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Title and Subtitle: INVESTIGATION OF THE "HANGOVER" EFFECTS OF AN ACUTE DOSE OF ALCOHOL ON PSYCHOMOTOR PERFORMANCE

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Abstract:

This report presents a review of the literature and reports the results of an investigation of the acute and hangover effects of ethanol on driving related skills, as well as focussing on 'event-related potential' data recorded in a sub-group of subjects participating in the project. An earlier study had noted an impairment of a relatively simple reaction time task 3 hours after a dose of alcohol, and the current study was attempting to determine whether this effect would be observed in the more typical 'morning after' situation. There were no statistically significant linear or higher order trends in the dose/response relationship during the hangover session in any of the tasks employed in this study. However this finding does not preclude the existence of a subjective 'hangover' effect nor that performance on other tasks is affected by hangover, for reasons which are discussed in the report.

Keywords: HANGOVER ALCOHOL EVENT-RELATED POTENTIAL RESIDUAL EFFECTS

FINAL REPORT OF RESULTS FROM CONSULTANCY 89/175

INVESTIGATION OF THE "HANGOVER" EFFECTS OF AN ACUTE DOSE OF ALCOHOL ON PSYCHOMOTOR PERFORMANCE

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EXECUTIVE SUMMARY

This study is presented in two parts, the first part reviewing the literature and reporting the results of the investigation of the acute and hangover effects of ethanol on driving-related skills, while the second part focusses on the literature and the event-related potential data recorded in a sub-group of the subjects participating in the project. The decision to present the findings in two separate sections of the report was made for a number of reasons. Firstly, electroencephalographic (EEG) recordings were obtained for approximately half of the subjects performing the behavioural tests, and therefore the number of subjects is considerably reduced in the analysis of the event-related potential data. Secondly, the behavioural data relevant to the interpretation of the event-related potential data is restricted to the task during which the recordings were obtained, namely the Mackworth Clock. Therefore, the behavioural data from this task is analysed in Part 2 for the sub-group of subjects taking part in the EEG recording phase of the experiment, whereas the behavioural data from the complete sample is analysed for all tasks included in the test battery in Part 1. Where behavioural outcomes are considered in Part 2 of the report, equivalent analyses are reported in Part 1 for the entire sample to provide comparability. Thirdly, the literature reviewed in Part 2 includes reports on the effects of ethanol on the event-related potential, which is of primary relevance to Part 2, whereas the literature reviewed in Part 1 includes reports on the hangover effects of ethanol on driving-related skills which is of primary relevance to the data presented in this section of the report. Finally, different conventions exist in the two areas for the collection, manipulation and analysis of experimental data, and this format allows uniformity of reporting within each area of interest.

Part 1 - Hangover and driving related skills

While ethanol induced hangover is a common phenomenon, the study of its effects upon human performance has found few consistent effects. Typically, performance on more complex and difficult tasks, and those which are less well

learned, are negatively affected for a short time after an acute dose of alcohol has been cleared from the system. However, Dauncey, (1989) noted an impairment of a relatively simple reaction time task three hours after a dose of alcohol. Performance at this time was at the same level as at the peak BAC, although the BAC was at or near zero. Additionally, the reported level of drinking was found to covary with the size of the effect, heavier drinkers exhibiting a greater degree of impairment. The present study used a replication of the original task in an attempt to determine whether this effect would be observed in the more typical "morning after" testing situation, as well as testing visuomotor coordination (divided attention) and vigilance (Mackworth clock) with tests known to be sensitive to the acute effects of alcohol.

A linear dose/response relationship was observed for the simple reaction time and divided attention in the acute test session. The other two tasks showed a similar pattern of means, with performance decreasing as dose of alcohol increased, but these results were not statistically significant. There were no statistically significant linear or higher-order trends in the dose/response relationship during the hangover session in any of the tasks employed in this study.

The results are discussed in relation to current hypotheses of hangover, and the differences in method between Dauncey (1989) and the present study.

