Linguistic and neural correlates of attachment dysregulation in Borderline Personality Disorder

Anna Buchheim, PhD [1*]; Susanne Erk, MD [2*]; Carol George, PhD [3]; Horst Kächele, MD [1]; Tilo Kircher, MD [4]; Philipp Martius, MD [5]; Dan Pokorny, Dr. [1]; Martin Ruchsow, MD [6]; Manfred Spitzer, MD, PhD [7]; Henrik Walter, MD, PhD [2]

[1] *University Clinic for Psychosomatic Medicine and Psychotherapy, University Ulm*

[2] *Department of Psychiatry, Division of Medical Psychology, University Bonn*

[3] *Department of Psychology, Mills College, Oakland CA*

[4] *Neuroimaging - L&F Experimental Psychopathology, Clinic for Psychiatry and Psychotherapy, University RWTH Aachen*

[5] *Psychosomatic Hospital Dr. Schlemmer, Bad Wiessee*

[6] *Hospital for Psychiatry and Psychotherapy Christophsbad, Göppingen*

[7] *Department of Psychiatry III, University Ulm*

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* *Both authors contributed equally to this study*

Address for correspondence:

Anna Buchheim, Ph.D., University Clinic for Psychosomatic Medicine and Psychotherapy, University of Ulm, Am Hochstraess 8, D-89081 Ulm, Germany,

Phone: +49-731-50025709, Fax: +49-731-50025662; e-mail: anna.buchheim@uni-ulm.de

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Abstract:

Context: Borderline personality disorder (BPD) is characterized by extreme and enduring emotional instability, multifaceted emotional pain, and fear of abandonment. BPD frequently report unresolved, traumatic, abusive experiences with their caregiver. Several studies investigated neural correlates of dysfunctional emotional reactivity in BPD linked to trauma history. No study has examined neural correlates when attachment trauma was activated representationally during overt speech. We were interested in linguistic and neural reponses to "monadic" and "dyadic" attachment pictures; that is, stimuli portraying individuals as facing attachment threats alone versus in the presence of potential attachment figures.

Objective: To explore linguistic and neural correlates of dysregulation in attachment narratives using a valid attachment measure feasible for functional magnetic resonance imaging environment. We predicted that "monadic" attachment pictures would activate brain areas concerned with fear and pain in patients with BPD.

Design: Case control study.

Setting: A university hospital.

Participants: 11 female patients with BPD, 17 healthy female age- and education-matched controls.

Main Outcome Measures: Attachment classification, linguistic traumatic dysregulation, blood oxygen level-dependent functional magnetic resonance imaging signal changes.

Results: BPD patients were classified significantly more often as unresolved with respect to attachment trauma, associated with higher amount of linguistic traumatic dysregulation in "monadic" narratives than controls. BPD patients showed significantly more dorsal anterior cingulate cortex activation than controls when responding to "monadic" pictures. Furthermore patients showed hyper-activation of the right superior temporal sulcus and hypo-activation of the right parahippocampal gyrus when responding to “dyadic” attachment pictures.

Conclusion: These findings suggest evidence for possible mechanisms related to core clinical features of BPD, like fearful and painful intolerance of aloneness, hypersensitivity to social environment, and reduced positive memories of dyadic interactions. One clinical implication is to highlight the nature of dysregulated attachment representation in patients with BPD, which might be a useful target of psychotherapeutic intervention.

Borderline personality disorder (BPD) is characterized by extreme and enduring emotional instability involving a range of intense affects, including rage, panic, emptiness, loneliness [1] and, characteristically multifaceted emotional pain and fear of abandonment [2]. Childhood maltreatment (neglect, physical and sexual abuse) is one of the central psychosocial risk factors for BPD [3, 4].

Clinically, it is generally agreed that an essential dimension of BPD is a dysfunction of emotion regulation systems [5, 6, 7, 8]. Neuroimaging studies reported metabolic abnormalities in frontal cortex and subcortical areas [9, 10, 11]. Two recent fMRI studies showed limbic hyper-reactivity in BDP patients while viewing aversive pictures [12, 13]. PET studies showed prefrontal dysfunction in BPD patients in response to listening to biographically-based scripts of abandonment and abuse [14, 15]. Importantly, no patient study to date has examined neural activation patterns in relation to attachment, the basic neural system that processes relationship-based experience, including pain and abuse.

Adult attachment research is based on the linguistic analysis of attachment narratives, called "attachment representation," produced during interview assessments [16, 17, 18, 19, 20]. Attachment status based on such narrative discourse patterns falls in two attachment groups: organized (secure, dismissing, preoccupied) and disorganized/"unresolved." Disorganized/"unresolved" individuals are flooded with painful affect related to attachment trauma during the interview, often evidenced through descriptions of intense fear or linguistic disorientation [20]. Studies concur that the "unresolved" attachment classification predominates in BPD patients, related particularly to lack of resolution of physical and sexual abuse [21, 22, 23, 24, 25]. Moreover, borderline patients have severe problems in "mentalizing," as attachment-related abuse is associated with unbearable pain [26, 27, 28]. We conclude, then, that attachment disorganization and traumatic dysregulation is central to understanding BPD.

The development of an attachment relationship is an essential biological mechanism that influences motivational and emotional processes related to survival and reproductive fitness [29, 30, 31]. We know from animal studies that, in particular, limbic structures are involved in the formation of attachment and responses to separation and deprivation [32, 33, 34]. Accordingly, structural neuroimaging studies show reduced volumes of hippocampus and amygdala in patients with trauma and difficult attachment histories [35, 36].

Functional imaging studies on human attachment have focused on healthy subjects. Stimuli used included pictures of loved ones (e.g., relationship partner versus friends; own versus an unknown baby) [37, 38, 39, 40, 41, 42], evoking responses in a network of cortical and subcortical areas, including the cingulate cortex, insula, basal ganglia, and orbitofrontal cortex None of these fMRI-studies, however, specifically examined brain activation patterns while subjects were talking about attachment.

Reliable analyses of fMRI data gathered during continuous overt speech have been shown recently in healthy controls as well as in schizophrenic patients with severe formal thought disorder [43, 44, 45]. We recently demonstrated the feasibility of using an established attachment measure, the Adult Attachment Projective [17, 18, 19], in an fMRI environment while healthy subjects were telling stories to attachment pictures [46, 47].

The Adult Attachment Projective (AAP) assesses discourse patterns as a response to a set of attachment pictures. “Unresolved” stories are characterized by the failure to contain or “resolve” frightening story events, as evidenced by descriptions of desperation, fear, or helplessness. Moreover, the coding system delineates specific linguistic signs of “traumatic dysregulation,” allowing for the differentiation of trauma contained in “unresolved” narratives [48]. These linguistic “traumatic dysregulation markers” are central in the present study (see Methods).

This study investigated the neural correlates of attachment dysregulation in borderline patients using our fMRI-adapted version of the AAP [47]. We were especially interested in responses to “monadic” and “dyadic“ attachment situations; that is, responses to stimuli portraying individuals as facing attachment threats alone versus in the presence of potential attachment figures. Given that one of the key features of BPD patients is their intolerance of aloneness [49], we predicted that AAP stimuli representing traumatic contents, such as aloneness, desperation, and physical threat, would elicit a significantly greater association with linguistic traumatic (and not only normative) dysregulation markers in the BPD group than controls. On the neural level, we hypothesized that BPD patients, as compared with controls, would show increased activation of brain regions associated with fear and pain (e.g. amygdala, anterior cingulate cortex) during narration in response to these stimuli.

