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Event-Related Brain Potentials to High Incentive Stimuli in Abstinent Alcoholics[1]

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PORJESZ, B., H. BEGLEITER, B. BIHARI AND B. KISSIN. *Event-related brain potentials to high incentive stimuli in abstinent alcoholics.* ALCOHOL 4(4) 283–287, 1987.—We have previously found that the P3 component of the event-related potential (ERP) is reduced in alcoholics in visual target-selection paradigms. P3 voltage depends on the "subjective significance" of the stimulus (e.g., task relevance, probability of occurrence, motivational factors). We were interested in assessing P3s in alcoholics to motivationally significant stimuli that did not differ with respect to other aspects of significance. Equiprobable, task-relevant visual stimuli with different acquired incentive values were presented to alcoholics under baseline and two incentive conditions. Alcoholics manifested similar lower P3 voltages without P3 latency delays to all stimuli, regardless of incentive values. Latency Corrected Averages indicated that these results were not due to latency jitter in the averages. These results suggest multiple system deficits in alcoholics, perhaps in involving frontal and/or medial temporal lobe, the brain sources implicated in the generation of P3. Our results perhaps reflect a deficit in motivational-cognitive systems in alcoholics, possibly affecting their ability to actively sustain information processing.

Event-related brain potentials Alcoholism Electrophysiology P3 Incentive

WE have previously found that the P3 component of the event-related potential (ERP) is reduced in alcoholics in visual target-selection paradigms [2, 10–13]. P3 voltage depends on the "subjective significance" of the stimulus (e.g., task relevance [17], probability of occurrence [18], motivational factors [3]). In our previous paradigms, subjective significance was determined by task relevance and probability of stimulus occurrence.

In the present experimental design we assessed P3s in alcoholics to motivationally significant stimuli. Equiprobable task-relevant visual stimuli that differed in terms of their acquired incentive value were presented to alcoholics under baseline and under two incentive conditions in a paradigm previously investigated in healthy volunteers [3].

METHOD

Subjects

Two groups of age- and- education-matched right-handed male subjects were tested: 16 alcoholics (mean age 34.69, S.D. 7.16) and 16 controls (mean age 31.75, S.D. 7.11). All subjects were medication-free and ranged in age between 18 and 46. The alcoholics were diagnosed according to the Research Diagnostic Criteria (RDC). They had been drinking an average of 12 years and were abstinent a minimum of two weeks (mean 20.38 days). Only alcoholics who were free of other medical problems and without histories of abuse of other psychoactive drugs were included. Alcoholics with histories of head injury, seizures unrelated to withdrawal, signs of polyneuropathy or liver disease (cirrhosis, hepatic encephalopathy) were excluded. The control subjects were healthy paid volunteers who were screened for medical problems. They were occasional "social drinkers" who drank an average of ~10 glasses of wine or beer a month (or the equivalent). They were asked to refrain from drinking alcohol for 24 hours prior to testing.

Procedure

Subjects were seated in a sound- and light-attenuated chamber with their heads resting on a chin rest looking directly at a viewing hood 44 cm away. They were instructed to fixate on a fixation point in the center of the screen. The stimuli consisted of visually presented numbers: 0.00 and 1.00. They were generated by a PDP 11/40 computer and displayed in the center of a CRT (visual angle 3.8 degrees) one at a time for 20 msec.

All subjects participated in three experimental conditions. In each condition they were randomly presented with the number stimuli for a total of 30 times each, with a random interstimulus interval varying between 2–5 seconds. The three conditions were always presented in the same order (Table 1).

In the first condition (baseline), subjects were informed

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TABLE 1
EXPERIMENTAL PARADIGM

Run 1 Baseline	Run 2 Incentive (S) (Accuracy)	Run 3 Incentive (S) (Speed-Accuracy)
0.00—Press B	0.00—Press A	0.00—Press A
1.00—Press A	1.00—Press B	1.00—Press B

they would see the numbers 0.00 or 1.00 on the screen; following each presentation of a number stimulus, they were to press the corresponding microswitch. The instructions to the subjects stressed accuracy of responding without mentioning speed of response.

