Impact of Alexithymia on treatment outcome: a naturalistic study of short-term cognitive-behavioral group therapy for panic disorder

Rufer, M; Albrecht, R; Zaum, J; Schnyder, U; Mueller-Pfeiffer, C; Hand, I; Schmidt, O
Impact of Alexithymia on treatment outcome: a naturalistic study of short-term cognitive-behavioral group therapy for panic disorder

Abstract

Background: It is often suggested in the literature that alexithymic patients are less responsive to psychotherapy than nonalexithymic patients. However, few empirical studies have examined this issue. Furthermore, it is unclear whether or not alexithymia itself may improve during psychotherapy.

Methods: Fifty-five consecutive outpatients with panic disorder received short-term cognitive-behavioral group therapy (CBGT) and were followed up 6 months later. Nineteen patients (35%) were on concomitant antidepressant medication. Alexithymia was measured using the 20-item Toronto Alexithymia Scale (TAS-20). Both completers and intention-to-treat analyses were calculated, taking into consideration the potentially confounding effect of comorbid conditions. Results: Baseline alexithymia did not predict outcome of CBGT, neither at posttreatment nor at follow-up. The presence of comorbid axis I disorders predicted nonresponse at posttreatment but not at follow-up. TAS-20 total scores decreased over time, with the TAS-20 factors 1 (difficulty identifying feelings) and 2 (difficulty describing feelings) decreasing significantly, while factor 3 (externally oriented thinking) remained largely stable. Conclusions: These findings are encouraging for cognitive-behavioral therapists working with patients with alexithymia who suffer from panic disorder: CBGT outcome does not appear to be negatively affected by alexithymia, and some alexithymic characteristics may even be reduced following CBGT. Assessing alexithymia at treatment onset may be useful for individually tailoring therapeutic interventions.
Impact of Alexithymia on Treatment Outcome: A Naturalistic Study of Short-Term Cognitive-Behavioral Group Therapy for Panic Disorder

Michael Rufer\textsuperscript{a} Renate Albrecht\textsuperscript{c} Johanna Zaum\textsuperscript{c} Ulrich Schnyder\textsuperscript{a} Christoph Mueller-Pfeiffer\textsuperscript{a, b} Iver Hand\textsuperscript{c} Oliver Schmidt\textsuperscript{c}

\textsuperscript{a}Department of Psychiatry, University Hospital of Zürich, Zürich, and \textsuperscript{b}Center of Education and Research, Psychiatric Services of the County of St. Gallen-North, Wil, Switzerland; \textsuperscript{c}Department of Psychiatry and Psychotherapy, University Hospital of Hamburg, Hamburg, Germany

Key Words
Alexithymia · Panic disorder · Psychotherapy

Abstract
Background: It is often suggested in the literature that alexithymic patients are less responsive to psychotherapy than nonalexithymic patients. However, few empirical studies have examined this issue. Furthermore, it is unclear whether or not alexithymia itself may improve during psychotherapy.

Methods: Fifty-five consecutive outpatients with panic disorder received short-term cognitive-behavioral group therapy (CBGT) and were followed up 6 months later. Nineteen patients (35\%) were on concomitant antidepressant medication. Alexithymia was measured using the 20-item Toronto Alexithymia Scale (TAS-20). Both completers and intention-to-treat analyses were calculated, taking into consideration the potentially confounding effect of comorbid conditions.

Results: Baseline alexithymia did not predict outcome of CBGT, neither at posttreatment nor at follow-up. The presence of comorbid axis I disorders predicted nonresponse at posttreatment but not at follow-up. TAS-20 total scores decreased over time, with the TAS-20 factors 1 (difficulty identifying feelings) and 2 (difficulty describing feelings) decreasing significantly, while factor 3 (externally oriented thinking) remained largely stable. Conclusions: These findings are encouraging for cognitive-behavioral therapists working with patients with alexithymia who suffer from panic disorder: CBGT outcome does not appear to be negatively affected by alexithymia, and some alexithymic characteristics may even be reduced following CBGT. Assessing alexithymia at treatment onset may be useful for individually tailoring therapeutic interventions.

Introduction
Alexithymia as a construct refers to deficits in the regulation of emotions [1–3]. It is related to a variety of physical and mental health problems [3, 4]. The typical features of alexithymia include difficulties in identifying and describing feelings, difficulties in differentiating between emotional states and physical sensations, a constricted imaginative activity, and an externally oriented cognitive style [1, 5]. Similarly, individuals with high anxiety sensitivity also show a proneness to focus on bodily sensations, based on their alertness to anxiety-related
Impact of Alexithymia on Psychotherapy Outcome

Psychopathology 2010;43:170–179

stati [6]. Empirically, there is evidence for a close relation between alexithymia and anxiety sensitivity [6–8].

