Impaired Central Processing of Emotional Faces in Anorexia Nervosa

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Objectives: To elucidate the potential relationship between classification of emotional faces and impaired central processing in eating disorders and to investigate the potential mediatory role of alexithymia and depression in this relationship. **Methods:** Visual-evoked potentials (VEPs) to emotional faces and classification performance were assessed in 12 anorexic females and matched healthy controls. **Results:** Patients with anorexia nervosa showed no modulation of emotional face processing and displayed significantly increased N200 amplitudes in response to all emotional categories and decreased VEPs in response to unpleasant emotional faces in the P300 time range as compared with healthy controls. They also made more mistakes in emotional face recognition, in particular, for neutral, sad, and disgusted content. **Conclusions:** There are marked differences in evoked potentials and emotion recognition performances of patients with anorexia nervosa and controls in facial processing. Differences in brain dynamics might contribute to difficulties in the correct recognition of facially expressed emotions, deficits in social functioning, and in turn the maintenance of eating disorders. **Key words:** anorexia nervosa, emotional faces, emotional processing, VEPs, EEG, alexithymia.

ED = eating disorder; **ERP** = event-related potential; **VEP** = visual-evoked potential; **AN** = anorexia nervosa; **MDD** = major depressive disorder; **EEG** = electroencephalography; **TAS** = Toronto Alexithymia Scale; **BMI** = body mass index; **BDI** = Beck Depression Inventory; **STAI** = State Trait Anxiety Inventory.

INTRODUCTION

Eating disorders (EDs) are the most prevalent psychiatric disorders in females aged 14 to 26 years and are associated with considerable physical and psychological morbidity (1–4). With prevalence rates of up to 0.3% to 1% among young females (5), these disorders represent a great challenge for physicians of various specialties and significantly affect health care in the female population (6).

There is evidence that the recognition of emotional states is affected in eating disorders. The human face represents a powerful medium for social signaling and the ability to decode complex facial expressions is essential to social behavior (7). Kucharska-Pietura and co-workers (8) reported difficulties in recognizing emotions from facial expression in patients with anorexia nervosa (AN) and suggested that this impairment may contribute to poor interpersonal communication and a lack of empathy, both of which have been shown to be associated with AN. Zonnevijlle-Bender and colleagues (9) also demonstrated that patients with eating disorders perform worse on an emotion recognition test as compared with healthy controls. However, results are not univocal. Still other studies (10,11) have found comparable facial recognition performance in patients with EDs. To date, the question as to whether possible deficits are accompanied by differences in the central processing of emotional faces as measured by visual-evoked potentials (VEPs) and their components in response to emotional faces remains open.

An important variable in the possible interrelation between eating disorders and emotion recognition is alexithymia, a syndrome marked by the inability to identify, describe, regulate, and express one's emotions (12,13). Several studies have shown that ED patients are characterized by high alexithymia scores (9–11,14,15). This variable might contribute to or, to some extent, help to explain the observed difficulties in recognizing emotional states in EDs and should therefore be taken into account when investigating the processing of emotional faces in EDs.

Brain processes accompanying the perception of emotional faces can be studied by means of event-related potentials (ERPs). Especially components like the N200 and the P300 are of interest and were studied in relationship to EDs (16). The N200 has an anterior scalp distribution (17,18) and is sensitive to deviations from the long-term context that renders a stimulus unfamiliar and difficult to encode (18). Furthermore, the N200 is related to the inhibition of executive functions (17,19). The P300 amplitude is one index of attention and processing capacity (20) and reflects cognitive resources allocated to the evaluation of relevant information (21,22), the performance of cognitive processing (23) and target detection (24). It covaries with both reported arousal (25–27) and peripheral indices of cardiovascular reactivity (28) to emotional stimuli. Previous research has also indicated that differences between emotional and neutral faces and between different emotional facial expressions are reflected in brain activity in the time range of 200 to 500 milliseconds (2,29) post stimulus, which includes the N200 and the P300 time window. These components might therefore represent a possible indicator of differences in processing of emotional faces in EDs.

The objective of the present study was to investigate emotional face processing and recognition in patients with AN by means of ERPs. More specifically, we hypothesized that anorexic patients would show differential central processing, as measured by the N200 and the P300, and decreased emotion recognition performance of emotional faces in comparison with healthy controls. The second aim was to investigate the potential mediatory role of alexithymia and depression in this hypothesized relationship.

