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ALCOHOL AND BILATERAL EVOKED BRAIN POTENTIALS

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Man is cerebrally unique in that he is the only primate with marked functional asymmetry, where the two halves of the brain are specialized to serve separate functions. In most individuals (95%), the left hemisphere plays a dominant role in all language function - speech, verbal perception and thinking, while the right hemisphere dominates non-verbal contents, e.g., form perception and feelings.

Responses to non-meaningful blank flashes are mediated by the non-dominant right hemisphere. Experiments performed at Beck's laboratory indicate that right lobe evoked responses are typically larger than left lobe responses to flashes in normal subjects. This finding of interhemispheric asymmetry was significant at central locations (C_3 and C_4) in normal subjects, but was not present in alcoholics, who also displayed lower visual evoked potential amplitudes at all electrode locations, namely occipital, frontal and central (Schenkenberg et al., 1970, 1972).

In another experiment at the same laboratory, Lewis, Dustman and Beck (1970) report that cross-hemispheric asymmetry that was manifested in central areas before alcohol ingestion, disappeared after each alcohol intake. Visual evoked potentials from the right central area were significantly reduced, while those on the left remained unimpaired.

Although visual evoked potential interhemispheric asymmetry has been reported to exist in some subjects, the results of systematic studies of large populations of subjects are equivocal. Harmony

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et al. (1973) assessed visual evoked potential asymmetry at central, occipital and temporal locations using both monopolar and bipolar recordings in 139 normal subjects. No significant differences in peak-to-peak amplitudes were obtained between the two hemispheres, and amplitude differences were found to be less than 40%. Ninety percent of the correlations between waveforms obtained simultaneously from the two hemispheres exceeded .85 from all electrode locations. The experimenters therefore concluded that the two hemispheres are strikingly symmetrical in the normal population.

Evoked potentials have been useful in investigating the differential effects of alcohol in the central nervous system, as they are highly susceptible to its suppressive influence by becoming concomitantly reduced in amplitude.

In a study of the effects of 100 cc's of alcohol on the auditory evoked response, Gross et al. (1966) established that alcohol significantly reduces the auditory evoked response. The maximal effects were obtained 15-30 minutes after alcohol ingestion.

Salamy and Williams (1973) investigated the effects of varying concentrations of alcohol on somatosensory evoked potentials recorded from bipolar leads in the post-Rolandic, parasaggital plane, and monopolar vertex recordings. They reported an inverse relationship between the dose of alcohol, the blood alcohol level, and the amplitude of the evoked potential. Early components were found to be more resistant to the effects of alcohol than later components. The major effect was obtained at vertex, particularly amplitude N_1-P_2, which was markedly reduced with increasing blood alcohol levels.

Lewis, Dustman and Beck (1970) studied the effects of two doses of alcohol mixed with grapefruit juice (.41 g/kg and 1.23 g/kg) on the visual and somatosensory evoked responses of moderate drinkers, recorded at occipital and central cortical locations. No effect was obtained with the low dose of alcohol. With the high dose they found a significant decrease in amplitude of both visual and somatosensory evoked responses recorded from central areas, particularly of the later components. Visual evoked response amplitudes were only significantly reduced from the right central electrode placement. No significant changes in amplitude of any component were obtained at occipital locations.

The purpose of the present experiment is to: first, ascertain whether or not bilateral asymmetry is apparent in the normal visual evoked potential and, second, to assess the effects of a moderate dose of alcohol on this interhemispheric asymmetry and, thirdly, to investigate whether there are differential effects of alcohol at different bilateral locations on visual evoked potential amplitude.

METHODS

The subjects were 13 healthy right-handed, adult male graduate students, with a mean age of 24, and a mean weight of 169 pounds. All subjects were occasional drinkers.

They were required to come to two recording sessions on non-consecutive mornings in the same week, and were requested to eat large identical breakfasts on both mornings. On one morning they received 95% ethyl alcohol mixed with orange juice, while on the other morning an equivalent amount of only orange juice was administered.

The amount of alcohol each subject received was dependent on his body weight in the ratio of 1.04 g/kg (1.3 ml/kg) of 95% alcohol. This amount of alcohol comprised 1/4 of his total drink, and to it were added 3 parts of orange juice.