Part 2 - Hangover and event-related potentials

A linear dose/response relationship was observed during the acute session on the components of the event-related potential which reflect the time taken to evaluate a stimulus, with a corresponding increase in reaction time. Stimulus evaluation time was not significantly delayed on the morning after ingestion of alcohol. However, there was a linear dose/response relationship on a different component of the event-related potential suggesting an increased difficulty to selectively attend to a location in space on the following morning, which may be related to fatigue. There were no overt performance measures which paralleled

the event-related potential results on the following morning in the present study.

PART 1

THE ACUTE AND HANGOVER EFFECTS OF ETHANOL ON DRIVING RELATED SKILLS

INTRODUCTION

Hangover is a term commonly used to denote the adverse after effects of consuming alcohol, especially after doing so to excess. Within this report, "hangover" will also be used to indicate alterations in perception, cognition and performance due to the prior consumption of ethanol, as indicated by the tasks which were used to test performance and the measures taken of electroencephalographic (EEG) activity.

Although the symptoms of "hangover" were described over 2000 years ago and case reports of the hangover phenomenon are common in modern folk wisdom, there has been surprisingly little documented research on the topic. Hangover occurs when a raised blood alcohol concentration begins to decline and increases in intensity as it approaches zero (Ylikahri, Huttenen, Eriksson & Nikkilä, 1974). Under these conditions, the drinker may experience a variety of symptoms which are aversive in nature and which may last for several hours. These symptoms are more severe in drinkers who have become dependent upon alcohol. The symptoms include: headache, fatigue, sweating, disturbances of balance and gait, pallor, tremor, nystagmus, nausea and vomiting, general malaise and disturbances of mood, with anxiety and depression. With increasing levels of dependence, emotional suffering and withdrawal symptoms also occur and can include cardiac disturbances, hallucinations, sleeplessness, severe depression and delirium. Under these conditions, feelings of psychological distress become predominant (Chapman, 1970).

Numerous pathophysiological explanations of hangover have been postulated. Some of these include: overactivity of the vestibular system (the system which regulates balance or equilibrium); accumulation of acetaldehyde, a toxic metabolite of alcohol; potassium retention; lactacidaemia; disturbed fluid balance; gastrointestinal irritation; hypoglycaemia; dilatation of intra- and extra-cranial blood vessels; and disturbed sleep regulatory mechanisms (Chapman, 1970).

Studies of the effect of hangover on driving related skills

Although there have been many investigations of the effects of alcohol on human performance, the majority of these have focussed on the acute effects of alcohol when the BAC is at its peak after consumption (Mitchell, 1985). Few researchers have investigated the effects of alcohol on performance over extended periods of time. Ekman, Frankenhaeuser, Goldberg, Hagdahl and Myrsten (1964) conducted a study of the subjective and objective effects of alcohol as a function of dosage and time. They repeatedly tested subjects at intervals between 10 and 290 minutes after consumption of 0.33, 0.44, and 0.66 g/kg doses of alcohol taken as whiskey. As in most studies of this type, subjects served as their own controls. Different dosage conditions were counterbalanced and separated by a minimum of 1 week. Peak performance decrement was observed in an arithmetic test 30-40 minutes after consumption but returned to control levels within 100 minutes. Self- and other-ratings of intoxication peaked between 30 and 60 minutes. These experimenters noted that subjective estimates of intoxication were much more affected than objective ones.

In a similar study by Idestrom and Cadenius (1967), performance on a variety of psychomotor and perceptual tasks (reaction time, tapping speed, coordination, critical fusion frequency, Rombourg and Bourdon tests) was monitored over 3 hours and measured again 13 hours after consumption of 0.0, 0.2, 0.4, 0.6, and 0.8 g/kg of alcohol. The highest dose impaired performance on all tasks except the Critical Fusion Frequency Test and showed its peak effect at 30-60 minutes after consumption. After 2 hours, performance decrement was very slight and there were no measurable effects of alcohol on performance at the 13 hour measurement time.

