Personality and the effects of acute alcohol intake. A contingent negative variation study in healthy subjects

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Summary
This study investigates the relationship between blood alcohol concentrations (BACs) and contingent negative variation (CNV). Fourteen healthy subjects were divided on the basis of their personality profiles – the Minnesota Multiphasic Personality Inventory (Hs+Hy+D/3) – into a high score (HS) and low score (LS) subgroup. The CNV was recorded using a choice-reaction time (RT) task. CNV recording was performed in two conditions: inter-stimulus intervals (ISIs) of 1500ms and 2500ms at three different BACs (0.3, 0.5 and 0.8 g/L) after acute alcohol administration. At the high BAC (0.8 g/L), both subgroups showed a reduced CNV amplitude area and a longer RT (p<.05) in both ISI conditions. No effects either on the CNV or on the RT were observed at the low BAC (0.3 g/L). At the intermediate BAC (0.5 g/L), the HS subgroup displayed an increased CNV amplitude (p<.05), not accompanied by a significantly longer RT (short ISI), and a reduced late CNV (p<.05) with a longer RT (p<.05) (long ISI). In the LS group, only a longer RT was observed in the long ISI condition.

CNV modifications point to an individual, apparently personality-related, threshold of sensitivity to different alcohol levels.

KEY WORDS: blood alcohol concentration, contingent negative variation, personality.

Introduction
Contingent negative variation (CNV) (1) is a slow, negative, cerebral potential evoked in subjects whenever there is a strict temporal relationship between two stimuli (“warning” and “imperative”). Over the years, several studies (2,3) have suggested that when the inter-stimulus interval (ISI) is longer, the CNV waveform consists of at least two distinct components: an early wave, thought to be related to the orienting response to the warning stimulus (labelled early CNV or “E” wave), and a late component (late CNV or “L” wave), which (like the Bereitschaftspotential) is related to the cerebral activity preceding voluntary movement (4). Numerous studies have shown not only that this bioelectric phenomenon is dependent on mental state changes, but also that it is linked to various endogenous factors, such as expectancy, attention, preparation, motivation and readiness (1), and sensitive to levels of vigilance, anxiety and stress (5). Furthermore, CNV appears to be modified in some mental illnesses (6) and its waveform may vary according to exogenous factors such as psychoactive drugs, e.g., benzodiazepines (7), delta-9-tetrahydrocannabinol (8) and cocaine (9).

Alcohol is without doubt the world’s most commonly used psychoactive drug. Findings on the effects of alcohol on CNV, both in chronic alcoholics (10) and after acute alcohol intake, have been contrasting. Some authors (11) have found a CNV amplitude decrease in association with ethanol, while others (12) have reported that CNV amplitude is not affected by alcohol. Relatively few data exist on modifications in CNV following acute alcohol consumption. However, one drawback of all these studies is that none has involved accurate measurement of blood alcohol concentrations (BACs).

A crucial role in the relationship between psychoactive drug use and mental functioning is played by a subject’s personality characteristics (8). Münte et al. (13) observed that emotional lability can strongly influence the nature and time course of a drug’s effects (diazepam, caffeine) on CNV. Moreover, CNV has been shown to be a sensitive measure of the complex interaction between personality traits and drug-induced conditions (14). In view of these considerations, this study was conducted to investigate whether acute ethanol intake, in social drinkers may, according to a subject’s personality profile, modify the associative functions underlying CNV.

Materials and methods
Population
Fourteen healthy volunteers (8 men and 6 women), all right-handed, aged between 24 and 37 years (mean
age 30.89 ± SD 4.2), who were neither teetotallers nor alcoholics, were recruited from amongst the medical staff in our department. No subject had a history of drug abuse, while all were moderate social drinkers (averaging 75.7 g ethanol/week). None of the participants were smokers and none had a family history of alcoholism or other drug abuse. Only subjects without serious physical or medical impairment and not on psychotropic medication were enrolled in this study. Informed consent to the experiment was obtained from each participant.

Psychometric evaluation

The Minnesota Multiphasic Personality Inventory (MMPI) (Form R) was administered to all the subjects prior to the CNV recording session. The subjects were all normal from a psychopathological point of view, as shown by their T scores, which ranged from 30 to 70 in all the clinical scales. In order to analyse the role that psychometric variables might play in the relationship between alcohol level and CNV, the mean of the scores obtained in the three neurotic scales of the MMPI (Hypochondriasis-Hs, Depression-D and Hysteria-Hy) was first calculated for each subject. This value was obtained using the following formula: (Hs+D+Hy)/3. In accordance with other studies in the literature, a new variable, expressing the "neurotic" MMPI profile of each subject, was thus arrived at (15).

