CONTRACT REPORT



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# ETHYL ALCOHOL AND THE ELECTROENCEPHALOGRAM: IMMEDIATE AND LONGER-TERM EFFECTS


Submitted to

National Road Safety Council

NATIONAL INSTITUTE FOR PERSONNEL RESEARCH
COUNCIL FOR SCIENTIFIC AND INDUSTRIAL RESEARCH

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by

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Neuropsychology Division

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SUMMARY

Electroencephalograms (EEGs) were recorded from 72 ethyl alcohol (EA) subjects and 22 subjects given a placebo containing no EA on three different occasions : before treatment, immediately after treatment and the morning after treatment. For statistical purposes the EA group was subdivided into high (>0,08 mg %) and low (≤0,08 mg %) EA, and high (>10,42Hz) and low (≤10,42Hz) alpha frequency groups - group and subgroup comparisons showed that mean alpha frequency, amplitude and index measures and abnormality ratings all responded to EA, both immediately and on the morning after its administration. The effects of high and low concentrations of EA in the blood were not marked in relation to EEG measures, while high and low alpha frequency subgroups responded differentially to EA.

OPSOMMING

Die elektroenkefalogramme (EEGs) van 72 etielalkohol-(EA)proefpersone en 22 proefpersone, aan wie 'n placebo wat geen EA bevat het nie, toegedien is, is by drie geleenthede opgeneem : voor behandeling, kort na behandeling en die daaropvolgende oggend. Statistiese vergelykings is op dié groepe en die volgende subindelings van die EA-groep uitgevoer : hoë (>0,08 mg %) en lae (≤0,08 mg %) EA en hoë (>10,42Hz) en lae (≤10,42Hz) alfafrekwensie groepe. Gemiddelde alfafrekwensie, -amplitude en -indeksmetings en bepalings van abnormaliteit het almal veranderings kort na en op die oggend na toediening van EA getoon. Hoë en lae EA-bloedkonsentrasies het geen noemenswaardige uitwerking op die EEG-metings gehad nie, terwyl hoë en lae alfafrekwensie subgroepe verskillende response op EA getoon het.

CONTENTS

# INTRODUCTION

There is a considerable literature detailing the changes occurring in the electroencephalogram (EEG) after the ingestion of ethyl alcohol (EA). It is not within the scope of this report to provide an exhaustive review of this literature. However, an attempt will be made to give the consensus of findings previously obtained, and to indicate their possible relevance to the driving situation.

Nelson[1] (1969) reviewed the effects of EA on the EEG to that date. In general, EEG frequency measures were most affected by the ingestion of EA : initially, alpha (8-13Hz) frequency showing a decrease, and after additional EA, theta rhythms (4-7Hz) becoming prominent. Amplitude measures also showed some change after ingestion of EA, in the form of an initial increase in alpha amplitude. Nelson also referred to studies suggesting EEG abnormalities after EA, but considered that these results should be interpreted with caution. This is necessary as excessive slow activity in the EEG after EA may reflect diminished arousal rather than cortical dysfunction. In Nelson's view, "probably the only classes of EEG abnormalities which are unequivocal in this situation are focal and paroxysmal disturbances" (p. 27).

The majority of studies encompassed by Nelson's review relied on visual inspection and analysis of EEG measures. However, more recent investigations employing more objective techniques of analysis (for example, that of Salamy and Williams[2], 1973, using major and intermediate period analysis, and amplitude integration) have not changed in essence the findings outlined above.

The finding of an initial reduction in alpha frequency upon ingestion of EA, followed by augmentation of theta frequencies with increasing EA intake, appears to relate to the following subjective states, respectively. Firstly, to a diminished level of alertness, and then to progressively reduced arousal. More specifically, a blood alcohol content (BAC) up to 0,05 mg per 100 ml induces some sedation or tranquility. BACs between 0,05 and 0,15 mg per 100 ml produce lack of co-ordination and behavioural changes such as aggression and

talkativeness resulting from depression of the cortical centres normally inhibiting such behaviour. Between 0,15 and 0,20 mg per 100 ml BAC intoxication is obvious and inco-ordination, confusion and disorientation occur. Between 0,30 and 0,40 mg per 100 ml BAC unconsciousness, stupor and anaesthesia appear (Buttiglieri et al,[3] 1972). The relationship between these states and deterioration in driving performance is obvious and well-documented.