A number of studies have been specifically designed to examine the hangover effects of ethanol in a variety of laboratory or other controlled situations. These have generally had two principal aims: 1) to determine whether measurable decrements in performance occur after complete or partial clearance of ethanol, which can be attributed to a hangover effects, and 2) to

discover a mechanism which might cause such decrements. One possibility is that the dysphoria of a hangover itself is sufficiently debilitating to reduce performance. If this is the case, performance should be negatively related to the intensity of the hangover, and presumably to subjective reports of this. Another is that neural or humoural effects of the ethanol persist beyond its presence in the system, and directly alter the ability to perceive, process and respond to, the relevant stimuli in the tasks affected (see Ylikahri, Leino, Huttonen, Pōsō, Eriksson & Nikkilä, 1976). In this case, a biochemical marker of these effects might be identified which would show a relationship to performance.

Karvinen, Mettinen and Ahlman (1961) found that about one half of their male subjects were unable to perform as much physical work the morning after drinking between 1 and 2.4 g/kg of ethanol. At this level of consumption, approximately one third of the subjects reported severe discomfort from the hangover. However, a moderate reduction in the maximal amount of effort of which the individual is capable is unlikely to be a major factor in motor vehicle driving performance.

Coordination and speed of response would seem more crucial to driving performance. Takala, Siro and Toivainen (1958) required male subjects to perform a battery of spatial, numerical, perceptual and motor tests while sober, and 12.5 hours after drinking. Two "hangover" test sessions were conducted, one after drinking beer, and the other after brandy. While no consistent pattern of deterioration of performance was observed, the authors surmised that only difficult performance tasks, and those involving "higher intellectual functions" would be affected by the hangover induced by a dose of 1.4 g/kg of ethanol. Similarly, Seppälä, Leino, Linnoila, Huttunen and Ylikahri (1976), measuring performance 10, 12 and 14 hours after a 1.75 g/kg dose of ethanol, found that only the number of mistakes on a choice reaction time task was elevated. Performance on coordination and attention tasks was unaffected. They also measured blood acetaldehyde and glucose levels in an attempt to

discover biochemical correlates of hangover, but found no relation of these to the perceived intensity of the hangover. Performance was not related to hangover intensity in this study, but Myrsten, Rydberg, Ideström and Lamble (1980) did find that deterioration in performance was related to reported intensity of hangover. These decrements in performance were on an arithmetic task, and the Bourdon Test (a paper and pencil checking task) 14 hours after a 1.43 g/kg dose of ethanol.

Obviously the most appropriate task for assessing driving related performance is automobile driving itself. Laurell and Törnros (cited in Franck, 1983) reported a test on a closed course driving task when the BAC of subjects had dropped to 0 on the day after an experimental "party" which ended at midnight. The driving maneuvers were quite difficult, and the pay which the subjects received for participation was reduced according to the number of errors made. Significant reductions in performance were observed both at the first 0 BAC reading, and three hours thereafter. In this study, no relationship was found between reported severity of hangover and performance.

Within the aviation industry, regulations state that pilots may not operate an aircraft within a specified number of hours of consuming alcohol. These limits have been established on the basis of limited research and vary considerably (from 8 to 24 hours (Yesavage & Leirer, 1986)). The industry has been particularly interested in the potential long-lasting effects of alcohol and several studies have been conducted on the psychomotor performance of pilots the morning after consuming high doses of alcohol (Collins, 1980; Collins & Chiles, 1980; Yesavage & Leirer, 1986). Of the three studies cited, only one found evidence of impairment of performance 14 hours after consuming enough alcohol to produce a peak BAC of at least 0.10 g% (Yesavage & Leirer, 1986). The task involved a simulated flight where pilots were required to perform two flight emergency maneuvers. Their performance was compared with that shown during a control condition where no alcohol was consumed for 48 h prior to testing. Under the hangover condition, pilot performance was worse on most

measures but significantly worse on three of six variance measures and one of six performance measures. The variance in performance increased significantly in the hangover condition for heading errors during takeoff and landing and in vertical distance from the glidescope during landing. There was also an increase in average yaw (rotation about the vertical axis) during takeoff.

