Magnetic Resonance Imaging Volumes of the Hippocampus and the Amygdala in Women With Borderline Personality Disorder and Early Traumatization

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Background: Based on findings of stress-induced neural disturbances in animals and smaller hippocampal volumes in humans with posttraumatic stress disorder, we hypothesized that patients with borderline personality disorders (BPD), who often are victims of early traumatization, have smaller volumes of the hippocampus and the amygdala. We assumed that volumes of these brain regions are negatively correlated with traumatic experiences and with neuropsychological deficits.

Methods: We studied 21 female patients with BPD and a similar group of healthy controls. We performed clinical assessments, a modified version of the Childhood Trauma Questionnaire, and magnetic resonance imaging volumetric measurements of the hippocampus, amygdala, temporal lobes, and prosencephalon. Neuropsychological testing included scales on which disturbances in BPD were previously reported.

Results: The patients with BPD had nearly 16% smaller volumes of the hippocampus ($P<.001$) and 8% smaller volumes of the amygdala ($P<.05$) than the healthy controls. The results for both hemispheres were nearly identical and were controlled for the volume of the prosencephalon and for head tilts. The volumes of the hippocampus were negatively correlated with the extent and the duration of self-reported early traumatization only when BPD and control subjects were considered together. Levels of neuropsychological functioning were associated with the severity of depression but not with the volumes of the hippocampus.

Conclusion: In female patients with BPD, we found reduction of the volumes of the hippocampus (and perhaps of the amygdala), but the association of volume reduction and traumatic experiences remains unclear.

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Research in animals has revealed a substantial impact of stress on regions of the brain that are rich in type II glucocorticoid receptors, ie, highly binding mineralocorticoid-like receptors. In animals under inescapable and uncontrollable psychosocial stress, functional and structural neural disturbances up to neural death were found. Similar changes were observed after artificial glucocorticoid exposition of the brain. Many of these findings refer to the hippocampus and, in agreement with these observations, memory dysfunctions were found in animals that were exposed to stressful conditions.

Research in humans mainly refers to long-term sequelae of traumatic stress (months to years). Nevertheless, some findings are in agreement with findings in animals, eg, smaller hippocampal volumes were observed by means of magnetic resonance imaging (MRI) measurement in patients with posttraumatic stress disorder (PTSD) and in female survivors of early sexual or physical abuse. Smaller volumes of this brain structure correlated with combat exposure and dissociative symptoms, while the associations with declarative memory functions remain unclear.

Unfortunately, the volumetric findings varied considerably with regard to the quantity of significant reductions in the volume of the right and/or left hippocampus (5% to 26%), and, apart from one study, were not controlled for the total brain volume. The amygdala was rarely investigated in clinical studies, although it is known for its associations with the hippocampus and memory functions, and the results are not in agreement. Smaller volumes of the amygdala were found by Bremner and colleagues, although neither of these findings was significant.
SUBJECTS AND METHODS

SUBJECTS

Twenty-one white female patients, aged 21 to 40 years, who were treated for BPD in the Department of Psychiatry, Luebeck School of Medicine, Germany, during 1997 to 1998 (18 inpatients, 3 outpatients) were included. All of them met DSM-IV criteria for BPD, as assessed by the treating psychiatrists within the first 3 days after admission. A history of traumatization was not an inclusion criterion for the study and was not assessed before patients were included in the study. Comparative subjects were healthy women who had never sought psychiatric or psychotherapeutic help. They were students or employees of the medical school or employees of local factories. They were included if they were similar to the patients according to the following requirements: sex (perfect match), race (perfect match), handedness (perfect match), age (±3 years), and years of education (±1 year). There were no significant differences between the groups on any of these variables (Table 1). However, patients with BPD reported fewer years of employment, were less frequently married, and more often lived alone than the healthy subjects. These differences are probably associated with the developing disorder.

Participants were neither pregnant nor currently or previously had any of the following medical conditions, which were assessed by the medical history, a careful clinical examination, and laboratory means: endocrine system disorders, malignant diseases, liver cirrhosis, neurological diseases, loss of consciousness for more than 10 minutes, or mental retardation. Further exclusion criteria were current infectious diseases, anorexia, schizophrenia, schizoaffective disorders, and major depressive disorder with psychotic symptoms. Patients with clinically diagnosed alcohol and/or drug dependence during the 6 months before the study were also excluded. Nine patients occasionally had received psychotropic medication during the 7- to 1-week period prior to the study, but all subjects had been drug free for at least 7 days before the assessments.