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MATERIALS AND METHODS

Participants

Fifteen female patients with AN were recruited from patient self-help groups (Anorexia Nervosa and Associated Disorders (ANAD) e.V., Pathways, Cinderella e.V., Caritas Self-Help Centre, Max-Planck-Institute of Psychiatry) in Munich between October 2006 and May 2007. All female patients met the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV) (30) criteria for AN as assessed by the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID) (31). Exclusion criteria included past or present psychotic disorders, any current medication (except contraceptives), and substance abuse (tobacco use was allowed). A systematic evaluation of past neurological problems (e.g., head injuries) was not performed.

Mean ± standard deviation age in the AN group was 22.6 ± 5.6 years. Mean body mass index (BMI) was 16.3 ± 1.1 kg/m^2 and mean duration of illness was 3.7 ± 3.2 years. Four (27%) patients received an additional Axis I diagnosis. Of these, two had major depressive disorder (MDD) with no comorbid anxiety disorder, one had MDD with a comorbid anxiety disorder (panic disorder), and one had social phobia with no other comorbid anxiety or depressive disorder. Due to the possible impact of MDD on the results, the three patients with MDD were excluded from all further analyses. All other participants with AN were of the restricting subtype.

Patients with AN were matched for gender, age, and educational level with healthy controls. Controls had a mean age of 24.1 ± 5.5 years, a mean BMI of 22.0 ± 4.8 kg/m^2; they were recruited in universities, vocational schools, and technical colleges. They were also assessed using the SCID (31). None of the control participants had an Axis I diagnosis or currently received medication (except contraceptives). Participants were paid 30 Euros for taking part in the study.

Procedure

Experiments were conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from an Institutional Review Board. Participants were provided with written information about the experiment and informed consent was obtained. Height and weight were measured and all participants completed a series of questionnaires including the Beck Depression Inventory (BDI), the State Trait Anxiety Inventory (STAI), the Toronto Alexithymia Scale (TAS), and a questionnaire assessing personal data, such as age and schooling.

A total of 240 emotional faces selected from the Karolinska Directed Emotional Faces (32) were presented. This set of 240 comprised 40 faces in each of the following categories: neutral, sad, happy, fearful, angry, and disgusted. All pictures were presented in random order and the subjects were requested to classify the faces according to the six emotional categories. To this end, a forced-choice procedure with six possible categories was presented after each picture presentation. For each emotional category, half of the faces depicted were female and half were male. A single trial began with a fixation cross followed by an emotional face which was presented for 2 seconds. Participants were instructed to avoid exploratory eye movements and eye blinks and to attentively observe the pictures. Immediately after picture offset, participants were given 10 seconds in which to classify the emotional quality of the stimuli by pressing one of six response buttons corresponding to the six emotional categories. They were instructed to be as accurate as possible but to also carry out classification in the case of uncertainty. If a response was not provided within 8 seconds, a second command appeared on the screen which prompted the subject to enter his/her classification. After the response or a maximal interval of 10 seconds, a variable time interval of 1.5 to 3 seconds followed before the next trial commenced. Over the course of the experiment, two short 5-minute breaks took place. The experiment lasted approximately 1 hour.

Electroencephalography (EEG) Recording and Reduction

EEG activity was recorded using 64 Ag-AgCl leads according to the 10 to 10 system with a band-pass of 0.01 to 100 Hz (SynAmps, Compumedics Neuroscan, Charlotte, North Carolina) and digitized at a sampling rate of 1000 Hz. Electrode positions were determined using an electrode cap (Falk Minow Services, Herrsching-Breitbrunn, Germany). The recordings were referenced to Cz and re-referenced offline to linked mastoids. Horizontal and vertical eye movements were recorded using electrodes placed at the outer canthus of each eye (EOG_H) and above and below the left eye (EOG_V). Electrode resistance was maintained below 5 K Ω.

Blinks were corrected using the Gratton and Coles algorithm implemented in the analysis software (Brain Vision Analyzer, Brain Products, Gilching, Germany). The EEG was examined for muscle activity and other sources of artifacts. Trials contaminated by artifacts were eliminated before averaging, and accounted for approximately 8% of the trials. There were no systematic differences in the amount or distribution of artifacts between patients and controls. Finally, EEG was filtered (30 Hz) and averaged for each category with onset of picture presentation. Epochs extended from 200 milliseconds before trigger onset to 1000 milliseconds after trigger onset. Only epochs with correct recognition of face category were included in the VEPs analyses.

Data Analysis

Sociodemographic and questionnaire data were entered into analyses of variance (ANOVAs) with the between-factor Group.

Task performance was examined as the proportion of correct responses for each emotional category and entered into a mixed design analysis of covariance (ANCOVA) with six levels of Emotional Condition as repeated measure and two levels of *Group* as between-subject measure. Alexithymia and Depression were included as covariates. In the Results section, uncorrected F values are reported together with Greenhouse-Geiser epsilon values and corrected probability levels.

