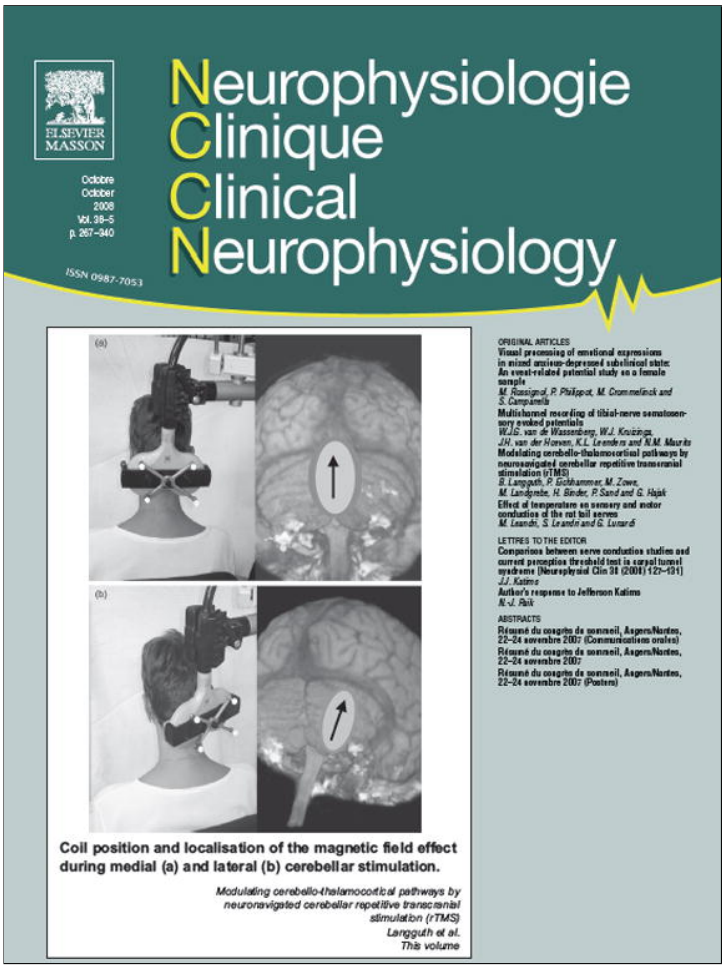

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ARTICLE ORIGINAL/ORIGINAL ARTICLE

Visual processing of emotional expressions in mixed anxious-depressed subclinical state: An event-related potential study on a female sample

Le traitement visuel des expressions émotionnelles dans les états sous-cliniques d'anxiété-dépression : une étude en potentiels évoqués dans un échantillon de sujets de sexe féminin

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KEYWORDS
Affective neurosciences;
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Event-related potentials

Summary
Aims. — Controversy remains about the existence and the nature of a specific bias in emotional facial expression processing in mixed anxious-depressed state (MAD).
Material and methods. — Event-related potentials were recorded in the following three types of groups defined by the Spielberger state and trait anxiety inventory (STAI) and the Beck depression inventory (BDI): a group of anxious participants ($n = 12$), a group of participants with depressive and anxious tendencies ($n = 12$), and a control group ($n = 12$). Participants were confronted with a visual oddball task in which they had to detect, as quickly as possible, deviant faces amongst a train of standard neutral faces. Deviant stimuli changed either on identity, or on emotion (happy or sad expression).

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Results. — Anxiety facilitated emotional processing and the two anxious groups produced quicker responses than control participants; these effects were correlated with an earlier decisional wave (P3b) for anxious participants. Mixed anxious-depressed participants showed enhanced visual processing of deviant stimuli and produced higher amplitude in attentional complex (N2b/P3a), both for identity and emotional trials. P3a was also particularly increased for emotional faces in this group.
Conclusion. — Anxious state mainly influenced later decision processes (shorter latency of P3b), whereas mixed anxious-depressed state acted on earlier steps of emotional processing (enhanced N2b/P3a complex). Mixed anxious-depressed individuals seemed more reactive to any visual change, particularly emotional change, without displaying any valence bias.

MOTS CLÉS
Neurosciences affectives ;
Anxiété ;
Dépression ;
Émotions ;
Visages ;
Potentiels évoqués