Patients and healthy subjects had given their written informed consent and were given financial remuneration for their efforts (maximum US $50 to $100, with the amount depending on the distance from home). The study was approved by the institutional review board (by the Ethics Commission of the Luebeck School of Medicine).

CLINICAL ASSESSMENTS

Participants completed the Structured Clinical Interview for DSM-IV Personality Disorders (SCID-II) according to the guidelines, and all BPD items were assessed by the interviewers in all cases. The SCID-I sections covering PTSD and all substance-related disorders were also administered. In addition, subjects were screened for alcohol-related disorders using the Luebeck Alcoholism Screening Test, which was shown to be highly sensitive (91%) and specific (87%). In addition, γ-glutamyltransferase levels were obtained and breath alcohol testing was performed at least 3 times in a 4-week period prior to the MRI and neuropsychological testing in the BPD group, and at least once in the healthy group. None of these test results were positive in any subject or at any point of measurement. All subjects reported not having used any illegal drugs during the 3 months before measurements. Urinary screening test results (Triage-Test; Merck, Darmstadt, Germany) were negative at least twice in the BPD patients and at least once in the control subjects and not positive in any subject at any time. Current psychopathologic status was assessed by means of the Beck Depression Inventory, and the State-Trait Anxiety Inventory.

The traumatization history was assessed by means of the 70-item version of the Childhood Trauma Questionnaire (CTQ), which includes all items of the 28-item version recently published. Data were analyzed according to the 4 dimensions (physical and emotional abuse, emotional neglect, physical neglect, and sexual abuse) and the total score. We added 4 corresponding items (time schedules) to obtain the periods (duration) of abuse or neglect according to the 4 subscales of the CTQ and with regard to the first 20 years of life.

In all subjects, a venous blood sample was obtained and red and white blood cells, thrombocytes, C-reactive protein, γ-glutamyltransferase, aspartate aminotransferase, alkaline phosphatase, and cholinesterase values were measured. No abnormal findings were found in any participant.

NEUROPSYCHOLOGICAL TESTING

Neuropsychological tests were administered, which in extended test batteries were found to indicate disturbances in BPD and which in part cover functions in which the hippocampus (and in parts the amygdala) are directly or indirectly involved. The Digit Symbol Test of the HAWIE-R measures psychomotor performance and speed and thereby gives some indications of the attention level. The Logical Memory Test of the Wechsler Memory Scale (German version by Calabrese et al, unpublished version) measures verbal (declarative) memory functions, i.e., immediate and delayed recall, separately, and allows analysis of the retention ratio. The Rey-Osterrieth Complex Figure Test was used to measure visual-perceptual integration (copying a figure) and visual-spatial memory functions by assessment of immediate and delayed recall. Visual-perceptual integration was also tested by means of the Embedded Figures Test. Time scores, the number of errors, and the number of representations needed were rated. Both variables (number of errors and number of representations needed) give information about the certainty with which the subject makes decisions. Aspects of intelligence were assessed by means of the 4 subscales of the 1986 version of the Reduced Wechsler Intelligence Scale.

MRI ACQUISITION AND PROCEDURES

All MRI scans were acquired by a 1.5-T Siemens Magnetom SP 4000 (Siemens, Erlangen, Germany). The 3-plane localizer and a sagittal localizer (with a mediosagittal right and left paramediosagittal slice) were acquired for representation of the individual anatomy and the position of the head (repetition time [TR], 300 milliseconds; echo time [TE], 15 milliseconds; 3 slices; thickness of slices, 5 mm;
field of view [FOV], 220 mm; distance factor, 5; matrix, 164×256 pixels). A MPRage (magnetization preparation rapid gradient echo) was plotted on the scans of the 3-plane localizer. It yielded a highly T1-weighted complete data set of the cranial volume of the brain in contiguous slices (TR, 10 milliseconds; TE, 4 milliseconds; 128 slices; time of inversion, 300 milliseconds; thickness of slices, 1.25 mm, interslice gap, 0 mm; matrix, 256×256 pixels; FOV, 250 mm; distance factor, 0; 3-dimensional partitions, 128; flip angle, 10°).

