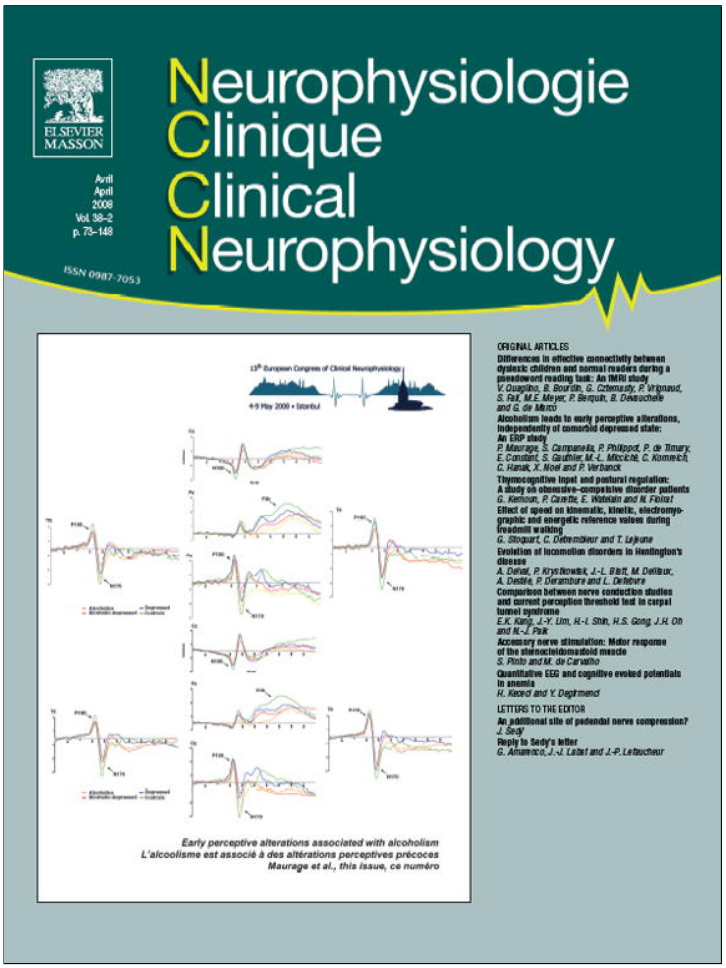

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ORIGINAL ARTICLE/ARTICLE ORIGINAL

Alcoholism leads to early perceptive alterations, independently of comorbid depressed state: An ERP study

L'alcoolisme est associé à des altérations perceptives précoces, indépendamment de la comorbidité avec un état dépressif : une étude en potentiels évoqués

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KEYWORDS
Alcoholism;
Depression;
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EFE

Summary
Introduction. — Alcoholism is associated with a deficit in the processing of emotional facial expressions (EFE) and with a delayed P3b component, partially mediated by earlier perceptive deficits (P100, N170). Since alcohol dependence often occurs with depression, we aim at investigating whether classical event-related potentials (ERP) alterations observed in alcoholism are modulated or not by depression.
Methods. — Four groups (controls; alcoholics; depressed; alcoholics-depressed) of 12 participants performed two different discrimination tasks, a gender and an emotional one. They had to decide as quickly as possible about the gender or the emotion displayed by facial stimuli during an ERP recording session (32 channels). Reaction times (RTs), P100, N100, N170 and P3b were recorded.
Results. — At the behavioural level, control participants discriminated EFE (but not gender) more rapidly than the three other groups. At the ERP level, the differences observed on RTs for emotional task were neurophysiologically indexed by a delayed P3b component. This delay was

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associated with earlier ERP alterations (P100, N100, N170), but only in participants suffering from alcohol dependence, in association or not with depression.
Discussion. — On the one hand, individuals with alcoholism, associated or not with a comorbid depression, were impaired in the processing of EFE. This deficit was neurophysiologically indexed by early perceptive (P100, N100, N170) and decisional (P3b) alterations. On the other hand, non-alcoholic patients with depression only exhibited P3b impairment. These results lead to potential implications concerning the usefulness of the ERP for the differential diagnosis in psychiatry, notably concerning the comorbidities in alcoholism.

MOTS CLÉS
Alcoolisme ;
Dépression ;
Comorbidité ;
PE ;
EFE

Résumé
Introduction. — L'alcoolisme est associé à un déficit du traitement des expressions faciales émotionnelles (EFE) et à une altération de la composante P3b, altération partiellement modulée par des déficits perceptifs antérieurs (P100, N170). Étant donné que la dépendance à l'alcool est souvent associée à des troubles dépressifs comorbides, cette étude a pour objectif d'investiguer si les altérations classiquement observées en potentiels évoqués dans l'alcoolisme sont modulées ou non par la dépression.
Matériel et méthodes. — Quatre groupes (Sujets témoins ; alcooliques ; dépressifs ; alcooliques-dépressifs) de 12 participants ont effectué deux tâches de discrimination, respectivement basées sur un jugement de genre ou d'émotion. Les sujets devaient prendre une décision aussi rapide que possible concernant le genre ou l'émotion présentés par un visage, durant une session d'enregistrement des potentiels-évoqués (32 canaux). Les temps de réaction ainsi que les composantes P100, N100, N170 et P3b ont été enregistrés.
Résultats. — Au niveau comportemental, les sujets témoins ont discriminé les EFE (mais pas le genre) plus rapidement que les trois autres groupes. Au niveau électrophysiologique, les différences observées pour les temps de réaction dans la tâche émotionnelle ont été indexées au plan neurophysiologique par une composante P3b retardée. Ce ralentissement était en outre associé à des altérations précoces des potentiels évoqués (P100, N100, N170), mais uniquement chez les sujets présentant une dépendance à l'alcool, en association ou non avec des troubles dépressifs.
Discussion. — D'une part, les sujets atteints d'alcoolisme, en lien ou non avec une dépression comorbide, étaient déficitaires pour le traitement des EFE. Ce déficit était indexé au plan neurophysiologique par des altérations perceptives (P100, N100, N170) et décisionnelles (P3b). D'autre part, les sujets dépressifs non-alcooliques présentaient uniquement une altération de la P3b. Ces résultats ont des implications potentielles pour ce qui concerne l'utilité des potentiels évoqués dans le diagnostic différentiel en psychiatrie, notamment pour ce qui est des comorbidités dans l'alcoolisme.

Introduction

Over the last decades, the recognition of emotional facial expressions (EFE) has been widely explored in psychological and neuroscience studies and it is now largely accepted that this ability is subserved by several brain regions including amygdala, insula, occipitotemporal lobes and orbitofrontal cortex [1]. EFE recognition thus consists in a diverse array of processes and strategies, which, depending on the experimental task, will recruit different neural networks that current experiments are still trying to precise [2].

However, the ramifications for clinical diagnosis and therapy are not less important than these issues of basic research. Several neuropsychiatric disorders involve alterations in the ability to perceive, recognize, express or experience emotions [3]. One of these most investigated pathologies is the alcohol dependence. Alcoholism leads to various social and interpersonal dysfunctions, notably for the decoding of EFE [4]. As the appropriate processing of EFE is an essential skill for the development and maintenance of satisfactory interpersonal relations [5], the failure of this skill among alcoholic individuals has deleterious consequences on their social integration [6,7]. The behavioural deficit for EFE processing in alcoholism (namely lower performance and longer response latencies than healthy controls) has recently been investigated by means of event-related potentials (ERP). By monitoring cerebral electrical activity during cognitive tasks with a high temporal resolution, ERP allow to identify the electrophysiological component representing the onset of a dysfunction and then to infer the corresponding impaired cognitive stages [8]. The great majority of ERP studies on alcoholism used basic visual or auditory stimuli (e.g. flashes or bursts) and focused on measuring the P3b component, a long-lasting positive potential, maximally recorded at more or less 300 ms over parietal sites, functionally related to decisional processes and closure of cognitive processing before starting the motor response [9]. Overall, alcoholic individuals have reduced amplitude and delayed latency for the P3b in comparison with non-alcoholic individuals [10].