The present study, therefore, was undertaken with the following questions in mind : Do measures of EEG activity apart from alpha frequency, amplitude and abnormality, show changes after the ingestion of alcohol? Further, are the changes in EEG measures reflected in the period immediately following ingestion of EA still discernible after a twelve-hour interval (the so-called "hangover" effect) ?

## SUBJECTS

All subjects were male volunteers who were contacted at work and invited to participate in a study of the effects of EA on tests related to driving performance. The EEG examination formed part of this broader investigation. Subjects were required to possess a current driver's licence and to participate in social drinking. The final group selected numbered 94. These were separated at random into a control group (N = 22) who were administered a placebo containing no EA, and the experimental group (N = 72) who were given EA in various beverages. Mean age of the control group was 35,045 years (SD 11,178 years), of the experimental group 30,722 years (SD 10,37). The difference in mean age between the groups was just significant ($t = 1,66$ ; $p = 4,99$).

## PROCEDURE

Subjects were tested in groups of five. Each group was tested in the late afternoon between 16h00 and 17h30 and in the evening between 19h00 and 20h30, and the "hangover" effect measured the following morning between 08h00 and 09h30. On occasion all five individuals

comprising a group were given EA, on others some group members were given the placebo. Placebo or EA administration was not done according to any definite sequence, and EEG recording personnel were not informed whether any individual subject had been given the placebo or EA. All subjects were instructed to have lunch on the day of testing, and administration of placebo or EA took place in an informal, relaxed atmosphere in the staff room of the NIPR. Subjects were allowed to choose from a range of mixers to be given with the placebo or EA. The placebo consisted of a highly concentrated essence of gin, brandy, rum or whisky from which all EA had been distilled. EEGs were recorded, using the bipolar technique, on a portable Galileo E8b 8-channel electroencephalograph, with a single, wide-spaced electrode montage for ease of application. Recordings were carried out in a quiet laboratory environment. Three recordings were made from each subject ; before the administration of placebo/EA ; about 30 minutes after the ingestion of the final dose of placebo/EA (this took place over 60 minutes in three instalments) and the morning following ingestion of placebo/EA. Blood samples were taken from each subject in the EA group after the second EEG.

All EEGs were analysed by eye with the aid of a millimetre cursor by the same electroencephalographer. Analysis was made without a knowledge of whether the EEG had been obtained from a placebo or EA subject. The groups were compared in terms of the following EEG measures :

1) Alpha frequency : this was the mean of at least three measures made from representative, artefact-free samples of alpha activity recorded from the centro-occipital and temporo-occipital areas.

2) Alpha amplitude : as alpha amplitude is considerably more variable than alpha frequency, the electroencephalographer's judgment of representative alpha amplitudes was more subjective than that applied to the frequency measure. Amplitude measurements were made from the same areas and under the same conditions as those pertaining to the frequency measure.

3) Alpha index : this was rated on a four-point scale during the initial 100 seconds of recording, which was made at 1,5 cm/sec. Zero on the scale indicated that alpha activity was absent, 1 that alpha activity occupied between one and 33% of the record, 2 that alpha rhythms were present between 34 and 67 % of the time and 3 that alpha occupied 68 - 100% of the EEG.

4) Alpha organisation : this was based, to some extent, on the alpha frequency, amplitude and index measures, and is an indication of the "goodness" of the alpha activity. A rating of zero on this measure signifies a disorganised alpha activity, with considerable variation in both frequency and amplitude and a low index rating. A rating of 3 indicates a mono-rhythmic, unmodulated alpha rhythm, present for most of the EEG.

5) Abnormality : rating of abnormality of the EEG (on a four-point scale, with zero indicating a normal and three a severely abnormal recording) was made, bearing Nelson's[4] (1969) previously quoted strictures in mind, in terms of focal and paroxysmal dysfunctions and, in addition, sharp wave and spike activity. The latter would appear to be reasonably unequivocal.