Methods

Partcipants: Thirteen female BPD patients were recruited from an inpatient psychiatric hospital in Bad Wiessee, Germany and compared to a group of 21 healthy female volunteers, matched for age and education. Exclusion criteria were serious medical or neurological illness, including comorbid psychotic disorders, bipolar disorder, left handedness, metal in body, and deficits in German language. Six subjects were excluded. The two patients coded as “resolved” were excluded from analysis, because the frequency in this group was too small to count as a separate group. Four control subjects were excluded because of excessive head movement during the fMRI procedure. Psychiatric diagnoses, including diagnostic criteria for BPD, were assessed by a trained psychiatrist (PM) using the Structured Clinical Interview I and II for DSM-IV [50, 51]. Comorbidity in our patient sample included depression (69%), anxiety or panic disorder (23%), and somatoform disorder (15%). 62% of the patients were treated pharmacologically, including antidepressants (4), major tranquilizer (neuroleptics, 3), and other non-psychotropic medication (4). All subjects gave written informed consent. The protocol was approved by the local institutional review board. Clinical characteristics of the sample are shown in Table 1.

Insert Table 1 here

Attachment coding: Subjects were administered the fMRI-adapted version of the AAP [46, 47]. The AAP set consists of eight pictures, one neutral and seven attachment scenes.

Insert Figure 1a, b here

The pictures are simple line drawings that depict theoretically derived attachment events (illness, solitude, separation, abuse) and are administered in the following order: #2 “Child at Window”; #3 “Departure”; #4 “Bench”; #5 “Bed”; #6 “Ambulance”; #7 “Cemetery”; #8 “Corner”.
The attachment pictures include four “monadic” scenes (individuals depicted alone) and three dyadic scenes (individuals depicted in potential attachment dyads). Individuals are instructed to tell a story about the scene, including the character(s)’ thoughts, feelings and, outcome of the story [17]. Individuals were classified into one of two main attachment groups: “resolved” and “unresolved”. “Unresolved” attachment in the AAP coding system is defined as an individual’s failure to contain or re-organize linguistic “markers” or indicators of dysregulated attachment including, for example, evidence of danger, threat, fear, helplessness, or isolation. This failure leaves story characters haunted or threatened by dysregulation [17]. AAP stories are considered

"resolved" when dysregulating markers are contained. Containment occurs when characters are described as capable of utilizing internal, behavioral, or relationship resources to block dysregulation (see Table 2).

Insert Table 2 here

AAP classifications were coded by two independent reliable judges based on the transcribed verbatim AAP narratives, that subjects produced in the scanner. Inter-rater agreement was 100% (kappa=1,00). Judges were blind to any identifying information about the subjects. The scanner-administered validity of the AAP was examined based on convergent classifications with Adult Attachment Interviews (AAI) [16] administered outside the fMRI-environment one month after fMRI acquisition, classified by an independent trained AAI judge also blind to all information about subjects. The AAI, a validated semi-structured interview assessment of attachment, asks individuals to describe childhood experiences with caregivers and autobiographic attachment events (e.g. separations, loss, abuse) [16, 20]. There was a high correspondence between the AAP and AAI "resolved" vs. "unresolved" categories (kappa = .70).

Both AAP judges also coded the AAP stories differentiating between "normative" and "traumatic" markers [48]. "Normative" dysregulation markers are defined as those typically present because of AAP picture "pull" for the topic, as based on evaluations of several hundred AAP transcripts in normative and clinical samples [17]. For example, in "Cemetery," it is common for the character "to talk to the deceased." In "Bench," and "Corner," it is common for characters to be "frightened" because of a fight or break up with a friend (see Table 2). These "normative" markers do not have the same terrifying or dysregulating quality as "traumatic" markers, such as the “deceased talking back to the living” in “Cemetery” or the girl in “Bench”, described as “suicidal, abandoned, homeless and incarcerated”. There was 100% inter-rater agreement in coding “normative” and “traumatic” markers (kappa=1.00). The results presented below focuses on the “traumatic” markers because of the specific link between BPD and traumatic childhood experience [26, 27, 28].

fMRI-Attachment-Paradigm: The detailed procedure has been described elsewhere [46, 47]. In short, subjects were trained using two neutral non-AAP pictures before scanning. They were given the standard AAP instructions for story telling (“What led up to that scene; what are the characters thinking or feeling; what might happen next?”). They were asked to talk about each picture for 2 minutes, keeping their head as still as possible while speaking. Each picture trial during scanning consisted of the following sequence: Standard instruction (10 sec); fixation cross (10 sec), AAP picture (120 sec), fixation cross (15 sec).

Data Acquisition: 1.5 Tesla Siemens Magnetom Symphony scanner (Siemens, Erlangen, Germany), image size: 64 × 64 pixel, FoV of 192 mm, slice thickness 4 mm with 1 mm gap, 25 slices, TE/TR 40 ms/2500 ms, total acquisition time about 25 minutes (= 598 volumes, one session). Instructions and pictures were presented with fMRI compatible video-goggles (Resonance Technologies, Northridge, CA). Speech was digitally recorded using an fMRI compatible microphone and saved digitally with respect to picture onset on a computer using Cool Edit Pro (Syntrillium Software Cop. Phoenix, Arizona). Participants wore customized headphones.

Data Analysis:

Behavioral scales. Differences between two or three study groups were analyzed using the exact Mann-Whitney U-test and the Kruskal-Wallis H-test (SPSS version 14). Non-parametric test procedures were preferred because of the non-normal distribution of the dependent variables. The magnitude of the group differences was expressed by the effect size (ES, Cohen's d). Group characteristics (medians, 1st and 3rd quartiles, minima, maxima and outliers) were represented by box plots.

Neuroimaging data. Preprocessing and statistical analysis of fMRI data were carried out with SPM2 (www.fil.ion.ucl.ac.uk) and MATLAB 6.1 (MathWorks, Natick, Massachusetts). The first four images were discarded to account for equilibration effects. Individual functional images were corrected for motion artifacts by realignment to the first volume of each session. Four control subjects with excessive head movement were excluded (> 2 mm within a trial cycle) in order to minimize movement effects. Further preprocessing included spatial normalization (3x3x3 mm) and smoothing (FWHM 8 mm).

The regression model for each subject was as follows: The hemodynamic response was modeled for every picture using 2 or 3 regressors, depending on the duration of the narrative: Regressor 1 modeled the time period from onset of picture to onset of speech; regressor 2 modeled the picture during speaking; and regressor 3 modeled the time from offset of speech to end of picture presentation (if the subject did not talk for 2 minutes). Three more regressors were built, each modeling all nine pictures: Regressor 4 modeled the onset of each and every word of all pictures as a stick function; regressor 5 modeled instructions; and regressor 6 modeled all fixation crosses = base line. Regressors of interest were convolved with a function that modeled a prototypical hemodynamic response before inclusion into the regression model. Finally, six more regressors modeled residual motion. The variance of each voxel was estimated for each trial according to the General Linear Model. Individual regionally specific effects of interest were calculated for each participant using linear contrasts, resulting in a t-statistic for every voxel.

The effects of interests for the analysis in this study were “monadic” and “dyadic” picture narratives. Based on the coding results, picture 8 was excluded (see Results section). The contrast “monadic pictures” included pictures #2, #4 and #7; the contrast “dyadic pictures” included #3, #5 and #6. Both contrasts contained three pictures. We calculated for each subject the contrast [picture presentation before the subjects started speaking (regressor 1) + picture presentation during speech (regressor 2)] versus baseline (fixation cross, regressor 6), thereby, including potential mental processes before the actual speaking phase started.

Group differences were assessed at a second level using random effects analysis. One-way ANOVAs were calculated to evaluate differential group effects (diagnosis) and attachment classification, one each for the “monadic” and “dyadic” pictures. The three attachment groups were “resolved” controls, “unresolved” controls and “unresolved” patients; t-statistics for each voxel were set at a threshold of $p < 0.001$ uncorrected for multiple comparisons. Results were corrected for extent threshold resulting in $p < 0.05$ at the cluster level. Talairach & Tournoux [55] and Duvernoy [56] atlases were used to identify brain areas.