The median value (T score = 53.5) was taken as the basis on which to split the experimental group into two subgroups: seven subjects formed the group with low neurotic MMPI profile scores (LS), whilst another seven formed the group with high neurotic MMPI profile scores (HS).

Procedure

The subjects were instructed to eat a light, low-fat breakfast on the morning of the experiment and not to consume alcohol during the 24 hours immediately prior to it. On arriving at the laboratory, each subject was briefed about the nature of the experiment and the testing procedures involved. The experimental design consisted of four electrophysiological recordings (CNV), starting three hours after breakfast: in the first recording session, none of the participants had a detectable BAC; subsequently, CNV was recorded at definite BACs (0.3 - 0.5 - 0.8 g/L), which were obtained by administering three alcohol doses in succession. The beverage drunk was a mixture of orange juice and pure ethanol (20% alcohol by volume). The volume of beverage required to achieve the aforementioned BACs was determined by each subject's body weight, according to the following formula: a dose of 1.7 cc/kg was administered to achieve a BAC of 0.3 g/L; a further 1.1 cc/kg was given to reach a BAC of 0.5 g/L; finally, another 1.7 cc/kg was required to achieve a BAC of 0.8 g/L. BAC readings were taken immediately before (15 minutes post-beverage) and after each recording via breath analysis with a Siemens Alcomat (M52052-A). CNV recordings were performed in each subject, as soon as the aforementioned BACs were reached.

CNV recording

Subjects sat in a comfortable chair in a faradized room with attenuated sound and dimmed lights. CNV was recorded by means of the classical two-stimuli paradigm (a choice-reaction time task) in which the "warning" stimulus (S1) is followed by the "imperative" stimulus (S2): the S1 was a light flash (100 ms, 1.5 Joule), while the S2 consisted of either a 250 or a 2000 Hz tone, which lasted 400 ms and had a rise/decay time of 0.5 ms. The S2 was presented binaurally via earphones at an intensity of 70 dB. A pushbutton device was placed in the right hand of the subject, who was instructed to press the button immediately upon hearing the 2000 Hz sound (correct execution of the task stopped the sound signal). Delivery of the target stimulus, which had an overall probability of occurrence of 35%, was randomised. The EEG was recorded through Ag/AgCl electrodes fixed with collodion to the scalp. Electrodes were placed at Fpz, Fz, Cz, C3 and C4 according to the 10-20 International System. Linked mastoids were used as reference, while a ground electrode was placed on the forehead. The electro-oculogram (EOG) was recorded using two electrodes, placed directly above and below the right eye. Electrode impedance was kept constant at below 3 kOhm. EEG was amplified with Physio-Amp Marazza preamplifiers: event-related potentials were filtered using a bandpass of 0.005-30 Hz, while the EOG bandpass used was 0.02-30 Hz. Data acquisition was performed by means of a PC-based system interfacing an A/D board (Analog Device 812). The sample rate was 250 Hz per channel. On-line analysis, data processing and data management were performed by means of a software package specifically developed for the application.

Trials with eye movements (including blinks) were automatically excluded from storage through the on-line analysis of EOG-recorded electrical activity. A further selection was performed by the operator through an offline analysis of the activity at Fpz in order to eliminate trials contaminated by low-voltage artifacts not detected by the automatic rejection procedure.

The recording epochs lasted 4 seconds. The baseline (mean value of EEG voltage) referred to the 750 ms prior to S1. Two different ISIs of 1500 and 2500 ms were used. Two successive CNV recordings were performed at the same BAC: the first with a short ISI (1500 ms), the second with a longer ISI (2500 ms), after verification that the BAC had not changed.

The duration of the inter-trial interval varied randomly between 6 and 10 seconds. For each recording, a minimum of 20 consecutive artifact-free trials per subject were selected and analysed.

CNV data

All CNV parameters were measured in relation to the baseline. CNV amplitude was measured as the total area (negative shift between S1 and S2) in the 1500 and 2500 ISI conditions. Moreover, only in the long ISI (2500 ms) condition was it possible to measure two temporal "windows": the early CNV, corresponding to the 200 ms between 500 and 700 ms post-S1, and the late CNV, corresponding to the 200 ms pre-S2 (16). The reaction
time (RT) was measured upon delivery of the imperative stimulus (S2).

**Statistical analysis**

Due to the emergence of somatic complaints, electrophysiological data recording could not be completed in one LS group subject. In spite of this, the original division into subgroups, to which the 14 participants were submitted on the basis of their psychometric T scores, was maintained for the statistical analysis, even though the analysis of the electrophysiological data involved only 13 of the subjects.