For statistical purposes the subjects were divided into a number of groups and subgroups. The effects of EA on the EEG were assessed by comparing the cortical activity of control and experimental groups. As a BAC of 0,08 mg % is the legal limit below which driving in South Africa is permitted, the EA group was subdivided into high and low groups. The former comprised all those individuals in the EA group with a BAC of more than 0,08 mg % (mean BAC for this group was 0,101 mg % ; SD 0,012). The latter was made up of all EA subjects whose BAC was equal to or less than 0,08 mg % (mean group BAC 0,067 mg % ; SD 0,01). As indicated, frequency measures appear to be most closely related to increased BAC, and therefore all subjects who had received EA were divided into high and low alpha groups (with alpha frequencies higher than, and equal to or less than, the EA group mean of 10,42Hz respectively). Both subgroups were further subdivided into high and low EA groups, on the same basis as mentioned previously. One way

analysis of variance and student's t test were used to compare all group and subgroup mean alpha frequency, amplitude, index, organization and abnormality level scores before, immediately after and the morning after treatment, and mean changes in these measures from before to immediately after treatment ; from immediately after to the morning after treatment ; and from before to the morning after treatment. Chi-square in 2x2 or 3x2 form was used to assess group and subgroup incidence differences in abnormality level before, immediately after and the morning after treatment, and changes in group or subgroup abnormality level incidences from before to immediately after treatment, from immediately after to the morning after treatment and from before to the morning after treatment.

## RESULTS

Significant ($p < 0,05$) group and subgroup differences in respect of EEG measures are presented in Tables I to VI. Figures 1 to 4 give the mean alpha frequencies of the groups and subgroups before, immediately after and the morning after treatment.

Table I

Significant differences between placebo (N = 22) and EA (N = 72) groups in respect of EEG measures

| | Mean | | SD | | | |
|---|---|---|---|---|---|---|
| Measure | Placebo | EA | Placebo | EA | t | p |
| Alpha frequency immediately after treatment | 10,489 | 9,868 | 0,887 | 0,924 | 2,752 | <0,05 |
| Change in alpha frequency from before to immediately after treatment | -0,08 | -0,531 | 0,586 | 0,574 | 3,18 | <0,01 |
| Change in alpha frequency from immediately after to the morning after treatment | -0,045 | 0,417 | 0,615 | 0,68 | 2,822 | <0,01 |
| Change in alpha amplitude from before to immediately after treatment | -1,225 | 2,126 | 5,627 | 8,619 | 2,097 | <0,05 |
| Change in alpha amplitude from immediately after to the morning after treatment | 4,402 | -1,499 | 7,932 | 9,431 | 2,633 | <0,01 |
| Change in abnormality from before to the morning after treatment | 0,045 | -0,174 | 0,475 | 0,521 | 1,741 | <0,05 |

Table II

Significant placebo and EA group changes in incidence of EEG abnormality from before to immediately after treatment

| Group | Incidence of less abnormal EEGs | Incidence of EEGs showing no change in abnormality level | Incidence of more abnormal EEGs | N |
|---|---|---|---|---|
| Placebo | 0(2,34) | 18(12,40) | 4(7,26) | 22 |
| EA | 10(7,66) | 35(40,60) | 27(23,74) | 72 |

Expected frequencies for each cell are indicated in brackets.
$\chi^2 = 8,258$ ; $p<0,05$

Table III

Significant EEG differences between high (N = 33) and low (N = 39) EA and placebo (N = 22) groups

| Measure | Mean | | | SD | | | F | p | Significant ($p<0,05$) group differences* |
|---|---|---|---|---|---|---|---|---|---|
| | Placebo (P) | Low EA (LEA) | High EA (HEA) | Placebo (P) | Low EA (LEA) | High EA (HEA) | | | |
| Alpha frequency immediately after treatment | 10,489 | 9,994 | 9,72 | 0,908 | 0,962 | 0,883 | 4,60 | 0,01 | P x HEA |
| Alpha index immediately after treatment | 2,0 | 2,308 | 2,167 | 0,724 | 0,295 | 0,346 | 3,39 | 0,04 | P x LEA |
| Change in alpha frequency from before treatment to immediately after treatment | -0,08 | -0,50 | -0,568 | 0,60 | 0,55 | 0,616 | 5,13 | 0,01 | P x LEA<br>P x HEA |
| Change in alpha frequency from immediately after treatment to the morning after treatment | -0,045 | 0,333 | 0,515 | 0,63 | 0,677 | 0,69 | 4,65 | 0,01 | P x HEA |
| Change in alpha amplitude from immediately after treatment to the morning after treatment | 4,402 | -2,46 | -0,362 | 8,118 | 8,251 | 10,809 | 3,93 | 0,02 | P x LEA |

* Scheffe's Multiple Comparisons Test (Brownlee, [5] 1960, p. 252).

