraphobia. *Br J Psychiatry* 1995;**166**:87–92.
 37. KAPMAN M, KEIJERS GP, HOOGDUIN CA, VERBRAAK MJ. Addition of cognitive behaviour therapy for obsessive-compulsive disorder patients non-responding to fluoxetine. *Acta Psychiatr Scand* 2002;**106**:314–319.
 38. SIMPSON HB, GORFINKLE KS, LIEBOWITZ MR. Cognitive-behavioral therapy as an adjunct to serotonin reuptake inhibitors in obsessive-compulsive disorder: an open trial. *J Clin Psychiatry* 1999;**60**:584–590.
 39. WARREN R, THOMAS JC. Cognitive-behavior therapy of obsessive-compulsive disorder in private practice: an effectiveness study. *J Anxiety Disord* 2001;**15**:277–285.