Monopolar recordings were obtained from electrodes secured to the scalp with collodion. Electrodes were placed at central and occipital locations, bilaterally, using the linked ears as reference, and the naseum as ground. The central leads were placed 4 cm on either side of the midline on the inter-aural line, corresponding to C_3 (left) and C_4 (right) of the 10-20 International System. Occipital placements were 2.5 cm anterior to the inion and 2.5 cm on either side of the midline, corresponding to O_1 (left) and O_2 (right) of the 10-20 International System. Resistances were maintained below 5000 ohms.

Each subject was seated in a sound-attenuated, electrically shielded enclosure, with his head resting on an adjustable chin-rest, so that he was looking directly into a viewing hood, fixating in the center of his visual field. A Grass photostimulator set at an intensity of 16, delivered flashes at a random rate of 1-5 seconds apart, for a total of 32 flashes. The visual stimulus was a 5 cm square neutral density filter, that cut down the amount of light being transmitted by 80%.

The drink was administered after the first (baseline) run, and the subject was required to drink it in 10 minutes, spacing it as evenly as possible over the 10 minutes. Each recording session consisted of seven runs, one before the administration of the drink (baseline), and the others at 15, 30, 45, 60, 90, and 120 minutes after the drink had been finished, respectively. Blood alcohol levels were monitored with Breathalyzer readings which were obtained throughout the experiment immediately preceding each run.

Visual evoked potentials for each electrode placement were recorded on a Grass polygraph and summated simultaneously in

4 channels of a Hewlett Packard Signal Analyzer, for a 500 msec epoch.

Four amplitude measures were obtained from the occipital leads as the perpendicular distance in (μV) between successive peaks. Specifically, these are: the negative-going Amplitude A (approximately 60-90 msecs), the positive-going Amplitude B (90-120 msecs), the negative Amplitude C (120-165 msecs), and positive Amplitude D (165-220 msecs after the flash).

Two amplitude measures were also obtained from the central electrodes: namely, P_1-N_1, occurring at approximately 100-140 msecs and N_1-P_2, occurring from 140-200 msecs after the flash.

RESULTS

In order to assess cortical interhemispheric asymmetry, paired t-tests were performed on differences between visual evoked potential amplitudes from the right and left hemispheres. Consistent interhemispheric asymmetry was found in Amplitude B (N_1-P_2) and C (P_2-N_2) at occipital sites, but not from central areas. Neither the early components of the central response (P_1-N_1) nor the late components (N_1-P_2) displayed any significant bilateral asymmetry.

As can be seen in Figure 1 of group means for Amplitude B, the right hemisphere occipital recordings (O_2) were significantly larger than the responses simultaneously evoked by the left hemisphere (O_1) throughout the non-alcohol day.

The occipital asymmetry that was present in Amplitude B throughout the recording session on the control day, disappeared completely with the ingestion of alcohol. (See Figure 2)

Significance tests of the same amplitude under identical conditions but on the alcoholization day, indicate that while interhemispheric asymmetry is present in Run 1, baseline ($t = 3.2746$, $p < .01$), before alcohol administration, it disappears immediately after alcohol ingestion. There are no significant amplitude differences between right and left hemispheres, even two hours after the completion of drinking, although the two curves are just beginning to diverge at that time. The right hemisphere response was reduced to a much greater degree than the left hemisphere response, which was relatively unaffected by alcohol.

Figure 3 illustrates typical right and left hemisphere occipital evoked potentials, recorded from the same subject, comparing the same run on the alcohol and control days. The top two traces were obtained from left and right leads respectively, 45 minutes after