Table IV

Significant group changes in incidence of EEG abnormality from before to immediately after treatment for high and low EA and placebo groups

| Group | Incidence of less abnormal EEGs | Incidence of EEGs showing no change in abnormality level | Incidence of more abnormal EEGs | N |
|---|---|---|---|---|
| Placebo | 0(2,34) | 18(12,40) | 4(7,26) | 22 |
| Low EA | 8(4,15) | 17(21,99) | 14(12,86) | 39 |
| High EA | 2(3,51) | 18(18,61) | 13(10,88) | 33 |

Expected frequency for each cell is indicated in brackets.

$\chi^2 = 12,214$ ($p<0,05$)

Table V

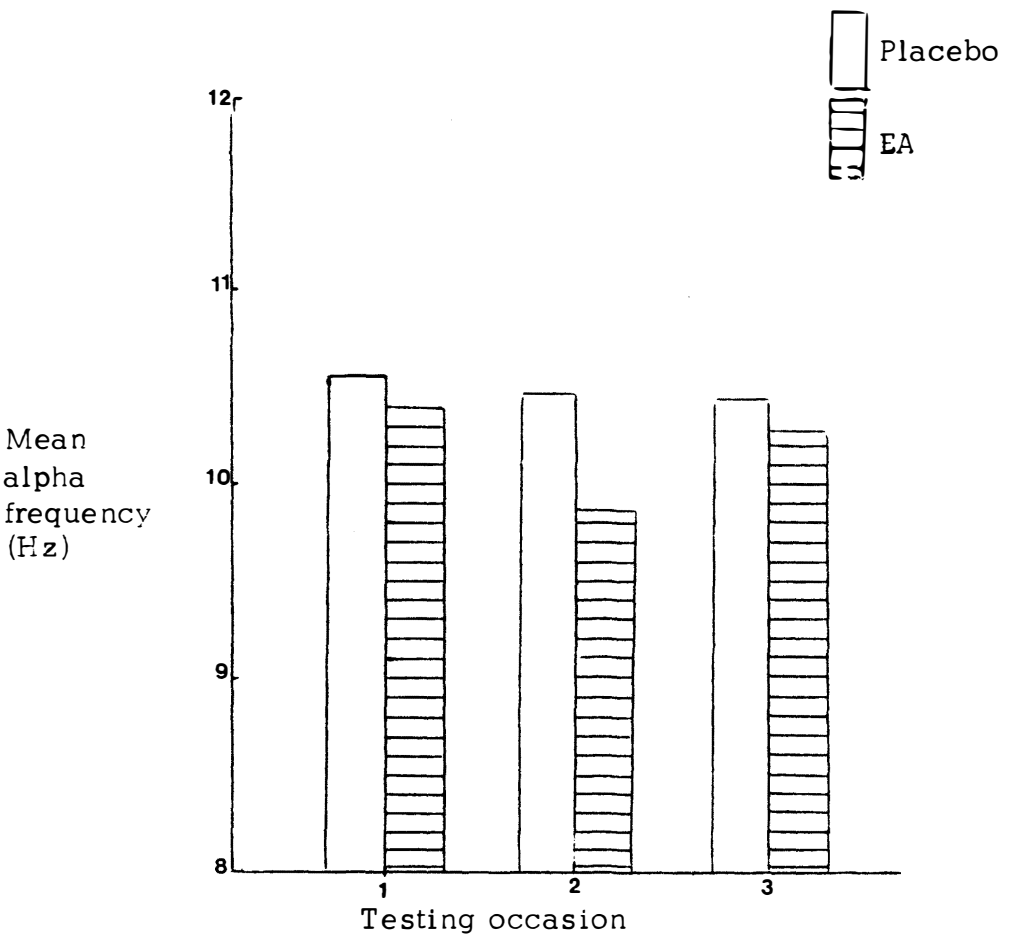
Significant EEG differences between high (N = 40) and low (N = 32) alpha frequency subgroups of the EA group