To control for neurological diseases T1-weighted (TR, 600 milliseconds; TE, 15 milliseconds; 20 slices; thickness of slices, 3 mm; FOV, 240 mm; distance factor, 8; matrix, 234×256 pixels) and T2-weighted sequences were obtained that ran perpendicular to the temporal lobe axis and to the longitudinal axis of the hippocampus. The T2-weighted (TR, 3000 milliseconds; TE, 90 milliseconds) and the proton-density-weighted sequences (TR, 3000 milliseconds; TE, 15 milliseconds [proton-density-weighted]; 20 slices; thickness of slices, 3 mm; FOV, 240 mm; distance factor, 0; matrix, 234×256 pixels) were plotted at the sagittal localizer. The 2×2 fold recalculation via paraxial images allowed the corrections of both sagittal and axial deviations.

The hippocampal longitudinal axis was directly located at the sagittal slice of the MPRage that met the gyrus dentatus and axial images were calculated perpendicular to this axis. This procedure allowed to control deviations of the head from the normal zero position caused by inclination or reclinasion. Axial deviations caused by head rotation were controlled by calculating fifteen 100% axial images (thickness of slices, 1 mm) covering the internal auditory canal and the seventh and eighth cranial nerves. These structures were used as landmarks for head rotation on the axial plane.33 Deviations were corrected by plotting 2-mm paraxial scans parallel to the longitudinal axis of the hippocampus throughout the whole temporal lobe. As the result of this procedure, parasagittal 2-mm slices of the midtemporal regions with adjacent structures could be calculated, which were corrected for axial deviation. The 2-fold recalculation via paraxial images allowed the correction of both sagittal and axial deviations.

Paracoronal scans, perpendicular to the longitudinal axis of the hippocampus, were used for volumetry of the hippocampus, the amygdala, and the temporal lobe. All calculations from the MPRAge were performed by the NUMARIS–3-D processing (version 2.02; Siemens). All data were saved on optical disks (Pioneer 502 A; Pioneer Electronic Europe, Beveren, Belgium).

MRI VOLUMETRIC MEASUREMENTS

The volumetric procedure was similar to that reported by Bremner et al.10,11 but with some substantial modifications. The prosencephalon was measured to exclude false-positive group differences of the volumes of interest due to global cerebral atrophy. The thickness of slices was 6 mm, and these slices ran parallel to the cranial basis. The volumetry of the prosencephalon included the cerebrum and most parts of the diencephalon as well as the lateral and the third ventricles, while excluding the rhombencephalon, the pons, the medulla oblongata, and the cerebellum. The prosencephalon was circumscribed on the border between the brain and the cerebrospinal fluid using the mouse cursor-driven system under NUMARIS.

Volumes of the temporal lobes were measured beginning with the first slice posterior to the superior colliculus including all slices (thickness, 4 mm) up to the most rostral aspects of the temporal lobe. The temporal lobe’s caudal and lateral demarcation at the coro-nal sites was distinctly visible because of the high contrast of the liquor-cortex transition. The demarcation line passed over the gyrus temporalis superior. At the lower rim of the lateral sulcus, it bent in a medial direction over the transverse temporal gyrus and reached the circular sulcus of the insula lobe. This point was connected with the most lateral edge of the bottom of the temporal cornu of the lateral ventricle by a straight line. This demarcation line met the hippocampus at the bottom of the lateral ventricle, following this structure at its caudal transition between gray and white matter up to the medial cortex of the temporal lobe. The choice of these borders and landmarks allowed the volumes of the temporal lobes to be measured without measuring the hippocampal and ventricular volumes. Thus, structures were included that are known to contain only few glucocorticoid receptors compared with the amygdala and the hippocampus.39

The demarcation of the hippocampus sometimes is associated with almost isointense signals of the neighboring structures, a problem of MRI-based volumetry that has been intensively discussed in previous studies.10,33,35,36 The outline of the hippocampus in our study enclosed 11 or 12 slices (thickness, 2 mm), beginning with the first slice posterior to the superior colliculus and ending at the most dorsal slice of the interpeduncular cistern. Thus, the whole hippocampal body and most of the tail was included in the measurement.34

A line was drawn from the inferior medial edge of the temporal horn following the white matter band that divides the gyr hippocampalis and parahippocampalis to the cortical surface of the hippocampal gyr.37 The demarcation line pulls over the cortex of the gyrus dentatus and the alveus. This white matter structure made an excellent visible boundary between the cerebrospinal fluid of the lateral ventricle and the CA3 region. By this boundary, the demarcation line met the point of the beginning of the outline at the bottom of the lateral ventricle.