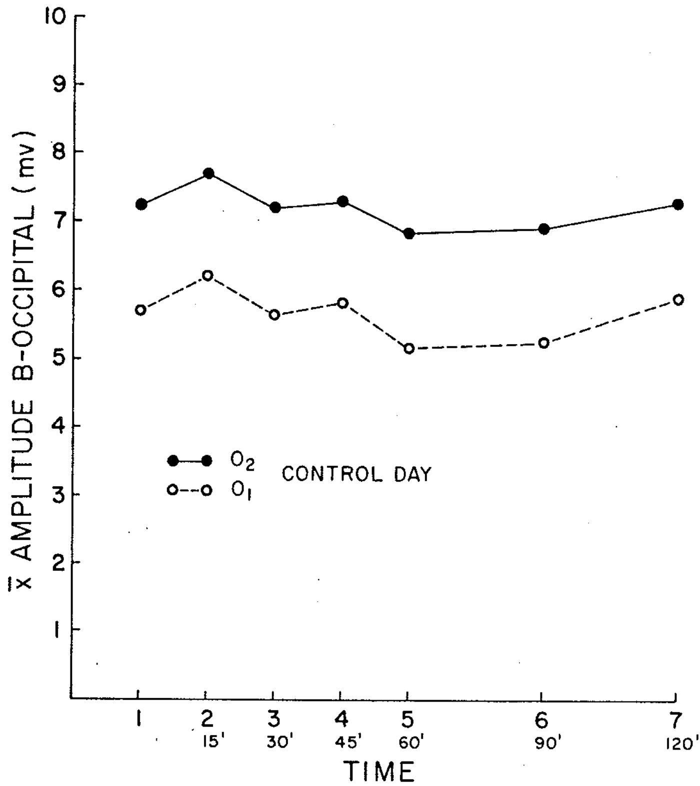


Figure 1. Mean Amplitude B (N_1-P_2) for all subjects (N = 13) recorded simultaneously from the right (O_2) and left (O_1) occipital area on the control day. Amplitudes were significantly larger from O_2 than O_1 as follows:

Run 1 (baseline) $t = 2.2968$, $p < .05$
Run 2 (time 15) $t = 3.0783$, $p < .01$
Run 3 (time 30) $t = 2.7452$, $p < .02$
Run 4 (time 45) $t = 2.2869$, $p < .05$
Run 5 (time 60) $t = 3.7159$, $p < .01$
Run 6 (time 90) $t = 3.2653$, $p < .01$
Run 7 (time 120) $t = 2.6636$, $p < .05$