Résumé
But de l'étude. — Des controverses subsistent quant à l'existence et à la nature de biais de traitement relatifs aux expressions faciales émotionnelles dans les états mixtes anxieux-dépressifs (MAD).
Materiel et méthode. — Nous avons comparé les réponses cérébrales de trois groupes de volontaires constitués selon leur niveau d'anxiété (mesuré avec l'échelle STAI) et de dépression (BDI) : un groupe de volontaires sains ($n = 12$), un groupe de participants à tendance anxieuse ($n = 12$) et un groupe à tendance anxieuse et dépressive. Les participants se voyaient proposer une tâche visuelle de type *oddball* lors de laquelle ils devaient détecter, aussi rapidement que possible, des visages déviants parmi une série de visages standard. Les stimuli déviants différaient des stimuli fréquents d'expression neutre soit sur le plan de l'identité du visage, soit sur celui de l'émotion affichée (joie ou tristesse).
Résultats. — l'anxiété facilite le traitement émotionnel et les deux groupes de sujets anxieux répondaient plus rapidement aux stimuli rares que le groupe témoin. Cet effet était corrélé à une P300 plus précoce. Les participants anxieux-dépressifs manifestaient un traitement visuel accru des stimuli déviants et produisaient un complexe attentionnel (N2b/P3a) d'amplitude plus élevée, tant pour les stimuli déviants sur l'identité que sur l'émotion. L'onde P3a apparaissait aussi particulièrement ample dans ce groupe.
Conclusion. — Un état anxieux influence principalement les processus décisionnels tardifs alors qu'un état mixte anxieux-dépressif agit sur des étapes plus précoces du traitement émotionnel. Les individus manifestant à la fois des symptômes anxieux et dépressifs semblent plus réactifs aux changements visuels et tout particulièrement aux changements émotionnels, sans pour autant manifester de biais de valence spécifique.

Introduction

The recognition and production of emotional facial expressions (EFE) appear prominent in human's social interactions. They have been extensively investigated among normal individuals in the last few decades [1].

Due to the optimal temporal resolution of event-related potentials (ERP), several visual ERP components were shown to be modulated by emotional category:

- the P100/N100 waves, which are typically described as reflecting primary visual analyses [2];
- the N170, recorded at occipitotemporal sites and reflecting the structural encoding of faces [3,4];
- the N2b/P3a occipitofrontal complex, which is maximally recorded around 250 ms and reflects the voluntary switch of attention operated by a subject attending to deviations [5];
- the P3b, which peaks at parietal sites around 450 ms, and indexes different functions such as cognitive closure and premotor decisional response-related stages [6—8].

ERP studies reported alterations in both anxiety and depression, respectively. On the one hand, anxiety studies suggest the presence of vigilance biases throughout different paradigms [9,10]. For instance, when subclinically anxious individuals (without depressive tendencies) had to detect deviant emotional faces among neutral ones, early waves and attentional stage were not modified, but anxiety was associated with an earlier P3b component [11]. Conversely, individuals with social phobia demonstrated enhanced right temporoparietal N170 amplitudes in response to angry faces in an emotion identification task [12]. On the other hand, ERP studies often reported a delay or a reduction of P3b in clinical depression [13,14]. Lately, Cavanagh and Geisler [15] found that P3b was reduced in amplitude and latency when individuals with depressive tendencies processed happy faces as compared to fearful faces, suggesting that happiness was less salient and more difficult to classify than fear.

Recently, researchers outlined the frequent comorbidity between anxiety and depression, developing the concept of ''mixed anxiety-depression'' (MAD) [16,17]. Increasing attention is being given to the cognitive features of this disorder, and a major question pertains to the way emo-

tions are processed in mixed depressed-anxious states. For example, Bouhuys et al. [18] postulated that anxiety coupled with depression leads to an increased perception of negative EFE in ambiguous faces. More recently, Suslow et al. [19] found that depressed people without comorbid anxious disorder were not impaired in the detection of negative faces, but that MAD patients were slower to respond to positive faces, suggesting that anxiety disorder may lower the efficiency of visual search for positive faces. ERP studies have also stressed the role of comorbid anxiety, by showing differential activation patterns and P300 specific alterations by anxiety cumulated to depression [20,21].

These recent observations stress the importance of considering how two major clinical dimensions — depression and anxiety — might interact in their impact on EFE processing, even at a subclinical level. In order to investigate this issue, we used an emotional oddball paradigm, in which participants are confronted with series of frequent standard stimuli and have to detect infrequent deviant stimuli.

The first aim of this study was to test whether ''subclinical'' MAD individuals showed an impaired processing of emotional faces in general, or whether the impairment of emotional processing is specific to either positive or negative emotions. To this aim, we compared target stimuli depicting the same identity as the neutral frequent ones but displaying a sad or a happy expression with target stimuli portraying a different identity. A general impairment would give rise to both identity and emotional changes, whereas an emotion-specific deficit would be observed only for positive and/or negative emotions. Moreover, we aimed to investigate the specific role of mixed anxious-depressed state in cognitive processing by comparing differences between anxious state and anxious-depressed state at a cognitive level. We observed three groups of participants in order to isolate the effect of anxiety alone and MAD state: individuals with anxiety alone, individuals with anxiety coupled with depression, and individuals without anxiety or depression. Because threatening stimuli were not used, we expected a quicker detection of all deviant stimuli in individuals with anxiety alone compared to the two other groups, due to general speeded processing [22]. In MAD state, we hypothesized a wider alteration of ERP components, appearing since attentional stage and persisting until decisional closure processes, forasmuch as perceptual processes have been involved in anxious-depressed patients [21], but also a more specific alteration of emotional process.