In the second condition (Accuracy/Incentive) the subjects were informed that each presentation of the 1.00 stimulus now could signify the earning of one dollar if they pressed the correct microswitch. The 0.00 stimulus signified that no money could be earned in that trial when the correct microswitch was pressed. However, an error in button-pressing to either stimulus resulted in the loss of one dollar for that trial. The correct microswitches that corresponded to the 1.00 and 0.00 stimuli respectively were reversed in this condition from the baseline condition. Subjects were allowed a maximum of two seconds in which to make a response.

In the third condition (Incentive/Speed-Accuracy), the subjects were again told they would win a dollar for every 1.00 stimulus to which they responded correctly; however, now in order to win the dollar, they had to respond to each presentation of the 1.00 stimulus as quickly as possible, within a criterion time (350 msec). Every stimulus (both 0.00 and 1.00) responded to incorrectly or exceeding the criterion time resulted in the loss of a dollar. The same buttons were used during this run as in the Incentive/Accuracy run (Run 2).

Electrodes

Gold Grass electrodes were affixed to the scalp with collodion at four midline (Fz, Cz, Pz and Oz) and two lateral (P3 and P4) scalp locations according to the 10–20 International System. Beckman biopotential leads were positioned above and below the right eye in order to monitor eye movement artifacts. Trials with excessive eye-movements (>50 μV) were automatically rejected. All scalp leads were referenced to gold linked ear electrodes. A biopotential ground electrode was placed in the center of the forehead. Resistance of all scalp electrodes was maintained below 3 kΩ.

The EEG was amplified 20,000 times (Grass model P511J) with a bandpass of 0.1–100 Hz. A/D sampling began 49 msec prior to the onset of the stimulus (baseline) and continued for a 700 msec epoch following stimulus presentation (sampling rate 142.8 points/sec). Stimulus presentation and data acquisition were controlled by a PDP 11/40 computer. All single ERPs, reaction time values and behavioral responses (button press) were stored on disk and digital magnetic tape for subsequent analysis.

DATA ANALYSIS AND RESULTS

All ERPs were subjected to eye movement artifact rejection procedures (>50 μV). ERPs obtained to trials with correct behavioral responses were subjected to two kinds of data analysis: (1) Principal Component Analysis with Varimax Rotation (PCAV) using the covariance matrix (BMDP-4M), (2) Latency Corrected Averages (LCA) followed by baseline-to-peak measures.

In the first data analysis method, averaged ERPs for all experimental conditions (3) and stimuli (2) across electrodes (6) were subjected to PCAV for each group of subjects (Con-

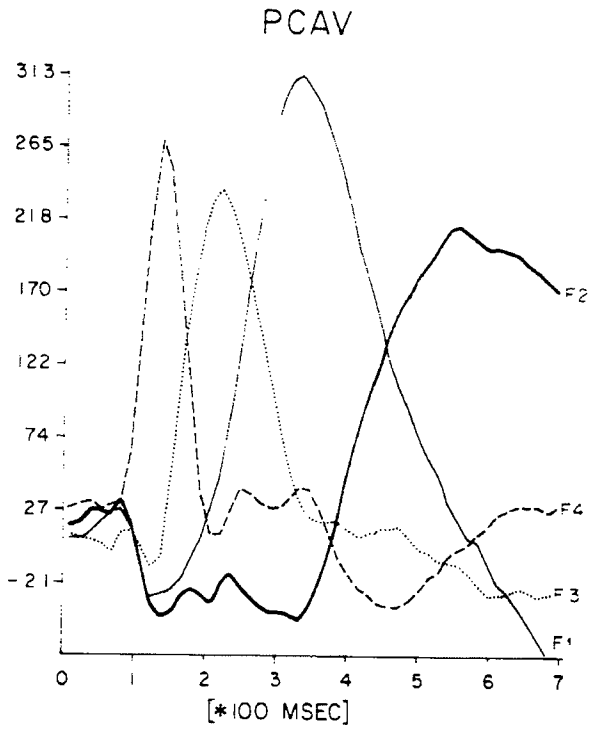

FIG. 1. Factor loadings of first four factors obtained from Principal Components Analysis with Varimax Rotation.