Nevertheless, the processing of a stimulus can be roughly separated into different stages, each having electrophy-

siological correlates. Although the impairment of the P3b among alcoholics is well-established [11], it does not necessarily mean that this deficit originates at the decisional level, as a deficit at earlier stages cannot be excluded. Indeed, a recent study [12] has suggested that the P3b deficit may be associated with earlier perceptive deficits: the P100 component, a positive potential recorded around 100 ms at occipital sites and reflecting primary visual analyses [13], is delayed in latency, while the N170 component, a negativity maximally recorded around 170 ms at occipito-temporal sites and particularly sensitive to face processing [14], is delayed in latency and reduced in amplitude. As these data confirm previous findings showing delayed latency for the P100 component on alcoholism [15,16], and as that P3b deficit described in other psychiatric populations, such as in schizophrenia, is also associated with earlier P100 and N170 impairments [17—19], many authors have called for reconsidering the interpretation of P3b impairments at a fundamental and clinical level, by taking earlier ERP components into account in current studies, in order to define at which stage of the cognitive processing a deficit originates [20]. In alcoholism as well as in other psychiatric diseases, reconsidering the P3b deficit by investigating its potential association with earlier impairments is thus necessary to improve the understanding of the pathology, which in turn will help to optimize therapy.

Moreover, another important feature deserves specific attention, i.e. comorbidity. Alcohol dependence and affective disorders (particularly depression) co-occur at significantly higher rates than it could be expected by chance within the general population [21,22]. This comorbidity, which is still too often not taken into consideration among studies on alcoholism, is further elevated in samples of people seeking treatment for alcohol dependence. This suggests that the co-occurrence of affective disorders may be an important determinant of treatment seeking [23]. Tiet and Mausbach [24] reviewed both the psychosocial and medication treatments for those diagnosed with a substance-related disorder and one of the following disorders: depression, anxiety, schizophrenia, bipolar disorder, severe mental illness and non-specific mental illness. This review described that:

- existing efficacious treatments for reducing psychiatric symptoms also tend to work in dual-diagnosis patients;
- existing efficacious treatments for reducing substance use also decrease substance use in dually diagnosed patients;
- the efficacy of integrated treatment is still unclear.

This review called for conducting more and more methodologically rigorous research in this area, for studies investigating effects of medical treatments on comorbid disorders, but also for studies exploring the cognitive deficits of these populations.

Indeed, the importance of comorbidities is confirmed by a brief overview of the current literature:

- in ERP studies using simple auditory and visual stimuli [25], female alcoholics showed reduced P3b amplitude, but only when a comorbid lifetime diagnosis of depression was present. Nevertheless, Malone et al. [26] showed that ERP alterations appear to be a general characteristic of alcoholism, although the presence of other comorbid disorders results in greater reductions of P3b amplitude;
- at a behavioural level, alcoholism [6,7], but also depression [3], have been related to a deficit in EFE processing. These deficits associated with alcoholism or depression are well-known, but the potential cumulative effect of the comorbidity between alcoholism and depression on cognitive functions has only recently been investigated. Several studies [27] suggested a deleterious influence of comorbidity on the cognitive deficits in alcoholism. Nevertheless, contradictory results have also been found, showing no difference between alcoholic and alcoholic-depressed patients concerning the executive functions [28]. This question is thus still under debate and, to our knowledge, there has not been any study investigating EFE processing in alcoholism by taking into account the role of comorbid depressive disorder;
- at the ERP level, to our knowledge, there has not been any study investigating the cumulative effect of depression on alcoholism in the processing of EFE.

The present study was thus designed to reach the following aims: first, testing the specificity of the EFE deficit, on the basis of two different tasks: emotional and gender tasks. If the deficit is purely ''emotional'', it should be constrained to the emotional task. On the contrary, if patients are deficient in both tasks, the deficit should be considered as ''general to faces'' and not ''specific to emotions''. Second, testing the differential deficit between groups across emotions: the use of EFE depicting different emotions (namely anger, happiness and sadness) will allow us to test the potential behavioural and ERP group differences according to the emotional valence of the stimulus. Indeed, it has been suggested that the intensity of the EFE decoding deficit among alcoholic and/or depressed subjects could vary across the different emotions depicted in EFE [10]. Third and more importantly, taking into account the effect of comorbid depressive state in the well-known deficit of EFE processing in alcoholism, on the basis of four groups (namely controls, alcoholics, depressed and alcoholic-depressed subjects). Our main hypothesis is that the effect of comorbidity should be observable on subjects' performance, i.e., the alcoholic-depressed group should show the higher impairment. Finally, ERP were used to define, if a behavioural deficit is found, where this deficit originates from. Indeed, as suggested above, alcoholism has been related to early perceptive impairments (P100 and N170) in the processing of EFE (associated with the classical P3b alteration). Conversely, recent ERP studies on clinical depression [29], using EFE stimuli, only showed decisional impairments, as indexed by P3b modifications. Therefore, the locus (perceptive versus decisional) and/or the intensity of the deficit (in latency and amplitude) may vary according to the presence or not of comorbid depression in alcoholism.

Methods

Participants

Four groups of twelve right-handed subjects (five women per group) took part in the study:

Table 1 Characteristics of the alcoholic (A), depressed (D), alcoholic-depressed (AD) and control (C) groups: mean (S.D.).

	C ($n = 12$)	A ($n = 12$)	D ($n = 12$)	AD ($n = 12$)
Number of drinks per day	0.4 (0.36)	27.36 (6.52)	0.28 (0.15)	24.82 (13.22)
Number of days since last drink	4.7 (3.42)	17.42 (3.71)	16.5 (8.49)	20.34 (4.37)
Number of anterior treatments	NA	2.78 (1.53)	1.96 (1.08)	2.01 (1.13)
Number of previous depressive episodes	NA	NA	2.23 (1.13)	0.85 (0.98)
Mean duration of the present depressive episode (in months)	NA	NA	11.3 (4.74)	9.6 (2.34)
Mean disease duration (in months)	NA	113 (34.21)	36 (13.24)	106 (41.73) 27 (10.87)[a]

NA: not applicable.
[a] The first number concerns alcoholism, the second concerns depression.

- control group (C), composed of healthy volunteer participants who had no personal or familial history of psychiatric disorder (including depression) or drug/substance abuse (including alcoholism);
- alcoholic group (A), composed of subjects diagnosed with alcohol dependence according to DSM-IV criteria;
- depressed group (D), composed of subjects diagnosed with major unipolar depression according to DSM-IV criteria;
- alcoholic-depressed group (AD), composed of subjects diagnosed with alcohol dependence and major unipolar depression according to DSM-IV criteria. Groups characteristics are presented in Table 1.

The four groups were matched for age, gender and education. Education level was assessed according to the number of years of education completed since the beginning of primary school. All patients were recruited during the third week of their treatment (mean duration of hospitalization: 16.3 days, S.D. 2.65) in a psychiatric centre (St-Luc Hospital and Brugmann Hospital, Brussels, Belgium). Exclusion criteria for all groups included major medical problems, neurological disease (including epilepsy), visual impairment, other psychiatric diagnosis (including clinical anxiety, as assessed by an exhaustive psychiatric examination) and polysubstance abuse. Each participant had normal-to-corrected vision. Patients and control participants were assessed for psychological control measures (using validated self-completion questionnaires), namely State and Trait anxiety (State and Trait Anxiety Inventory, form A and B [30]) and depression (Beck Depression Inventory, [31]). Although controls were free of any medication, some patients were still medicated: six alcoholic and five alcoholic-depressed subjects received low doses of benzodiazepines (A [mean dose: 20.42 mg per day, S.D. 8.67], AD [mean dose: 12.54 mg per day, S.D. 10.38]), eight depressed subjects and three alcoholic-depressed subjects were taking antidepressants, namely selective serotonin reuptake inhibitors (D [mean dose: 48.34 mg per day, S.D. 26.17], AD [mean dose: 34.35 mg per day, S.D. 18.13]). Participants were provided with full details regarding the aims of the study and the procedure to be followed. After receiving this information, all participants gave their informed written consent. The study was approved by the ethical committee of the medical school.

Tasks and procedure

Control measures

The Benton Face Recognition Test [32], a discrimination and pairing task requiring subjects to select a target face from a set of faces shown: full-face, in profile or in shadow, was used to test the ability to process correctly the identity attributes of emotionally neutral faces. This test consisted in 22 items, with a maximal score of 51.

Moreover, in order to test the integrity of the motor and visual abilities, a simple reaction time task was used, in which subjects had to decide as quickly as possible if the presented stimulus was a human or animal face. Five neutral faces (selected from the standardized set of Ekman and Friesen pictures [33], two males) and five animal pictures (namely lion, frog, dog, duck and horse selected from the Internet) were chosen. This task consisted in two blocks of 80 stimuli (40 faces, 40 animals) randomly presented.