Given that there were three conditions to explore (the 3 different BACs vs the no alcohol condition) in each subgroup and that the subject number was relatively low, a Friedman ANOVA was used as a non-parametric alternative to one-way repeated measures ANOVA. Post-hoc analysis was performed using the Wilcoxon matched pairs test. A significance level of p<.05 for a two-tailed hypothesis was selected.

**Results**

1. **MMPI**

As the MMPI profiles of the LS and HS subgroups (Fig. 1) clearly show, the L,F, and K scores are almost identical in the two subgroups. There was no difference either in the male to female ratio (4/3 in both LS and HS subgroups).

2. **RT**

In the 1500 ms ISI condition, both subgroups showed some significant RT changes (LS: Friedman ANOVA Chi-square=11.4, df=3, p<.01; HS: Friedman ANOVA Chi-square =13.7, df=3, p<.005). At the BAC of 0.8 g/L, slower RTs were observed in both the LS (Wilcoxon T=0, p<.05) and the HS subgroups (Wilcoxon T=0, p<.05) when compared with those observed in the basal condition. At BACs of 0.3 g/L and 0.5 g/L, RTs did not significantly differ from the basal condition in either subgroup (Table I).

Both subgroups revealed significant RT modifications in the 2500 ms ISI condition (LS: Friedman ANOVA Chi-square=15.0, df=3, p<.005; HS: Friedman ANOVA Chi-square =17.9, df=3, p<.0005). At the BAC of 0.8 g/L, slower RTs were observed in both the LS (Wilcoxon T=0, p<.05) and the HS subgroups (Wilcoxon T=0, p<.05). Similarly, at the intermediate BAC (0.5 g/L), slower RTs were observed in both the LS (Wilcoxon T=0, p<.05) and the HS subgroups (Wilcoxon T=0, p<.05). By contrast, at the BAC of 0.3 g/L, RTs did not differ significantly from those observed in the basal condition in either experimental subgroup (Table II).

3. **Electrophysiological data**

CNV was clearly detectable in all the sites explored. As no asymmetric distribution was found and the highest

![Figure 1 - Mean MMPI scale scores in the experimental sample highlighting the differences in the personality profiles of the low and high score subgroups.](image-url)
Table I - CNV recording with 1500 ms ISIs.

<table>
<thead>
<tr>
<th>BLOOD ALCOHOL CONCENTRATION (g/L)</th>
<th>SUBJECTS</th>
<th>0.0</th>
<th>0.3</th>
<th>0.5</th>
<th>0.8</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNV Areas (± SD)</td>
<td>LOW SCORE</td>
<td>15821.5 (4943.21)</td>
<td>14320.7 (5884.07)</td>
<td>17698.5 (8716.16)</td>
<td>10873.3 (3612.15)</td>
</tr>
<tr>
<td>Reaction Times (± SD)</td>
<td>LOW SCORE</td>
<td>240.3 (10.84)</td>
<td>241.3 (10.78)</td>
<td>245 (9.44)</td>
<td>281.3 (12.82)</td>
</tr>
<tr>
<td>CNV Areas (± SD)</td>
<td>HIGH SCORE</td>
<td>10281.9 (2684.78)</td>
<td>11462.4 (2553.04)</td>
<td>18927.9 (8848.56)</td>
<td>6308.86 (2877.66)</td>
</tr>
<tr>
<td>Reaction Times (± SD)</td>
<td>HIGH SCORE</td>
<td>243.7 (6.77)</td>
<td>248 (9.66)</td>
<td>252 (12.80)</td>
<td>297.7 (15.34)</td>
</tr>
</tbody>
</table>

Mean integral CNV amplitudes and reaction times for both high (HS) and low score (LS) subjects at the different BACs in the 1500 ms ISI condition. Each CNV area and RT values were expressed as ms·µV and ms, respectively. Values in brackets indicate standard deviation. * Statistical significance compared with basal level 0.0 g/L.

Table II - CNV recording with 12500 ms ISIs.