Our second goal was to locate the origin of potential emotional biases in the information processing stream. Studies reported a deficit in P300 in psychopathological states [8,13—15], but if the subjacent deficit is circumscribed to response-related stages, it might also originate from lower level of processing, and extend to the overall process [23]. Because depression and anxiety alone are known to affect the processing of emotional stimuli, we hypothesized a modification of ERP components and, more specifically, the P3b wave in MAD states. These modifications could be sustained either by an attentional alteration as in anxiety [24], with a deficit starting at the voluntary attentional level on the N2b component, or by a disturbed elaborative process as in depression [25], with a perturbation restricted to the P3b component.

Table 1 Psychological characteristics of MAD, HA and CS participants.

	CS	HA	MAD
Age	22.08 (2.61)	22.25 (2.95)	21.66 (1.97)
Beck Score	1.58 (2.06)	6.08 (1.73)	14.58 (4.46)
STAIE	42 (9.56)	57.91 (11.18)	64 (9.29)
STAIT	39.41 (6.07)	61.08 (7.21)	63.5 (8.92)

Materials and methods

Participants

Thirty-six students from the faculty of psychology of University of Louvain took part in the experiment. They were selected from a sample of 180 students according to their scores on French versions of the Spielberger state and trait anxiety inventory (STAI) [26], and of the 13-items Beck inventory scale (BDI) [27]. Given the higher prevalence of anxiety and depression in women [28] and the fact that gender is known to affect EFE processing [29], we decided to select only female participants. All participants were right-handed, between 18 and 28 years of age, with normal/corrected vision, and without neurological disease.

Participants scoring above 56 on trait anxiety (STAI-T [1], [26]) were classified as anxious, and the BDI cut-off scores used to define the depressive tendencies were 10 and higher [30]. On these bases, we constituted three groups of 12 participants: normal control participants (CS), high anxious participants (HA), and high anxious participants with depressive tendencies (MAD). Group characteristics are reported in Table 1.

The experimental procedure used an ''emotional oddball paradigm'' [29,31]. Stimuli consisted of six faces (selected from a highly standardized set of pictures from the Ekman and Friesen series, [32]) with neutral, happy, and sad expressions. Participants had to detect as quickly as possible the occurrence of a deviant face among the presentation of standard faces. Standard faces always presented neutral expressions, whereas deviant faces were either the same face displaying an emotion (sad or happy) or a different neutral face (change in identity).

Stimuli, sizing 6 cm horizontally and 8 cm vertically and subtending a visual angle of $3 \times 4^{\circ}$, were presented one by one on a black background, and a black screen was displayed as an inter-trial interval, lasting randomly between 1300 and 1600 ms. Eighteen blocks were composed, each defined by 100 stimuli (e.g. 80 frequent stimuli with face A neutral; five deviant face A happy, five deviant face A sad and 10 face B neutral). The order of the eighteen blocks varied across participants.

[1] Spielberger Scale scores group the participants as follows: less than 36: very low; 36-45: low; 46-55: normal; 56-65: high; more than 65: very high.

Recording

The EEG recordings were performed with 32 electrodes mounted in an electrode Quick-Cap with the standard 10—20 International System and intermediate positions. Recordings were made with a linked mastoid physical reference, but were re-referenced by using a ''common average'' [33]. The EEG was amplified by battery-operated SYNAMPS amplifiers with a gain of 30 K and a band-pass of 0.01—100 Hz. The impedance of all electrodes was kept below 20 kΩ. EEG was continuously recorded (sampling rate 500 Hz, NeuroScan software) and vertical electrooculogram (VEOG) was recorded in a bipolar manner from electrodes placed on the supraorbital and infraorbital ridges of the left eye. Trials contaminated by EOG artifacts (mean of 15%) were eliminated off-line by computing an average artifact response based on a percentage (in this case, 20%) of the maximum eye movement potential. The EOG response is therefore subtracted from the EEG channels on a sweep-by-sweep, point-by-point basis in order to obtain ocular artifact-free data. Epochs beginning 150 ms prior to stimulus onset and continuing for 850 ms were created. Codes synchronized with stimulus delivery were used to average selectively the epochs associated with different stimulus types. Data were off-line filtered with a 30 Hz low-pass filter.

Procedure

During the ERP recording, participants sat in a chair in a dark room with their head placed 1 m from the screen and restrained in a chin rest. The participants were instructed to signal as quickly as possible the occurrence of deviant stimuli by pressing a mouse button with their right index finger. Faces were presented for 500 ms, and participants had 1500 ms to answer since stimulation onset. The entire experiment took approximately 50 minutes per participant.