VEPs were averaged for 12 regions, formed by crossing hemisphere (right/left) with horizontal plane (anterior, medial, posterior), and vertical plane (inferior, superior) (33,34). Concerning the N200, peak amplitudes (peak ±20 sampling points) and peak latencies in the time range between 180 and 300 milliseconds were calculated. With regard to the P300 and its broad waveform, mean voltages were assessed in the time window of the P300 (280–450 milliseconds) as well as peak latencies. EEG data were analyzed using ANCOVAs with two levels of Hemisphere (right/left), six levels of Region (antero-inferior, antero-superior, medial-inferior, medial-superior, postero-inferior, postero-superior), six levels of Emotional Condition (neutral, happy, sad, fearful, angry, disgusted), and two levels of Group (anorectic versus control) with Alexithymia and Depression as covariates.

Finally, correlations between questionnaire data and both task performance and VEPs were assessed, using Pearson correlation coefficients and partial correlation coefficients.

RESULTS

Sample Description and Questionnaire Data

Sociodemographic features (age, BMI) and questionnaire data obtained for each of the two participant groups are presented in Table 1. Group comparisons revealed a significantly lower BMI for participants with AN and no differences with respect to schooling or age (Table 1). Patients with AN scored significantly higher in depression (BDI), trait anxiety (STAI), and alexithymia (in subscores 1: "ability to identify feelings" and 2: "ability to describe feelings" as well as in total score). To rule out possible interactions between emotional face processing and alexithymia, the TAS subscores 1 and 2, in which significant differences between both groups occurred, were included as covariates in further analyses. Also, depression measured by the BDI score was included as further covariate.

Face Recognition Performance

Figure 1 summarizes performance on the face recognition task for patients with AN and controls separately.

TABLE 1. Comparison Between the Two Groups With Respect to Sociodemographic and Questionnaire Data

	Anorexics (mean ± SD)	Controls (mean ± SD)	F (df = 1,28)	p
Age	23.86 ± 4.25	22.39 ± 4.78	0.93	NS
Schooling	4.09 ± 0.95	4.27 ± 0.46	0.53	NS
BMI	16.34 ± 1.14	22.95 ± 4.52	26.09	***
BDI	18.42 ± 8.25	3.07 ± 3.10	22.03	***
STAI-state	41.42 ± 5.68	37.73 ± 9.65	2.26	NS
STAI-trait	49.58 ± 10.02	36.73 ± 10.04	14.69	**
TAS 1	21.16 ± 5.40	14.91 ± 4.63	18.21	***
TAS 2	18.92 ± 3.99	13.81 ± 5.60	25.17	***
TAS 3	17.67 ± 3.42	18.67 ± 4.64	1.22	NS
TAS total	66.52 ± 13.44	47.39 ± 11.65	15.43	**

SD = standard deviation; BMI = body mass index; BDI = Beck Depression Inventory; STAI = State Trait Anxiety Inventory; TAS = Toronto Alexithymia Scale.
** $p < .01$; *** $p < .001$.

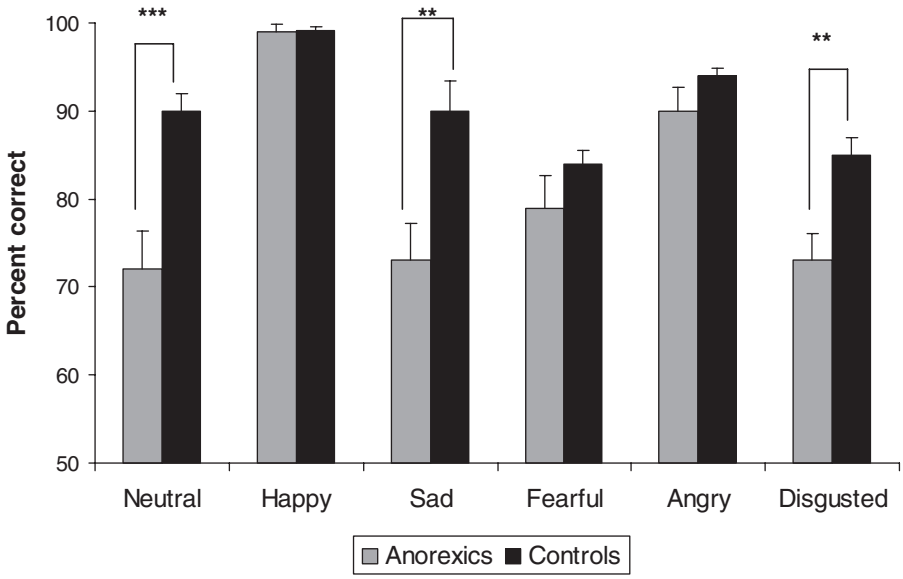

Figure 1. Recognition performance contrasting controls and anorexic patients. Bars represent standard error of means. * $p < .05$; ** $p < .001$.