Many patients with panic disorder (PD), which is associated with anxiety sensitivity [8], show disturbances in emotion regulation, have the tendency to misinterpret nonthreatening sensations of autonomic arousal as symptoms of bodily diseases, and are unable to relate such symptoms to psychological factors. Thus, a strong association of PD with alexithymia was hypothesized and empirically confirmed [8–15]. The prevalence of alexithymia in patients with PD was estimated in the range of 29–58% [e.g., 11, 15].

Alexithymia has also been shown to negatively influence treatment outcome in various conditions such as posttraumatic stress disorder [16], functional gastrointestinal disorders [17], and eating disorders [18]. None of these studies, however, specifically investigated the impact of alexithymia on the outcome of psychotherapy, although it has been suggested that alexithymic patients are less responsive to psychotherapy [19]. This view is mainly based on clinical experiences and theoretical assumptions. Proposed explanations include the difficulty of alexithymic patients to elaborate their emotions, their little interest in introspective and analytical cognitive activity, and their capacity to trigger negative therapist reactions [18, 20–22]. As a consequence, some authors recommended a supportive rather than change-oriented psychotherapeutic approach to alexithymic patients [23, 24]. Thus, the question of whether or not alexithymia predicts psychotherapy outcome is highly clinically relevant. Despite this, relatively few studies empirically investigated the role of alexithymia in predicting psychotherapy outcome. We electronically searched PubMed and PsycINFO databases for articles on this topic. The search terms ‘alexithymia’, ‘psychotherapy’, and ‘outcome’ were used, and additional articles were obtained from reference list searches. Summarizing these studies, there seems to be increasing evidence supporting the view that alexithymic patients respond poorly to psychodynamic psychotherapy (table 1). Although it has been suggested that alexithymia has a less negative impact on the outcome of cognitive-behavioral therapy (CBT) as compared to psychodynamic treatment [25], very few studies examined the predictive value of alexithymia for the outcome of CBT, yielding inconsistent results (table 2).

We therefore conducted a prospective study of outpatients who received CBT for PD (including exposure therapy), and evaluated (1) the predictive value of alexithymia for the outcome of CBT, taking into specific consideration the possible confounding effects of comorbid depressive symptoms, and (2) the degree to which alexithymia can change over time. Based on the results of previous studies on CBT for obsessive-compulsive disorder [26, 27], we hypothesized that (1) the outcome would not be negatively affected by alexithymia, and (2) CBT with exposure therapy, during which patients were asked to describe precisely the intensity and the different qualities of their emotions, would go along with improvements in the typical feature of alexithymia ‘difficulties in identifying and describing feelings’.

Method

Patients

The study was approved by the ethics committee of the Ärztekammer Hamburg, Germany. Patients were consecutively recruited between February 2004 and October 2005 from the Behaviour Therapy Outpatient Unit of the Department of Psychiatry and Psychotherapy (University Hospital of Hamburg, Germany). To be included in the study, patients had to be diagnosed by an experienced clinician with the primary diagnosis of PD with or without agoraphobia and to score 7 or higher on the clinician-rated version of the Panic and Agoraphobia Scale (PAS), an empirically derived cutoff indicating at least mild symptom severity [28]. The Mini-International Neuropsychiatric Interview [29] for DSM-IV [German version: 30], a reliable and valid, structured diagnostic interview, was used to confirm the diagnosis of PD with or without agoraphobia and to establish comorbid diagnoses. Exclusion criteria were current or past psychotic disorders, organic mental disorders, current substance dependence, and an acute risk of suicide (as assessed by an experienced clinician). All other comorbid disorders were allowed as we intended to evaluate a clinically representative sample of PD outpatients.

A total of 55 consecutive patients were eligible for this study, none of whom refused to participate. Written informed consent was obtained from all patients prior to study enrollment. Thirty-two (58%) out of 55 patients had a diagnosis of PD without agoraphobia, 23 patients (42%) PD with agoraphobia. These subgroups were not analyzed separately because of the small sample size. One or two comorbid axis I disorders were diagnosed in 22 patients (40%). The most frequent diagnosis was major depression (n = 15, 28%); others were obsessive-compulsive disorder (n = 4), dysthymia (n = 3), social phobia (n = 2), anorexia nervosa (n = 1), alcohol abuse (n = 1), and generalized anxiety disorder (n = 1).

Posttreatment data were provided by 50 patients (91%). Noncompleters either finished treatment but refused to participate in posttreatment assessment (3 patients) or dropped out of treatment due to noncompliance (2 patients, both after the second session). The latter were reassessed after they dropped out; these data were used for intention-to-treat analyses (see ‘statistical analyses’). Six months after the end of therapy, all treatment completers were asked to participate in a follow-up assessment. Those who refused to come for a face-to-face interview were offered an interview by telephone. Forty-three patients (78% of all originally enrolled in the study) participated in the 6-month follow-up (36 by face-to-face interview and 7 by telephone) while the remaining 7
Comparisons of treatment completers with noncompleters and follow-up completers with noncompleters showed no significant differences in gender, age, pretreatment scores of PAS, 20-item Toronto Alexithymia Scale (TAS-20), and Beck Depression Inventory (BDI) (data not shown, all p > 0.19).