Data analysis

Two parameters were coded for every stimulus:

- the type of stimulus (rare happy; rare sad; rare identity; in order to have the same number of averaged frequent stimuli, only the frequent stimuli preceding the deviant ones);
- the type of response (keypress for deviant stimuli, no keypress for frequent ones).

This coding allowed for the computation of different averages of ERP target stimuli. The averages were calculated for each participant individually. Peaks of interest are described subsequently. Statistical analyses were computed using Statistical Package for Social Sciences, 14th version (SPSS 14.0). We conducted repeated-measures ANOVAs (more details will be provided in section ERP Results). Greenhouse-Geiser epsilon correction was used to compensate for violation of sphericity when appropriate. Simple effects analyses of these factors were explored throughout; Bonferroni Post-hoc tests were used when appropriate. The sources of significant interactions were systematically examined through simple effects. The alpha level of significance was set at 0.05 throughout. Eta-squared were equally calculated in order to certify statistical power.

Results

Psychometrical features

Statistical analysis confirmed that groups were not significantly different with respect to age ($F[2,33] = .167$, ns), but BDI scores differed significantly between the three groups ($F[2,33] = 57.77$, $p < .001$). Analyses confirmed that the anxiety level obtained for the STAIT ($F[2,33] = 35.812$, $p < .001$) and the STAIS ($F[2,33] = 15.343$, $p < .001$) did not differ between HA and MAD ($p = .443$ and 1.000, respectively), but that these two groups were significantly different from CS ($p < .001$).

Behavioral data

Behavioral data are presented in Fig. 1. The performance was at 98% correct; analyses were thus conducted only on correct response latencies. We observed a main effect of trial type ($F[2,66] = 10.715$, $p < .001$): All groups detected slower sad stimuli as compared to happy or identity trials (both $p < .001$) without difference between these last stimuli ($p = 1.00$). We also obtained a main effect for group ($F[2,33] = 5.86$, $p = .007$): HA detected rare deviant stimuli more quickly than did CS (respectively: $p = .007$) whereas MAD did not differ from HA ($p = 1.000$) and tend to have quicker detection than CS ($p = .063$).

Correlation analyses demonstrated significant relationships between reaction time and psychological variables. When all groups were merged together, state and trait anxiety was negatively correlated with reaction time to happy ($r = -.493$, $p = .002$; $r = -.398$, $p = .016$), sad ($r = -.476$, $p = .003$; $r = -.400$, $p = .016$) and identity trials ($r = -.493$, $p = .002$; $r = -.401$, $p = .015$), confirming an accelerated detection of deviant stimuli in people with higher level of anxiety. However, these correlations were nonsignificant inside individual groups.

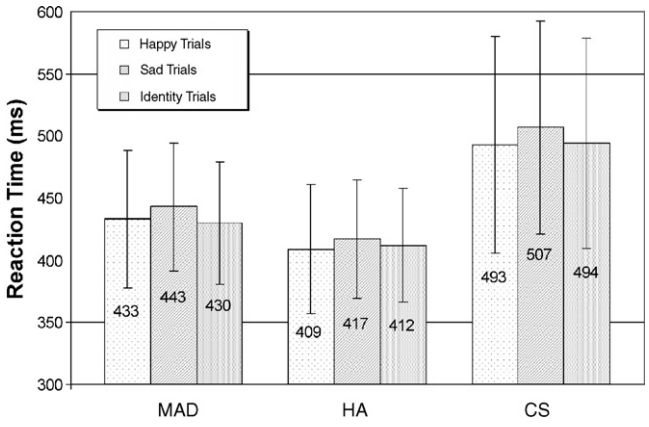

Figure 1 Reaction time (ms) for deviant stimuli detection as a function of group.

ERP results

P100/N100 and N170 components

Initial visual inspection of the non-subtracted ERP waves suggested different consecutive phases of attention effects. There were: an occipital P1 enhancement (90—120 ms); a frontal N1 enhancement (90—120 ms); a parieto-temporal N170 effect (140—200 ms).

The P1 and N1 attention waves were manually identified on the basis of their latency range, topographical distribution and reproducibility from the channels Oz for the P100 and Cz for the N100, in waveforms evoked by frequent and deviant identity, happy and sad stimuli. These measures were submitted to omnibus repeated-measures MANOVA on latency and amplitude, with group (MAD, HA or CS) as between-subjects factor, and trial type (frequent, identity, happy, sad) as within-subject factors. We did not observe any significant effects on early P100/N100 waves.

N170 was manually identified at the TP7 and TP8 electrodes. These measures were submitted to two repeated-measures MANOVAs on latency and amplitude, with group (MAD, HA or CS) as between-subjects factor, and trial type (frequent, identity, happy, sad) and laterality (left or right) as within-subject factors. Statistical analysis did not reveal any significant effect of latency on this wave.