Results

Behavioral scales: The AAP classifications for the sample were as follows: Controls: 10 “resolved” and 7 “unresolved”; borderline patients: 2 “resolved” (excluded from further analysis, see above) and 11 “unresolved”. The comparison of the “unresolved” subjects classified by the Adult Attachment Interview content [16] showed that healthy controls were predominantly unresolved for loss whereas the “unresolved” patients were unresolved for sexual abuse and loss (see Table 1).

Analyses of the linguistic “traumatic” markers showed greater “traumatic” dysregulation in the AAP stories of BPD patients as compared to controls in response to the “monadic” pictures, independent of overall attachment classification (Table 3). Significant differences were found for all “monadic” pictures. The differences for pictures “Window”, “Bench”, and “Cemetery” were highly significant ($p< 0.01$). Therefore, these three “monadic” pictures were selected for the fMRI analysis.

There was no significant difference between the groups for any of the “dyadic pictures”. The comparisons of the overall scores in “monadic” and “dyadic” pictures in the three groups (“resolved” controls, “unresolved” controls, and “unresolved” patients) are shown in the figures 2 and 3. These pictures demonstrate that the difference between both the “resolved” and “unresolved” subjects in the control group is small and not significant compared to the differences between the two groups of control subjects and patient group (“unresolved” patients). This interpretation was confirmed by results of the non-parametric tests for the three groups (see Table 4).

Insert Table 3 here

Insert Figures 2, 3 here

Insert Table 4 here

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fMRI analysis: BPD and control group comparisons showed the following: 1. BPD patients’ responses to “monadic” pictures showed significantly stronger activation of the dorsal anterior cingulate cortex (ACC, x=3, y=18, z=24, Z= 4.43) than controls. 2. BPD patients’ responses to “dyadic” pictures showed significantly stronger activation of the right superior temporal sulcus

(STS, (x=60, y=-45, z= 24; Z= 4.52) than controls. 3. Control subjects' responses to "dyadic" pictures showed significantly higher activation than BPD patients of the right parahippocampal gyrus (GH, x=33, y=-39, z= -15; Z=4.31).

We calculated the same contrasts between the patient group (all "unresolved") and each of the two subgroups of control subjects ("resolved," "unresolved") in order to test whether this effect was due to diagnosis or attachment status. The results were the same; that is, the difference was present between BPD and each of the control groups. This indicates that the effect is related to diagnosis. This is also apparent from the plots in Figure 4 a, b, c, which show the parameter estimates at the locations mentioned for all three groups.

Insert Figure 4 a, b, c here

Comment

The present study investigated the neural correlates of traumatic attachment dysregulation during subjects' narration of stories while viewing independently validated AAP pictures of attachment-activating events. Our main hypothesis, derived from core clinical features of BPD (e.g., affective dysregulation, intolerance of aloneness), was confirmed. The presentation of "monadic" pictures triggered traumatic dysregulation, evidenced in the narratives (behavioral level) and corresponding activation in brain regions associated with pain and fear (brain level). BPD patients showed significantly more "traumatic" markers in their "monadic" stories and significantly more activation in the dorsal anterior cingulate cortex. We interpret this activation as a neural correlate of the activation of emotional pain. Patients and non-patients did not differ in the number of "traumatic" markers in dyadic stories; however, patients showed hyper-activation of the right STS and hypo-activation of the right parahippocampal gyrus. We interpret this finding as a neural signature of hyper-vigilance to social relations and reduced positive memories of interaction with attachment figures. We now discuss these findings in detail.

Linguistic attachment dysregulation: The majority of our BPD patients were classified as "unresolved" on the AAP. The fMRI-AAP classifications were validated by the significant classification-match to the AAI administered outside the fMRI. This convergence confirms that the fMRI-AAP procedure, first developed by examining healthy controls [46], is applicable to fMRI administration for BPD patients. The predominant prevalence of "unresolved" attachment among the BPD patients in this study is comparable to other studies investigating clinical populations [21, 57, 58] whereas the number of "unresolved" control subjects (38%) is greater than the average percentage (19%) previously reported in healthy populations [59].

Whereas both the BPD patients and the normal controls had "unresolved" attachment classifications, the source of their unresolved status appears to be linked to differences in past experience. On the AAI, unresolved patients showed significantly higher scores for loss and abuse; whereas unresolved were only somewhat dysregulated with respect to loss clearly less so for abuse. We believe that this pattern suggests that the combination of unresolved loss and abuse is more likely to contribute to pathological distress than experiences of loss in and of itself, an interpretation that is consistent with recent clinical studies of attachment [60, 61].

Our AAP analysis of dysregulating linguistic marker prevalence provides a more detailed level of understanding regarding the organization and threats to attachment in BPD than exists in the literature to date [62]. Unresolved BPD patients manifested more "traumatic" than "normative" attachment dysregulation, whereas "normative" dysregulation predominated in unresolved controls. However, this finding was only found in response to "monadic" pictures, especially, “Window”, “Bench”, and “Cemetery.” This result is consistent with clinical descriptions of BPD patients’ intolerance to aloneness and fear of abandonment [49, 63]. Flooded and overwhelmed, the BPD patients in this study were not able to integrate organizing narrative elements (i.e., productive thinking, safety provided by an attachment figure, constructive action) into their alone stories and they remained dysregulated when attachment was activated. Therefore, patients responded to “monadic” scenes with stories including elements of fear, helplessness and abandonment. For example, in “Cemetery,” BPD stories included themes of isolation, helplessness, abandonment, murder, suicide, and dissociated imagery (e.g., figures floating above the ground), whereas the narratives of non-patient “unresolved” controls described normal graveyard “contact” with the deceased or grief talk (i.e., explanations of the grief process).

Consistent with the reported incidence of childhood trauma in the literature on BPD patients, the patients in this study showed high levels of sexual abuse and unresolved characteristics on both the AAP and the AAI. Note, however, that none of the BPD patients in the study met the criteria for a comorbid Posttraumatic Stress Disorder or Dissociation Disorder.

Neural correlates of attachment dysregulation:

<u>Anterior Cingulate Cortex</u>

We found higher activation of the ACC in our (unresolved) BPD patients compared to non-patient controls in response to the “monadic” attachment pictures, representing frightening aloneness. ACC activation in response to pain and unpleasantness has been shown to undermine the ability to integrate affect, cognition, and response selection [64]. ACC activation is associated in normative samples with social relationship stimuli, including intimate relationships [42], mother’s listening to neonate´s cries [65], college-age women’s responses to hypothetically distressing scenes with their romantic partners [38], social exclusion [66], perceiving pain of others [67], and pictures evoking grief as a response to the loss through death of the beloved partner [68]. The anterior cingulate cortex (ACC) is a large region comprising several sub-regions. Bush et al. [69] distinguish between a ventral affective and a dorsal cognitive division of the ACC. Vogt [70] divides the ACC into four divisions with further subdivisions. Consistent with Bush et al., they view the subgenual part of the ACC as mainly concerned with emotions, in particular, the representation of autonomic afferences. The dorsal region posterior to the genu of the corpus callosum, however, is divided in two subsections, the anterior and posterior midcingulate cortex (aMCC, pMCC). According to Vogt [70], the pain and fear sites of the ACC overlap in the aMCC. The aMCC is innervated by the midline and intralaminar thalamic nuclei belonging to the medial pain system [71] and also receives direct input from the amygdala [72]. Thus, the aMCC is linked to aspects of pain, especially fear avoidance.

The ACC activation in the present study was located in the aMCC. Our finding may represent dysregulation or imbalance in the ACC's integrative function in pain and emotion processing and may be a neural signature of the emotional pain associated with fear of aloneness. Abandonment concerns and intolerance of aloneness are reported to be the most persistent symptoms in BPD patients six years after prospective follow up [73].