| Measure | Mean | | SD | | t | p |
|---|---|---|---|---|---|---|
| | Low alpha frequency group | High alpha frequency group | Low alpha frequency group | High alpha frequency group | | |
| Alpha frequency before EA | 9,563 | 11,069 | 0,500 | 0,645 | 10,708 | <0,01 |
| Alpha frequency immediately after EA | 9,227 | 10,381 | 0,574 | 0,825 | 6,889 | <0,01 |
| Alpha frequency the morning after EA | 9,711 | 10,744 | 0,552 | 0,885 | 5,972 | <0,01 |
| Alpha amplitude before EA | 30,727 | 26,204 | 9,479 | 10,539 | 1,865 | <0,05 |
| Alpha amplitude immediately after EA | 33,720 | 27,636 | 10,138 | 9,152 | 2,634 | <0,01 |
| Alpha amplitude the morning after EA | 31,973 | 26,336 | 11,321 | 10,823 | 2,122 | <0,05 |
| Alpha index the morning after EA | 2,250 | 2,050 | 0,331 | 0,534 | 1,921 | <0,05 |
| Abnormality rating immediately after EA | 0,875 | 0,400 | 0,612 | 0,614 | 3,219 | <0,01 |
| Change in alpha frequency from before to immediately after EA | -0,336 | -0,688 | 0,495 | 0,586 | 2,672 | <0,01 |
| Change in alpha frequency from before to the morning after EA | 0,148 | -0,325 | 0,511 | 0,603 | 3,491 | <0,01 |
| Change in abnormality rating from before to immediately after EA | 0,547 | 0,012 | 0,700 | 0,754 | 3,041 | <0,01 |

Table VI

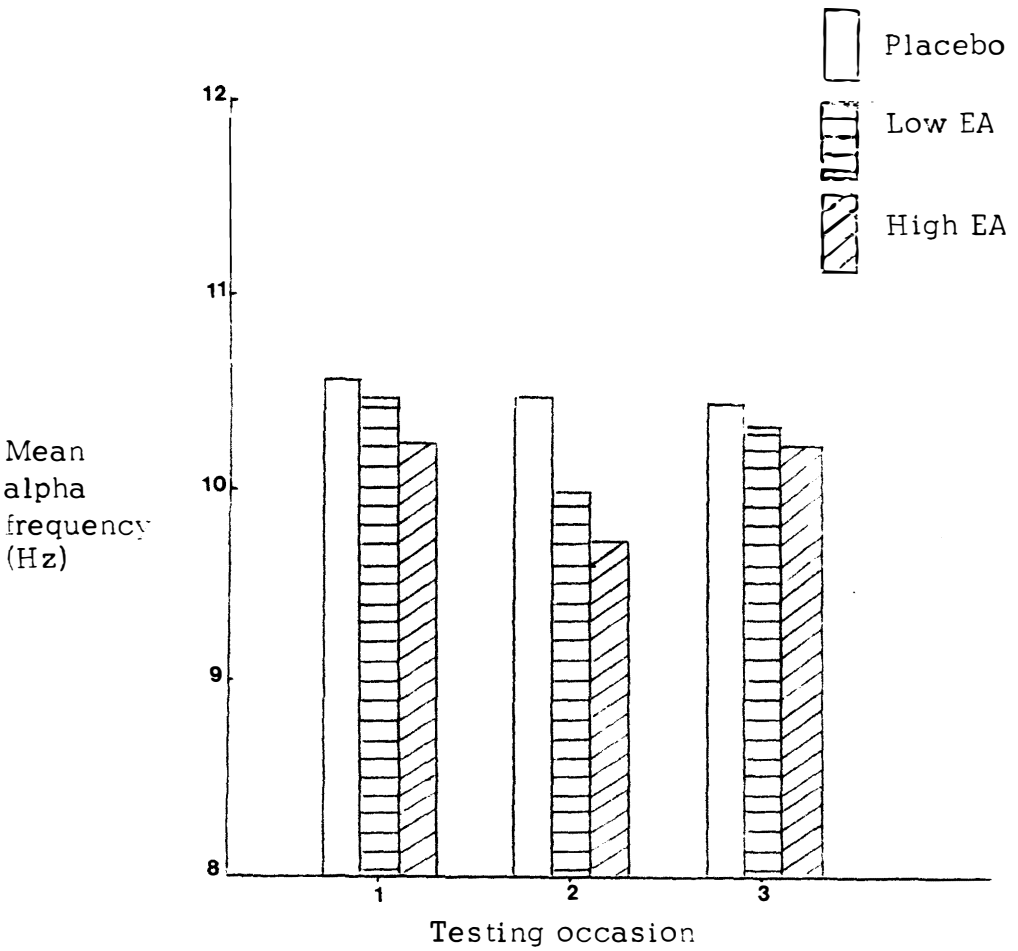
Significant EEG differences between high and low EA groups of the high (N = 40) and low (N = 32) alpha frequency groups