TABLE 2
REPEATED MEASURES ANOVA OF FACTOR SCORES FROM PCA WITH VARIMAX ROTATION USING COVARIANCE MATRIX (CONSERVATIVE *df*—JENNINGS AND WOOD 1.31)

	F1	F2	F3
Group	8.08†	11.22†	
Run	21.21‡		
Run × Group	12.31†		
Stimulus	4.38	7.24*	
Stimulus × Group			
Run × Stimulus			
R × S × G			
Electrode	19.89‡	18.05‡	
E × G	6.88*		
R × E	5.68*		5.21*
R × E × G			
S × E	19.65‡		15.81‡
S × E × G	6.55*		
R × S × E	5.37*		
R × S × E × G			

*$p<0.05$; †$p<0.01$; ‡$p<0.001$.

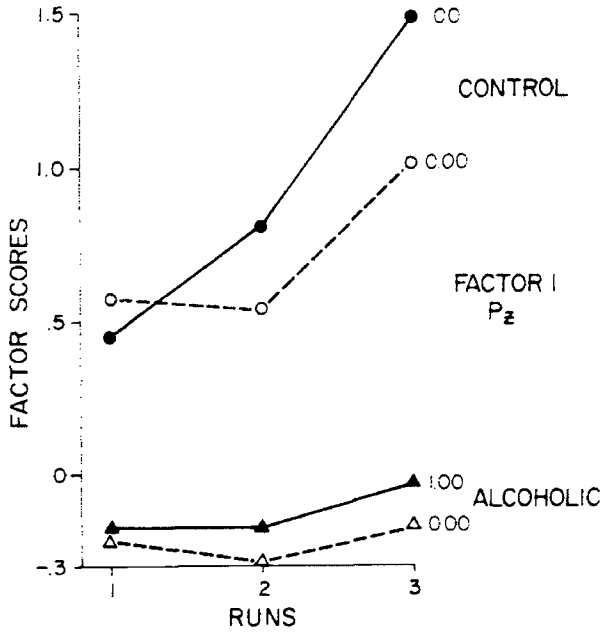

FIG. 2. Mean Factor 1 scores obtained at Pz electrode for the 1.00 stimulus (solid line) and 0.00 stimulus (dashed line) in the control and alcoholic groups.

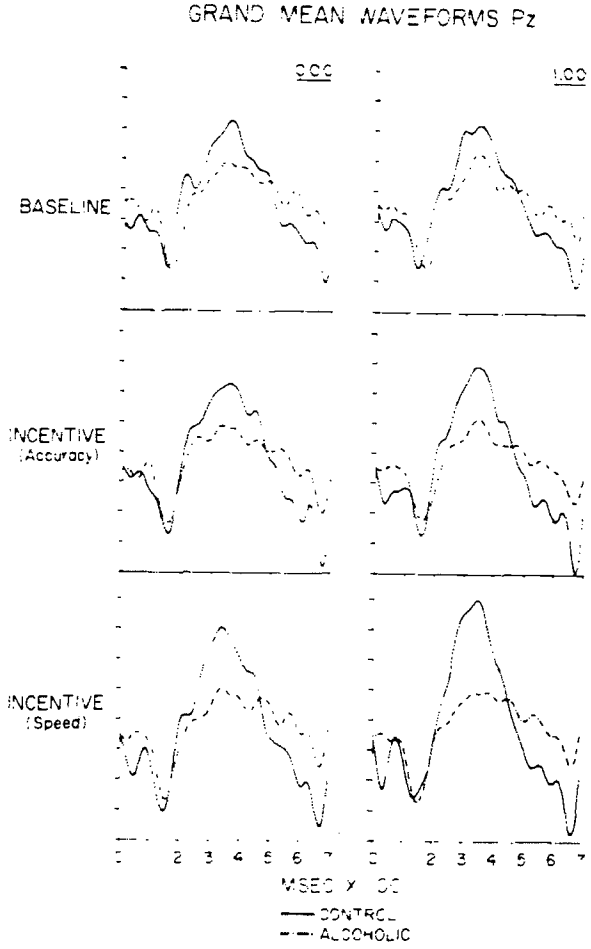

FIG. 3. Grand mean ERP waveforms to 0.00 and 1.00 stimuli at the Pz electrode for the control (solid line) and alcoholic (dashed line) group under the three experimental conditions: baseline, accuracy-incentive and speed-accuracy incentive.

trol and Alcoholic) separately. As the factor structures were similar in the control group and alcoholic group, the data from the two groups (N=16/group) were combined to perform one PCAV based on 1152 waveforms.