Our results are consistent with a FDG-PET study demonstrating increased baseline ACC metabolism in BPD patients as compared to healthy controls [11]. However, they contradict findings of two recent functional PET studies [14, 15] investigating BPD patients while listening to scripts describing their own experiences of abandonment [14] or trauma [15]. Women with BPD and a history of sexual abuse showed significantly less activation in ACC (and DLPFC) when compared to women with sexual abuse without BPD. Although, these studies and our study also differ in methodology (PET versus fMRI), presumably the most important difference is the experimental design, which may account for the opposite findings. Subjects in the Schmahl et al. studies listened to pre-processed, scripted memories that were then reintroduced during their neuroimaging experiment. In contrast, subjects in the present study had no preparation for experimental stimuli. The AAP scenes are specifically designed to activate attachment spontaneously, thus, preventing the subjects from anticipating or consciously engaging in self-regulating mechanisms. Therefore, our study design taps the immediate, unprepared activation of the attachment system.

Superior Temporal Sulcus

Patients showed significantly more activation of the right superior temporal sulcus (STS) than controls in response to the “dyadic” pictures. These pictures depict interaction with potential attachment figures and pull for stories describing social interaction (the “monadic” pictures do not require the individual to acknowledge the presence of others). The challenge, then, when presented with dyadic pictures is to tell a story that integrates interaction in the context of a potential attachment relationship (departure, bedtime, injury/death).

Functional imaging studies show right STS activation in tasks requiring theory-of-mind capacities [74, 75, 76, 77, 78, 79, 80]. Saxe and co-workers [81, 82] propose that the right STS is the most important part of the brain involved in the function of mentalizing. Fonagy et al. [27] propose that the abusive childhood experiences has forced BPD individuals to avoid thinking about the caregiver’s wish to harm them, leading to the inhibition of constructive mentalization capacities. “Theory of mind” studies confirm that, when asked to describe attachment experiences, BPD patients show disturbed reflective function that is often identified as a “hyper-analytical mode.” Borderline patients often demonstrate a misleading hypersensitivity for the mental state of the other during interaction (non-conscious mind-reading capacity) in order to manipulate and control potentially threatening relationships [27, 83].

Following this research, we suggest that the AAP "dyadic" picture STS activation pattern in BPD patients found in the present study indicates fear-based hyper-arousal in relationships and interactive contexts; these individuals are compelled to pay attention to social interactive cues. A history of traumatic experience requires individuals to be hyper-attentive to their surroundings and, more generally, unresolved attachment has been linked to fear-based hypervigilance in significant relationships [84, 85, 86].

Parahippocampal gyrus

The parahippocampal gyrus is the only structure in which controls show significantly more activation than BPD patients while responding to "dyadic" AAP pictures. This finding suggests an important input pathway to the hippocampal region in relation to memory processing. The hippocampus is known to mediate the convergence of perceptual, motor and cognitive neocortical information for memory representations [87]. We have recently shown that this region is associated with a subsequent memory effect for neutral items encoded in positive emotional context in healthy subjects [88]. Taken together, this suggests that the parahippocampal gyrus may mediate information about positive emotional information. Interestingly, in our AAP narratives of "dyadic" pictures, the "unresolved" control subjects describe overall positive dyadic interactions; interactions characterized by emotional warmth and mutuality. This impression was evidenced by higher scores on the AAP subscale "Synchrony," the scale that evaluates dyadic stories for relationship mutuality (care for others or enjoyment). Therefore, our finding regarding parahippocampal activation confirms that the control subjects' narratives are more associated with positive emotional memories compared to BPD patients, who show reduced activations. Our interpretation is also consistent with the fact that, on a descriptive level, the "resolved" control subjects showed the highest level of parahippocampal activation (i.e., more activation than "unresolved" controls).

Limitations

Several limitations should be considered when interpreting our findings. First, the influence of lifetime psychiatric conditions in the patient group cannot be ruled out, although we excluded patients with current psychosis and substance abuse. Thus, the neural results of attachment dysregulation may not be specific to BPD, but rather a feature of patients with multiple Axis I and Axis II disorder. Secondly 62% of our patients were medicated, though low dosed, which might be a confounding factor in comparing patient and control subjects. Third, the results of this study are based on a small sample of BPD patients, all of whom were "unresolved." Questions regarding "resolved" BPD patients cannot be examined in our study. Fourth, overt speech is necessarily accompanied by movement. However, we took a series of measures to eliminate this influence as much as possible: exclusion of subjects, inclusion of movement parameters as covariate of no interest, modeling the onset of every spoken word. Moreover, our results are not activations in regions, which typically are found for movement artifacts.

Conclusions

In conclusion, this study demonstrates that the fMRI-adapted version of the Adult Attachment Projective [47] paradigm can be used with BPD patients. Narrative analysis confirms that BPD is associated with dysregulating attachment fear related to abuse and loss. In particular, the "monadic" AAP pictures representing aloneness differentiated both between the linguistic and neural patterns of patients and controls. These findings on the behavioral and neural levels were coherent and provide evidence for possible mechanisms related to core clinical features of BPD: fearful intolerance of aloneness [49], hypersensitive attention to their social environment [27], as well as lack of contextualization of positive memories of past and current relationships [89]. One possible clinical implication of this research is to highlight the nature of traumatic attachment dysregulation in patients with BPD, which is considered as a necessary target of psychotherapeutic intervention [90]. Modulation of BPD patients' emotional responses in attachment situations during psychotherapy to patterns similar to normal controls, even with an unresolved attachment status, might be an important indication of their increasing capacities for modulating attachment distress in the present and not continue to be haunted by the past.

References

1. Kernberg OF. The management of affect storms in the psychoanalytic psychotherapy of borderline patients. *J Am Psychoanal Assoc*. 2003; 51:517-45.
2. Lieb K, Zanarini MC, Schmahl C, Linehan MM, Bohus M. Borderline personality disorder. *Lancet.* 2004; 364:453-461.
3. Zanarini MC, Frankenburg FR. Pathways to the development of borderline personality disorder. *J Personality Dis*. 1997; 11:93-104.
4. Zanarini MC. Childhood experiences associated with the development of borderline personality disorder. *Psychiatr Clin North Am*. 2000; 23:89-101.
5. Linehan MM, Tutek DA, Heard HL, Armstrong HE. Interpersonal outcome of cognitive behavioral treatment for chronically suicidal borderline patients. *Arch Gen Psychiatry*. 1994; 151:1771-76.
6. Corrigan F M, Davidson A, Heard H. The role of dysregulated amygdalic emotion in borderline personality disorder. *Med Hypotheses*. 2000; 54:574-9.

7. Stiglmayr CE, Shapiro DA, Stieglitz RD, Limberger MF, Bohus M. Experience of aversive tension and dissociation in female patients with borderline personality disorder: A controlled study. *J Psychiatr Res*. 2001; 35:111-18.

8. Posner, M. I., Rothbart MK. Vizueta N, Levy KN, Evans DE, Thomas KM, Clarkin JF. Attentional mechanisms of borderline personality disorder. *PNAS*. 2002; 99:16366-16370.

9. De la Fuente JM, Goldman S, Stanus E, Vizuette C, Morlan I, Bobes J, Mendlewicz J. Brain glucose metabolism in borderline personality disorder. *J Psychiatry Res*. 1997; 31: 531-541.

10. Tebartz van Elst L, Thiel T, Hesslinger B, Lieb K, Bohus M, Henning J. Subtle prefrontal neuropathology in a pilot magnetic resonance spectroscopy study in patients with borderline personality disorder. *J Neuropsychiatry Clin Neurosci.* 2001; 13: 511-514.