The ANCOVA revealed a significant main effect of *Face Category* ($F(5,110) = 2.79$, $p = .04$, $\eta^2 = 0.11$, $\varepsilon = 0.69$), indicating differential performance in the six face categories. Post hoc least significant difference tests showed that happy (correct response rate: 99.3%) and angry faces (93.0%) were recognized significantly better than sad (81.7%), fearful (82.3%), neutral (81.6%), and disgusted faces (78.9%; all comparisons, $p < .05$). Furthermore, a significant main effect of Group ($F(1,22) = 5.31$, $p = .03$, $\eta^2 = 0.19$, $\varepsilon = 0.59$) showed that females with AN made significantly more mistakes (mean correct response rate = 81.6%) than healthy controls (90.8%). These main effects were qualified by a significant interaction of Face Category × Group ($F(5,110) = 4.06$, $p = .008$, $\eta^2 = 0.16$, $\varepsilon = 0.86$). Post hoc t tests revealed significantly lower recognition performance in anorexic females for neutral ($p < .001$), sad ($p = .02$), and disgusted ($p = .007$) faces (Figure 1).

With respect to the covariates alexithymia and depression, no significant main or interaction effects were observed. TAS subscores 1 ("ability to identify feelings") and 2 ("ability to describe feelings") as well as depression were therefore excluded as covariates from the detailed error analyses.

The misclassifications of emotional faces were analyzed, using repeated-measures ANOVAs. No significant interaction effects occurred, indicating that patients with AN and controls made the same types of errors within all emotional categories.

Correlation coefficients were computed for emotion recognition performance and alexithymia subscales 1 and 2 as well as with depression score of the BDI. Significant negative correlation coefficients were observed between correct response rate and the degree of alexithymia for neutral faces (TAS 1, $r = -.61$, $p = .001$; TAS 2, $r = -.55$, $p = .003$) as well as between the depression scores and the correct response rate for neutral ($r = -.61$, $p = .001$) faces.

To investigate the role of the clinical variables like the BMI and the duration of illness on the face identification performance, correlation analyses were performed. These two variables did not correlate significantly with the correct response rates for emotional faces.