| | | Mean | | SD | | | |
|---|---|---|---|---|---|---|---|
| Group | Measure | Low EA group | High EA group | Low EA group | High EA group | t | p |
| High alpha frequency | Alpha frequency immediately after EA | 10,602 | 10,111 | 0,741 | 0,843 | 1,91 | <0,05 |
| Low alpha frequency | Alpha amplitude immediately after EA | 36,571 | 30,49 | 8,947 | 10,436 | 1,718 | <0,05 |
| Low alpha frequency | Alpha index immediately after EA | 2,441 | 2,10 | 0,161 | 0,374 | 3,165 | <0,01 |
| Low alpha frequency | Alpha index the morning after EA | 2,353 | 2,133 | 0,285 | 0,34 | 1,924 | <0,05 |



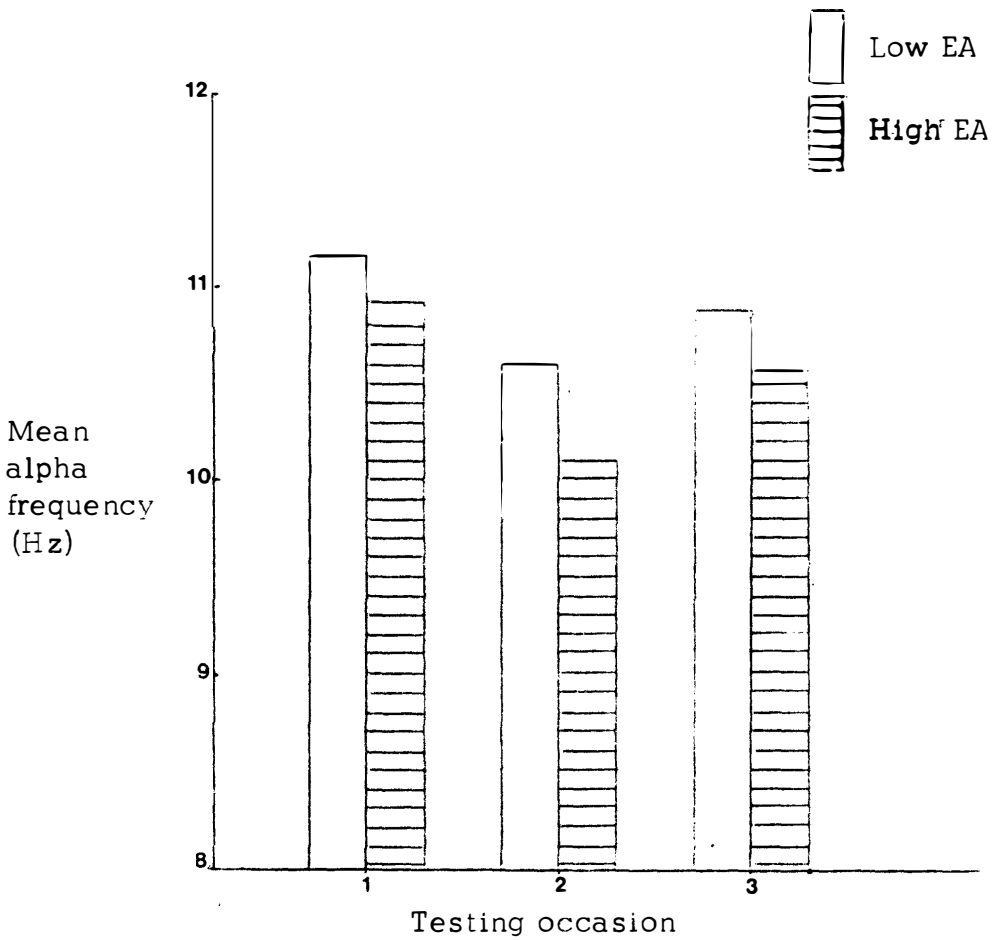

1 Before treatment