11. Juengling F, Schmahl C, Heßlinger B, Ebert D, Bremner JD, Gostomzyk J, Bohus M, Lieb K. Positron emission tomography in female patients with borderline personality disorder. *J Psychiatr Res*. 2003; 37:109-15.

12. Herpertz, S., T. M. Dietrich, TM, Wenning B, Krings T, Erbereich SG, Willmes K, Thron A, Sass H. Evidence of abnormal amygdala functioning in borderline personality disorder: A functional MRI study. *Biol Psychiatry*. 2001; 50:292-298.

13. Donegan NH, Sanislow CA, Blumberg HP, Fulbright RK, Lacadie C, Skularski P, Gore JC, Olson IR, McGlashan TH, Wexler BE. Amygdala hyperreactivity in borderline personality disorder: Implications for emotional dysregulation. *Biol Psychiatry*. 2003; 54: 1284-93

14. Schmahl CG, Elzinga BM, Vermetten E, Sanislow C, McGlashan TH, Bremner DJ. Neural correlates of memories of abandonment in woman with and without borderline personality disorder. *Biol Psychiatry*. 2003; 54:142-151.

15. Schmahl C G, Vermetten E, Elzinga BM, Bremner DJ. A positron emission tomography study of memories of childhood abuse in borderline personality disorder. *Biol Psychiatry*. 2004; 55:759-765.
16. George C, Kaplan N, Main M. The Adult Attachment Interview. Unpublished Manuscript, University of California, Berkley; 1984/1985/1996.
17. George C, West M, Pettem O. The Adult Attachment Projective: Disorganization of Adult Attachment at the level of representation. In: Solomon J, George C, eds. *Attachment disorganization*. New York, Guilford; 1999: 462-507.
18. George C, West M. The development and preliminary validation of a new measure of adult attachment: The adult attachment projective. *Attach Hum Dev*. 2001; 3:30-61.
19. George C, West M. *The Adult Attachment Projective: A New Assessment of Adult Attachment.* New York: Guilford Publications; in press
20. Main M, Goldwyn R. *Adult Attachment Classification System*. Unpublished Manuscript. University of California, Department of Psychology, Berkeley; 1985-2005.
21. Fonagy P, Steele M, Steele H, Target M, Gerber A, Leigh T, Kennedy R, Mattoon G. The relation of attachment status, psychiatric classification, and response to psychotherapy. *J Consult Clin Psychol.* 1996; 64:22-31.
22. Patrick M, Hobson R, Castle D, Howard R, Maughn B. Personality disorder and the mental representation of early social experiences. *Dev Psychopathol*. 1994; 6:375-388.
23. Stalker CA, Davies F. Attachment organization and adaptation in sexually abused women. *Can J Psychiatry*. 1995; 40:4-10.
24. Barone L. Developmental protective and risk factors in borderline personality disorder: A study using the Adult Attachment Interview. *Attach Hum Dev*. 2003; 5:84-77.

25. Levy KN, Meehan KB, Weber M, Reynoso J, Clarkin JF. Attachment and borderline personality disorder: Implications for psychotherapy. *Psychopathology*. 2005; 38:64-74

26. Fonagy P, M. Target, Gergely G. Attachment and borderline personality disorder: A theory and some evidence. *Psychiatr Clin North Am* 2000; 23:103-122.

27. Fonagy P, Gergely G, Jurist EL, Target M. *Affect regulation, mentalization, and the development of the self*. Other Press, New York; 2003.

28. Gabbard G. Mind, Brain, and Personality Disorders. *Am J Psychiatry*. 2005; 162: 648-655.

29. Bowlby J. *Attachment and Loss*. Vol. 1: *Attachment*. New York, Basic Books; 1969.

30. Bowlby J. *Attachment and Loss*. Vol. 2: *Separation*. Anxiety and Anger. New York, Basic Books; 1973.

31. Bowlby J. *Attachment and loss*. Vol. 3: *Loss, sadness and depression*. London, Hogarth Press; 1980.

32. Insel TR. A neurobiological basis of social attachment. *Am J Psychiatry*. 1997; 154: 726-735.

33. Insel TR, Young LJ. The neurobiology of attachment. *Nat Rev Neurosci*. 2001; 2: 129-136.

34. Bauman MD, Lavenex P, Mason WA, Capitanio JP, Amaral DG. The development of mother-infant interactions after neonatal amygdala lesions in rhesus monkeys. *J Neurosci.* 2004; 24: 711-721.

35. van Elst LT, Hesslinger B, Thiel T, Geiger M, Haegele K, Lemieux L, Lieb K, Bohus M, Henning J, Ebert D. Frontolimbic brain abnormalities in patients with borderline personality disorder: A volumetric magnetic resonance imaging study. *Biol Psychiatry*. 2003; 54: 163-171.

36. Wignall EL, Dickson JM, Vaugh P, Farrow T, Wilkinson ID, Hunter MD, Woodruff P. Smaller hippocampal volume in patients with recent-onset posttraumatic stress disorder.

Biol Psychiatry. 2004; 56: 832-836.

37. Bartels A, Zeki S. The neural basis of romantic love. *Neuroreport.* 2000; 11: 3829-3834.
38. Bartel, A, Zeki S. The neural correlates of maternal and romantic love. *Neuroimage*. 2004; 21: 1155-66.
39. Nitschke JB, Nelson EE, Rusch BD, Fox AS, Oakes TR, Davidson RJ. Orbitofrontal cortex tracks positive mood in mothers viewing pictures of their newborn infants. *Neuroimage*. 2004; 21: 583-92.
40. Leibenluft E, Gobbini MI, Harrison T, Haxby JV. Mothers' neural activation in response to pictures of their children and other children. *Biol Psychiatry*. 2004; 15: 225-232.
41. Gobbini I, Leibenluft E, Santiago N, Haxby JV. Social and emotional attachment in the neural representation of faces. *Neuroimage*. 2004; 22: 1628-35.
42. Gillath O, Bunge SA, Shaver P, Wendelken C, Miculincer M. Attachment style differences in the ability to suppress negative thoughts: Exploring the neural correlates. *Neuroimage.* 2005: 28; 835-47.
43. Kircher TT, Brammer MJ, Williams SC, McGuire PK. Lexical retrieval during fluent speech production: An fMRI study. *Neuroreport.* 2000; 11: 4093-6.
44. Kircher TT, Liddle PF, Brammer MJ, Williams SC, Murray RM, McGuire PK. Neural correlates of formal thought disorder in schizophrenia: preliminary findings from a functional magnetic resonance imaging study. *Arch Gen Psychiatry.* 2001; 58:769-74.
45. Kircher TT, Liddle PF, Brammer MJ, Williams SC, Murray RM, McGuire PK. Reversed lateralization of temporal activation during speech production in thought disordered patients with schizophrenia. *Psychol. Med.* 2002; 32: 439-49.
46. Buchheim A, George C, Erk S, Kächele H, Walter H. Measuring attachment representation in an fMRI environment: Concepts and assessment. *Psychopathology*. 2006; 39: 136-143.

47. Buchheim A, Erk S, George C, Kächele H, Ruchsow M, Spitzer M, Kircher T, Walter H. Measuring attachment representation in an fMRI environment: A pilot study. *Psychopathology*. 2006; 39: 144-152.

48. George C, West M. *Traumatic dysregulation coding*. Unpublished coding instructions. Mills College, Oakland, CA; 2004.

49. Gunderson JG. The borderline patient's intolerance of aloneness: Insecure attachments and therapist availability. *Am J Psychiatry*. 1996; 153:752-758.

50. First MB, Spitzer RL, Gibbon M, Williams J, Benjamin L. *Structured clinical interview for DSM-IV-Axis I disorders*. New York: Biometrics Research Department; 1995.

51. First MB, Spitzer RL, Gibbon M, Williams J. *Structured clinical interview for DSM-IV-Axis II disorders*. New York: Biometrics Research Department; 1996.