Pretest and stimuli selection

In order to make sure that the stimuli selected for the gender and emotional tasks were reliably recognizable, and to control the difficulty across tasks and conditions, a pretest phase was conducted. The visual stimuli used in the pretest, namely emotional facial expressions (EFE), were selected from the standardized set of Ekman and Friesen pictures [33] and two types of continuum were computed on the basis of these faces, using the morphing software ''Morph 5.2.1.'' (see [34,35] for technical details about the morphing procedure). For the gender task, eight neutral pictures (four males) were selected, and four female-male pairs were formed. On this basis, a continuum was created in each pair, going from the female picture to the male one. For each pair, eight pictures were then created, varying according to the proportion of female-male characteristics contained in the stimulus. Namely, the pictures were (% of female-% of male): 5-95, 20-80, 35-65, 45-55, 55-45, 65-35, 80-20, 95-5. These pictures were grouped in four dyads with growing difficulty (respectively ''5-95'' and ''95-5'', ''20-80'' and ''80-20'', ''35-65'' and ''65-35'', ''45-55'' and ''55-45'') and 32 gender stimuli (four pairs X 8 morphing levels) were thus created.

For the emotion task, four faces (two males) were selected and four pictures were used for each face: angry, happy, sad and neutral facial expression. On the basis of these pictures, three continuum were created for each face, going

Table 2 Pretest results.

(a) Gender task

	5-95[a]	20-80	35-65	45-55	55-45	65-35	80-20	95-5
Performance	93.7 (3.41)	90.9 (2.98)	74.4 (4.23)	64.7 (3.16)	52.9 (6.82)	76.1 (4.91)	87.2 (3.67)	91.7 (2.34)
RTs	582 (31.6)	599 (37.2)	630 (42.2)	664 (40.5)	661 (51.4)	630 (34.7)	607 (38.9)	594 (23.4)

(b) Emotional task

	A[b]35[c]	A65	A95	H35	H65	H95	S35	S65	S95
Performance	56.7 (6.23)	87.2 (3.21)	91.4 (5.83)	72.7 (4.52)	89.2 (3.29)	90.6 (5.42)	74.8 (6.43)	85.7 (2.56)	86.3 (3.91)
RTs	721 (44.3)	671 (39.2)	649 (45.6)	710 (51.1)	638 (42.5)	630 (35.8)	722 (45.2)	681 (47.6)	673 (39.7)

Performance (% of correct response) (S.D.) and reaction times (ms) (S.D.) for (a) each morphing level in the gender task; (b) each emotion and morphing level in the emotional task.

[a] The first number represents the percentage of female face depicted in the stimulus, the second number representing the percentage of male face.

[b] A: anger; H: happiness; S: sadness.

[c] The number represents the percentage of the concerned emotion depicted in the stimulus.

from the neutral face to one emotional face (namely anger, happiness or sadness). Three pictures were then created for each continuum, containing 35, 65 or 95% of the selected emotion (and thus 65, 35 and 5% of the neutral face, respectively). Thirty-six emotional stimuli (four faces X 3 emotions X 3 morphing levels) were thus created.

Thirty-four participants (24 females, mean age = 19.65, S.D. 1.31) took part in the pretest phase. These participants had a normal-to-corrected vision, were free of any history of psychiatric disorder or drug/substance abuse (including binge drinking habits) and their personal alcohol consumption was lower than six standard drinks per week. Two tasks were proposed: firstly, gender task: the 32 gender stimuli were presented 24 times each (12 blocks of 64 stimuli) and participants had to determine as quickly as possible which gender was mainly displayed in the stimulus; secondly, emotion task: the 36 emotional stimuli were presented 18 times each (nine blocks of 72 stimuli) and participants had to determine as quickly as possible which emotion was mainly displayed in the stimulus. In both tasks, a fixation cross was presented for 300 ms at the beginning of each trial, and then the stimulus for 800 ms. A black screen was displayed between stimuli for 300 ms. From the stimulus onset, participants had 1500 ms to answer. The results are presented in Table 2. On this basis, three dyads of gender stimuli (namely ''35-65'' — ''65-35'', ''20-80'' — ''80-20'' and ''5-95'' — ''95-5'') were selected for the gender task, as they represented the best matching (in performance and RTs) with the 35%, 65 and 95% stimuli of the emotional task, respectively. Nevertheless, it should be noted that, as expected, the gender task was easier and led to faster RTs than the emotion one. The experimental phase was thus based on 24 stimuli (four faces X 6 morphing levels) for the gender task and 36 stimuli (four faces X 3 emotions X 3 morphing levels) for the emotional task. These stimuli are illustrated in Fig.1.

Gender task

The gender task was based on the detection of the gender mainly displayed on the face. Six experimental conditions were used (''G'' for gender followed by a number representing the percentage of female face depicted in the stimulus), grouped in three dyads: G5-G95, G20-G80, G35-G65. Participants were confronted with a total of three blocks, each defined by 80 stimuli, so that the emotional task consisted of 240 stimuli (40 per condition). The order of the three blocks varied across participants.

Emotional task

The emotional task consisted in the detection of the emotion displayed in the face. Nine experimental conditions were used: three emotions (anger, happiness, sadness) X 3 morphing levels (35, 65 and 95%). Participants were confronted with a total of nine blocks, each defined by 80 stimuli, so that the emotional task consisted of 720 stimuli (80 per condition). In order to facilitate the task, only two emotions were displayed in each block, and the study contained three blocks for each pair of emotions (anger—happiness, anger—sadness, happiness—sadness). The order of the nine blocks varied across participants. At the beginning of each block, participants were told which pair of emotions would be presented in that particular block (for example, ''anger—happiness'').

During the ERP recordings, participants were sitting in a dark room on a chair placed at 50 cm from the screen with their head restrained in a chin rest. Visual stimuli subtended a visual angle of $6 \times 8^{\circ}$. At the beginning of each trial in each task (simple reaction time task, gender task, emotional task), a fixation cross was presented for 300 ms, and then the stimulus for 800 ms. A black screen was displayed between stimuli for 300 ms. From the stimulus onset, participants had 1500 ms to answer by pressing the response button corresponding to the stimulus (e.g., ''male'' button in the gender task; ''anger'' button in the emotional task) with their right forefinger. Response times and error rates were recorded. Participants were told that speed was important but not at the cost of accuracy. Only correct responses were considered for analysis of Reaction Times (RTs) and ERP.

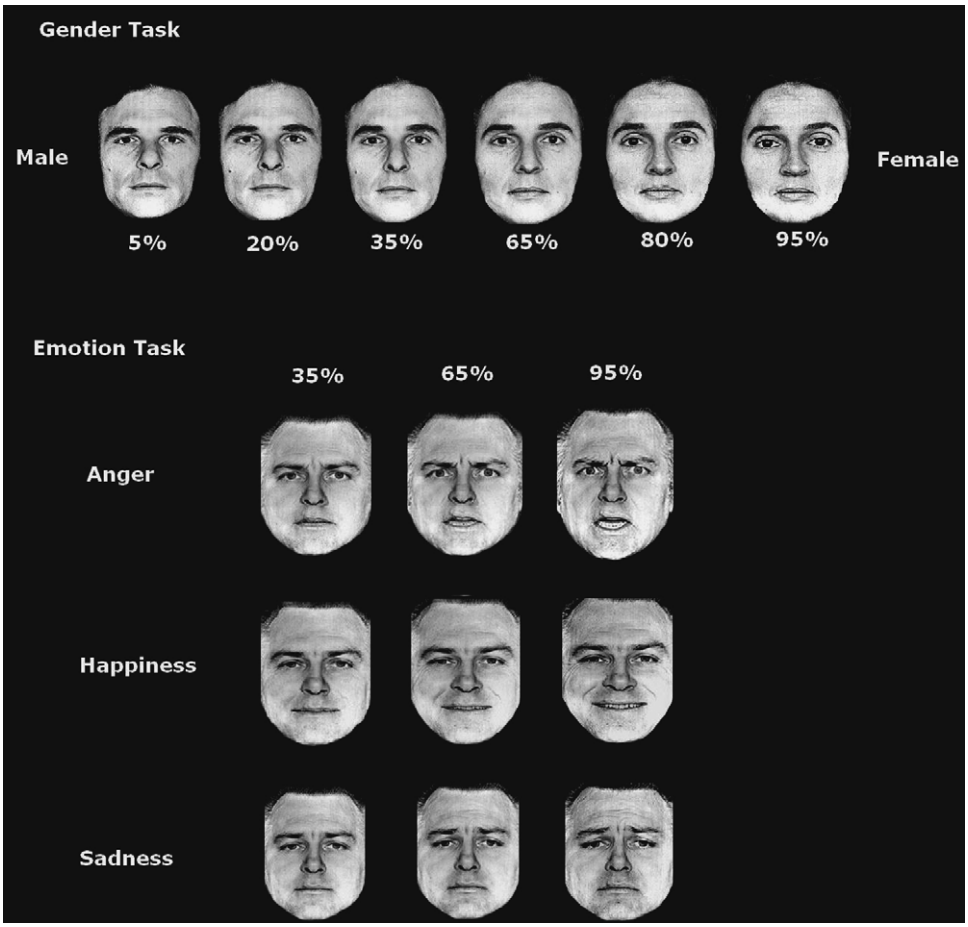

Figure 1 Illustration of the stimuli used in this study. For the gender task (above), this figure offers an example of continuum from male to female face (the number represents the percentage of female face depicted in the stimulus). For the emotion task (below), the three morphing levels (35, 65 and 95%) are depicted for each emotion used (namely anger, happiness and sadness).