The first four factors obtained from PCAV account for 80.8% of the variance (Fig. 1). Factor 1 is maximum at Pz and peaks at 332.5 msec: it corresponds to the P3 component. Factor 2 is maximum at Pz and represents a rather broad component, namely the so-called slow wave (SW) peaking at 556.5 msec. Factor 3 peaks at 220.5 msec and may represent the P2 component. Factor 4 peaks at 136.5 msec and may reflect the N1 component.

The factor scores for each of the four factors were subjected to a four way repeated measures analysis of variance (ANOVA); namely: 2 groups (Alcohol and Control) × 3 runs (Baseline, Incentive Accuracy, Incentive-Speed/Accuracy) × 2 stimuli (0.00 and 1.00) × 6 electrodes (Fz, Cz, Pz, Oz, P3, P4). Degrees of freedom were reduced to 1 and N-1 (1,31) according to the method outlined by Jennings and Wood [5] to take into account unequal variance-covariance matrices in repeated measure designs (Table 2).

The ANOVAs for Factor 1 (P3) and Factor 2 (SW) yielded significantly different main effects between groups, stimuli and electrodes. However, only the ANOVA for Factor 1 was statistically significant for runs and run × group as well (Table 1). While both Factors 1 (P3) and 2 (SW) were significantly different for groups, they behaved quite differently from each other. Our results indicate that SW is larger in alcoholics than controls, while P3 is smaller.

The factor scores for Factor 1 at Pz indicated that in the baseline condition, Factor 1 was not different between the 1.00 and 0.00 stimulus in either group of subjects (Fig. 2). However, with the introduction of incentive in Runs 2 and 3, the factor scores were significantly larger to the 1.00 when compared to the 0.00 stimulus in the control but not the alcoholic group. With the addition of speed (Run 3), the factor scores to both stimuli increased in the control group. In the alcoholic group on the other hand, the Factor 1 scores remained at the same low level regardless of our experimental manipulation. They were significantly lower than in the control group under all conditions including baseline. These results are also apparent in the grand mean waveforms (Fig. 3).

The second data analysis procedure involved the use of Latency Corrected Averages (LCA) on raw single trial ERPs [1,8] in order to reduce latency jitter in the average. The baseline-to-peak amplitude and latency measures obtained from these averages were then subjected to a four-way analysis of variance (ANOVA) with repeated measures: 2 groups (Control, Alcoholic) × 3 conditions (Baseline, Incentive/Accuracy, Incentive-Speed/Accuracy) × 2 stimuli (0.00, 1.00) × 6 electrodes (Fz, Cz, Pz, Oz, P3, P4). Again conservative degrees of freedom according to Jennings and Wood [5] were used. The results were very similar to those obtained with PCAV procedures. No differences in the latency of P3 were obtained between groups. The results of these LCA and baseline-to-peak measurement procedures indicated that alcoholics manifested significantly lower amplitude P3 components than controls to the 1.00 stimulus in both incentive conditions. Furthermore, the alcoholics maintained the same low amplitude P3 to both stimuli regardless of condition.

In terms of RT, both groups responded more quickly to

both stimuli in Run 3 compared to the other two runs. In addition, both groups responded significantly faster to the 1.00 than the 0.00 in both incentive conditions. In the third run (speed), the controls responded significantly faster than the alcoholics to both stimuli. Both groups made very few errors under any of the conditions and did not differ significantly from each other. However, the controls made significantly more correct responses within the criterion time than the alcoholics.

DISCUSSION

Our results replicate our previous findings of reduced P3 amplitudes and no P3 latency delays in abstinent alcoholics [2, 10–13]. These decreased P3 voltages in alcoholics were demonstrated both with PCA (Factor 1-P3) and P3 amplitude measures following Latency Corrected Averaging (LCA). It should be noted that these reduced P3 amplitudes in abstinent alcoholics cannot be due to latency jitter in the averages, as LCA procedures take fluctuations in latency into account [1]. While we have previously reported lower P3 amplitudes in alcoholics, this is the first time we have demonstrated these results using LCA procedures.