Amplitude was only influenced by trial type ($F[3,93] = 19.288$, $p < .001$) in such a way that N170 was larger for deviant stimuli, emotional or not, than for neutral frequent stimuli ($p \leq .001$ in all cases). However, amplitudes of rare stimuli did not differ according to displayed emotions.

N2b/P3a and P3b effects

Fig. 2 represents the regular oddball components observed in all groups for the rare minus frequent stimuli condition. Firstly, a negativity was maximally recorded around 230 ms at Oz, with a positive counterpart recorded around 220 ms at Fz. Both their morphology and topography allow to identify this bipolar complex as the well-described N2/P3a complex, as observed for instance by Campanella et al. [31]. Secondly, a positivity was recorded around 450 ms, and was identified as the P3b component.

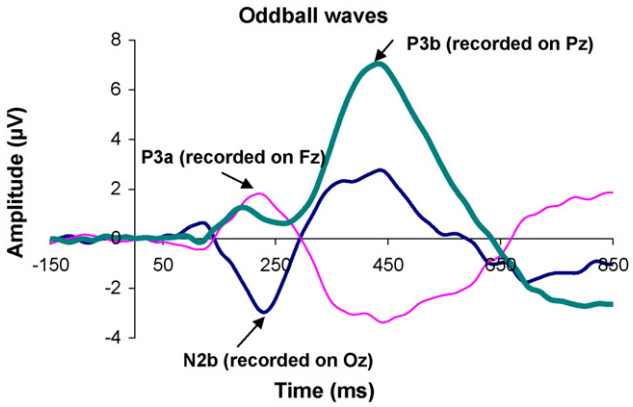

Figure 2 Classical oddball waves, N2/P3a and P3b, recorded in the three groups on Oz, Pz and Fz, and obtained by means of the subtraction RARE stimuli — FREQUENT stimuli.

For N2b (peaking between 200 and 300 ms at Oz), P3a (same time distribution at Fz), and P3b (peaking around 450 ms at Pz) components, individual peak amplitudes and latencies were manually identified on the basis of their latency range, their topographical distribution, and their reproductibility from median channels from the ERP resulting from the subtraction of waveforms evoked by standard and deviant stimuli (Figs. 3 and 4). Theses values were tested using omnibus repeated-measures MANOVA on latency and amplitude, with group (MAD, HA or CS) as between-subjects factor, and trial type (identity, happy, sad) as within-subject factors.

A significant main effect of type of stimulus was observable on N2b latency ($F[2,66] = 16.224$, $p < .001$): sadness (245 ms) evoked later N2b peak than happiness (220 ms; Bonferonni comparison: $p < .001$) or identity (225 ms; Bonferonni comparison: $p < .001$), without difference between these last stimuli ($p = .804$). N2b amplitude was affected by group membership ($F[2,33] = 3.93$, $p = .029$), and post-hoc tests (Bonferroni) showed that N2b was maximally recorded in MAD (−5.41 μV), as compared to HA (−2.87 μV, $p = .047$), without difference as compared CS (−3.13 μV, $p = .085$), these two last groups not differing from each other.

On P3a component, a main trial-type effect was observed on latency ($F[2,66] = 4.642$, $p = .013$) with a later P3a for sad trials (240 ms) as compared to happy (222 ms, $p = .047$) or identity trials (219 ms, $p = .041$), without difference between these last type of trials.

Trial type had an equal impact on amplitude ($F[2,66] = 5.756$, $p = .007$): P3a was globally reduced for identity trials (2.313) compared to emotional ones (happy: 3.2 μV, $p = .05$; sad: 3.4 μV, $p = .001$; no difference between emotional trials: $p = 1.000$). This effect is not surprising, given that the emotional-trial category was composed of both sad and happy trials; the number of identity trials in the whole experiment was equal to the sum of these two categories. Since the P3a has been associated with a novelty effect, we expected a larger wave in response to emotional trials than for identity trials, two times as frequently [31]. The results confirmed this hypothesis.

More interestingly, a group effect ($F[2,33] = 5.307$, $p = .01$) was observed: MAD globally produced enhanced P3a (4.3 μV) as compared to HA (2.2 μV, $p = .019$) or CS (2.4 μV, $p = .032$), but there was no difference between these last groups. MAD also produced significantly higher P3a for sad rare face than HA ($p = .001$) or CS ($p = .006$). Finally, the most interesting result was a group X type interaction effect ($F[4,66] = 5.756$, $p = .022$): only the MAD group produced different amplitudes in response to the different categories of trials ($F[2,22] = 7.705$, $p = .008$); HA ($F[2,22] = .247$, NS) and CS ($F[2,22] = 1.119$, NS) did not. More precisely, individuals presenting both anxious and depressed symptoms showed enhanced P3a for happy (4.6 μV,) and sad (5.4 μV) trials relative to identity trials (2.9 μV, LSD contrast: respectively $p = .048$ and $p < .001$). Indeed, this group is entirely responsible of the previously described Trial type effect, since the other groups did not display amplitude differences among trial categories. Correlation analyses showed that state anxiety was positively associated with P3a amplitude, both for happy ($r = .430$, $p = .009$) and sad trials ($r = .419$, $p = .011$) but not for identity trials ($r = .304$, *NS*). P3a amplitude for sad trials was also correlated to Beck score ($r = -.428$, $p = .009$).