EEG recording and analyses

The electroencephalogram (EEG) was recorded using 32 electrodes mounted in an electrode Quick-Cap. Electrode positions included the standard 10—20 system locations and intermediate positions. Recordings were taken with a linked mastoid physical reference but re-referenced using a common average. The EEG was amplified by battery-operated A.N.T.® amplifiers with a gain of 30000 and a band-pass of 0.01—100 Hz. The impedance of all electrodes was always kept below 10 kΩ. The EEG was recorded continuously (sampling rate 500 Hz, A.N.T. Eeprobe software) and the vertical electrooculogram (VEOG) was recorded bipolarly from electrodes placed on the supraorbital and infraorbital ridges of the left and right eyes. Trials contaminated by EOG artifacts (mean of 6%) were manually eliminated off-line. Epochs were created starting 200 ms prior to stimulus onset and lasting for 1500 ms. Data were filtered using a 30 Hz low-pass filter. In order to compute different averages of ERP target stimuli for each subject individually, three parameters were coded for each stimulus:

- the emotion-gender type (anger, happiness, sadness, gender);
- the morphing intensity (35, 65 and 95% for the emotion; 65, 80 and 95% for gender);
- the response type (correct or incorrect).

For each participant and each component of interest (namely P100, N100, N170 and P3b), individual peak amplitudes and maximum peak latencies were obtained from several electrodes separately for the ERPs evoked in response to stimuli: Oz, O1, O2, T5 and T6 for P100 and N170; C3, Cz and C4 for N100; P3, Pz and P4 for P3b. These values were tested (with the statistical software SPSS 13.0), using repeated measures of analysis of variance (ANOVA — a Greenhouse-Geisser correction was applied when appropriate), paired sample t-tests and Scheffe post-hoc tests. The results section will only present the statistically significant results. Moreover, as the location results (i.e., the results associated with the amplitude and latency differences between electrodes) were not the central focus of this study and as no significant interaction effect was found for location, these results will not be reported here.

Table 3 Results of the alcoholic (A), depressed (D), alcoholic-depressed (AD) and control (C) groups concerning the control and psychological measures: mean (S.D.).

	C ($n = 12$)	A ($n = 12$)	D ($n = 12$)	AD ($n = 12$)
Age [NS]	41.75 (9.12)	41.83 (6.65)	41.67 (8.37)	41.67 (6.48)
EL[a] [NS]	12.92 (2.10)	12.67 (2.38)	12.58 (2.27)	12.83 (1.99)
BDI[b] [*]	3.67 (4.35)	6.17 (3.27)	21.33 (4.14)	20.58 (4.73)
Stai[c] A [NS]	48.12 (12.58)	49.00 (7.06)	57.75 (10.31)	52.67 (10.36)
Stai[c] B [*]	46.25 (9.21)	51.75 (9.42)	62.33 (10.51)	62.42 (11.17)

NS: not significant; *: $P < 0.001$.
[a] EL: education level.
[b] BDI: Beck Depression Inventory [31].
[c] STAI: State and Trait Anxiety Inventory [30].

Results

Control measures

Psychological measures

As shown in Table 3, all groups were similar in terms of age ($F[3,44] = 0.01$, N.S.), gender, and education ($F[3,44] = 0.05$, N.S.). As expected, differences between groups were observed for depression ($F[3,44] = 60.23$, $P < 0.001$) as shown by Scheffe post-hoc tests: the D and AD groups did not differ ($P = 0.97$) but led to higher BDI scores than C (AD-C: $P < 0.001$; D-C: $P < 0.001$) and A ones (AD-A: $P < 0.001$; D-A: $P < 0.001$), which did not differ ($P = 0.54$).

Concerning anxiety, groups did not differ on state anxiety ($F[3,44] = 2.21$, N.S.), but the results found for trait anxiety ($F[3,44] = 7.59$, $P < 0.001$) were similar than those found for depression as shown by Scheffe post-hoc tests: the D and AD groups did not differ ($P = 0.99$) but led to higher STAI-B scores than C (AD-C: $P < 0.01$; D-C: $P < 0.01$) and A ones (AD-A: $P < 0.05$; D-A: $P < 0.05$), which did not differ ($P = 0.62$).

Benton test and simple reaction times

The results are presented in Table 4 (part a). Groups did not differ for the Benton test ($F[3,44] = 0.41$, N.S.) and for the simple reaction times task, neither on RTs ($F[3,44] = 1,28$, N.S.) nor on performance ($F[3,44] = 1.01$, N.S.).

Gender task

It should be noted that, as there were no differences inside each pair of gender stimuli, neither for the Reaction Times (G5-G95: t[47] = 0.03, N.S.; G20-G80: t[47] = 1.67, N.S.; G35-G65: t[47] = 0.26, N.S.) nor for Performance (G5-G95: t[47] = 1.7, N.S.; G20-G80: t[47] = 1.27, N.S.; G35-G65: t[47] = 0.54, N.S.), it has been decided to collapse (for every statistical analysis) the two conditions of each pair into one single condition, namely G65 (grouping G35 and G65 results), G80 (grouping G20 and G80 results) and G95 (grouping G5 and G95 results).

Behavioural data

Performance. A 4 x 3 ANOVA with group as between-factor and morphing as within-factor was computed. The results are shown in Table 4 (part b). A main effect of morphing ($F[2,88] = 125.7$, $P < 0.001$, $\eta^2 = 0.74$) was found — as expected, higher performance was found for ''95%'' morphing level as compared to ''80%'' (t[47] = 4.76, $P < 0.001$) and ''65%'' (t[47] = 12.73, $P < 0.001$) ones, and for ''80%'' as compared to ''65%'' (t[47] = 11.16, $P < 0.001$).

Reaction Times. A 4 x 3 ANOVA with group as between-factor and morphing as within-factor was computed. The results are shown in Table 4 (part b). The only significant effect was a main effect of Morphing ($F[2,88] = 41.43$, $P < 0.001$, $\eta^2 = 0.48$) — as expected, faster RTs were found for ''95%'' morphing level as compared to ''80%'' (t[47] = 2.71, $P < 0.01$) and ''65%'' (t[47] = 8.04, $P < 0.001$) ones, and for ''80%'' as compared to ''65%'' (t[47] = 6.06, $P < 0.001$).

ERP data

For each component of interest, 4 X 3 X 5 (3) ANOVAs were computed separately for latencies and amplitudes, with group (C, A, D, AD) as between-factor, morphing (65, 80 and 95%) and location (Oz, O1, 02, T5, T6 for P100-N170; C3, Cz, C4 for N100; P3, Pz, P4 for P3b) as within-factors. These results are illustrated in Fig. 2.

P100.

- Latencies: a main effect of group ($F[3,44] = 4.02$, $P < 0.05$, $\eta^2 = 0.21$) was found — Scheffe post-hoc tests showed that C and D subjects had significantly shorter P100 latencies than A (C-A: $P = 0.037$; D-A: $P = 0.024$) and AD subjects (C-AD: $P = 0.020$; D-AD: $P = 0.014$), which did not differ.
- Amplitudes: no significant main effect nor interaction were found.

N100.

- Latencies: the only significant effect concerned group ($F[3,44] = 3.35$, $P < 0.05$, $\eta^2 = 0.18$) — Scheffe post-hoc tests showed that AD subjects had significantly longer N100 latencies than C ($P = 0.016$) and D ($P = 0.045$) subjects, but not than A ones.
- Amplitudes: no significant main effect nor interaction were found.