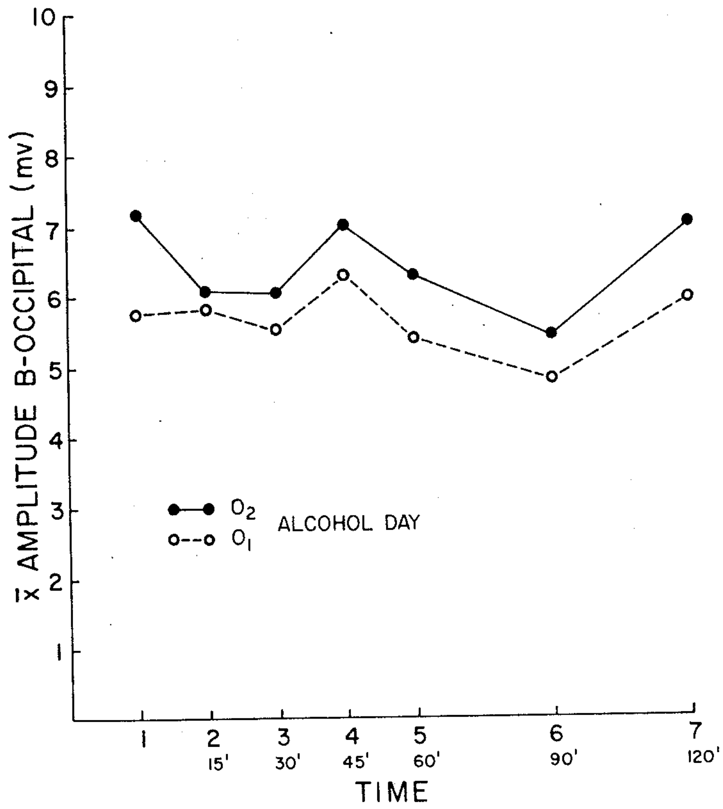


Figure 2

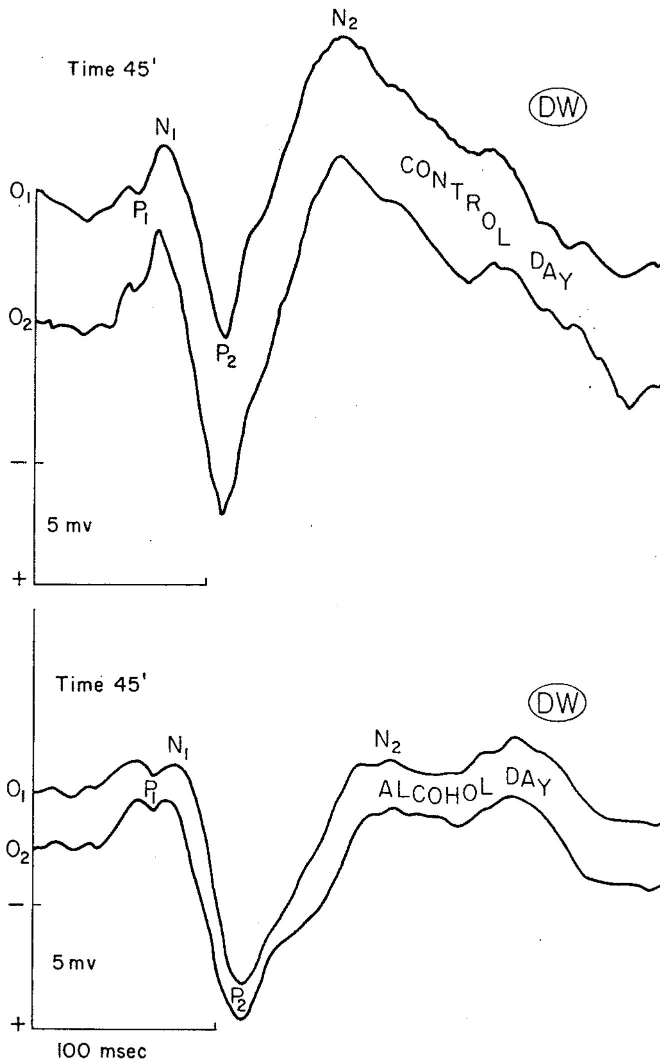


Figure 3

only orange juice was ingested. The marked asymmetry in amplitude of the positive-going wave N_1-P_2 (Amplitude B) and the negative-going P_2-N_2 (Amplitude C) can be seen. The bottom two traces illustrate how this asymmetry between O_1 and O_2 has virtually vanished when alcohol is administered.

In order to determine the effect of alcohol on the visual evoked potential, a 2-factor analysis of variance with repeated measures on one factor was performed for each amplitude, comparing the 6 alcohol with 6 control runs. Only the late components of all electrode placements were found to be significantly decreased by alcohol. The major depressant effect of alcohol was found in the large late positive-going wave N_1-P_2 of the central area.

This very striking reduction in visual evoked potential amplitude with alcohol for all subjects at the right hemisphere can be seen in Figure 4; $F (1,24) = 19.6554$, $p < .01$. Alcohol immediately depresses this amplitude, and its effects are sustained, even outlasting the 2-hour post-alcohol testing session.

As can be seen in Figure 5, a similar decrease in amplitude was obtained from the left hemisphere central response and the Analysis of Variance was also significant at $p < .01$, $F (1, 24) = 13.7836$.

Typical visual evoked potentials from central electrodes of both hemispheres can be seen in Figure 6 for the same subject on the control day and alcohol day, 15 minutes after drinking. The large late component (N_1-P_2) is strikingly reduced by alcohol in both hemispheres, while the first component (P_1-N_1) is affected much less.

In addition to the major decrease in late component amplitude at the central locations, the late component of the occipital response was also significantly depressed by alcohol bilaterally.

Figure 7 demonstrates the depressant effect of alcohol on mean magnitude of the late component (Amplitude D) of the right occipital. This amplitude was essentially identical for Run 1 (baseline) on both days, but became significantly reduced on the alcohol day, beginning immediately after alcohol ingestion; however, it is almost fully recovered 2 hours after the intake of alcohol.

The same result was obtained at the left occipital (Figure 8), which also almost returned to its original magnitude 2 hours after alcohol ingestion. There was no longer any significant difference between alcohol and control days at this time. An illustration of this occipital late component decrease can be seen in Figure 3, which is a typical record taken 45 minutes after liquid intake; there is a marked difference in amplitude of the large late component between control and alcohol days.