52. Laux L, Glanzmann P, Schaffner CD, Spielberger D. *Das Stait-Trait-Angstinventar*. Weinheim: Beltz; 1981.

53. Barratt ES. Impulsiveness subtraits: Arousal and information processing. In: Spence JT, Izard CE, eds. *Motivation, emotion and personality*. North-Holland: Elsevier; 1985:137-146.

54. Bernstein EM, Putnam FW: Development, reliability and validity of a dissociation scale. *J Nerv Ment Dis*. 1986; 174:727-735.

55. Talairach J, Tournoux P. *Co-Planar Stereotactic Atlas of the human brain*. Thieme, Stuttgart; 1988.

56. Duvernoy HM. *The human brain*. Springer, New York; 1999.

57. Agrawal HR, Gunderson J, Holmes BM, Lyons-Ruth K. Attachment studies with Borderline Patients. A review. *Harv Rev Psychiatry*. 2004; 12:94-104.

58. Dozier M, Stovall KC, Albus KE. Attachment and psychopathology in adulthood. In: Cassidy J, Shaver P, eds. *Handbook of Attachment: Theory, research and clinical applications*. New York: Guilford; 1999:497-519.
59. Van IJzendoorn MH, Bakersman-Kranenburg MJ. Attachment representations in mothers, fathers, adolescents and clinical groups: A meta-analytic search for normative data. *J Couns Clin Psychol*. 1996; 64:8-21.
60. Lyons-Ruth K, Yellin C, Melnick S, Atwood G. Childhood experiences of trauma and loss have different relations to maternal unresolved and Hostile-Helpless states of mind on the AAI. *Attach Hum Dev*. 2003; 5:330-352.
61. Wallis P, Steele H. Attachment representations in adolescence: Further evidence from psychiatric residential settings. *Attach Hum Dev*. 2001; 3:259-268.
62. Buchheim A, George C. The representational and biological function of attachment disorganization in borderline personality disorder and anxiety disorder. In: Solomon J, George C, eds. *Disorganization of Attachment and Caregiving.* New York: Guilford Press; in press.
63. Herpertz S, Kunert HJ, Schürkens A, Steinmeyer EM, Saß H, Freese R, Flesch M, Müller-Isberner R, Osterheider M. Impulskontrolle und Affektregulation bei Persönlichkeitsstörungen. *Psychother Psychosom med Psychol*. 2000; 50: 435-442.
64. Schnitzler A, Ploner M. Neurophysiology and functional neuroanatomy of pain perception. *J Clin Neurophysiol*. 2000; 17:592-603.
65. Lorbeerbaum JP, Newman JD, Dubno JR, Horwitz AR, Nahas Z, Teneback CC, Bloomer CW, Bohning DE, Vincent D, Johnson MR, Emmanuel N, Brawan-Mintzer O, Book SW, Lydiard RB, Ballenger JC, George MS. Feasibility of using fMRT to study mothers responding to infant cries. *Depress Anxiety*. 1999; 10:99-104.

66. Eisenberger N, Lieberman MD, Williams KD. Does rejection hurt? An fMRI study of social exclusion. *Science*. 2003; 302:290-292.

67. Jackson PL, Meltzoff AN, Decety J. How do we perceive the pain of others? A window into neural processes involved in empathy. *Neuroimage*. 2005; 24: 771-779

68. Gündel H, O'Connor MF, Littrell L, Fort C, Lane R. Functional neuroanatomy of grief: An fMRI study. *Am J Psychiatry*. 2003; 160: 1946-1953

69. Bush G, Luu P, Posner M. Cognitive and emotional influences in anterior cingulate cortex. *Trends Cogn Sci*. 2000; 4:215-222.

70. Vogt BA. Pain and emotion interactions in subregions of the cingulate gyrus. *Nature* 2005; 6:533-544.

71. Vogt BA, Finch DM, Olson CR. Functional heterogeneity in cingulate cortex: the anterior executive and posterior evaluative regions. *Cereb Cortex*. 1992; 2:435-43.

72. Vogt BA, Pandya DN. Cingulate cortex of the rhesus monkey: II. Cortical afferents. *J Comp Neurol*. 1987; 262:271-89.

73. Zanarini MC, Frankenburg FR, Hennen J, Silk KR. The longitudinal course of borderline psychopathology: 6 year prospective follow up of the phenomenology of Borderline Personality Disorder. *Am J Psychiatry*. 2003; 160:274-283.

74. Gallagher HL, Happe F, Brunswick N, Fletcher PC, Frith U, Frith CD. Reading the mind in cartoons and stories: An fMRI study of 'theory of mind' in verbal and nonverbal task. *Neuropsychologia*. 2000; 38:11-21.

75. Gallagher HL, Frith CD. Functional imaging of “theory of mind”. *Trends Cogn Sci.* 2003; 7:77-83.

76. Brunet E, Sarfati Y, Hardy-Bayle MC, Decety J. A PET investigation of the attribution of intentions with a nonverbal task. *Neuroimage*. 2000; 11: 157-166.

77. Frith C. Brain mechanisms for 'having a theory of mind'. *J Psychopharmacol*. 1996; 10:9-15.

78. Frith C. What do imaging studies tell us about the neural basis of autism? *Novartis Found Symp*. 2003; 251:149-66.

79. Saxe R, Kanwisher N. People thinking about thinking people. The role of the temporo-parietal junction in "theory of mind". *Neuroimage*. 2003; 19:1835-1842.

80. Walter H, Adenzato M, Ciaramidaro A, Enrici I, Pia L, Bara BG. Understanding intentions in social interaction: The role of the anterior paracingulate cortex. *J Cogn Neurosci.* 2004; 16:1854-63.

81. Saxe R, Carey S, Kanwisher N. Understanding other minds: Linking developmental psychology and functional neuroimaging. *Annu Rev Psychol*. 2004; 55:87-124.

82. Saxe R, Wexler A. Making sense of another mind: The role of the right temporo-parietal junction. *Neuropsychologia*. 2005; 43:1391-9.

83. Fonagy P, Target M, Steele H, Steele M. Reflective-functioning manual: For application to Adult Attachment Interviews. Confidential document (Version 5.0). London, University College; 1998.

84. George C, Solomon J. Attachment and caregiving: The caregiving behavioral system. In: Cassidy J, Shaver P, eds. *Handbook of Attachment: Theory, research and clinical applications*. New York: Guilford; 1999: 649-670.

85. Solomon J, George C. Toward an integrated theory of caregiving. In: Osofsky J, Fitzgerald H, eds. *WAIMH Handbook of Infant Mental Health* New York: Wiley; 2000: 323-368.

86. Perry BD, Pollard RA, Blakley TL, Baker WL, Vigilante D. Childhood trauma, the neurobiology of adaptation, and "use-dependent" development of the brain: How "states" become "traits." *Infant Mental Health Journal.* 1995; 16: 271-289.

87. Eichenbaum H. A cortical-hippocampal system for declarative memory. *Nat Neurosci.* 2000; 1:41-50.

88. Erk S, Kiefer M, Grothe J, Arthur P, Wunderlich A, Spitzer M, Walter H. Emotional context modulates subsequent memory effect. *Neuroimage*. 2003; 18:430-447.

89. Levy K Clarkin JF, Yeomans F. Mechanisms of change in the TFP treatment of borderline patients. *J Clin Psychol*. in press.

90. Fonagy P, Bateman A. Progress in the treatment of borderline personality disorder. *Br J Psychiatry*. 2006; 188: 1-3.