N170.

- Latencies: the only significant effect concerned group ($F[3,44] = 3.05$, $P < 0.05$, $\eta^2 = 0.17$) — Scheffe post-hoc tests showed that C subjects had shorter N170 latencies

Table 4 Behavioral results of the alcoholic (A), depressed (D), alcoholic-depressed (AD) and control (C) groups.

(a) Simple reaction time task and Benton test

Group	Reaction Times	Performance	Benton test
C ($n = 12$)	572 (83.1)	97.7 (1.82)	41.50 (2.93)
A ($n = 12$)	607 (54.2)	97.1 (2.06)	41.58 (3.47)
D ($n = 12$)	651 (97.6)	95.2 (5.78)	41.25 (3.16)
AD ($n = 12$)	628 (79.7)	96.3 (3.75)	41.17 (3.90)

(b) Gender task

Group	Performance			RTs		
	G[a]65[b]	G80	G95	G65	G80	G95
C ($n = 12$)	82.4 (8.1)	91.2 (7.8)	94.2 (8.8)	667 (68.4)	636 (73.9)	622 (77.6)
A ($n = 12$)	85 (7.5)	93.1 (5.1)	96.2 (3.5)	746 (80.1)	705 (65.7)	694 (71.2)
D ($n = 12$)	80.9 (12.6)	90.4 (12.3)	90.5 (14.4)	755 (119.1)	727 (132.1)	710 (118.2)
AD ($n = 12$)	78.3 (6.5)	88.3 (7.2)	93.3 (6.1)	769 (104.7)	736 (100.1)	725 (96.5)

(c) Emotional task

(c1) Performance

Group	A[c]35[b]	A65	A95	H35	H65	H95	S35	S65	S95
C ($n = 12$)	58.7 (10.1)	88.3 (10.9)	94.5 (6.5)	75.8 (12.2)	91.5 (9.5)	92.5 (7.1)	81.5 (9.4)	90.2 (6.3)	91.1 (5.4)
A ($n = 12$)	48.5 (14.1)	84.1 (7.9)	91 (7.7)	77.3 (11.1)	89.1 (7.6)	89.1 (10.9)	73.9 (12.7)	86.5 (11.2)	85.4 (11.5)
D ($n = 12$)	54.3 (8.6)	84.1 (10.53)	88.1 (11.2)	65.9 (14.6)	85.2 (18.5)	88.1 (17.3)	76.5 (12.6)	84.3 (10.6)	85.5 (9.5)
AD ($n = 12$)	51.2 (12.5)	77.9 (17.1)	85.5 (16.7)	72 (18.6)	82.1 (15.1)	82.9 (14.8)	67.1 (16.3)	75.4 (17.8)	77.3 (18.5)

(c2) RTs

Group	A[c]35[b]	A65	A95	H35	H65	H95	S35	S65	S95
C ($n = 12$)	746 (97.9)	701 (89.9)	691 (104.8)	746 (89.2)	675 (82.5)	660 (93.9)	738 (87.8)	723 (87.4)	716 (89.9)
A ($n = 12$)	853 (112.3)	796 (95.3)	764 (79.6)	848 (110.1)	785 (92.1)	760 (86.6)	838 (102.7)	802 (103.7)	798 (104.8)
D ($n = 12$)	827 (122.3)	797 (117.9)	774 (120.3)	846 (134.4)	786 (128.6)	767 (122.1)	842 (129.6)	811 (115.1)	823 (113.1)
AD ($n = 12$)	866 (104)	825 (102.4)	809 (114.8)	875 (106.5)	804 (105.6)	780 (107.1)	890 (120.1)	855 (114.6)	844 (113.2)

(a): simple reaction time task (RTs and performance) and Benton test (mean score) (S.D.); (b): gender task: performance (% of correct response) (S.D.) and Reaction Times (ms) (S.D.); (c): emotional task; (c1): performance (% of correct response) (S.D.); (c2): Reaction Times (ms) (S.D.).

[a] G: gender.

[b] The number represents the morphing level.

[c] A: anger; H: happiness; S: sadness.

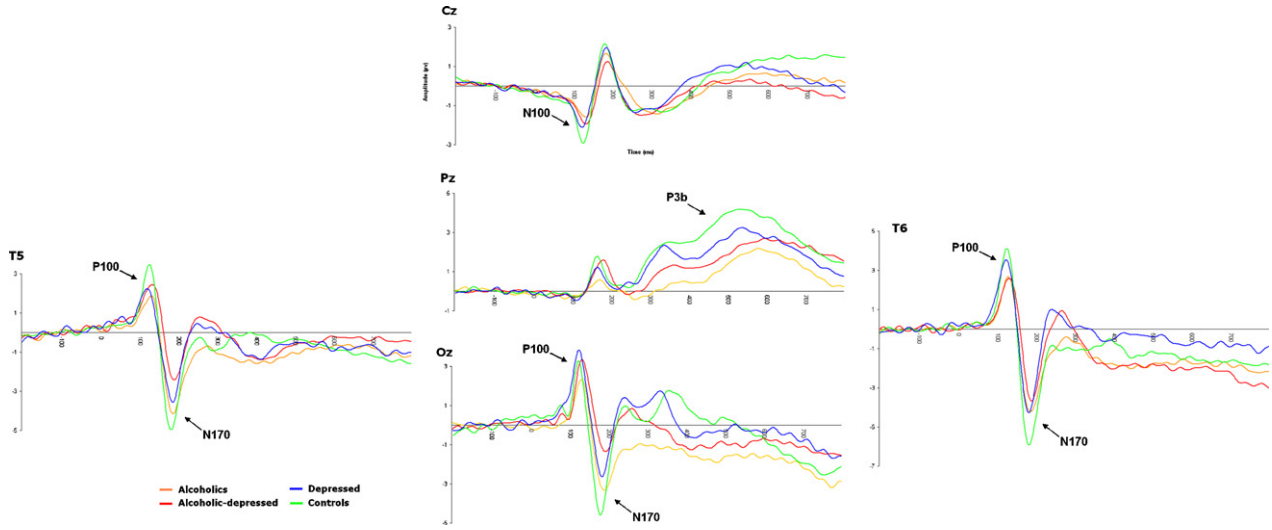

Figure 2 Electroencephalographic results for the gender task on Oz, T5, T6 (P100 and N170 component), Cz (N100 component) and Pz (P3b component) and for each group: alcoholics (in orange), controls (in green), depressed (in blue) and alcoholic-depressed subjects (in red).

than A ($P = 0.034$) and AD ($P = 0.031$) subjects, but not than D ones.
- Amplitudes: no significant main effect nor interaction were found.

P3b.

- Latencies: no significant main effect nor interaction were found.
- Amplitudes: the only significant effect concerned group ($F[3,44] = 2.93$, $P < 0.05$, $\eta^2 = 0.17$) — Scheffe post-hoc tests showed that C subjects had significantly higher P3b amplitudes than A ($P = 0.034$), D ($P = 0.027$) and AD ($P = 0.022$) subjects, which did not differ.

Overall. At the behavioural level, the four groups showed identical performance and RTs. The only common effect is a ''morphing effect'', suggesting that the more the distance from a female (male) prototype is high, the more the response is difficult: this ''distance effect'' is classically observed in the literature [36].

At the ERP level, we observed that C and D groups did not differ on the P100 and N100 components, both showing shorter latencies than A and AD groups. For the P3b component, please note the congruence between RTs and ERP latencies data, as the absence of behavioural difference is neurophysiologically indexed by the absence of P3b latency effect. Nevertheless, this congruence was not found for amplitudes, as A, AD and D groups all showed decreased P3b amplitude as compared to the C group. Thus, while patients have a preserved P3b latency (which is in line with behavioural results), the amplitude is globally reduced in every clinical group. This reduced amplitude was expected as it has been repeatedly shown to index a globally reduced cerebral activity among alcoholics and depressed patients [10].

Emotional task

Behavioural data

Performance. A 4 X 3 X 3 ANOVA with group as between-factor, emotion and morphing as within-factors was computed. The results are shown in Table 4 (part c1). Two main effects were found as follow:

- emotion ($F[2,88] = 14.87$, $P < 0.001$, $\eta^2 = 0.25$) — subjects had lower performance for anger than for sadness ($t[47] = 5.41$, $P < 0.001$) and happiness ($t[47] = 4.87$, $P < 0.001$) stimuli;
- morphing ($F[2,88] = 564.28$, $P < 0.001$, $\eta^2 = 0.92$) — as expected, a higher performance was found for ''95%'' morphing level in comparison with ''65%'' ($t[47] = 6.3$, $P < 0.001$) and ''35%'' ($t[47] = 25.99$, $P < 0.001$) ones, and for ''65%'' as compared to ''35%'' ($t[47] = 24.01$, $P < 0.001$).