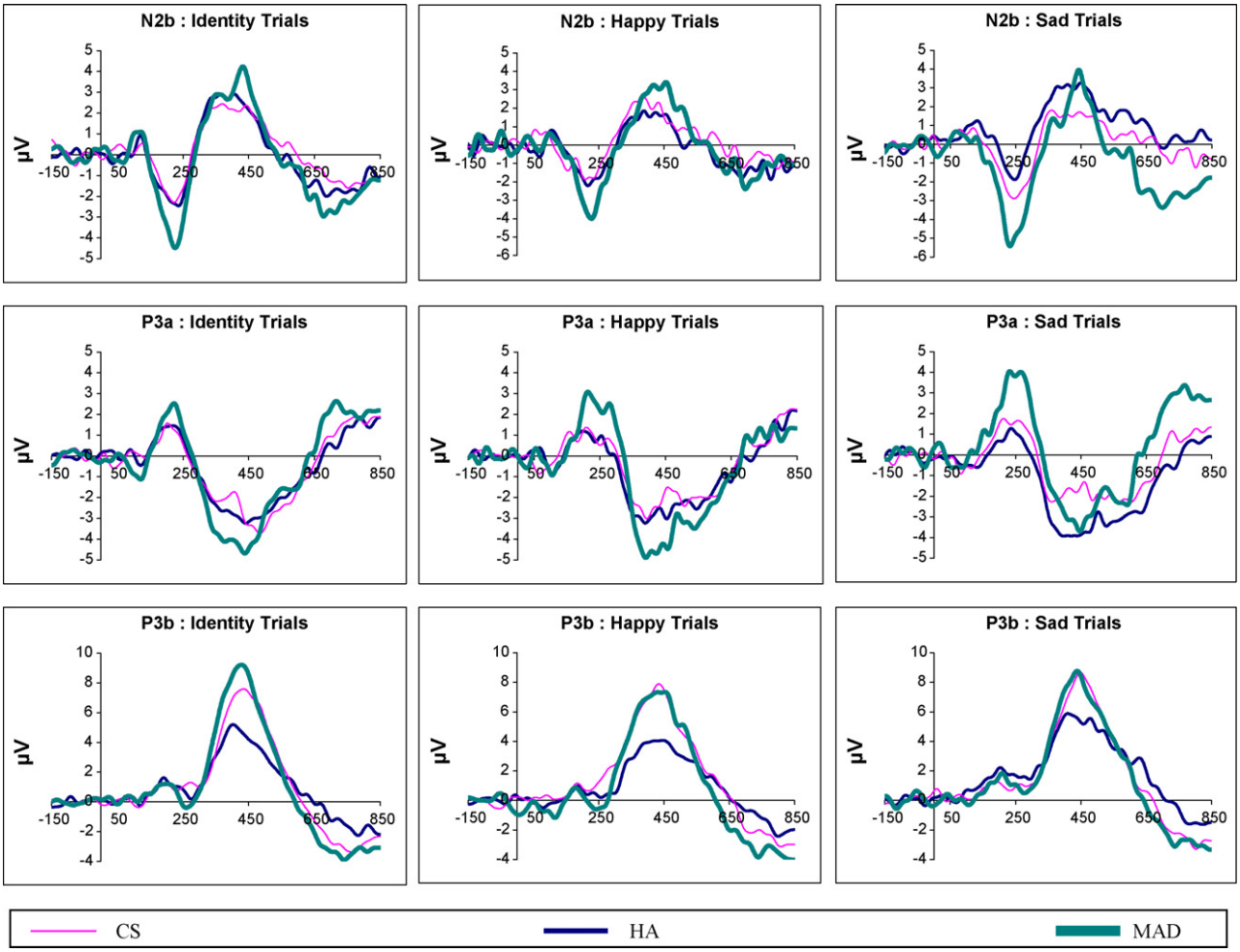

Figure 3 Subtraction of waveforms evoked by standard from those evoked by deviant stimuli in response to the two groups of emotional stimuli (happy, sad and identity trials). Negative is down.