Table 1. Group comparison of clinical scales and attachment trauma scales

Variable	BPD		Control		ES	Exact U-test		
	(n=11)		(n=17)					
	M	SD	**M**	SD		Z	p	
Clinical scales								
State anxiety [52] T1 (before fMRI)	**49.8**	10.9	**37.4**	5.3	1.58	2.92	.002	**
State anxiety [52] T2 (after fMRI)	**46.5**	9.5	**35.7**	4.9	1.54	3.11	.001	**
GSI (SCL-90 General Symptom Index)	**1.47**	.56	**.22**	.22	3.18	4.20	.000	***
Barrett Impulsivity Scale [53] Total Score	**84.8**	11.3	**67.4**	10.0	1.66	3.39	.000	***
Dissociative Experience Scale [54] Total Score	**16.0**	17.6	**4.2**	3.9	1.02	3.22	.001	***
Attachment trauma scales in the Adult Attachment Interview [19]								
Highest score for loss experiences	**5.10**	2.08	**3.18**	2.24	.88	2.16	.030	*
Highest score for abuse experiences	**5.55**	3.11	**1.94**	1.78	1.51	3.11	.001	**

Note. p = exact two-way significance

Table 2. Transcript example of a “Resolved” and “Unresolved” story to a “monadic” AAP picture “Bench”

Resolved AAP story	Unresolved AAP story
Normative Dysregulation (Example of a control subject) “A women is *afraid*, feels bad, had a fight with a friend, sits on a bench to be alone and by herself. She is sitting and crying. Her friend was very disappointed that she has not told him the truth for several times, so he *broke up with her*. Now she *feels abandoned* and is *afraid* of the future. She thinks about the fight and realizes that she has to say sorry. But she is *afraid* that her friend would not talk to her, like her mother often did when she was young. She is *afraid*. She is sitting there for a long time, thinking about the problem. After a while she gets up and is trying to get in contact with the friend to talk about everything.”	Normative Dysregulation (Example of a control subject) “She is very sad, wants to *hide herself under the bench*, she is very *frightened*, feels *abandoned* by everybody. Life can be so cruel. Her friend does not love her anymore, because she has overweight. Her mother *broke up* contact with her because she is not interested in her life anymore. She is *frightened* about the future and she doubts that she ever will meet someone who finds her attractive. I have no idea how this could end. I think she sits there for ever, I really don’t know.”
	Traumatic Dysregulation (Example of a borderline patient) “She feels **homeless**, it seems that she is **incarcerated** in jail, wants to **escape** from this **isolation**, she thinks about **suicide**. It is also possible that she is in a **mental institution**, because she has already tried to commit **suicide** and now she has to be alone in an empty room. Nobody helps her, and she has no relatives or friends. I have no idea. (Long pause) I think she only dreams of **running away**.”

Note. “Normative dysregulation markers” are underlined italics, “traumatic dysregulation markers” are bold

Table 3. Two-group-comparison of Traumatic Dysregulation Marker in the AAP

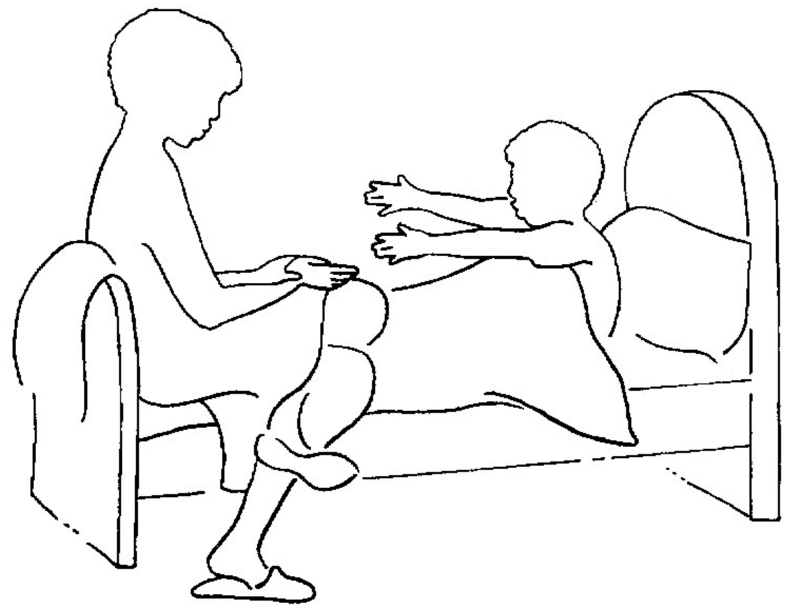
Frequency of Traumatic Marker in the AAP	BPD (n=11)		Control (n=17)		ES	Exact U-test		
	M	SD	M	SD		Z	p	
Total in all monadic (alone) pictures	**9.73**	8.42	**1.53**	1.97	1.51	3.66	.000	***
Window	**2.27**	3.50	**.53**	1.50	.71	3.22	.001	***
Bench	**2.82**	3.19	**.29**	.69	1.23	2.78	.005	**
Cemetery	**2.27**	2.28	**.29**	.77	1.28	3.05	.002	**
Corner	**2.36**	2.80	**.41**	.80	1.06	2.06	.040	*
Total in all dyadic pictures	**2.09**	3.21	**.53**	1.12	.72	1.45	.138	n. s.
Departure	**.18**	.60	**.12**	.49	.12	.32	1.000	n. s.
Bed	**.64**	1.50	**.29**	.99	.28	1.02	.422	n. s.
Ambulance	**1.27**	2.37	**.12**	.49	.76	1.66	.167	n. s.

<u>Note.</u> p = exact two-way significance

Table 4: Three-group-comparison: Occurrence Frequency of Traumatic Dysregulation Marker in the AAP

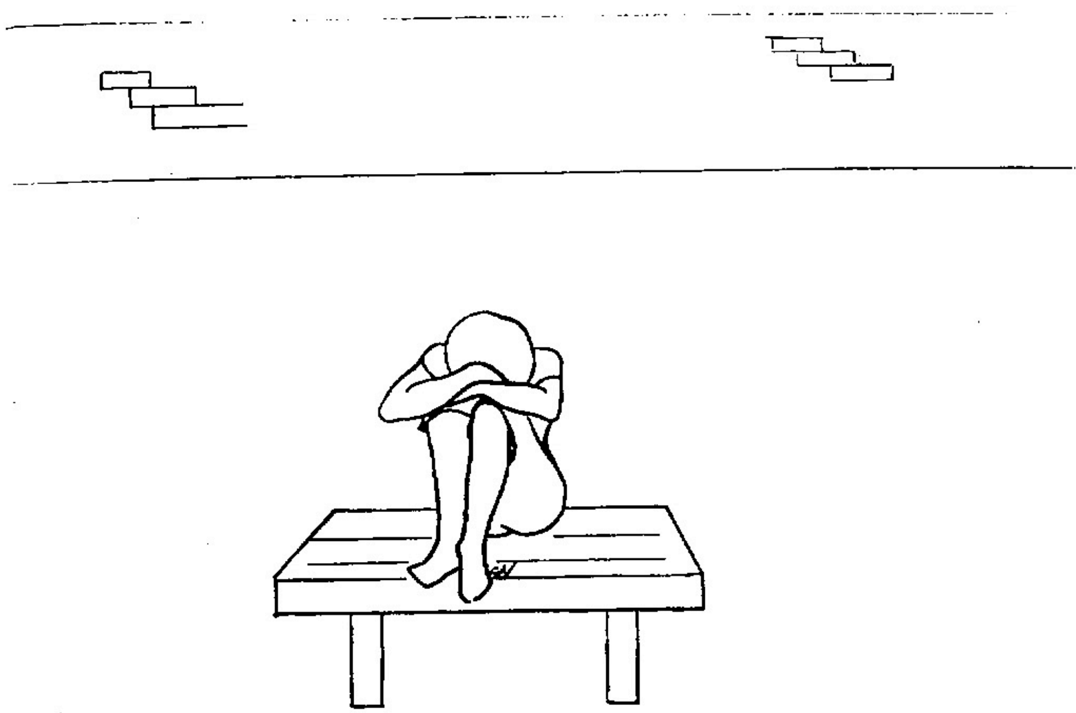
AAP picture	R Control Resolved (n=10)		U Control Unresolved (n=7)		B Borderline Unresolved (n=11)		R×U U test	R×B U test	U×B U test	R×U×B H test
	M	SD	M	SD	M	SD	p	p	p	p
Monadic (alone) pictures	**1.00**	2.21	**2.29**	1.38	**9.73**	8.41	.012	.000	.002	.000
Window	**.40**	1.26	**.71**	1.89	**2.27**	3.50	.743	.004	.020	.003
Bench	**.10**	.32	**.57**	.98	**2.82**	3.19	.331	.007	.117	.011
Cemetery	**.30**	.95	**.29**	.49	**2.27**	2.28	.537	.016	.032	.006
Corner	**.20**	.63	**.71**	.95	**2.36**	2.80	.250	.061	.305	.064
Dyadic pictures	**.70**	1.34	**.29**	.76	**2.09**	3.21	.515	.173	.083	.290
Departure	**.00**	.00	**.29**	.76	**.18**	.60	.412	1.000	1.000	.709
Bed	**.50**	1.27	**.00**	.00	**.64**	1.50	.485	.404	.245	.359
Ambulance	**.20**	.63	**.00**	.00	**1.27**	2.37	1.000	.325	.245	.251