In a study by Collins (1980), pilots performed a two-dimensional tracking task under static (stationary) and dynamic (during angular acceleration) conditions at ground level and simulated altitude of 12,000 feet. The peak BAC reached at midnight averaged 0.091 g%. Testing at this time revealed performance deficits under alcohol for tracking and visual reaction time. In the morning, however, there were no significant differences between subjects' performance when they had drunk alcohol 8 hours before and when they had not. The average BAC of subjects when they had drunk alcohol the night before was 0.012 g% prior to testing in the morning. Overall performance during the static condition was better than during the dynamic condition and tracking showed a circadian effect of improvement in the morning. There were no significant interaction effects of alcohol and altitude. Although subjects rated the degree of hangover higher and mood as poorer in the morning after alcohol, performance on the tasks was not affected. The subjects in this study were motivated to perform well and under all test conditions rated their effort as consistently high. It is possible that subjects overcame potential detrimental effects of hangover through effort.

In a study by Collins and Chiles (1980), pilots were repeatedly tested on a Multiple Test Performance Battery and a tracking task (static and dynamic conditions) before drinking, at midnight, after drinking, and 8 hours later. On the evening when alcohol was drunk, sufficient vodka or bourbon was consumed to achieve an average BAC of 0.093 g%. Eight hours later BACs had declined to 0.007 g% for the vodka drinkers and 0.005 g% for the bourbon drinkers. Compared with placebo and sleep control conditions, alcohol impaired performance acutely during the midnight test but had no adverse effects on

performance when subjects were tested in the morning. As in the previous study, subjects rated the magnitude of hangover, anxiety, fatigue and sleepiness highest and vigour lowest in the morning after drinking. Yet there was no measurable impairment of performance.

Overall, the experimental analysis of performance during hangover leads to some tentative conclusions. Hangover does appear to have a detrimental effect on performance, although this effect is usually observed only on the more difficult tasks, whether these test visuomotor or cognitive skills. Most studies reviewed here failed to find a relationship between the reported intensity of the hangover and performance. While biochemical changes reliably accompany the absorption and metabolism of ethanol (Myrsten *et al.*, 1980; Ylikhari *et al.*, 1976), no reliable relationship between these changes and performance has emerged.

Possible effects of hangover on driving

Restrictions on the use of alcohol in certain situations (e.g. automobile driving) are considered necessary due to its strong association with road accidents. Therefore, public education campaigns and legal penalties have been introduced to minimise the adverse effects of alcohol consumption upon road safety. There is obviously an assumption that the acute effects of alcohol dissipate as the substance is removed from the body, and that an individual who has "sobered up" is essentially no different from one who has not been intoxicated.

Detailed analysis of the results of a study concerned with the effects of alcohol and cannabis upon performance (Chesher, Dauncey, Crawford & Horn, 1986; Dauncey, 1989) has shown that one of their measures, simple reaction time, remained increased three hours after consumption of alcohol. Since the alcohol had largely been cleared by that time, it may be argued that such a result was due to a longer lasting effect of alcohol which persists beyond the metabolic cycle of the substance.

If this is a reliable effect of alcohol, reassessment of the duration of this effect of is required. As Dauncey (1989) report that the increase in reaction time was no different from that recorded at about the time of peak BAC, it is clear that this would alter thinking about the appropriate restrictions on driving for those consuming alcohol. While it is not immediately clear how this might be administered, the observed effect certainly deserves further investigation.