Table 1. Empirical studies on the predictive value of alexithymia for the outcome of psychodynamic therapy

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>Diagnoses</th>
<th>Assessment of alexithymia</th>
<th>Therapy approach</th>
<th>Mean duration of therapy (weeks)</th>
<th>Assessment of outcome</th>
<th>Time point of outcome assessment</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>McCallum et al. [61]</td>
<td>251</td>
<td>Complicated grief and different axis I and II disorders</td>
<td>TAS-20</td>
<td>(1) Interpretive outpatient therapy (individual and group therapy) (2) Supportive outpatient therapy (individual and group therapy)</td>
<td>(1) 12 (2) 20</td>
<td>Different scales, including TRIG</td>
<td>Post-treatment</td>
<td>Alexithymia predicted poor outcome in all treatment groups</td>
</tr>
<tr>
<td>Ogrodniczuk et al. [62]</td>
<td>33</td>
<td>Major depression (subsample of Ref. 61; all patients were therapy responders)</td>
<td>TAS-20</td>
<td>Interpretive and supportive individual outpatient therapy</td>
<td>20</td>
<td>BDI</td>
<td>Post-treatment</td>
<td>Difficulty in identifying feelings predicted the persistence of depressive residual symptoms</td>
</tr>
<tr>
<td>Ogrodniczuk et al. [21]</td>
<td>107</td>
<td>Complicated grief and different axis I and II disorders (subsample of Ref. 61)</td>
<td>TAS-20</td>
<td>Interpretive outpatient therapy (individual and group therapy)</td>
<td>12</td>
<td>Different scales, including TRIG</td>
<td>Post-treatment</td>
<td>Alexithymia predicted poor treatment outcome</td>
</tr>
<tr>
<td>Spitzer et al. [63]</td>
<td>149</td>
<td>Different axis I and II disorders</td>
<td>TAS-20</td>
<td>Psychodynamic inpatient therapy</td>
<td>Not available</td>
<td>IIP</td>
<td>Post-treatment</td>
<td>Alexithymia did not predict changes in interpersonal problems, but high-alexithymic patients had still more interpersonal problems at the end of treatment than low-alexithymic patients</td>
</tr>
<tr>
<td>Simson et al. [64]</td>
<td>48</td>
<td>Different axis I and II disorders</td>
<td>LEAS; TAS-20</td>
<td>Psychodynamic inpatient therapy</td>
<td>8.5</td>
<td>GAF</td>
<td>Post-treatment</td>
<td>High scores in emotional awareness (LEAS) predicted good treatment outcome, but TAS-20 scores were not associated with the treatment outcome</td>
</tr>
<tr>
<td>Grabe et al. [65]</td>
<td>297</td>
<td>Different axis I and II disorders</td>
<td>TAS-20</td>
<td>Psychodynamic inpatient therapy</td>
<td>8–12</td>
<td>GSI of SCL-90-R</td>
<td>Post-treatment</td>
<td>Alexithymia did not predict poor treatment outcome, but alexithymic patients suffered more from psychopathological distress at discharge than nonalexithymic patients</td>
</tr>
<tr>
<td>Leweke et al. [66]</td>
<td>480</td>
<td>Different axis I disorders</td>
<td>TAS-26</td>
<td>Psychodynamic inpatient therapy</td>
<td>Short-term (limited to 4 weeks) and long-term therapy (8–12 weeks)</td>
<td>GSI and depression subscale of SCL-90-R</td>
<td>Post-treatment</td>
<td>Alexithymia predicted less favorable outcome, especially in patients with somatoform disorders</td>
</tr>
</tbody>
</table>

TRIG = Texas Revised Inventory of Grief; IIP = Inventory of Interpersonal Problems; LEAS = Levels of Emotional Awareness Scale; GAF = Global Assessment Scale of Function; GSI = Global Severity Index.

patients could not be traced or refused to participate. Comparisons of treatment completers with noncompleters and follow-up completers with noncompleters showed no significant differences in gender, age, pretreatment scores of PAS, 20-item Toronto Alexithymia Scale (TAS-20), and Beck Depression Inventory (BDI) (data not shown, all p > 0.19).

**Treatment**

All patients were treated with short-term cognitive-behavioral group therapy (CBGT) for PD. Each group comprised 5–7 patients, and treatment was conducted by 2 psychotherapists. Therapists were either licensed cognitive-behavioral therapists, skilled and experienced in treating patients with anxiety disorders, or
had reached an advanced level in their CBT training. They all received supervision on a weekly basis.