<table>
<thead>
<tr>
<th>BLOOD ALCOHOL CONCENTRATION (g/L)</th>
<th>SUBJECTS</th>
<th>0.0</th>
<th>0.3</th>
<th>0.5</th>
<th>0.8</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNV Areas (± SD)</td>
<td>LOW SCORE</td>
<td>29918.3 (8708.34)</td>
<td>30748.8 (13881.2)</td>
<td>32546.2 (15062.5)</td>
<td>19863.3 (5043.53)</td>
</tr>
<tr>
<td>Early CNV (± SD)</td>
<td>LOW SCORE</td>
<td>2309.63 (908.264)</td>
<td>2379 (1727.7)</td>
<td>6870.83 (6296.3)</td>
<td>2387.83 (882.159)</td>
</tr>
<tr>
<td>Late CNV (± SD)</td>
<td>LOW SCORE</td>
<td>2883.83 (817.625)</td>
<td>2588 (1196.13)</td>
<td>1965.67 (699.626)</td>
<td>1885.17 (489.481)</td>
</tr>
<tr>
<td>Reaction Times (± SD)</td>
<td>LOW SCORE</td>
<td>232 (5.06)</td>
<td>235 (4.69)</td>
<td>244.83 (17.62)</td>
<td>300.3 (16.17)</td>
</tr>
<tr>
<td>CNV Areas (± SD)</td>
<td>HIGH SCORE</td>
<td>21021.6 (4194.12)</td>
<td>18917 (6297.16)</td>
<td>27297.1 (8677.47)</td>
<td>11788.3 (7020.98)</td>
</tr>
<tr>
<td>Early CNV (± SD)</td>
<td>HIGH SCORE</td>
<td>2066.14 (1234.35)</td>
<td>1958.43 (886.733)</td>
<td>3040.57 (3577.62)</td>
<td>1008.43 (405.751)</td>
</tr>
<tr>
<td>Late CNV (± SD)</td>
<td>HIGH SCORE</td>
<td>3824.14 (1441.38)</td>
<td>2386.43 (1333.01)</td>
<td>2078.43 (1040.63)</td>
<td>1835.43 (790.531)</td>
</tr>
<tr>
<td>Reaction Times (± SD)</td>
<td>HIGH SCORE</td>
<td>236.86 (7.01)</td>
<td>245.43 (9.78)</td>
<td>264 (12.81)</td>
<td>310 (11.01)</td>
</tr>
</tbody>
</table>

Mean integral CNV amplitudes and reaction times for both high (HS) and low score (LS) subjects at the different BACs in the 2500 ms ISI condition. Each CNV area and RT values were expressed as ms·mV and ms, respectively. Values in brackets indicate standard deviation. * Statistical significance compared with basal level 0.0 g/L.
amplitude was observed at Cz, all the data reported below refer to the Cz site.

3.1 CNV with 1500 ms ISI

In the LS subgroup, only very slight CNV amplitude differences were observed between the different BACs (Friedman ANOVA Chi-square=7.4, df=3, p=.06), while, a more marked, although still non significant, CNV amplitude decrease was apparent at the BAC of 0.8 g/L compared with the basal condition. By contrast, significant CNV changes at the different alcohol levels were observed in the HS subgroup (Friedman ANOVA Chi-square=15.34, df=3, p=.001). The post-hoc analysis revealed a significant increase in the CNV amplitude at the BAC of 0.5 g/L (Wilcoxon T=1, p<.05). This subgroup also revealed a significant decrease in the CNV amplitude at the BAC of 0.8 g/L (Wilcoxon T=0, p<.05) (Table I, Fig. 2).

3.2 CNV with 2500 ms ISI

In the LS subgroup (Friedman ANOVA Chi-square=7.6, df=3, p=.055), no significant changes were observed when the different BACs were compared with the basal condition, the only change of note being a slight CNV amplitude decrease observed at the BAC of 0.8 g/L. By contrast, a significant difference was found in the HS subgroup (Friedman ANOVA Chi-square=12.77, df=3, p=.005): the subjects in this group revealed a significant reduction in the CNV area at the BAC of 0.8 g/L (Wilcoxon T=1, p<.05).

No significant differences in early CNV component levels were found either in the LS (Friedman ANOVA Chi-square=2.6, df=3, p>.05) or the HS subgroup (Friedman ANOVA Chi-square =.086, df=3, p>.05). Both the LS (Friedman ANOVA Chi-square=6.8, df=3, p=.08) and the HS subgroups (Friedman ANOVA Chi-square=6.94, df=3, p=.07) showed some significant variations in the late CNV component, which was significantly lower in the LS subgroup at the 0.8 BAC (Wilcoxon T=0, p<.05), as well as in the HS subgroup at both the 0.5 (Wilcoxon T=0, p<.05) and the BAC of 0.8 g/L. (Wilcoxon T= -1, p<.05) (Table II, Fig. 2).