Analysis on P3b disclosed a main effect for group on latency ($F[2,33] = 3.807$, $p = .033$) showing earlier latency in HA as compared to CS ($p = .043$). Correlations revealed a global link between earlier P3b and production of shorter reaction times for happy trials (TR-Pz: $r = .370$, $p = .026$), and sad trials (TR-Pz: $r = .425$, $p = .010$), but not for identity trials. Moreover, a within-group correlation revealed that only HA showed these associations (happiness: TR-Pz: $r = .598$, $p = .04$; sadness: TR-Pz: $r = .745$, $p = .005$; identity: TR-Pz: $r = .652$, $p = .022$). Psychological variables were correlated P3b latency: latency for sad trials was influenced by state and trait anxiety ($r = -459$, $p = .005$; $r = -.335$, $p = .046$), and trait anxiety was also correlated with P3b latency for happiness ($r = -.470$, $p = .004$)

Finally, P3b amplitude was affected by trial type ($F[2,66] = 4.362$, $p = .023$): P3b was larger for sad trials (8.6 µV) than for happy (7.6 µV, $p = .016$) or identity trials (7.7 µV, $p = .03$).

Discussion

The aims of this study were:

- to examine the specificity of the modification of emotional processing in individuals with mixed anxious-depressive tendencies by investigating the role of isolated anxious and comorbid anxious and depressed tendencies;
- to investigate the level of occurrence of biases, if any, in the information processing stream.

First, P100 and N100 were not influenced by trial type, contrary to the N170, which appears sensitive to novelty, but not to emotion in facial expressions. This observation supports results reviewed by Eimer and Holmes [34], who have interpreted the N170 as insensitive to the emotional load of processed faces.

Although early waves did not differ between groups, there were clear between-group differences in behavioral performance. Behavioral detection of deviant trials was accelerated in both isolated-anxiety and MAD state, but the mechanisms underlying this faster processing seemed different in both anxious groups. Indeed, for subjects with isolated anxious tendencies, this behavioral reaction was only indexed by faster decisional processing (P3b), supporting the notion of a faster detection of any new information in anxiety [11].