The measurement of the amygdala complex began 1 slice dorsal of the recessus infundibularis and stretched in 2-mm contiguous slices to the most rostral slice, in which a connection between the white matter of the temporal lobe and the frontal lobe was definitely visible.33

Volumetric measurements were performed by two of us (J.H., K.S.) under the supervision of experienced radiologists (M.Z., M.O., D.P.), all of whom were unaware of the names and diagnoses of the subjects. Interrater reliability was sufficient, with average measure intraclass correlation coefficients ranging from 0.90 to 0.97 (median, 0.96) for the regions of interest. Test-retest reliability (J.H.) was also sufficient with intercorrelation coefficients ranging from 0.90 to 0.98 (median, 0.96).
DATA ANALYSES

The statistical analyses were performed using SPSS versions 7.01 and 8.0. The χ² test and the Mann-Whitney U test with z values corrected for ties were applied for the basic analyses of group differences. Analyses of covariance (ANCOVAs) were used to analyze group differences of MRI volumetric measurements of the volumes of interest with the volume of the prosencephalon as covariate (entered first). This procedure was necessary to control between-groups analyses for the interindividual variation of the brain sizes. ANCOVAs were also applied to control group differences in neuropsychological tests for depression (Beck Depression Inventory) and in the volumetric measures of the hippocampus and the amygdala. Test-retest and interrater reliabilities were tested by intraclass correlation analyses. Correlational analyses were performed using Spearman’s r. All statistical analyses are 2-tailed, and α levels of significance are P<.05.

To our knowledge, only Lyoo et al17 studied patients with BPD by means of MRI-based volumetric measurements, finding smaller frontal lobes compared with healthy controls (P=.03, not controlled for brain size). However, neither the hippocampus nor the amygdala were investigated in BPD patients by MRI-based volumetric measurements, although the majority of patients with BPD also report a history of traumatic childhood experiences.18-26 It is noteworthy that memory deficits in BPD have repeatedly been reported by independent work groups, who applied extensive neuropsychological test batteries.27-29 Furthermore, there is an ongoing debate as to whether traumatization is causally associated with BPD.30-33

The main purpose of this study was to investigate volumes of the hippocampus and the amygdala in female patients with BPD. Second, we studied neuropsychological functions and the history of childhood traumatization. Third, we analyzed the interrelationships between volumes and neuropsychological functioning, as well as between measured volumes and the extent of traumatic childhood experiences.

RESULTS

SAMPLE CHARACTERISTICS AND TRAUMATIZATION HISTORY

Our patients were found to represent a severely disturbed BPD sample with an average of 8.1 criteria fulfilled (Table 2). Although screening results for alcoholism in the patients were not significantly different from those in the healthy subjects, 1 patient was found to meet criteria of alcohol dependence. Because this was true only during the period 7 to 12 months prior to the study, she was not excluded. Blood levels of γ-glutamyltransferase were within the normal range in all participants. However, the BPD group had Beck Depression Inventory scores about 6-fold higher than the healthy controls (Table 2). The BPD group had substantially higher CTQ total scores and CTQ subscores and reported longer cumulative periods of traumatization than did the healthy control group (Table 3). Considering all participants, the correlation of the number of borderline criteria × CTQ total score was 0.81 (P<.001) and the correlation of the mean period of traumatization × CTQ total score was 0.79 (P<.001).