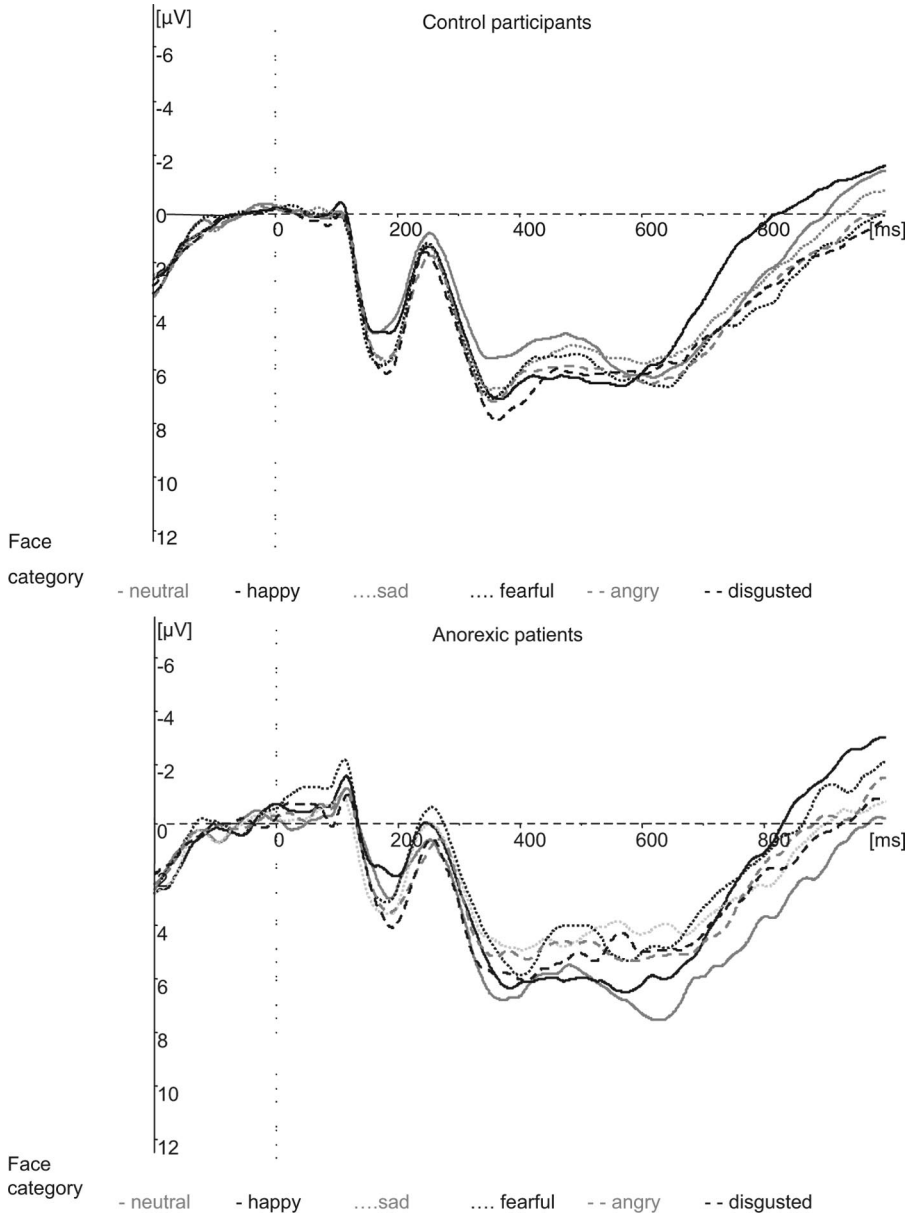

Figure 2. VEPs to the six emotional face categories within controls and anorexic patients.

VEPs to Emotional Faces

As seen in Figure 2, VEPs substantially differed across the six face categories as well as between healthy and anorexic participants in the time window starting at 200 milliseconds (depicted at right medial-superior electrode cluster). Figure 3 contrasts the two groups with respect to each of the six emotional categories (at medial-superior electrode cluster corresponding to the used statistical analyses based on 12-electrode clusters).

In accordance with latency ranges reported in earlier studies using emotional pictures (33–36) or emotional faces (3) and based on visual inspection of the grand average, peak amplitudes (peak ±20 sampling points) and peak latencies in the time window of 180 to 300 milliseconds for the N200, and mean amplitude and peak latencies in the time range of 280 to 450 milliseconds for the P300 were examined. Alexithymia (TAS subscores 1 and 2) and depression were included as covariates in subsequent analyses. Results are presented with a focus on the between-subject factor Group.

N200 Statistical Analyses

Peak Amplitude

A significant main effect of Group ($F(1,22) = 9.25$, $p = .006$, $\eta 2 = 0.30$, $\varepsilon = 0.83$) was observed indicating more negative N200 amplitudes for anorectic patients as compared with healthy controls (mean −1.11 versus 1.47 μV). Additionally, there was a significant effect of the covariate Depression ($F(1,22) = 7.42$, $p = .01$, $\eta^2 = 0.25$, $\varepsilon = 0.74$). The Group × Face Category interaction was not significant ($F(5,110) = 1.43$, $p = .23$).