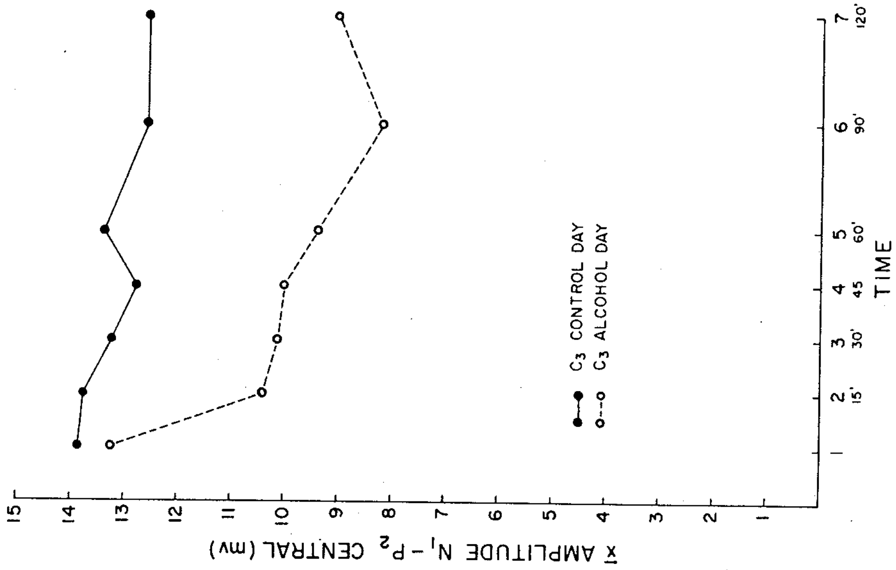


Figure 5

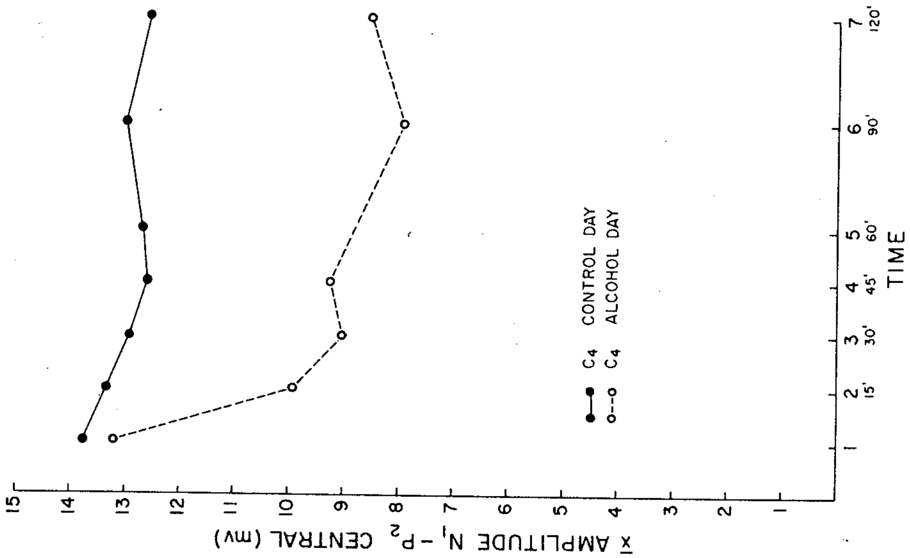


Figure 4

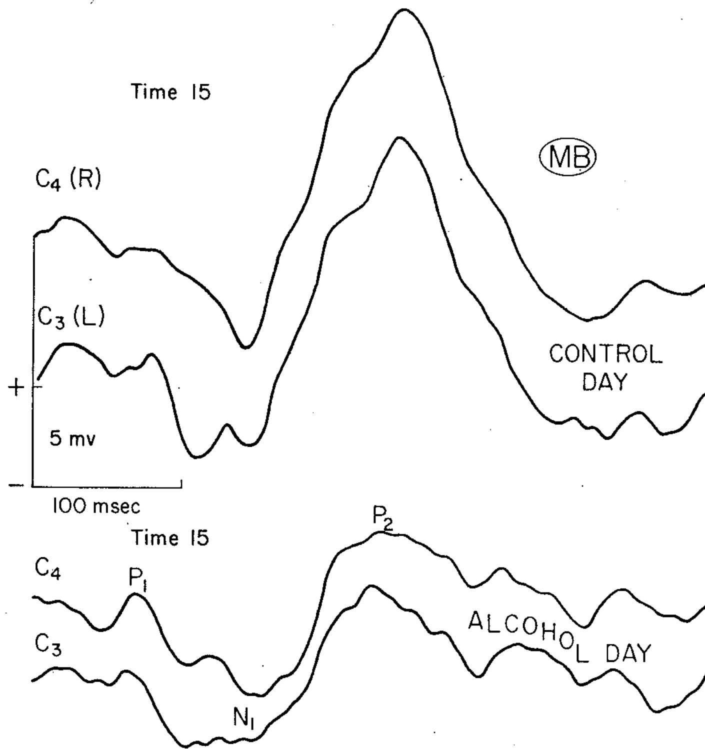
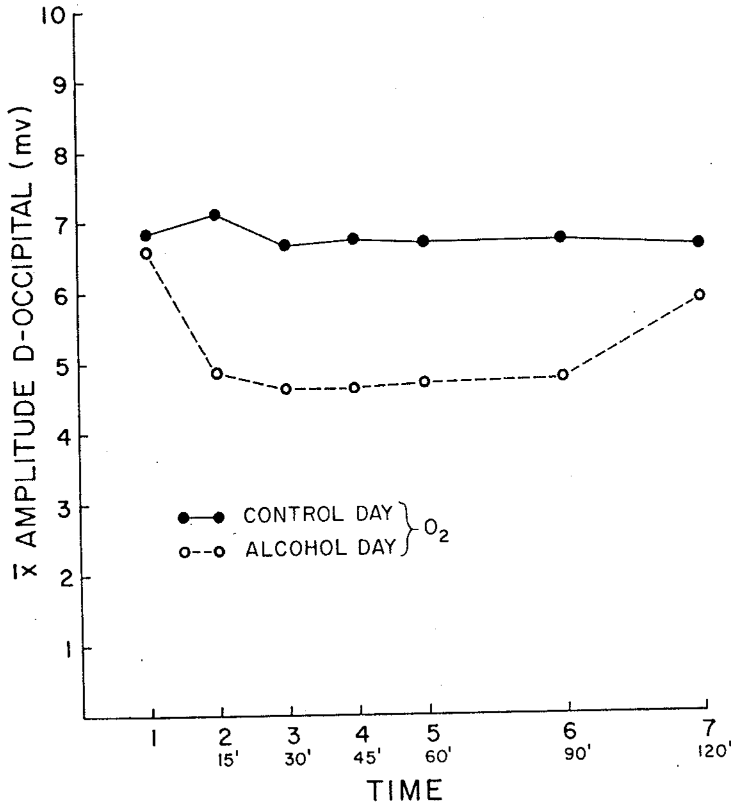


Figure 6



<u>Figure 7</u>. Mean Amplitude D for all subjects recorded from the right occipital (O_2) on alcohol and control days. Alcohol significantly reduces this amplitude as follows:
Run 1 (baseline) t = 0.4087, n.s.