VOLUMETRIC FINDINGS AND TRAUMATIZATION

Volumetric measurement revealed no group differences of the brain, ie, the prosencephalon, nor of the left and right temporal lobes. However, the BPD group had significantly smaller mean hippocampal volumes with reductions of 15.7% (left hemisphere) and 15.8% (right hemisphere) compared with the healthy control group (Table 4). Minor volumetric differences were found for the amygdala in both hemispheres with reductions of 7.9% (left) and 7.5% (right). Volumes in BPD patients with PTSD did not differ from those in BPD patients without PTSD (Table 4). Smaller volumes in BPD patients were even found when only those 9 BPD patients without PTSD were compared with their 9 matched control subjects: F=8.6 (P=.01) for the mean hippocampal volume and F=5.3 (P<.05) for the mean amygdaloid volume (controlled for the volume of the brain, ie, the prosencephalon).

The mean hippocampal volume × CTQ total score revealed a substantial correlation with r=−0.49 (P<.001) as well as the mean hippocampal volume × mean period

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of traumatization with $r = -0.53$ ($P < .001$), but correlations were not significant when analyses were performed separately for BPD patients and healthy controls.

All corresponding correlational analyses for the mean amygdaloid volume did not reach significance levels (amygdaloid volume × CTQ: $r = -0.24$, $P = .11$; amygdaloid volume × mean period of traumatization: $r = -0.24$, $P = .14$).

**NEUROPSYCHOLOGICAL TESTING**

None of the applied tests revealed any significant differences between the BPD group and the healthy control group when analyses were controlled for Beck Depression Inventory score, which itself moderately but significantly contributed to a variety of results

<table>
<thead>
<tr>
<th>Table 2. DSM-IV BPD Criteria Alcohol-Related Findings, Current Depression, and Anxiety in Female Patients With BPD and Healthy Controls*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td><strong>BPD Group</strong></td>
</tr>
<tr>
<td>(n = 21)</td>
</tr>
<tr>
<td>No. of BPD criteria</td>
</tr>
<tr>
<td>Posttraumatic stress disorder</td>
</tr>
<tr>
<td>Alcohol-related findings</td>
</tr>
<tr>
<td>LAST score</td>
</tr>
<tr>
<td>Alcohol dependence (1 y)</td>
</tr>
<tr>
<td>GGT level, U/L</td>
</tr>
<tr>
<td>BDI score</td>
</tr>
<tr>
<td>STAI-X1 score</td>
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<tr>
<td>STAI-X2 score</td>
</tr>
</tbody>
</table>

*Data are given as mean ± SD or number (percentage). BPD indicates borderline personality disorder; LAST, Luebeck Alcoholism Screening Test; GGT, γ-glutamyltransferase; BDI, Beck Depression Inventory; and STAI, State(X1)-Trait(X2) Anxiety Inventory.
†Mann-Whitney U test, $z$ values are given (corrected for ties) or $x^2$ test.

In contrast with previous reports, neuropsychological testing in the female patients with BPD did not yield disturbances compared with the healthy controls, when group differences were controlled for current depressive states, nor were the results associated with the volumes of the hippocampus and/or the amygdala. Although this was also true in tests investigating declarative memory functions (eg, the Logical Memory Test) and similar findings were reported in alcoholic subjects, we have to concede that we did not apply tests (eg, pair association tests), which are more adequate for investigating declarative memory functions of the hippocampus. Furthermore, a normal memory functioning might be only true with regard to the processing of neutral stimuli and if these stimuli are indeed interpreted as neutral. On the other hand, traumatic contents (memories)—under
The influence of the amygdala\textsuperscript{15,63}—may continue to be processed in a pathological way as known from PTSD.\textsuperscript{64-66} Further studies are proposed to focus on functional neuroimaging and nontrauma- vs trauma-specific memory tasks to clarify these issues in BPD.

Some limitations of this study should be considered. First, we did not perform volumetry of subcortical regions (e.g., caudatum) as comparison measures. Second, we did not assess major depressive disorders as a possible confounding variable of the volumetric results\textsuperscript{65}: major depressive disorders and even more the cumulative lifetime duration of major depression episodes were reported to be associated with hippocampal volume reductions by some\textsuperscript{68-71} but not all authors\textsuperscript{72,73} (for