2 Immediately after treatment
3 Morning after treatment

Figure 1 : Mean alpha frequencies EA and placebo groups



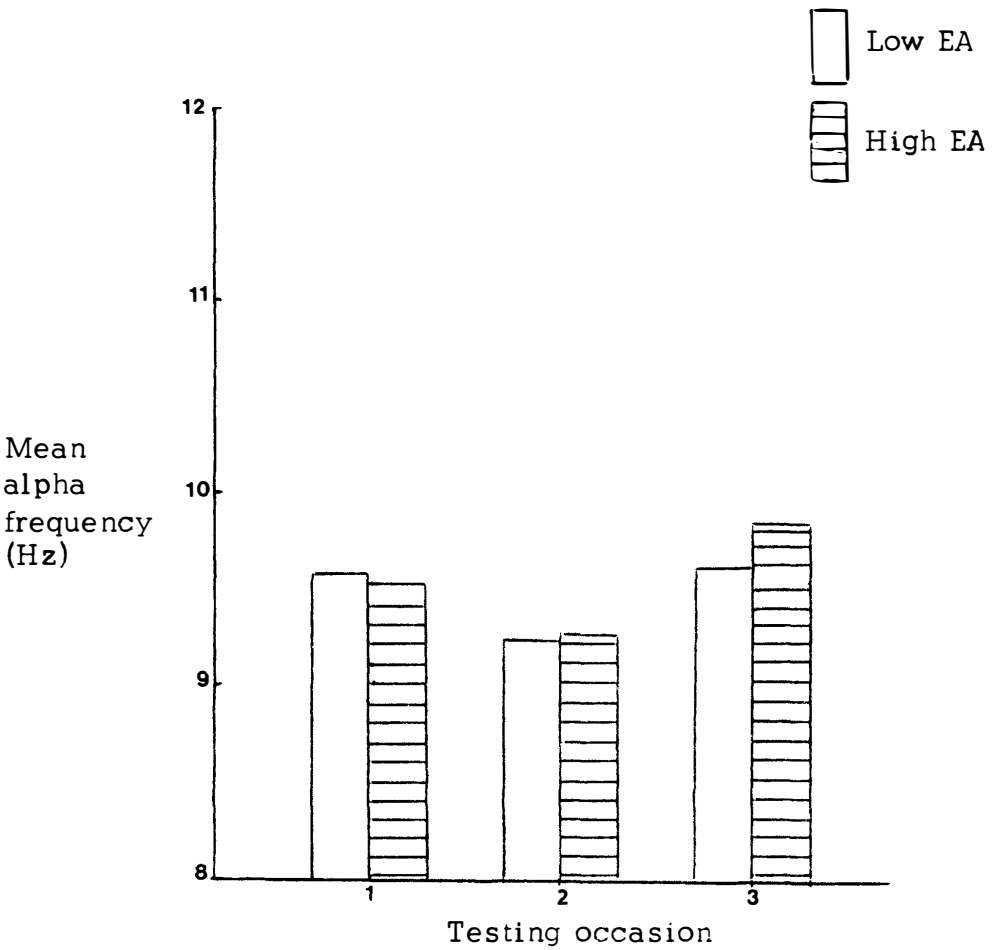

1 Before treatment
2 Immediately after treatment
3 Morning after treatment

Figure 2 : Placebo, high EA and low EA groups : mean alpha frequencies




1 Before treatment
2 Immediately after treatment
3 Morning after treatment

Figure 3 : High and low EA subgroups of the high alpha subjects of the EA group : mean alpha frequencies




1 Before treatment
2 Immediately after treatment
3 Morning after treatment

Figure 4 : High and low EA subgroups of the low alpha subjects of the EA group : mean alpha frequencies