Discussion

This study aimed to highlight possible modifications in the psychophysiological (CNV) profile of healthy subjects following acute alcohol intake. Two subgroups of subjects, a low score (LS) and a high score (HS) group, were formed on the basis of their neurotic profiles. The first observation concerns the basal (alcohol-free) condition, in which the HS subjects showed a lower CNV amplitude than the LS subjects, and in which RTs were unimpaired in both groups, in both the short and long ISI conditions (1500 and 2500 ms, respectively). These electrophysiological findings may reflect some differences in the psychophysiological profile of the two subgroups. The reduced CNV amplitude in the HS group might be due to a basal state of anxiety in these subjects that reduces the attention paid to the execution of motor tasks, which they probably perceive as stressful. CNV amplitude is, in fact, known to be closely related to attention and motivational levels and to decrease as a result of distraction (17). Proulx and Picton (18) suggested that anxiety reduces the CNV amplitude because it is a distracting factor which reduces the amount of attention paid to the experimental task. The same authors (19) had previously suggested that “normal high-anxiety subjects” have difficulty regulating their anticipation of events and that they are forced to search for appropriate response strategies.

The presence of a lower CNV amplitude in our HS subgroup thus seems to add weight to Proulx and Picton’s hypothesis, i.e., that the experimental condition may be considered as a source of anxiety and thus of distraction which is reflected in the cerebral activity, i.e. CNV. The lack of any marked modification in the CNV amplitude and RT values at the lower BAC in either of the ISI conditions suggests that the physiological effects on the central nervous system of a blood alcohol concentration of 0.3 g/L are negligible. The most interesting effects of alcohol were found, in both subgroups, at the intermediate BAC (0.5 g/L) and, in particular, in the short ISI condition. In this condition, only the HS subgroup showed a statistically significant CNV amplitude increase, with no RT variation emerging in either subgroup.

The reason for this trend is not clear. It is known that blood alcohol concentrations up to 0.6 g/L have a sedative, anxiolytic effect, accompanied by slight disinhibition and euphoria (20). From a biochemical point of view, blood ethanol concentrations of 0.5–0.6 g/L are known to increase dopaminergic synthesis through selective nucleus accumbens stimulation, which results in an anxiolytic effect, a feeling of greater relaxation and...
comfort, slight euphoria and locomotor activity stimulation (21). It is also known that both a reduced anxiety level (5) and dopamine increase (22) induce a CNV amplitude increase. Moreover, these biochemical and behavioural hypotheses are supported by clinical trials which suggest that the dopaminergic activity of the nucleus accumbens under stress is decreased more in HS than in LS subjects (25). Therefore, we may hypothesise that ethanol at a BAC of 0.5g/L, acting on the greater dopaminergic sensitivity shown by more anxious individuals, enhances the CNV amplitude more in HS subjects than in LS subjects.

As mentioned in the methods section, the use of a longer ISI (2500ms) permits the observation of two CNV subcomponents which are not detectable at shorter ISIs. At the 2500 ms ISI, there was no change in the first component, i.e., the early CNV, which has been interpreted as an orientation process (24), at the 0.5 g/L BAC in either group, whereas we observed a clear amplitude decrease, which reached statistical significance in the HS subgroup, in the second component, i.e., the late CNV, which reflects motor anticipation (25). This decrease in late CNV suggests that a BAC of 0.5 g/L renders subjects incapable of sustaining attention in the presence of ISIs longer than 1500 ms. This hypothesis is strengthened by the observation that both subgroups showed impaired RTs only in the 2500 ISI condition. It therefore appears that a BAC of 0.5 g/L does not reduce the ability to anticipate stimuli only when these are presented at short ISIs (1500 ms). In other words, a delayed RT points to an inability to generate correct S2 anticipation. The finding of a more pronounced late CNV reduction in the HS subgroup is probably due to a more marked effect of alcohol in subjects with anxious personality traits. In fact, HS subjects are less able to anticipate and control stimuli perceived as stressful (18), and consequently display a greater reduction in CNV amplitude during distraction. CNV modifications at the maximum BAC (0.8g/L) can be interpreted more easily. Both subgroups showed an evident reduction in CNV amplitude, associated with a further RT impairment, in both experimental conditions, even though this reduction was, once again, more marked in the HS subgroup. High doses of ethanol are, in fact, well known to perturb information processing in non alcohol-dependent men, and to result in poor performances (26). Moreover, high alcohol concentrations have a sedative effect on vigilance, inhibiting excitatory neurotransmitters receptors and inducing depression of neuronal activity through GABA (27).

Therefore, the global effects of alcohol are clearly capable of inhibiting cortical activity, and thus of bringing about a decrease in CNV.

In conclusion, our data suggest that not only high blood alcohol levels but also lower ones (i.e., BAC of 0.5 g/L) can impair cognitive and psychophysiological functions, above all in healthy subjects who, while they may only be social drinkers, give high anxiety scores in personality trait assessments.

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