Finally, a significant interaction effect was found between emotion and morphing ($F[4,176] = 55.97$, $P < 0.001$), explained by the fact that a higher performance for ''95%'' morphing level in comparison with ''65%'' ones was found for anger ($t[47] = 8.07$, $P < 0.001$) but not in the happiness ($t[47] = 1.52$, N.S.) and sadness conditions ($t[47] = 0.88$, N.S.).

Reaction Times. A 4 x 3 X 3 ANOVA with group as between-factor, emotion and morphing as within-factors and was computed. The results are shown in Table 4 (part c2). Three main effects were found as follow:

- group ($F[3,44] = 3.16$, $P < 0.05$, $\eta^2 = 0.18$) — Scheffe post-hoc tests showed that C subjects had shorter RTs than A ($P = 0.041$), D ($P = 0.037$) and AD subjects ($P = 0.011$), which did not differ;
- emotion ($F[2,88] = 9.32$, $P < 0.001$, $\eta^2 = 0.17$) — there were longer RTs for sadness than for anger ($t[47] = 3.24$, $P < 0.01$) and happiness ($t[47] = 3.82$, $P < 0.001$) stimuli;

Table 5 Electrophysiological results in the emotional task.

Components								
	P100		N100		N170		P3b	
Groups	Lat	Amp	Lat	Amp	Lat	Amp	Lat	Amp
C ($n = 12$)	128 (10.7)	4.54 (2.07)	126 (6.93)	−1.91 (1.04)	182 (7.05)	−6.10 (3.62)	466 (60.1)	4.03 (2.16)
A ($n = 12$)	138 (8.76)	3.23 (1.63)	133 (11.16)	−1.46 (0.45)	194 (11.55)	−5.14 (2.11)	535 (76.2)	2.10 (1.519)
D ($n = 12$)	129 (14.88)	4.83 (3.21)	129 (12.36)	−2.06 (1.02)	183 (15.26)	−3.94 (3.07)	543 (39.4)	2.01 (1.88)
AD ($n = 12$)	138 (10.62)	4.09 (2.29)	141 (13.66)	−1.80 (1.23)	194 (16.31)	−3.87 (3.08)	520 (62.6)	1.56 (2.85)

Mean latencies (ms (S.D.)) and amplitudes (μv (S.D.)) across experimental conditions and locations for P100, N100, N170 and P3b components, among alcoholic (A), depressed (D), alcoholic-depressed (AD) and control (C) groups.

- morphing ($F[2,88] = 124.64$, $P < 0.001$, $\eta^2 = 0.73$) — as expected, faster RTs were found for ''95%'' morphing level in comparison with ''65%'' ($t[47] = 4.91$, $P < 0.001$) and ''35%'' ($t[47] = 12.21$, $P < 0.001$) ones, and for ''65%'' in comparison with ''35%'' ($t[47] = 12.23$, $P < 0.001$).

Finally, a significant interaction effect was found between emotion and morphing ($F[4,176] = 14.34$, $P < 0.001$), explained by the fact that faster RTs for ''95%'' morphing level in comparison with ''65%'' ones were found for anger ($t[47] = 3.47$, $P < 0.01$) and happiness ($t[47] = 6.47$, $P < 0.001$) but not in the sadness condition ($t[47] = 0.48$, N.S.).

ERP data

For each component of interest, 4 X 3 X 3 X 5(3) ANOVAs were computed separately for latencies and amplitudes, with group (C, A, D, AD) as between-factor, emotion (anger, happiness, sadness), morphing (35%, 65%, 95%) and location (Oz, O1, 02, T5, T6 for P100-N170; C3, Cz, C4 for N100; P3, Pz, P4 for P3b) as within-factors. These electrophysiological results are described in Table 5 and illustrated in Fig. 3.

P100.

- Latencies: two main effects were found:
  - group ($F[3,44] = 2.86$, $P < 0.05$, $\eta^2 = 0.16$) — Scheffe post-hoc tests showed that C and D subjects had shorter P100 latencies than A (C-A: $P = 0.041$; D-A: $P = 0.048$) and AD subjects (C-AD: $P = 0.047$; D-AD: $P = 0.049$), which did not differ;
  - morphing ($F[2,88] = 4.8$, $P < 0.05$, $\eta^2 = 0.11$) — P100 latencies were significantly longer for ''65%'' than for ''35%'' ($t[47] = 2.84$, $P < 0.01$) and ''95%'' ($t[47] = 2.46$, $P < 0.05$) stimuli.
- Amplitudes: no significant main effect nor interaction were found.

N100.

- Latencies: the only significant effect concerned group ($F[3,44] = 3.77$, $P < 0.05$, $\eta^2 = 0.21$) — Scheffe post-hoc tests showed that AD subjects had longer N100 latencies

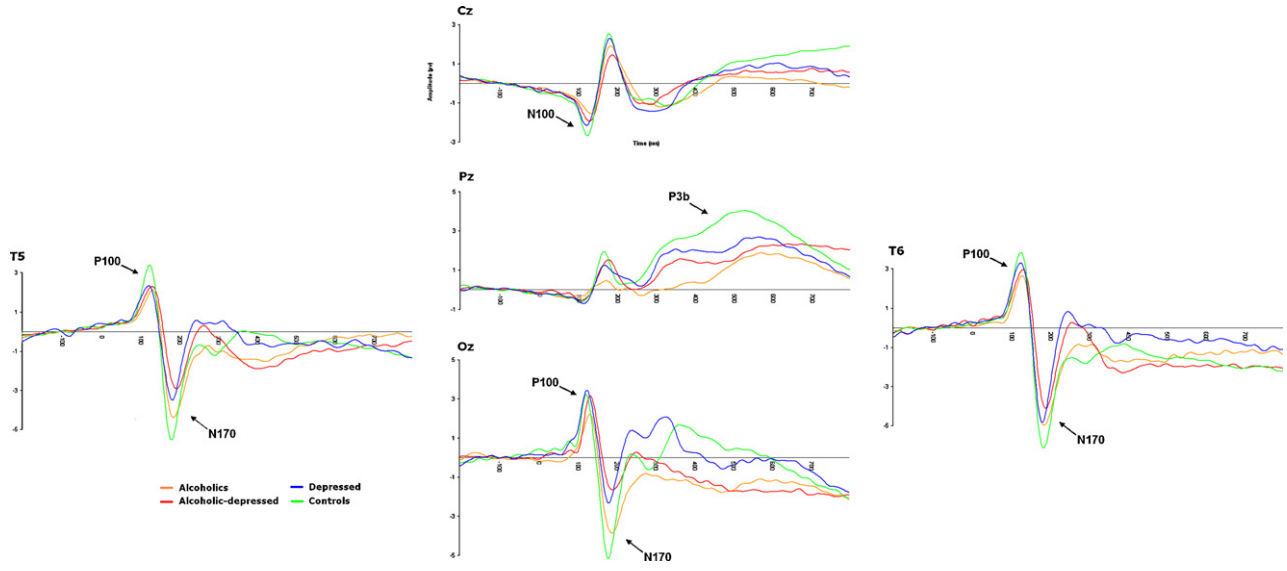

Figure 3 Electroencephalographic results for the emotion task on Oz, T5, T6 (P100 and N170 component), Cz (N100 component) and Pz (P3b component) and for each group: alcoholics (in orange), controls (in green), depressed (in blue) and alcoholic-depressed subjects (in red).

Table 6 Pearson's correlations (for the 48 subjects) between (a) psychological measures and mean behavioral-electrophysiological data for the emotional task and (b) behavioral and electrophysiological results for the emotional task.