The fully manualized CBGT protocol [31] consisted of 5 weekly sessions of 150 min each (including a 15-min break). Sessions 1 and 2 included psychoeducation about normal anxiety, anxiety disorders, coping strategies, and the treatment of anxiety disorders. The therapists presented and discussed a cognitive-behavioral model of panic attacks (‘vicious circle of anxiety’), the relationship of stress and anxiety, and emphasized the role of avoidance in maintaining anxiety. Both sessions finished with 20 min training in progressive muscle relaxation skills. As part of their assigned homework, patients were asked to self-monitor their symptoms and thoughts before, during and after panic attacks. In addition, they were required to practice relaxation at least once daily.

At the beginning of each therapy session, patients were asked about their success and possible difficulties with their assigned homework. From our clinical observation, the great majority of patients completed most of their homework. As pointed out by Lynam et al. [25], the compulsive nature and external focus of people with alexithymia possibly prompt good adherence to structured exercises and behavioral recommendations. However, the adherence to homework was not formally assessed in our study.

Session 3 focused on therapist-guided exposure to panic-like physical sensations by running up a flight of stairs to get patients’ hearts racing. Therapists used patients’ reports of symptoms of anxiety elicited during exposure to teach management of anxiety and other negative emotional states, in order to improve their ability to cope with distressing emotions [32]. Patients were encouraged to describe precisely the intensity and the different qualities of their emotions. In addition, therapists asked them for their accompanying cognitions. For example, when patients judged their physical sensations as dangerous, they were asked to evaluate the validity of their catastrophic thoughts and to try to change them into more rational ones. At the end of the session, patients compared their new experiences with their earlier expectations (cognitive restructuring). They were also asked to do self-exposure homework and apply the coping strategies learned during therapy. Sessions 4 and 5 focused on cognitive restructuring with an emphasis on the evaluation of patients’ self-exposure experiences and their individual beliefs and appraisals. The therapy finished with the discussion of relapse prevention strategies [for a more detailed description of the CBGT protocol, see the treatment manual, 31]. A recent study showed that quality of life of patients with PD improved following this short-term group therapy program [33].

Patients were allowed to continue psychopharmacological medication taken before entering the study if this therapeutic regimen had remained stable for at least 2 weeks prior to the first CBGT session. Nineteen patients (35%) received concomitant medication. Sixteen patients were treated with either a selective serotonin reuptake inhibitor (SSRI) or a tricyclic antidepressant, 2 patients with low-dose benzodiazepines, and 1 patient with two different antidepressants in combination with a low-dose benzodiazepine. Nearly all patients kept their medication stable throughout the group therapy; only 2 patients switched to another antidepressant and 3 patients stopped medication. None of the patients started new medication during group therapy. There were also relatively few medication changes during the 6-month follow-up period. Four patients started antidepressant medication, 1 patient added an SSRI to the existing tricyclic antidepressant medication, and another patient added a low-dose benzodiazepine to the existing SSRI medication.

### Table 2. Empirical studies on the predictive value of alexithymia for the outcome of cognitive-behavioral therapy

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>Diagnoses</th>
<th>Assessment of alexithymia</th>
<th>Therapy approach</th>
<th>Mean duration of therapy weeks</th>
<th>Assessment of outcome</th>
<th>Time point of outcome assessment</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bach and Bach</td>
<td>30</td>
<td>Panic disorder or somatoform disorder</td>
<td>TAS-26</td>
<td>Inpatient cognitive behavioral therapy</td>
<td>&gt;8</td>
<td>SCL-90-R</td>
<td>2-year FU</td>
<td>Alexithymia predicted poor treatment outcome</td>
</tr>
<tr>
<td>Ruder et al.</td>
<td>42</td>
<td>Obsessive-compulsive disorder</td>
<td>TAS-20</td>
<td>Inpatient cognitive behavioral therapy</td>
<td>10</td>
<td>Y-BOCS</td>
<td>Posttreatment and 6-year FU</td>
<td>No association of alexithymia with outcome, neither at posttreatment nor at FU</td>
</tr>
<tr>
<td>Rosenblum et al.</td>
<td>230</td>
<td>Alcohol and drug abuse</td>
<td>TAS-20</td>
<td>(1) Outpatient cognitive behavioral group therapy (2) Outpatient group motivational intervention</td>
<td>10</td>
<td>Time of abstinence from alcohol and drugs</td>
<td>15-week FU</td>
<td>Patients with more severe alexithymia derived greater benefit from cognitive behavioral therapy than from motivational intervention program</td>
</tr>
<tr>
<td>Spek et al.</td>
<td>201</td>
<td>Subthreshold depression</td>
<td>TAS-20</td>
<td>Cognitive behavioral therapy</td>
<td>Not available</td>
<td>BDI</td>
<td>Posttreatment and 1-year FU</td>
<td>Baseline alexithymia scores were not correlated with treatment outcome</td>
</tr>
</tbody>
</table>

FU = Follow-up; Y-BOCS = Yale-Brown Obsessive-Compulsive Scale.
None of the patients received additional psychotherapy during the CBGT period (ongoing psychotherapies were discontinued before CBGT started). At follow-up, 11 patients were receiving individual psychotherapy.