Note. Mann-Whitney U-test: p = exact two-way significance; Kruskal-Wallis H-test: p = significance estimated in 100 000 Monte-Carlo trials



5

Figure 1a. Dyadic AAP Picture: „Bed“ (George et al. 1999)



4

Figure 1b. Monadic (alone) AAP Picture: „Bench“ (George et al. 1999)

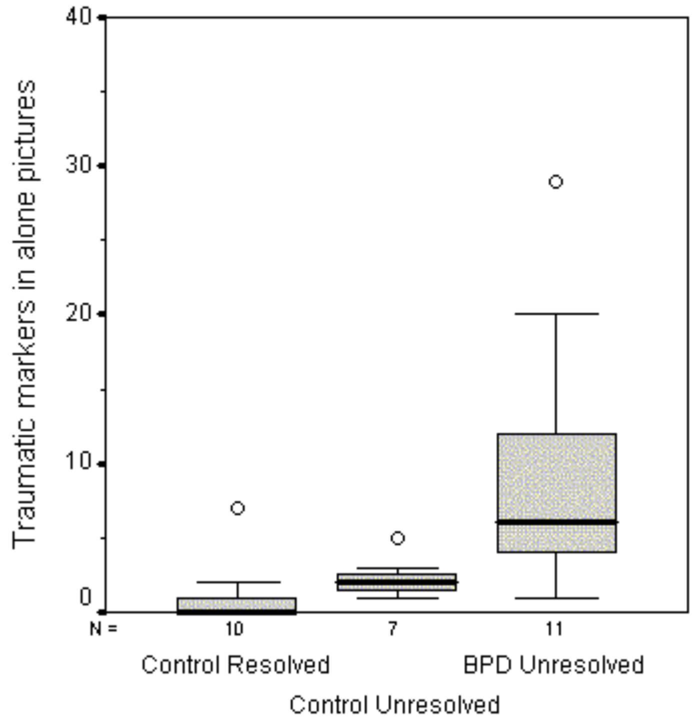


Figure 2. Occurrence frequency of traumatic dysregulation markers in monadic (alone) pictures

Note. The thick median line at the graph bottom signalizes the value 0 („no marker occurrence“) by the majority of subjects

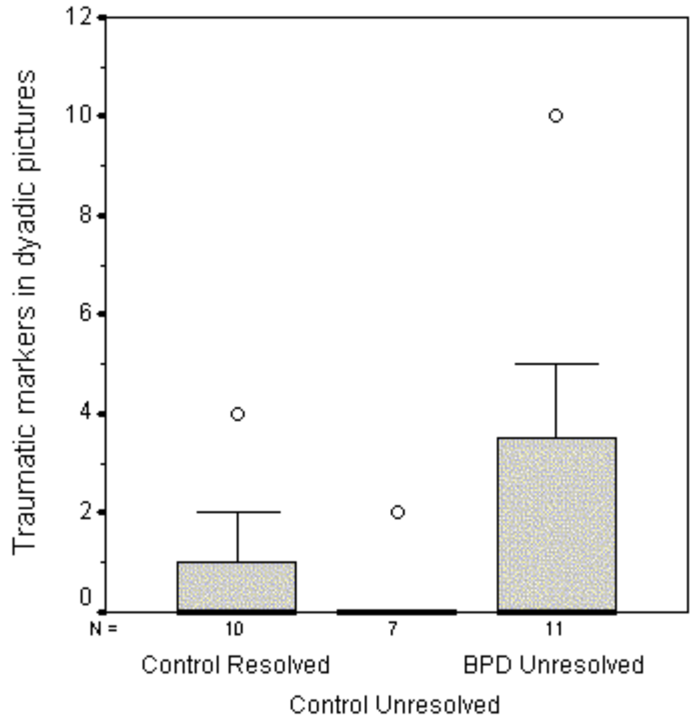


Figure 3. Occurrence frequency of traumatic dysregulation markers in dyadic pictures

Note. The thick median lines at the graph bottom signalize the value 0 („no marker occurrence“) by the majority of subjects.

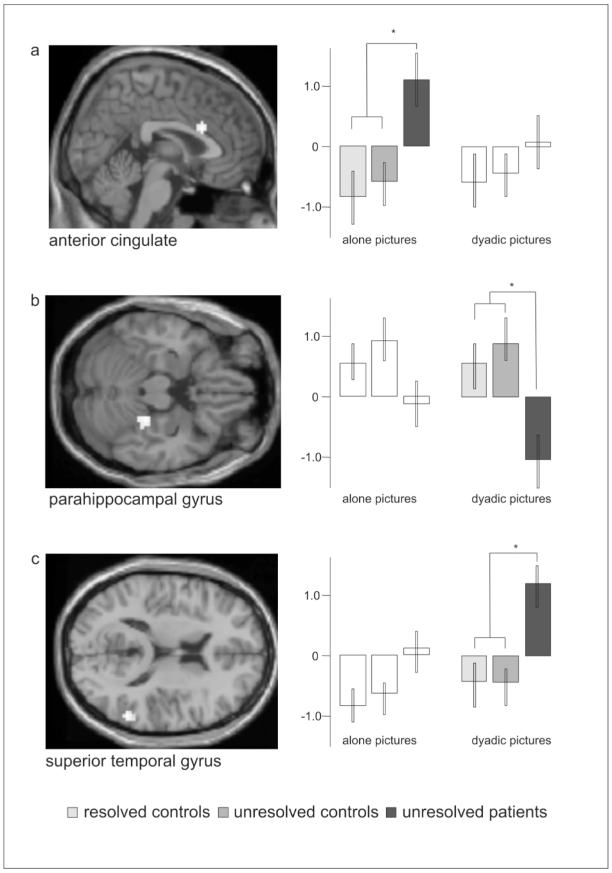


Figure 4 a, b, c

Group differences in brain activation during the narration of stories to attachment related pictures from the Adult Attachment Projective (AAP). The results are from the second level analysis for monadic (alone) pictures (n=3) or dyadic pictures (n=3), respectively, thresholded at $p<0.001$ at the voxel level and $p< 0.05$ at the cluster level (for exact location and z-values see text). The figure shows effect sizes with standard error of mean for each group (unresolved patients, n=11, unresolved control, n=7, and resolved controls n=10). Note, that the groups are grey scale coded only for the analysis in which the effect is significant at the chosen level of significance.