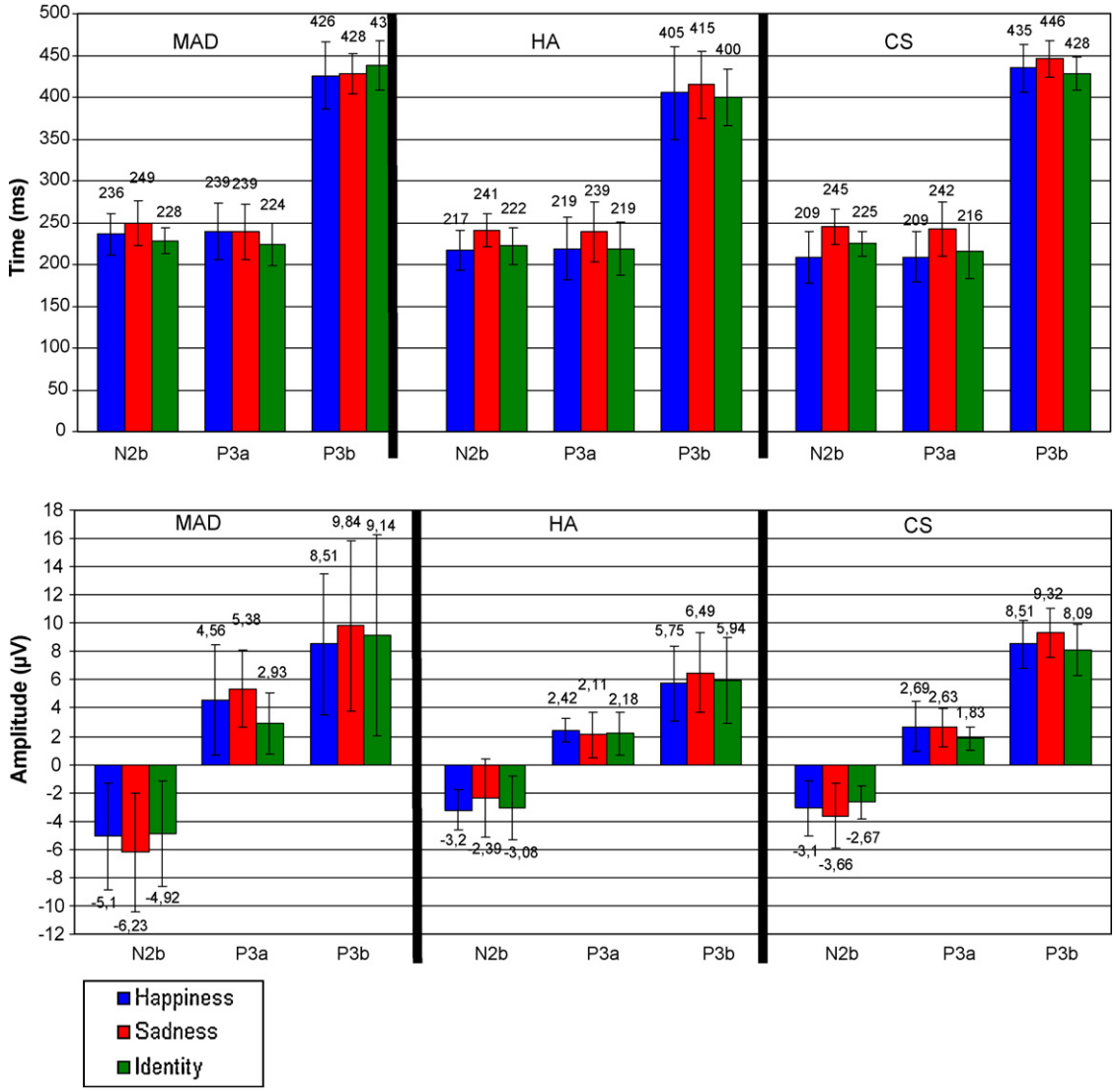

Figure 4 N2b (recorded on Oz), P3a (on Fz) and P3b (on Pz) latency (means in millisecond ± SE), and amplitude (means in microvolt ± SE) for happy (blue bar), Sad (red bar) and identity (green bar) trials (Subtraction of waveforms evoked by standard from those evoked by deviant stimuli). These figures are represented at midline electrodes for the three groups of participants.

Conversely, participants with comorbid anxious-depressive tendencies did not show modifications of the P3b wave, but rather an increased attentional processing of the deviant stimuli. Indeed, a global enhancement of N2b was associated with an increased P3a for emotional trials as compared to identity ones, especially for sad faces. Of course, identity trials were presented twice as much as sad or happy faces, but this effect of an enhanced P3a for emotion processing is only present in MAD group. This observation supports previous evidence of increased involuntary processing of emotional information in depression [25].

Consequently, it seems that the modification of facial processing in MAD originates in altered earlier attentive stages, and not in disturbed response-related processes. Studies investigating ERP components in depression often concentrated on P3b [8], and the most commonly reported result is a delay or a reduction of P3b in clinical depression [13,14]. Our results diverge from these observations, and could be attributed either to the subclinical level of depression displayed in our sample of subjects, or to comorbid anxiety. However, studies in subclinical participants found P3 modifications: For instance, Cavanagh and Geisler [15] observed reduced amplitude and delayed latencies of P3 when subclinical depressed participants had to detect happy faces, as compared to fearful faces, embedded in neutral faces. However, they did not control for comorbid anxiety, which could have enhanced the processing of threatening-fearful expressions. Conversely, since biases occurred when emotional material matched the specific vulnerability of each disorder, sadness did not elicit such more salient processing in anxiety, and the expressions used in our study could explain this discrepancy.

In spite of these observations, we defend the notion that comorbid anxiety plays a role in the differences in observed performance. As supported by different studies [17,35,36],

anxiety and depression are sustained by specific physiological characteristics that lead to different performance patterns, and comorbid anxious-depressed state results in particular biological and physiological correlates [20,21]. Therefore, we suggest that the enhanced attentional complex observable in MAD group results of the interaction between anxious and depressed states, since anxiety alone did not arouse such an effect. Our results thus support the hypothesis of a modified processing of affective stimuli in concurrent anxiety and depressed states.

To summarize, our study suggests that the visual emotional processing may be quite different in nature in anxiety and in MAD state, and we highlight two different ways to process visual emotional information in individuals with anxious versus anxious-depressive tendencies.

Other studies have been conducted using ERP in anxiety and depression. However, to our knowledge, our study is the first to explore the processing of emotional-faces stimuli in people with concurrent anxious and depressive tendencies using an emotional oddball paradigm. Although the present study provides some evidences for specific effects of mixed anxious-depressed state, it is important to recognize several limitations. First, the inclusion of a fourth group constituted by depressive participants without anxious tendencies would have been useful to address the exact contribution of anxious and depressive influence separately. However, in our investigations, anxiety was a correlate of depression, and participants showing depressive tendencies always displayed anxious behavior, whereas the reverse was not true. This problem may be related to the subclinical status of the population pool. Another limitation is that the scales used to define these tendencies did not allow for the exploration of specific features of anxiety symptoms, and high correlations between scores on the STAI and BDI scales have already been outlined in past literature [35]. Also, because the participants were only subclinical and selected according to criteria of the BDI and STAI, they were not grouped according to a clinical diagnosis, and therefore, we do not know whether one symptom, i.e. anxiety or depression, was more prominent that the other. Finally, because gender differences have been shown in emotional processes [29], and as depression is more prevalent in women [28], the present study concentrated on female participants; it would be useful to also conduct the experiment on male participants.

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