**Measures**

Experienced and specifically trained, independent interviewers who were not involved in the treatment process conducted all clinician ratings. A given patient was always assessed by the same interviewer. Alexithymia was evaluated with the German version [34] of the TAS-20 [35], a reliable and valid self-rating measure of the alexithymia construct. Empirically validated, this instrument contains the following factors: difficulty identifying feelings (factor 1), difficulty describing feelings (factor 2), and externally oriented thinking (factor 3). The TAS-20 is the most widely used measure of alexithymia, and the generalizability of its 3-factor structure was demonstrated across languages and cultures, including the German version used in the present study [36].

The clinician-rated version of the PAS [37, German version: 38] was used as the main outcome measure, together with the clinician-rated version of the global improvement item (ranging from 1 = ‘very much improved’ to 7 = ‘very much worse’) of the Clinical Global Impression Scale (CGI) [39, German version: 40]. The PAS contains 13 items with Likert-type scales (from 0 to 4). The scale was validated on a cross section of 452 patients with PD [38]. In addition, previous studies demonstrated that the PAS is appropriate for use in a variety of clinical and research environments and, because of its sensitivity to changes over time, it is a useful tool for assessing treatment efficacy in PD trials [37, 41, 42]. Secondary outcome measures were the Hamilton Anxiety Rating Scale (HARS) [43, German version: 40] and the Global Severity Index of the Symptom Checklist-90-Revised (SCL-90-R) [44, German version: 45]. Symptoms of depression were assessed with the BDI [46, German version: 47].

**Statistical Analyses**

For between-group comparisons, we used $\chi^2$ tests for categorical variables and $t$ tests for continuous variables. The scores of the TAS-20, PAS, HARS, SCL-90-R, and BDI were submitted to analyses of variance (ANOVA) with repeated measures (pretreatment, posttreatment, and follow-up). When the sphericity assumption was violated, $p$ values were corrected using the Greenhouse-Geisser correction. Effect sizes were estimated using partial effect sizes $\eta^2$. According to Cohen [48], $\eta^2$ values of 0.0099, 0.0588 and 0.1379 correspond to small, medium and large effect sizes, respectively. We did not use the international threshold criterion for alexithymia (TAS-20 $\geq$ 61) because of the small sample size and since this cutoff criterion may not be representative for the German population [49]. Since high test-retest correlations of the TAS-20 would underline the usefulness of this variable as a potential predictor [18], we calculated Pearson correlations, intraclass correlations and Cronbach’s $\alpha$. Regression analyses were performed to evaluate the predictive value of alexithymia for the posttreatment and follow-up outcome of CBGT. For all analyses of treatment outcome, both completers and intention-to-treat analyses (carrying the last observation forward) were calculated. In all analyses, the level of significance was set at $p < 0.05$ (two-sided). The Statistical Package for Social Sciences, version 12.0.1 (SPSS, Chicago, Ill., USA) was used for all calculations.

**Results**

**Pretreatment Characteristics and Their Relationship with Alexithymia**

At pretreatment, patients had a mean PAS score of 21.4 ($\pm$ 7.8), indicating moderate severity of PD symptoms. Further clinical and demographical characteristics are listed in table 3. Alexithymia did not correlate significantly with age ($r = –0.19$, nonsignificant) and duration of illness ($r = –0.13$, nonsignificant), and there were no significant differences in alexithymia scores between men and women ($t = 0.74$, d.f. = 53, nonsignificant). A significant positive correlation was found between alexithymia and BDI depression scores ($r = 0.30$, $p < 0.05$) but not between alexithymia and PAS scores ($r = 0.22$, nonsignificant).

**Change in Anxiety and Depression over Time**

In the group of follow-up completers ($n = 43$), the repeated measure ANOVA showed a significant PAS reduction from 21.1 ($\pm$ 8.0) at pretreatment to 14.6 ($\pm$ 9.7) at posttreatment and 11.5 ($\pm$ 10.5) at follow-up [$F(2, 84) = 30.31$, $p < 0.001$] with a large effect size ($\eta^2 = 0.42$). Post hoc analyses revealed significant differences from both