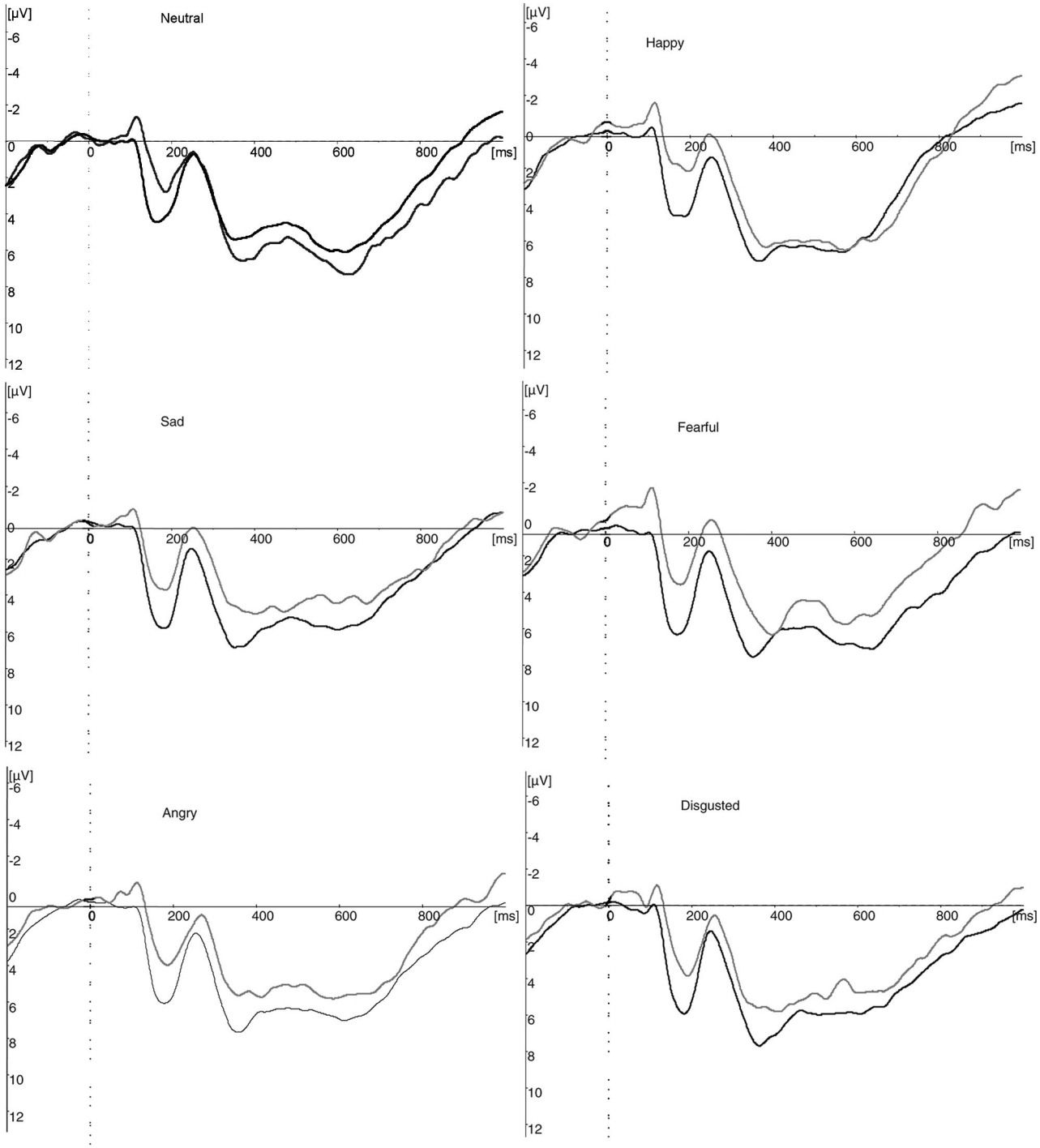

Figure 3. VEPs contrasting controls and anorexic patients for the six face categories.

Peak Latency

Mean N200 peak latencies did not differ significantly with regard to Group ($F(1,22) = 0.37$, $p = .55$) or the added covariates.

P300 Statistical Analyses

Mean Amplitude

The ANCOVA revealed a significant main effect of Group ($F(1,22) = 9.36$, $p = .006$, $\eta^2 = 0.30$, $\varepsilon = 0.83$) and significant interaction effects Group × Region ($F(5,110) = 3.18$, $p = .047$, $\eta^2 = 0.13$, $\varepsilon = 0.60$) and Group × Face Category ($F(5,110) = 2.81$, $p = .03$, $\eta^2 = 0.11$, $\varepsilon = 0.73$). Mean P300 activity was significantly higher in healthy controls as compared with females with AN (mean 4.53 μV versus 2.70 μV) whereas the observed Group × Face Category interaction indicated that this effect was only present for specific emotional categories (see subsequent analyses). This difference was most pronounced over antero-superior, antero-

inferior, and medial-superior regions. Neither main effects (TAS 1: $F(1,22) = 0.03$, $p = .86$; TAS 2: $F(1,22) = 0.01$, $p = .91$; BDI: $F(1,22) = 0.41$, $p = .53$) nor interactions effects, including the covariates Alexithymia and Depression, were observed and these variables were therefore excluded from further analyses.

Based on the significant Group × Face Category interaction effect, post hoc ANOVAs were calculated to determine in which of the emotional face categories the two groups significantly differed and whether VEPs were modulated as a function of face content for both healthy controls and patients.

ANOVAs computed separately for each emotional category revealed that females with AN exhibited lower P300 mean amplitudes in response to sad (mean 4.62 μV versus 2.44 μV; $F(1,25) = 10.33$, $p = .004$), fearful (mean 4.62 μV versus 2.44 μV; $F(1,25) = 12.78$, $p = .001$), angry (mean 5.20 μV versus 2.65 μV; $F(1,25) = 24.91$, $p < .001$), and disgusted (mean 5.32 μV versus 3.28 μV; $F(1,25) = 12.82$, $p = .001$) faces and significantly higher mean amplitudes in response to neutral faces (mean 2.56 μV versus 3.56 μV; $F(1,25) = 4.74$, $p = .04$).