Furthermore, this is our first demonstration that ERPs obtained to equiprobable, task-relevant stimuli with differential incentive values result in different Factor 1 (P3) scores and LCA P3 amplitudes in control, but not alcoholic subjects. While we have previously reported that the P3 amplitude is reduced in alcoholics, we have so far only demonstrated this with various target-selection paradigms. This is our first demonstration of low P3 voltages in alcoholics to equiprobable, task-relevant, motivationally significant stimuli.

In a target-selection design, subjects must identify a rare target stimulus embedded in a sequence of frequent standards. The voltage of the P3 component is related to a number of variables affecting the "subjective significance" of the stimulus, namely task-relevance [17], probability of occurrence [18], and motivational factors [3]. The more task relevant a stimulus is, the larger the P3 voltage; similarly, the more improbable a stimulus, the greater the amplitude of P3.

In the present paradigm, both stimuli (0.00 and 1.00) were task relevant, i.e., they both required a behavioral response (button press), and both stimuli were equiprobable. It should be noted that the amplitude of P3 was significantly lower in the alcoholics than the controls in the baseline condition prior to motivational manipulation of the stimuli. This result is evident in the grand mean waveforms (Fig. 3) and the factor 1 scores at Pz (Fig. 2). In fact, the amplitude of P3 remains at this low voltage level regardless of our experimental manipulations in the alcoholics. In the baseline condition, both stimuli are task relevant and equiprobable. Therefore they are of some significance to the subject and would be expected to generate P3s—even if of low-moderate amplitude; furthermore, they would be expected to elicit similar P3s to both stimuli. This is indeed apparent in the control group. With the addition of incentive, the stimuli become differentially significant to the subject and hence the P3 voltage would be expected to increase to the 1.00 but not the 0.00 stimulus. Again, these expectations were confirmed in the control, but not the alcoholic group. In fact the amplitude of P3 (and Factor 1 scores) remained at this low level regardless of experimental manipulations in the alcoholics.

The neural origin(s) of the P3 component are not clearly known at the present time. Some evidence with intracranial recording implicates the medial temporal lobe as contributing to the generation of the scalp P3 [4, 7, 15, 16, 19]. It has recently been suggested that source(s) within the frontal lobe are also involved in P3 generation [7]. This finding, coupled with the rather small effect of unilateral temporal lobectomy on scalp P3 during auditory discrimination tasks [6, 16, 19], suggests multiple brain sites contribute to the scalp P3 [7, 15, 16]. Thus our present results that alcoholics manifest low-voltage P3 components to motivationally significant stimuli may be indicative of limbic system and/or frontal lobe deficits in alcoholics.

Our PCAV results also indicated that Factor 2 (SW) was different between alcoholics and controls. SW often behaves differently from P3 in that it is larger with increases in equivocation or uncertainty, while P3 is smaller. SW decreases as detection accuracy increases, while P3 increases with detection accuracy [15]. In fact, our results indicate a larger SW in alcoholics than controls.

In conclusion, it seems that alcoholics are unable to generate P3 voltages comparable to those of healthy controls under various conditions designed to elicit P3s. We have previously reported low P3 amplitudes in alcholics in target-selection paradigms involving easy and difficult discriminations with rare visual stimuli. We now report low P3 voltages in alcoholics in a visual task involving equiprobable, task relevant, motivationally significant stimuli. Thus it seems that alcoholics are incapable of generating P3 voltages under any conditions we have examined, suggesting multiple system deficits, perhaps involving frontal lobe and medial temporal lobe functioning. The lack of differential responding to the rewarded stimulus, combined with their minimal level of responding to both stimuli under all conditions, perhaps reflects a deficit in the motivational-cognitive systems of alcoholics, possibly affecting their ability to actively sustain information processing [9]. This perhaps in part explains the tendency of alcoholics to perseverate old response patterns regardless of changes in reinforcement contingencies.

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