(a)

Results

Psychol. Measures	RTs	Perf	P100 Amp	N100 Amp	N170 Amp	P3b Amp	P100 Lat	N100 Lat	N170 Lat	P3b Lat
BDI[a]	**0.300 (.038)**	**−0.319 (.027)**	0.023 (.876)	−0.108 (.463)	0.224 (.126)	**−0.311 (.031)**	−0.137 (.352)	0.088 (.552)	−0.020 (.892)	**0.328 (.023)**
STAI A[b]	−0.041 (.780)	0.136 (.358)	0.104 (.480)	−0.166 (.258)	−0.039 (.795)	0.100 (.499)	−0.064 (.666)	−0.018 (.906)	0.092 (.533)	0.125 (.396)
STAI B[b]	0.196 (.183)	−0.100 (.501)	0.036 (.807)	0.055 (.712)	0.168 (.253)	−0.114 (.438)	0.067 (.650)	0.127 (.389)	0.170 (.249)	0.051 (.729)

(b)

ERP Results

Behav. Results	P100 Amp	N100 Amp	N170 Amp	P3b Amp	P100 Lat	N100 Lat	N170 Lat	P3b Lat
Mean Performance	0.086 (.559)	−0.108 (.464)	−0.162 (.271)	**0.301 (.038)**	**−0.399 (.005)**	**−0.464 (.001)**	**−0.412 (.004)**	**−0.328 (.023)**
Mean RT	−0.222 (.129)	−0.061 (.681)	**0.381 (.008)**	**−0.329 (.023)**	**0.451 (.001)**	**0.447 (.001)**	**0.487 (.0001)**	**0.476 (.001)**

ρ value (*P-value*); significant results are indicated in bold text.

[a] BDI: Beck Depression Inventory [31].

[b] STAI: State and Trait Anxiety Inventory [30].

than C ($P = 0.008$) and D ($P = 0.017$), but not than A ones.
- Amplitudes: no significant main effect nor interaction were found.

N170.

- Latencies: the only significant effect concerned group ($F[3,44] = 2.9$, $P < 0.05$, $\eta^2 = 0.16$) — Scheffe post-hoc tests showed that C subjects had shorter N170 latencies than A ($P = 0.041$) and AD subjects ($P = 0.039$), but not than D ones.
- Amplitudes: The only significant effect concerned morphing ($F[2,88] = 5.86$, $P < 0.01$, $\eta^2 = 0.12$): the N170 amplitude was higher for ''95%'' stimuli than for ''65%'' ($t[47] = 3.74$, $P < 0.01$) and ''35%'' ($t[47] = 2.24$, $P < 0.05$) ones.

P3b.

- Latencies: a main effect of group ($F[3,44] = 3.81$, $P < 0.05$, $\eta^2 = 0.21$) was found — Scheffe post-hoc tests showed that C had shorter P3b latencies than A ($P = 0.011$), D ($P = 0.009$) and AD subjects ($P = 0.043$), which did not differ. Moreover, an interaction was found between group and emotion ($F[6,88] = 2.54$, $P < 0.05$, $\eta^2 = 0.14$) — indeed, for anger and happiness stimuli, C had shorter P3b latencies than A (anger: $P = 0.0011$; happiness: $P = 0.031$), D (anger: $P = 0.008$; happiness: $P = 0.01$) and AD subjects (anger: $P = 0.011$; happiness: $P = 0.034$), while for sadness stimuli, the only significant difference was between C and D subjects ($P = 0.014$).
- Amplitudes: a main effect of group ($F[3,44] = 3.08$, $P < 0.05$,, $\eta^2 = 0.17$) was found — Scheffe post-hoc tests showed that C subjects had higher P3b amplitudes than A ($P = 0.037$), D ($P = 0.031$) and AD subjects ($P = 0.011$), which did not differ. Moreover, an interaction was found between group and emotion ($F[6,88] = 2.38$, $P < 0.05$, $\eta^2 = 0.14$) — for sadness stimuli, C had higher P3b amplitudes than A ($P = 0.023$), D ($P = 0.045$) and AD subjects ($P = 0.016$), while for anger and happiness stimuli, controls had higher P3b amplitudes than D (anger: $P = 0.023$; happiness: $P = 0.048$) and AD subjects (anger: $P = 0.0029$; happiness: $P = 0.045$), but not than A subjects.

Overall. At the behavioural level, as already observed in the literature, sadness seems to imply longer RTs (indexed by delayed P3b) than anger and happiness stimuli [37]. Moreover, the ''distance effect'' described above for the gender task is also observable. But the most compelling effect of this task consists in the fact that C group had shorter response latencies than D, A and AD groups. This is congruent with neurophysiological results, showing that C group presents a P3b of increased amplitude and shorter latency than A, D and AD groups. Interestingly, the difference between C, A, and AD groups is neurophysiologically indexed as soon as the P100 component, while C and D groups only differed on the P3b component.

Complementary analyses

Finally, complementary analyses were computed to:

Table 7 P-values of the post-hoc tests comparing the RTs observed in the control group with those of each patient group for the three thirds of the emotional task and for the whole task.

Comparison with controls				
Patient group	Third 1	Third 2	Third 3	Whole task
Alcoholics ($n = 12$)	.047	.027	.048	.038
Depressed ($n = 12$)	.035	.029	.044	.032
Alcoholic-depressed ($n = 12$)	.006	.007	.007	.006

For each part of the emotional task and each patient group, the difference with the control group is significant.

- test the potential participants' gender effect as well as the medication effect (in the A, D and AD groups): these variables were included as covariables in our ANOVA statistical analyses. We did not find any significant influence of the gender or medication doses on the behavioural or electrophysiological results ($P > 0.05$ for every test), neither any significant correlation between the medication level and any behavioural or electrophysiological result ($P > 0.05$ for every correlation);
- test the correlations between the psychological measures and the behavioural-electrophysiological data for the emotional task. The results, presented in Table 6 (part a), showed no influence of the State and Trait Anxiety on any behavioural and electrophysiological result. Moreover the BDI scores were significantly correlated with impaired behavioural (longer RTs, lower performance) and P3b (delayed latency and reduced amplitude) results, thus confirming ERP results showing a late influence (namely at the P3b level) of the depression level on electrophysiological data;
- test the correlations between the behavioural and electrophysiological data for the emotional task. The results are presented in Table 6 (part b), and confirm that a higher performance and shorter RTs are associated with (a) shorter latencies for each ERP component, and (b) higher amplitude of the P3b component.

Concerning the RTs, patients are, as shown above, specifically impaired for the emotion task. This result, and the observation that the emotion task led to globally higher RTs than the gender one ($t[47] = 11.29$, $P < 0.001$), could be linked to the fact that the emotional task had three times more trials than the gender one. Indeed, the deficit observed among psychiatric groups could be due to a general fatigue effect (linked to the length of the emotional task) or to a statistical bias (the higher number of trials leading to more significant differences between groups) rather than to an emotional deficit per se. To test this hypothesis, we separated the emotional task in three third, and performed for each a 4 X 3 ANOVA on the RTs with group as between-factor and morphing as within-factor. As shown in Table 7, the three third of the emotional task led to results similar to those of the whole task, namely a main group effect ($F[3,44] > 2.91$, $P < 0.05$), suggesting that the specific emotional deficit among patients is not linked to the length difference between tasks, but rather to the emotional factor.

Discussion

The present study was designed to answer four main questions:

- is the deficit specific to EFE or general to tasks implicating the processing of human faces?
- Is there a differential deficit across groups according to the type of emotion depicted in the EFE?
- Has the comorbid depressive disorder an effect on the well-known EFE deficit described for alcoholism?
- Where does this EFE deficit originate from on the cognitive stream, as indexed by ERP?

First, concerning the specificity of the deficit, the most compelling result is the absence of behavioural differences between groups in the simple reaction time task, in the Benton test, and more importantly in the gender task. This result suggests that each group performs identically when confronted to a ''face categorization gender task''. On the opposite, in the emotional task, while the four groups displayed equal performance, controls had shorter response latencies and thus performed the task more easily than patients of A, D and AD groups. These behavioural results lead to the hypothesis that the deficit observed among patients is specific for the emotional processing. ERP results confirmed the specificity of the emotional deficit, in the gender task, the absence of behavioural differences is in perfect agreement with the decisional P3b component [9], which did not show any difference in latency between the four groups; In the emotional task, C group displayed shorter P3b latencies in comparison with A, D and AD groups. Thus, behavioural and ERP results corroborated to underline the emotional specificity of the EFE deficit.

Second, with regard to the differential deficit according to emotion type, our results clearly indicate that the different EFE used in this study (namely anger, happiness and sadness) are not leading to similar results across groups.