### Table 4. Magnetic Resonance Imaging Volumetric Measurements in 42 Female Subjects\textsuperscript{*}

<table>
<thead>
<tr>
<th></th>
<th>BPD Group (n = 21)</th>
<th>Control Group (n = 21)</th>
<th>BPD – Controls, %</th>
<th>Statistics†</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prosencephalon</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td>1143.55 ± 68.836</td>
<td>1137.71 ± 89.119</td>
<td>...</td>
<td>z = 0.0, P = .99</td>
</tr>
<tr>
<td>Right</td>
<td>70.71 ± 56.68</td>
<td>71.60 ± 76.25</td>
<td>...</td>
<td>F = 0.0, P = .90</td>
</tr>
<tr>
<td><strong>Temporal lobe</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td>113.54 ± 70.59</td>
<td>121.10 ± 78.12</td>
<td>...</td>
<td>F = 0.2, P = .89</td>
</tr>
<tr>
<td>Right</td>
<td>71.81 ± 62.09</td>
<td>71.85 ± 75.64</td>
<td>...</td>
<td></td>
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<tr>
<td><strong>Hippocampus</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Left</td>
<td>1799 ± 164</td>
<td>2081 ± 230</td>
<td>-15.7</td>
<td>F = 21.5, P &lt; .001</td>
</tr>
<tr>
<td>Right</td>
<td>1799 ± 191</td>
<td>2083 ± 231</td>
<td>-15.8</td>
<td>F = 19.3, P &lt; .001</td>
</tr>
<tr>
<td><strong>Amygdala</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td>1383 ± 187</td>
<td>1492 ± 140</td>
<td>-7.9</td>
<td>F = 4.4, P = .04</td>
</tr>
<tr>
<td>Right</td>
<td>1395 ± 189</td>
<td>1500 ± 144</td>
<td>-7.5</td>
<td>F = 4.0, P = .053</td>
</tr>
</tbody>
</table>

\*BPD indicates borderline personality disorder; PTSD, posttraumatic stress disorder.

†Mann-Whitney U test (z) or analysis of covariance (F) (ANCOVA) with the volume of the prosencephalon as covariate entered first (df = 1).

‡Without the hippocampus and without the amygdala (see the “Subjects and Methods” section).

§Group difference between BPD with PTSD vs BPD without PTSD (ANCOVA): F1 = 0.2, nonsignificant.

||Group difference between BPD with PTSD vs BPD without PTSD (ANCOVA): F1 = 1.8, nonsignificant.