Run 2 (time 15) t = 2.9032, $p < .02$
Run 3 (time 30) t = 2.7295, $p < .02$
Run 4 (time 45) t = 2.405, $p < .05$
Run 5 (time 60) t = 2.5284, $p < .05$
Run 6 (time 90) t = 2.6371, $p < .05$
Run 7 (time 120) t = 0.8054, n.s.

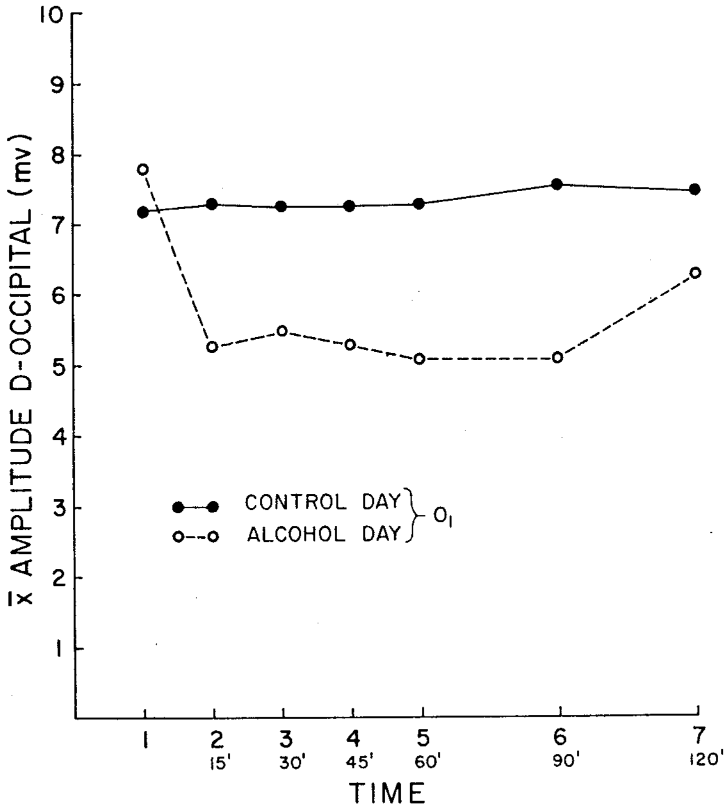


Figure 8. Mean Amplitude D for all subjects recorded at the left occipital (O_1) on alcohol and control days. Alcohol significantly reduces this amplitude as follows:

Run 1 (baseline) t = 0.7017, n.s.
Run 2 (time 15) t = 2.9537, p /.02
Run 3 (time 30) t = 2.9837, p /.02
Run 4 (time 45) t = 2.1797, p /.05
Run 5 (time 60) t = 2.3605, p /.05
Run 6 (time 90) t = 3.3501, p /.01
Run 7 (time 120) t = 1.269, n.s.

DISCUSSION

The results of the present experiment support the contention that responses to non-meaningful blank flashes are mediated by the non-dominant right hemisphere. Bilateral asymmetry was present at occipital sites, where the right lobe responses were significantly larger than left lobe responses. Despite the finding from Beck's laboratory that right central evoked potentials in normals are consistently larger than left central responses, we did not obtain asymmetry at that location. However, the asymmetry studies from Beck's laboratory are based on very small sample sizes. Our finding that neither the early nor late components of the central response demonstrate interhemispheric asymmetry is in agreement with Harmony et al.(1973), who report a remarkable degree of bilateral symmetry in the visual evoked response at identical electrode locations with large samples of subjects.

Therefore, great caution is suggested in evaluating the results of studies comparing interhemispheric amplitude asymmetry between normal and pathological groups. Until normative data can be established for the non-alcoholic population, no frame of reference exists with which to compare the alcoholic condition.

The ingestion of alcohol seems to have the effect of dissipating any existent bilateral asymmetry. In the present experiment, where VEP asymmetry was displayed at occipital locations, the ingestion of alcohol virtually abolished interhemispheric differences, decreasing responses from the right hemisphere, while leaving left hemisphere responses relatively unaffected. A similar dissipation of cross-hemispheric asymmetry with alcohol intake was reported by Lewis, Dustman and Beck (1970) for central areas. However, in contrast to our findings, they report that the left hemisphere was unaltered by alcohol intake at central locations. In the present experiment, although the right central area was found to be most susceptible to alcohol ingestion, the late components of central responses from both hemispheres were found to be particularly sensitive to alcohol.

The finding that the early components are more resistant to the depressant effects of alcohol than the later components supports the results of Salamy and Williams (1973) at a similar electrode location, namely vertex.

In addition to the major decrease in late component amplitude at both central locations, we found that the late component of the occipital response was also significantly depressed by alcohol bilaterally. This is in contrast to the findings by Lewis, Dustman and Beck (1970), who reported that all amplitudes of the occipital response are unaffected by alcohol. However, the results of the present study with visual responses are consistent with those of

Salamy and Williams (1973) with somatosensory responses. They found that the late component of the somatosensory evoked potential is significantly reduced in the primary receiving area, but that the early components are resistant to its effects. Similarly, in the present experiment, the early components of the visual evoked potential in the primary receiving area (occipital) are not susceptible to the depressant effects of alcohol, while the late components are markedly reduced.

The late components of the visual evoked potential are often considered to reflect the more cognitive aspects of perception, while the early components are taken to indicate sensory input. It seems, therefore, from the present findings that alcohol differentially affects cortical functioning, primarily impairing cognitive (or output) processes, while leaving incoming sensory process intact. Furthermore, the major change in visual evoked potential amplitude occurred at the central electrode placements which are adjacent to association cortex, suggesting that higher cortical functioning is more susceptible to alcohol than the primary sensory area (occipital).

There is evidence supporting this contention in information processing reaction-time experiments. In recent studies, Rundell et al. (1973) and Tharp et al. (this volume) report that stimulus pre-processing and encoding are unaffected by alcohol, but that alcohol affects the more central (output) stages of information processing, for example, response selection and organization.

In conclusion, the present experiment seems to indicate that interhemispheric asymmetry is present only at occipital locations, and is dissipated by the ingestion of alcohol. Furthermore, the present findings suggest that alcohol differentially affects central nervous system activity, primarily suppressing higher cortical functioning. However, the exact sites of action of alcohol, and the progression of its effects on the brain remain to be clarified.

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