On the one hand, at a behavioural level, two main results were found concerning emotion type: firstly, anger stimuli led in each group to lower performance than happiness and sadness stimuli. Moreover, 65% morphing level led to lower performance than 95% level for anger, but not for the other EFE. Anger is thus more difficult to detect than happiness and sadness, particularly for lower intensity levels. This is in line with the pretest phase and confirms earlier studies [38,39] showing that anger is more difficult to detect than other emotions, even for control subjects, when the stimuli

are ambiguous (as it is the case for lower morphing levels). Secondly, sadness stimuli led to longer RTs than anger and happiness ones. This clearly confirms earlier results among control [37,40] as well as psychiatric populations [12], showing that sadness leads to slower recognition than the other emotions, probably due to physical properties (for example, sadness expression implies less facial muscles contractions than anger and happiness, and is thus physically closer to the neutral expression).

On the other hand, at the ERP level, our results showed group differences for the P3b component according to the emotion depicted in the stimulus. Indeed, it was first shown that A and AD groups had delayed latencies for anger and happiness, but not for sadness. This preserved sadness decoding in alcoholism, associated with an impaired processing of anger and happiness, had already been suggested in earlier studies [4]. Moreover, non-alcoholic depressive subjects had a delayed P3b latency for anger, happiness but also sadness stimuli. This delayed processing of sadness confirms earlier results showing an important difficulty for sadness decoding in depression [41]. Interestingly, it has been suggested that the delayed processing of negative emotions (here anger and sadness) could participate in the persistence of depression [42], which underlines the clinical implications of this result. Concerning P3b amplitudes, the results globally confirmed the reduced amplitude among alcoholics and/or depressed subjects as compared to controls, but it was also shown that this reduced amplitude is present for each emotion among D and AD subjects, but only for sadness in the A group. The P3b amplitude has been repeatedly found as reduced in alcoholism and depression [10,11], but this result is to our knowledge the first to show a variation of the amplitude deficit according to the emotion type. Indeed, while it has to be confirmed in future studies, this result indicates that P3b amplitude deficit could be present for every emotion in depression, but only for some emotions (here sadness) in alcoholism. More generally, it shows the importance of controlling depression level during the exploration of EFE decoding in alcoholism, as some impairments (for example here the P3b amplitude deficit for anger and happiness stimuli) could be due to comorbid depression rather than to alcoholism per se (as it is present in AD but not in A group).

In conclusion, and while it was not the main focus of this study, the differences observed between groups according to the emotion depicted in the stimulus underline the importance for future studies to take into account the potential variation of the EFE decoding deficit in psychiatric populations according to the emotion type.

Moreover, ERP results also help answering the two last questions. In the emotional task as well as in the gender task, patients groups displayed some ERP modulations: On the one hand, C and D groups showed P100, N100 and N170 of earlier latencies than A and AD groups; On the other hand, C group presented enhanced P3b amplitude in comparison with A, D and AD groups. These results suggest two main considerations. Firstly, concerning the influence of depression on alcoholics' results, it appears that, independently of depressive disorder, alcoholism subtends early perceptive alterations (P100, N170). Thus, comorbid depression did not seem to increase the behavioural and ERP deficit observed in alcoholism. It should nevertheless be noted that a delayed N100 latency was observed in the alcoholic-depressed group, but not in the alcoholic group. While this study clearly showed no global influence of comorbid depression in alcoholism, this result for the N100, which has to be confirmed, could constitute an index of the influence of comorbid depression in alcoholism. Secondly, concerning the origin of the deficit, depression only influenced the ''late'' P3b amplitude, while alcoholism delayed earlier perceptive components (P100, N100, N170). It thus seems that, while alcoholic and depressed subjects did not differ concerning behavioural results, electrophysiological data clearly show that the deficit appears earlier in alcoholism than in depression on the cognitive stream.

Finally, one important question is: why did similar ERP differences (latency P100, N100, N170; amplitude P3b) imply behavioural differences in the emotional task but not in the gender task (where no P3b latency modulation was observed)? In the gender task, ERP modulations existed but did not lead to observable behavioural differences. We suggest that these modifications are neurophysiological markers of alcohol and/or depression disorder, but that the face gender categorization task is too easy to produce observable behavioural deficits. Indeed, behavioural data clearly show that the emotional task is harder than the gender task (global higher RTs). One possibility is that this effect of complexity is due to methodological aspects. We do not think that this is the case. Indeed, the pretest ensures us that participants were confronted in the two tasks with morphed faces of comparable complexity (see Table 2). Moreover, if the total number of trials is different in the two tasks, we showed that the three parts of the emotional task (each one presenting an identical number of trials than the gender one) display similar results (see Table 7), thus excluding any fatigue effect among patients in the emotional task. Another possibility is that the emotional task is more difficult per se. For example, a recent study of Foisy et al. [43] supports this idea. Indeed, participants performed two computer tasks on faces, one requiring to answer as rapidly as possible questions regarding non-emotional features of the face (gender, age range and cultural identity); the second one involving a different set of photographs implicating EFE decoding. Alcoholic and control participants showed similar results in both tasks in terms of response accuracy. Yet, in the emotional facial expression task, alcoholic participants were slower than controls to answer emotional questions on EFE, while no differences appeared on RTs in the control task.

In view of these results, this study investigating the role of alcoholism and comorbid depression on EFE processing, by comparing four groups on gender and emotional tasks, could be the first stage of a more ambitious evaluation of comorbidity influence in alcoholism. Indeed, we choose to study the effect of comorbid depressive disorder, as alcohol dependence and affective disorders frequently co-occur [21,22]. However, it clearly appears that other comorbid symptoms (such as antisocial personality disorder, schizophrenia, drug abuse...) also deserve specific attention. Moreover, it is difficult in clinical reality to have alcoholic patients with only depressive tendencies (and for example no comorbid anxiety). While no influence of anxiety level was found in this study, it would be interesting to test groups of ''alcoholics depressive but not anxious'' and ''alcoholics anxious but not depressive'' patients, in order to test fur-

ther the differential influence of depression and anxiety. It should also be noted that, even if our statistical results seem reliable (as shown by the significant p-values and effect sizes observed), samples are relatively small ($n = 12$; $n = 48$): our data should therefore be considered as preliminary and need to be replicated.

In conclusion, this paper mainly showed that:

- the behavioural deficit appears specific to EFE, as no differences were observable among participants in:
  - the simple RT task (no basic sensori-motor deficit)
  - the Benton task (suggesting that participants are all able to discriminate face identity)
  - the gender task, contrary to the emotional one;
- the EFE decoding deficit varies across groups according to the emotion depicted in the face. It has mainly been suggested that depression leads, in comparison with alcoholism, to a delayed P3b latency for sadness and to a lower P3b amplitude for happiness and anger. This first exploration of the differential EFE decoding deficit across psychiatric populations underlines the importance for future studies to use a wide range of EFE, in order to explore this differential deficit further;
- with the exception of a marginal comorbidity influence on N100 latency, this deficit for EFE processing did not significantly differ among alcoholic patients according to the presence of a comorbid depressive disorder. This result is in line with earlier studies [28] showing no influence of depressive comorbidity among alcoholic subjects;
- On the basis of ERP results, we suggest that the alcohol disorder (A and AD groups) is sufficient to induce early perceptive alterations, while the depressive disorder only induces late ''decisional'' modulation (on P3b)[1]. These data confirmed recent results obtained by Maurage et al. [12], showing that P3b deficit in alcoholism could be subtended by earlier perceptive deficits. Moreover, these data allowed us to show for the first time that, on the one hand, depression alone only affects decisional stages of EFE processing (in agreement with earlier ERP studies on EFE, [29]), and, on the other hand, that alcoholism is sufficient to induce early perceptive deficits in the information processing stream, independently of concurrent depressive disorder.

Importantly, as this differential deficit between alcoholism and depression was undetectable on the basis of behavioural measures (both groups being equally impaired concerning the RTs), these results underline the usefulness of the ERP for the differential diagnosis among psychiatric populations. Indeed, ERP could offer an interesting complement, besides the classical clinical diagnosis tools, to bring major data concerning the electrophysiological impairments among psychiatric subjects, and thus to add specific information undetectable on the basis of behavioural examination. For example, a reduced latency in early waves might help to confirm the existence of a drinking problem in a person presenting depressive symptoms. Future studies, exploring the respective influence of the various comorbidities frequently observed in alcoholism, could thus help to delimit the precise part that ERP could play in the differential psychiatric diagnosis.

[1] Please note that correlation analyses suggest that this effect of depressive disorder on P3b seems to be restricted to depressive symptoms, and not to anxious ones, as differences on ERP components were correlated to Beck scores, but not STAI ones.

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