### Table 5. Neuropsychological Testing\textsuperscript{*}

<table>
<thead>
<tr>
<th>Test</th>
<th>BPD Group (n = 21)</th>
<th>Control Group (n = 21)</th>
<th>BDI Score</th>
<th>Hippocampus Mean Volume</th>
<th>Amygdala Mean Volume</th>
<th>Group Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Digit-Symbol Test (age-corrected scores)</strong></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Copy</td>
<td>10.1 ± 2.7</td>
<td>12.4 ± 2.6</td>
<td>F = 5.4, P = .03</td>
<td>F = 0.0, P = .94</td>
<td>F = 0.6, P = .43</td>
<td>F = 2.3, P = .14</td>
</tr>
<tr>
<td>Immediate recall</td>
<td>33.7 ± 3.6</td>
<td>34.7 ± 1.1</td>
<td>F = 5.4, P = .03</td>
<td>F = 0.0, P = .93</td>
<td>F = 0.3, P = .58</td>
<td>F = 1.2, P = .23</td>
</tr>
<tr>
<td>Delayed recall</td>
<td>20.8 ± 7.5</td>
<td>22.5 ± 4.7</td>
<td>F = 2.3, P = .84</td>
<td>F = 0.4, P = .52</td>
<td>F = 0.5, P = .47</td>
<td>F = 0.4, P = .52</td>
</tr>
<tr>
<td>Retention: delayed recall/copy</td>
<td>21.3 ± 6.5</td>
<td>23.0 ± 5.6</td>
<td>F = 1.5, P = .23</td>
<td>F = 0.3, P = .61</td>
<td>F = 0.0, P = .94</td>
<td>F = 0.0, P = .87</td>
</tr>
<tr>
<td><strong>Rey-Osterrieth Complex Figure Test</strong></td>
<td></td>
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</tr>
<tr>
<td>Copy</td>
<td>13.0 ± 2.8</td>
<td>16.0 ± 3.5</td>
<td>F = 5.5, P = .02</td>
<td>F = 0.6, P = .46</td>
<td>F = 0.1, P = .81</td>
<td>F = 2.4, P = .13</td>
</tr>
<tr>
<td>Immediate recall</td>
<td>11.1 ± 3.2</td>
<td>13.9 ± 3.7</td>
<td>F = 4.7, P = .04</td>
<td>F = 1.1, P = .31</td>
<td>F = 0.3, P = .59</td>
<td>F = 1.3, P = .26</td>
</tr>
<tr>
<td>Delayed recall</td>
<td>0.9 ± 0.1</td>
<td>0.9 ± 0.1</td>
<td>F = 0.2, P = .67</td>
<td>F = 0.5, P = .47</td>
<td>F = 2.7, P = .11</td>
<td>F = 0.0, P = .99</td>
</tr>
<tr>
<td><strong>Wechsler Memory Scale, logical memory</strong></td>
<td></td>
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</tr>
<tr>
<td>Immediate recall</td>
<td>78.5 ± 41.5</td>
<td>44.2 ± 23.6</td>
<td>F = 8.9, P = .005</td>
<td>F = 0.7, P = .41</td>
<td>F = 0.1, P = .79</td>
<td>F = 0.4, P = .51</td>
</tr>
<tr>
<td>Delayed recall</td>
<td>5.4 ± 4.4</td>
<td>2.6 ± 1.9</td>
<td>F = 6.8, P = .01</td>
<td>F = 1.4, P = .25</td>
<td>F = 1.0, P = .33</td>
<td>F = 0.1, P = .74</td>
</tr>
<tr>
<td>No. of repeats</td>
<td>5.0 ± 4.0</td>
<td>3.1 ± 2.4</td>
<td>F = 6.5, P = .02</td>
<td>F = 0.5, P = .48</td>
<td>F = 1.0, P = .34</td>
<td>F = 0.0, P = .87</td>
</tr>
<tr>
<td><strong>Embedded Figures Test</strong></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Time/figure, s</td>
<td>10.3 ± 5.2</td>
<td>12.1 ± 4.9</td>
<td>F = 3.3, P = .08</td>
<td>F = 0.0, P = .74</td>
<td>F = 0.1, P = .74</td>
<td>F = 0.2, P = .65</td>
</tr>
<tr>
<td>No. of errors</td>
<td>16.7 ± 3.5</td>
<td>17.7 ± 3.1</td>
<td>F = 1.2, P = .28</td>
<td>F = 0.0, P = .84</td>
<td>F = 1.4, P = .24</td>
<td>F = 0.1, P = .82</td>
</tr>
<tr>
<td>No. of repeats</td>
<td>11.4 ± 1.8</td>
<td>13.2 ± 1.0</td>
<td>F = 10.4, P = .003</td>
<td>F = 0.4, P = .52</td>
<td>F = 0.0, P = .87</td>
<td>F = 2.4, P = .13</td>
</tr>
<tr>
<td><strong>Block design</strong></td>
<td>19.5 ± 5.4</td>
<td>27.6 ± 5.4</td>
<td>F = 6.6, P = .006</td>
<td>F = 0.0, P = .96</td>
<td>F = 0.1, P = .82</td>
<td>F = 5.6, P = .02</td>
</tr>
</tbody>
</table>

\*BPD indicates borderline personality disorder; BDI, Beck Depression Inventory; WIP 86, 1986 version of the Reduced Wechsler Intelligence Scale. Nine patients with BPD received psychotropic medication for sleep induction and 12 did not; Mann-Whitney U tests did not reveal any significant differences between these 2 subgroups (z values corrected for ties).

†Analysis of covariance with covariates entered first (df = 1).
review, see Soares and Mann\(^6\)). Third, we did not specifically test functions of the amygdala. Fourth, although we did not find differences of neuropsychological test results between BPD patients with and without occasional psychotropic medication, we cannot exclude that use of medication might have influenced the results.

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Error in Table Footnote. In the original article by Weickert et al titled “Cog-
nitive Impairments in Patients With Schizophrenia Displaying Preserved and
Compromised Intellect,” published in the September 2000 issue (2000;57:907-
913), Table 1 on page 910, the first part of the second footnote should have
read “a indicates healthy controls significantly different from deteriorated and
compromised patients; . . . ”