To this end, a study was designed in an attempt to both replicate the original finding, and to extend its validity. Mitchell (1985), in a review of the effects of alcohol upon performance, has noted that the more complex and highly integrated skills are those which tend to suffer the greatest decrements in performance after the administration of alcohol. Since simple reaction time is a task which is typically only affected at relatively high doses, the inclusion of a more complex task would allow comparison with performance that is thought to be more sensitive to the effects of alcohol. Finally, the duration of the testing sessions used by Dauncey (1989) was considerable, and some effect of fatigue or boredom might have differentially affected those subjects who consumed alcohol. The centrally depressive effects of alcohol are well known, and repetitive tasks with minimal levels of stimulation are particularly likely to lead to lapses in concentration, or even sleep, in subjects under its influence. A test which might assess the extent to which this may have contributed to the observed result would be useful.

The simple reaction time task used in the Dauncey (1989) study was recreated for this replication. The task consists of a repeated stimulus (an X) which appears in a rectangle centred on the visual display unit (VDU) of a microcomputer. The subject is required to press a button as quickly as they are able whenever the stimulus appears. The original test consisted of two types of trials, those in which the stimuli appeared at regular intervals, and others in which the interstimulus intervals were irregular.

The divided attention task of the Rozelle Test Battery (Lemon, 1990) is quite

sensitive to the effects of alcohol, and is able to detect the effect of moderate doses of alcohol with high reliability. Such a test has three advantages in this situation. First, the short duration of the test ensures that boredom or fatigue is unlikely to contaminate the results. Second, the sensitivity of the test to the acute effects of alcohol provides a standard of comparison for the visuomotor components of the other tests. Finally, it may indicate whether skills other than simple reaction time are affected after alcohol has been cleared from the system.

The Mackworth clock task (Mackworth, 1948) is recognised as a standard test of vigilance. Its long duration and minimal stimulation severely test the ability of the subject to maintain concentration. Any influence due to the soporific effects of alcohol should be apparent in the results provided by this test.

Dauncey (1989) also noted that an interaction between performance and the subject's history of alcohol consumption was apparent. Subjects reporting a current higher consumption level were more impaired on this task. Since this is the opposite of what would be expected from the known development of tolerance to alcohol, it is essential to assess the drinking history of the subjects to ensure that they fall within the range reported by the original subjects, and exhibit the same relationship with performance.

METHOD

Subjects

Sixty-four healthy male subjects were recruited from the nearby University and from the local community through posters and newspaper advertisements. The mean age was 24.6 (±6.6) years with a range of 18 to 53 years. Volunteers were not included in the study if they: (a) used other recreational drugs regularly (more than once/month) or were currently taking medication or being treated for a medical condition; (b) had a history of liver or kidney problems; (c) had previously sought help for alcohol problems; (d) had a history of psychiatric illness; (e) had never consumed a dose of alcohol equivalent to 7-8 standard drinks in one session (i.e. in approximately 3 hours), which is equivalent to the highest dose employed in this study; (f) had a history of epilepsy.

Instruments

Drug and Alcohol History - Several questionnaires were used to obtain drug and alcohol history information. The Lifetime Drinking History (Skinner, 1979) was used to obtain quantity/frequency measures for each of the different phases of alcohol consumption through which the individual passes, as well as other information such as relative proportions of the types of beverages consumed. These data were used in the calculation of a Lifetime Alcohol Consumption Measure in litres of pure alcohol.

The Last 30 Days of Drinking (Hesselbrock *et al.*, 1983) questionnaire examines in detail the recent drinking patterns of the individual, including minimum, maximum and average levels of consumption and frequency of each level, with respect to each of beer, spirits and wine.

These two questionnaires were individually administered through interview, whereas all other questionnaires were self-administered under the supervision of the experimenters.

The AUDIT (Saunders & Aasland, 1987), a World Health Organisation

Screening Instrument, was used to detect the presence of alcohol-related problems, such as injuries, health warnings, and subjective feelings of guilt/remorse or impaired control over drinking behaviour.

A modified version of the Recent Drug and Alcohol Use questionnaire was used to obtain a measure of alcohol consumption over the past week, and to screen for other recent drug use.