| Table 3. Pretreatment demographic and clinical characteristics of 55 patients with PD |
|---------------------------------|--------|--------|
| **Mean** | **SD** |
| Age, years | 39.7 | 11.3 |
| Illness duration, years | 3.5 | 5.0 |
| TAS-20 total score | 50.0 | 9.6 |
| Factor 1 (difficulty in identifying feelings) | 18.0 | 6.0 |
| Factor 2 (difficulty in describing feelings) | 12.9 | 3.3 |
| Factor 3 (externally oriented thinking) | 19.2 | 3.9 |
| PAS | 21.4 | 7.8 |
| HARS | 20.0 | 9.0 |
| BDI | 14.5 | 8.7 |
| SCL-90-R GSI | 0.9 | 0.6 |

| Patients |
|---------|--------|--------|
| Gender, female/male | 34/21 | 61.8/38.2 |
| Married/not married (or cohabiting) | 44/11 | 80.0/20.0 |
| Education level |
| No secondary school degree (<10 years) | 13 | 23.6 |
| Secondary school degree (10 years) | 12 | 21.8 |
| High-school diploma (13 years) | 19 | 34.5 |
| University degree | 11 | 20.0 |

Rufer/Albrecht/Zaum/Schnyder/Mueller-Pfeiffer/Hand/Schmidt

Psychopathology 2010;43:170–179
Impact of Alexithymia on Psychotherapy Outcome

Pre- to posttreatment (t = 5.98, d.f. = 42, p < 0.001) and posttreatment to follow-up (t = 2.41, d.f. = 42, p = 0.02). BDI scores also improved significantly, with a medium to large effect size ($\eta^2 = 0.14$), from pretreatment (14.0 ± 7.8) to posttreatment (12.6 ± 10.2) and follow-up (10.9 ± 9.1) [F (2,84) = 6.23, p = 0.003]. Post hoc analyses showed a trend toward reduction from pre- to posttreatment (t = 1.88, d.f. = 42, p = 0.07) and posttreatment to follow-up (t = 1.69, d.f. = 42, p = 0.098). Similarly, the secondary outcome measures were significantly reduced over time: HARS: F (2,84) = 14.46, p < 0.001; SCL-90-R: GSI F (2,84) = 14.26, p < 0.001. Repeating all ANOVAs with missing data being imputed by carrying the last observation forward did not substantially change these results (all p < 0.006).

**Change in Alexithymia over Time**

Results presented in table 4 show a significant decrease in alexithymia total scores over time. The significant positive correlation between alexithymia and depression at pretreatment suggested that improvements in alexithymia might be at least partly explained by improvements in depression. Therefore, we controlled for the potential influence of depression by using a linear mixed model with repeated-measures covariates. Following this procedure, improvement of alexithymia over time remained significant [F (2,61.8) = 3.98, p = 0.024]. A closer examination on the TAS-20 factor level showed that the decrease in alexithymia total scores was mainly due to changes in factor 1 (difficulty identifying feelings) and factor 2 (difficulty describing feelings), whereas factor 3 (externally oriented thinking) remained almost completely stable over time (table 4).

To examine the relative stability of alexithymia, we correlated TAS-20 scores both between pre- and posttreatment and between posttreatment and follow-up. As shown in table 5, the test-retest reliability was high for both time periods.

**Predictive Value of Alexithymia for Outcome**

Two different outcome criteria were defined: (1) focusing on the panic and agoraphobia symptoms, outcome

---

**Table 4. Changes of alexithymia scores from pre- to posttreatment and to 6-month follow-up (n = 43)**

<table>
<thead>
<tr>
<th></th>
<th>Pretreatment</th>
<th>Posttreatment</th>
<th>Follow-up</th>
<th>ANOVAs (d.f. = 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>TAS-20 total score</td>
<td>F (84) = 9.18</td>
<td>p = 0.001</td>
<td>$\eta^2$ = 0.18</td>
</tr>
<tr>
<td>Factor 1</td>
<td>17.7 (6.0)</td>
<td>16.5 (5.1)</td>
<td>15.0 (5.8)</td>
<td>F (84) = 5.64, p = 0.012</td>
</tr>
<tr>
<td>Factor 2</td>
<td>12.7 (3.0)</td>
<td>11.8 (3.7)</td>
<td>10.8 (3.8)</td>
<td>F (84) = 7.50, p = 0.001</td>
</tr>
<tr>
<td>Factor 3</td>
<td>18.8 (4.0)</td>
<td>18.8 (4.2)</td>
<td>18.4 (4.5)</td>
<td>F (84) = 0.31, p = 0.740</td>
</tr>
</tbody>
</table>

ANOVA = Analyses of variance (all p values Greenhouse-Geisser corrected).

* Pre and post comparison: t = 2.3, d.f. = 42, p = 0.03; post and follow-up comparison: t = 2.55, d.f. = 42, p = 0.02.

* Pre and post comparison: t = 2.09, d.f. = 42, p = 0.04; post and follow-up comparison: t = 2.96, d.f. = 42, p = 0.05.