ANOVAs conducted for each of the two participant groups revealed a significant main effect of Face Category ($F(5,70) = 8.17$, $p < .001$, $\eta^2 = 0.37$, $\varepsilon = 0.99$) in healthy controls with higher mean P300 activity for sad, angry, and disgusted faces as compared with neutral faces (all post hoc tests, $p < .05$). No significant modulation of the P300 according to Face Category ($F(5,55) = 2.05$, $p = .14$) was observed in the AN group.

Peak Latency

Neither Group ($F(1,22) = 0.16$, $p = .70$) nor the interaction effects Group × Region ($F(5,110) = 0.66$, $p = .49$) and Group × Face Category ($F(5,110) = 1.42$, $p = .24$) were significant.

Relationship Between Face Recognition Performance, N200 and P300 Amplitude

Correlation coefficients were computed between the correct response rate for each emotional face category and the corresponding mean P300 amplitudes as well as N200 peak amplitudes at antero-superior, antero-inferior, medial-superior, medial-inferior, postero-superior and postero-inferior electrode pools.

For the N2 component, significant correlations were observed between task performance and amplitude for sad faces at medial-superior ($r = .40$, $p = .04$) electrode location, for fearful faces at antero-superior ($r = -.40$, $p = .01$) and postero-inferior ($r = .50$, $p = .01$) locations, and for disgusted faces at antero-superior ($r = .54$, $p = .004$) and antero-inferior ($r = .49$, $p = .009$) electrode locations. In accounting for the degree of alexithymia (TAS subscales 1 and 2) and depression by assessing partial correlations, all observed correlation coefficients remained significant (sad: medial-superior, $r = .41$, $p = .04$; fearful: antero-superior, $r = -.54$, $p = .006$, postero-inferior, $r = .59$, $p = .002$; disgusted: antero-superior, $r = .48$, $p = .03$, antero-inferior, $r = .51$, $p = .01$).

Concerning the P300, significant correlations were observed between task performance and P300 amplitude for sad faces at antero-superior ($r = .46$, $p = .02$) and medial-superior ($r = .41$, $p = .04$) electrode locations, for fearful faces at antero-superior at ($r = -.39$, $p = .04$) and postero-inferior ($r = .49$, $p = .01$) locations, and for disgusted faces at antero-superior ($r = .54$, $p = .004$) and antero-inferior ($r = 0.51$, $p = .007$) electrode locations. In accounting for the degree of alexithymia (TAS subscales 1 and 2) and depression by assessing partial correlations, all observed correlation coefficients remained significant (sad: antero-superior, $r = .40$, $p = .04$, medial-superior, $r = .41$, $p = .03$; fearful: antero-superior, $r = -.54$, $p = .006$, postero-inferior, $r = .59$, $p = .002$; disgusted: antero-superior, $r = .49$, $p = .02$, antero-inferior, $r = .51$, $p = .01$).

DISCUSSION

Our data provide evidence of differential processing of emotional faces in subjects with AN as compared with healthy controls. Anorexic females not only made more mistakes in classifying neutral, sad, and disgusted faces, but they also exhibited increased N200 amplitudes to all face categories and decreased P300 amplitudes in response to unpleasant emotional faces. In the P300 time range, patients with AN showed less modulation of the VEPs in connection with the emotional valence of the facial expression as was the case in healthy participants. These effects are not attributable to differences in alexithymia and depression as these variables were controlled for. Possible shortcomings of the present study include its relative small sample size and a missing participants' evaluation of neurological history including head injuries.

With respect to the observed differences in N200 amplitudes, it can be suggested that patients with AN have greater difficulty to encode facial expressions. According to Dennis and Chen (37), enhanced N200 amplitudes may specifically reflect reduced resources available for attention performance and fewer attentional control resources. Consistent with this view, enhanced N200 amplitudes were correlated with lower task performance in the subsequent categorization task. However, another more likely explanation for the differences in N200 amplitudes might be a general increase in attentional demands for the classification of emotional faces for subjects with AN. It was shown that the frontocentral N200 is sensitive to visual novelty and mismatch between perceived and expected (template) information (17,18,38). Because the continuous presentation of faces in the present experiment is unlikely to provoke a novelty reaction, increased N200 amplitudes might rather reflect a general impairment in patients with AN to provide an adequate template for the efficient processing of facial expressions. Thus, processing of facial expression in these patients would lead to an increase in attentional demands or the need to shift attentional resources to the appropriate emotional category. The comodulation of the N2 component by depression as measured by the BDI points to an influence of depression on the visual-evoked N200 component in processing facial expressions. However,

ERP studies in individuals with depression are scarce and show equivocal results. Whereas a study by el Massioui and Lesèvre (39) showed a weak attention effect on the N200 component in patients with unipolar depression, more recent studies imply normal attention effects in depressed patients (40,41). Further studies with larger sample sizes have to systematically investigate the role of depression on the visual-evoked N200 component.