## DISCUSSION

### 1) Placebo and EA group differences

The results of previous investigations suggesting that EEG frequency and amplitude measures, and to a lesser extent ratings of abnormality, as reported by Nelson[6] (1969), show changes after EA, are confirmed by the present study. The immediate effects of EA are reflected in a decrease in alpha frequency (Figure 1) and an increase in alpha amplitude (Table I). It is of interest, also, that whereas the placebo group tended to show little difference in the incidence of EEGs displaying no change in abnormality rating before and immediately after administration of the placebo, the EA group had higher incidences of individuals whose EEGs were rated more and less abnormal after the ingestion of EA (Table VI). It is further of interest that a definite "hangover" effect is apparent in EEG measures (Figure 1). The difference in change in both alpha frequency and amplitude measures from immediately after to the morning after EA treatment is significantly different for EA and placebo groups (Table I). In addition, the average abnormality rating of the EA group decreases slightly, while that of the placebo group shows very little change, from before to the morning after treatment (Table I).

### 2) High and low EA and placebo group differences

In the Republic, the legal BAC below which driving is permitted is 0,08 mg per 100ml. The EA group was therefore divided into high and low EA subgroups about this limit in order to determine whether it corresponded to any physiological reality. Figure 2 and Tables III and IV suggest that this is, in fact, not the case, as although placebo and either high or low EA group differences in frequency and index exist before and immediately after treatment, there are no significant differences between high and low EA subgroups. As before, placebo and high or low EA subgroup differences in alpha frequency and amplitude changes from immediately after to the morning after treatment are apparent (Figure 2 and Table III). It is worthy of mention that the low alcohol subgroup

appears to be more responsible for the previously-noted effect of a greater difference in incidence of individuals who showed a change in abnormality rating (in both positive and negative directions) after EA than the high EA subgroup. This latter group did, however, show a tendency for more individuals to be rated more abnormal after EA than the placebo group (Table IV).

3) High and low alpha group differences

Table V indicates that groups selected on the basis of high and low pre-EA alpha frequencies show alpha frequency differences immediately after and the morning after EA, and also differ in terms of alpha amplitudes before, immediately after and the morning after EA and in terms of alpha index and abnormality rating immediately after EA. The frequency and amplitude differences obtained are, to some extent, predictable, as frequency and amplitude are inversely related to one another. It is surprising, however, that the low alpha frequency group is significantly more abnormal than the high alpha frequency group after EA, and also shows a significantly greater increase in mean abnormality rating from before to immediately after EA than the high alpha group. This may indicate that the low alpha frequency group possesses a less stable EEG than the high alpha group. Further, Table VI shows that the high alpha group shows a significantly higher decline in alpha frequency after EA (Figure 3) than the low alpha frequency group (Figure 4). This may reflect the operation of the law of initial value (Hord et al[7] 1964). Again, it is of interest that a "hangover" effect is apparent, and affects both high and low alpha frequency groups. Table VI indicates that high and low EA effects are operative within high and low alpha frequency groups. Alpha frequency appears to be the most effective indicator of differences in reaction to EA of the high alpha group, (Figure 3), while amplitude and index differentiate best between the effects of different amounts of EA in the low alpha frequency group.

## CONCLUSIONS

In common with the results of previous investigations, alpha frequency changes appear to be the most sensitive of the EEG indicators of reaction to EA. Other EEG measures showing a response to the effects of EA were amplitude and index measures, and ratings of abnormality. The effects of EA on these measures, and particularly on mean alpha frequency, are apparent at some distance in time (about 12 hours) after the administration of EA. The effect of the legal maximum BAC of 0,08 mg % in South Africa is not marked in relation to EEG measures. Previous suggestions of a differential response to EA of high and low alpha frequency individuals are upheld.

## RECOMMENDATIONS

1) The findings of this part of the investigation suggest a persistant but very mild depressant effect of EA on the EEG on the morning after drinking. The possible significance of this effect might emerge on completion of the analysis of the psychological and psychomotor tests applied to the same sample by the Psychometrics Division of NIPR. No recommendation for further research on the possible "hangover" effect is therefore made at this stage.

2) Attention is however again focussed on individual differences in response to EA. Controlled neuropsychological investigations of individual variations, especially in relation to maximum BAC prescribed by law, are recommended.

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