* Pre and post comparison: t = 1.83, d.f. = 42, p = 0.08; post and follow-up comparison: t = 2.18, d.f. = 42, p = 0.04.

**Table 5. Correlations between alexithymia scores at pretreatment, posttreatment and 6-month follow-up**

<table>
<thead>
<tr>
<th></th>
<th>Pre- to posttreatment (n = 50)</th>
<th>Posttreatment to follow-up (n = 43)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pearson's r</td>
<td>ICC r</td>
</tr>
<tr>
<td>TAS-20 total score</td>
<td>0.77**</td>
<td>0.75**</td>
</tr>
<tr>
<td>Factor 1</td>
<td>0.78**</td>
<td>0.75**</td>
</tr>
<tr>
<td>Factor 2</td>
<td>0.57**</td>
<td>0.55**</td>
</tr>
<tr>
<td>Factor 3</td>
<td>0.64**</td>
<td>0.63**</td>
</tr>
</tbody>
</table>

** p < 0.001. ICC = Intraclass correlation coefficient.
was defined as posttreatment PAS scores and follow-up PAS scores, respectively (controlled for baseline PAS scores) and (2) focusing on the global improvement, response to therapy was defined as CGI score of 1 or 2 (very much improved or much improved) at posttreatment and follow-up, respectively. Thus, according to the CGI, 25 out of 50 treatment completers (50%) were categorized as responders at posttreatment, while 19 of the 43 follow-up completers (44%) were rated as responders at follow-up.

Stepwise regression analyses were performed with the two outcome criteria as dependent, and pretreatment TAS-20 total scores as independent variables. The following pretreatment variables were also included as independent variables because of their potential impact on the therapy outcome: BDI scores, concomitant medication during therapy (0 = no medication; 1 = with medication), and the presence of comorbid axis I disorders (without comorbid disorder = 0; with comorbid disorder = 1).

Regarding the first outcome criterion (PAS scores), linear regression analyses were performed. Baseline PAS scores were first entered to control for baseline severity. The analyses revealed that alexithymia was neither related to the posttreatment nor to the follow-up outcome. Except for baseline PAS scores (p < 0.001, for both posttreatment and follow-up outcome), none of the independent variables reached significance and entered into the equation. Imputing missing data using the last-observation-carried-forward method did not change these results.

Regarding the second outcome criterion (CGI scores), logistic regression analyses for the posttreatment response revealed that the presence of a comorbid axis I disorder significantly predicted nonresponse to therapy ($R^2 = 0.11$, $\beta = -1.2$, $p = 0.04$). Neither alexithymia nor the other independent variables entered into the equation. None of the variables significantly predicted response at follow-up.

**Discussion**

The main aims of the present study were to evaluate (1) the predictive value of alexithymia for the outcome of CBGT for PD and (2) the degree to which alexithymia can change over time. The results showed (1) no significant impact of alexithymia on the posttreatment or follow-up outcome following CBGT and (2) a decrease in alexithymia over time, even after controlling for depression. This decrease was mainly attributable to changes in TAS-20 factors 1 (difficulty identifying feelings) and 2 (difficulty describing feelings), indicating that patients improved their ability to recognize their feelings and communicate them to others, while factor 3 (externally oriented thinking) remained largely stable.

Our finding that baseline alexithymia did not predict outcome of CBGT is in accordance with a study of patients with obsessive-compulsive disorder, showing that alexithymia, as assessed with the TAS-20, had no negative impact on the outcome of obsessive-compulsive disorder measured at posttreatment and at 6-year follow-up. In contrast, in a sample of inpatients suffering from somatoform disorder and PD, alexithymia, as assessed with the TAS-26, predicted persistent somatization following multimodal CBT. This study, however, suffered from a number of methodological shortcomings, including the lack of symptom assessment immediately after treatment. Thus, there seems to be increasing evidence suggesting that alexithymia has no negative impact on the outcome of CBT. This seems to be understandable because CBT does not primarily focus on insight, nor exclusively on verbal interventions, but on behavioral ‘experiments’ such as exposure. Alexithymic patients with PD may have new emotional experiences during exposure sessions (e.g., by doing physical exercises instead of avoiding activities because of their concerns about their physical health). This may encourage patients to challenge their dysfunctional beliefs and attitudes (e.g., ‘What did I learn from this exposure?’, ‘What does it tell me about my fears?’), which may ultimately lead to a reduction in symptoms of anxiety.

As an additional result, the presence of comorbid axis I disorders predicted posttreatment response as measured by the CGI. In earlier studies of PD, comorbidity with axis I disorders was also identified as one of a number of risk factors for poor treatment outcome. However, over the follow-up period, our patients with and without comorbid axis I disorders benefited comparably from the group therapy program. This is consistent with previous follow-up studies showing that group CBT targeting PD and agoraphobia is beneficial for patients both with and without comorbid conditions.