The present study revealed decreased P300 amplitudes in response to unpleasant emotional faces. This result is not in accordance with data from Dodin and co-workers (16), showing that subjects with AN had larger P300 amplitudes and longer P300 latencies in an oddball task, using body images and geometrical shapes. The discrepancy can be partly explained by differences in the task and differences in the stimuli used in both studies, namely, emotional faces as compared with geometric shapes and body images. In our opinion, the present study used a more complicated task as compared with that of Dodin and co-workers (16) in that we forced participants to attentively categorize the emotional content of each face presented. This very specific and attentional demanding task might interfere with the postulated nonspecific hyperarousal in AN as suggested by Dodin and co-workers (16). We argue that the observed differences in the P300 amplitude were not caused by decreased attentional capacity in patients with AN for the following reasons: first, patients with AN performed comparably with controls in several of the emotional categories; second, we did not observe a general decrease in P300 amplitude in the group with AN—their VEPs did not differ for happy faces and were even increased for neutral faces as compared with healthy controls. This result thus substantially extends former research reporting a decreased ability to recognize the expression of emotional faces in AN (8,9) by showing that central indices of negative emotional face processing are also affected in AN. Importantly, statistically significant modulation of the P300, according to the emotional expression of the presented faces, was absent in patients with AN. Bearing in mind that emotion recognition performance was related to the amplitude of the P300 for most of the unpleasant emotional categories, it is conceivable that the lack of modulation of this component in response to these emotional contents is accompanied by higher rates of facial misclassification in AN.

Empirical data suggest that emotional face recognition involves the amygdala for fear and anger, the insula and basal ganglia for disgust, and cingulate, medial frontal and parietal cortices for happiness (7). Although imaging and ERP data on emotional face perception in EDs are still lacking, other neuroimaging studies on AN have reported functional abnormalities in prefrontal, cingulated, and temporal structures during the processing of food, emotional or body images (42–45). It can be hypothesized that the observed reduced P300 response to negative facial expressions might reflect a dysfunctional activation pattern in underlying brain structures, such as the cingulate. Further data are required in examining the thus far unanswered question concerning differences in the central processing of emotional faces in AN.

In this context, it is initially astonishing that patients with AN exhibited greater P300 amplitudes to neutral faces as compared with controls. Across all six face categories, the VEPs to neutral faces were most pronounced in patients with AN whereas this category caused the least activity in controls, which in turn might explain the observed inverted difference between the two groups. Whereas healthy controls mobilize the least processing capacities to neutral faces as indexed by their P300 amplitude, patients with AN process neutral faces with a comparable or even higher amount of processing capacities as compared with happy and unpleasant faces. Whereas patients suffering from AN process neutral faces deeper as healthy controls do, they seem to fail in modulating their central activity in reaction to this "unharmful" category of facial information. It is plausible that social interactions in EDs are exhausting, with all facial information including neutral expressions which are frequently encountered in the social environment being connected with misunderstandings and misclassifications with respect to their emotional content.

The marked differences in ERPs between patients with AN and healthy controls cannot be explained by different levels of alexithymia as differences in the level of alexithymia were used as covariates. Interestingly, alexithymia as well as depression correlated negatively with the performance in identifying neutral faces, suggesting this category might be of special difficulty probably as these faces are of higher ambiguity concerning their emotional content. Concerning errors in the categorization of emotional faces, we observed no specific bias in the responses of females with AN. Thus, both groups made similar mistakes whereas patients with AN made quantitatively more of the same errors. An additional point of interest might be whether facial recognition becomes more precise when participants are allowed to take as long as they wish in providing their responses. It is possible that the deficits in correctly responding to emotional faces which were observed in the present study and not found in other studies are attributable to the fact that our participants were only given a certain amount of time in responding to the stimuli. This is a more difficult task than responding to emotional faces without a time constraint.

We conclude that there are marked impairments in the central processing and correct recognition of negative emotional faces in AN. The identification of facial information including the emotion expressed by the sender is a crucial component of social life and interpersonal communication. The observed reduced P300 response to negative emotional faces in AN can be interpreted as evidence of diminished cognitive processing ability with respect to negative emotional faces. This might lead to social difficulties and may thus contribute to the maintenance of EDs.

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