A further main finding was the significant decrease in TAS-20 total scores over time, and particularly in TAS-20 factors 1 and 2, even after controlling for depression. It is of note that the treatment protocol used in this study was clearly designed to reduce symptoms of PD and not alexithymia. Why then did alexithymia improve during this relatively short period of time? One reason could be that all patients received CBT including therapist-guided in vivo exposure with an emphasis on anxiety management.
A study of postmyocardial patients also found persistent therapeutic interventions. Alexithymia can be modified by different psychotherapeutic approaches other than CBT on alexithymia [55–57], controlled studies are needed to establish the degree to which alexithymia can be modified by different psychotherapeutic interventions.

Other elements of the group therapy program may also have led to reductions in alexithymia characteristics. By learning and practicing progressive muscle relaxation, including imagining a calm and comfortable place, and talking about their experiences to other group participants, alexithymic patients may have developed an increased awareness of the relation between their physiological arousal and emotions. This may have improved their ability to differentiate and regulate their affects by using psychological strategies, such as using imagination and communicating feelings to others. Friedlander et al. [54] reported that alexithymic individuals were no different from nonalexithymics in their ability to relax physiologically and subjectively during relaxation exercises. This supports our clinical observation that alexithymic patients are able to use relaxation as an affect regulation strategy, if they receive clear therapeutic guidance and instructions. Also psychoeducation about different components of anxiety (cognitive, somatic, emotional, and behavioral) and the training of a differentiation between these components during exposure may have contributed to the improved ability to identify and describe feelings.

However, because we did not include a control group, our data do not make it possible to draw any firm causal conclusions regarding our patients’ improvements in alexithymia. Therefore, and given that so far only few studies examined the effect of psychotherapeutic approaches other than CBT on alexithymia [55–57], controlled studies are needed to establish the degree to which alexithymia can be modified by different psychotherapeutic interventions.

On the one hand, the significant reduction of TAS-20 scores demonstrated a lack of absolute stability of alexithymia. The high test-retest correlations, which were comparable to those of several previous longitudinal studies of alexithymia in other mental disorders [e.g., 26, 58], however, indicated the relative stability of alexithymia. Thus, our findings are in line with the view that alexithymia should not be considered as either a trait or a state phenomenon but as a complex manifestation that includes both trait and state components [for a detailed discussion of the temporal stability of alexithymia, see 25].

In addition to the lack of a control group, some other important limitations of this study should be addressed. First, several factors may affect the generalizability of our results: all patients were recruited from a specialized CBT outpatient unit, were treated in groups, and had, on average, only moderate levels of PD. Moreover, the mean TAS-20 total score of 50.0 (±9.6) in our sample, although comparable to that reported by Bankier et al. [4], was considerably lower than those reported from other PD studies which had found TAS-20 total scores of 57.5 (±15.3) [10] and 61.3 (±8.0) [11]. Thus, our results may have been different, had we examined PD patients with higher levels of alexithymia. Second, it cannot be ruled out that with a larger sample size, we might have detected a significant relationship between alexithymia and CBGT outcome. A third limitation is that concomitant medication and individual psychotherapy (prior to CBGT and during the naturalistic follow-up) may have had a significant impact on therapy outcome. It is possible, for example, that individual psychotherapy has contributed to the reduction in PD symptomatology and the features of alexithymia ‘difficulty identifying feelings’ and ‘difficulty describing feelings’. However, in order to reduce the potentially confounding effect of additional individual psychotherapy, all patients who had psychotherapeutic treatment had to discontinue it before group therapy started.

Furthermore, we did not assess axis II comorbidity; therefore, the possible influence of comorbid personality disorders on treatment outcome could not be examined. Finally, the self-report nature of the TAS-20 is an important limitation. It is probably difficult for alexithymic patients to accurately rate their deficits [59]. Bagby et al. [60] recently reported on the reliability and validity of the Toronto Structured Interview for Alexithymia (TSIA), which is the first comprehensive structured interview with a specific focus on alexithymia. It includes specific prompts and probes, and may thus provide a more valid assessment of alexithymia. The German version of the
TSIA was recently evaluated [69]. It might be promising to use such a clinician-rated instrument in future studies to explore more in-depth the potential impact of alexithymia on psychotherapy outcome.

In conclusion, our results are encouraging for cognitive-behavioral therapists working with patients with alexithymia who suffer from PD: CBGT outcome does not appear to be negatively affected by alexithymia, and some alexithymic characteristics may even be reduced following CBGT. Future controlled studies should examine the degree to which alexithymia can be modified by different psychotherapeutic interventions.

Acknowledgment

The authors wish to thank Sabina Braun, Department of Psychiatry and Psychotherapy at the University Hospital of Hamburg, for her valuable help in patient recruitment.

References

Impact of Alexithymia on Psychotherapy Outcome

Psychopathology 2010;43:170–179