Sleep - A modification of the University of Sydney and the Department of Motor Transport and Road Safety Sleep Questionnaire, was used to assess typical sleep patterns and any associated problems. Also, a Sleep Log Diary was given to subjects to take home so that the number of hours slept each night and headaches or fatigue on awakening could be recorded over the course of the experiment, as well as an estimate of the typical number of hours slept.

Computerised Behavioural Measures

All computerised measures of psychomotor skills performance, excepting the Simple Reaction Time task, were taken from the Rozelle Test Battery (Lemon, 1990) and were presented on the visual display unit (VDU) of an Apple IIe microcomputer with attached response board.

Rozelle Divided Attention Task (2.5 minutes)

This dual task requires a combination of visual tracking and peripheral discrimination, and assesses the ability to perform simultaneously two effortful tasks, both of which require focussed concentration and attention. This task appears to be relevant to some of the skills used when driving and has previously been shown to be sensitive to an acute dose of alcohol, (Lemon, Mascord & Starmer, submitted for publication). In this task the subject is required to maintain a small box inside a pair of parallel vertical lines which move irregularly from side to side across the screen, using a steering wheel control. The error rate of the subject is monitored by the program, and the speed with which the lines move is adjusted to maintain an error rate of about

one error every 10 seconds. One measure of the task is the average speed of the display over the final 100 seconds of the test. Whilst performing this task, the subject must also watch for a target stimulus which appears periodically in one of four small (15 mm diameter) circles situated one at each corner of the screen, and must respond as quickly as possible by pressing a key corresponding to the appropriate corner of the screen where the target has appeared. At all times, each circle contains a diagonal line, but the orientation of each line changes every few seconds. One particular orientation (up/right diagonal) is the target stimulus to which the subject must respond. Only one target occurs at any one time.

Simple Reaction Time Task (8 minutes)

In this task, the subject is simply required to press a button as quickly as possible in response to an X which periodically appears in a small box (about 30 mm square) which is continuously displayed in the centre of the screen. In one section of this task, the stimulus presentations are evenly spaced at a rate of about 1 per 2 seconds, ('regular'), whilst in the other section the Xs appear at random time intervals with an average interval of about 15 seconds, ('irregular').

The Mackworth Clock (40 minutes)

This task measures performance on a vigilance task over an extended period, and requires sustained attention and concentration (Mackworth, 1948). In this task, 24 dots are continuously displayed in a circle on a VDU (Apple IIe). A moving rectangle flashes briefly (100 ms duration) on each dot, circling clockwise. Occasionally, the rectangle skips a dot, and the subject must respond by pressing a button when a dot is skipped. This is designated the infrequent target stimulus. All other stimuli are referred to as the frequent stimuli. The interstimulus interval (ISI) between successive flashes is 500 ms. The task continues for a duration of 40 minutes with 60 targets randomly presented in a total of 2400 stimuli. This represents a considerably shorter average interval between targets than is typically used in this test.

Self-Ratings of Subjective States

At the end of each test session, subjects were asked to complete 10 self-ratings of mood and signs of intoxication. Each rating was made on a 10-point scale with two anchor points (0 indicating "Not at all" and 9 indicating "A lot") for ratings of lethargy, sociability, jitteriness, alertness, detachment, assertiveness and being hung over. The anchor points for the question "RIGHT NOW, how drunk do you feel?" were 0 - "Not at all" and 9 - "Most ever". For the question "Would you drive a car right now?", 0 indicated "Definitely no" and 9 "Definitely yes". When asked "How well could you drive right now?, a rating of 0 indicated "Very badly" and 9 indicated "Very well".

Procedure

Subjects were screened via a telephone questionnaire, which included questions on the quantity and frequency of alcohol consumption in a **typical** week and questions relating to the exclusion criteria outlined above.

Session 1 (Baseline)

On arrival at the National Drug and Alcohol Research Centre, at approximately 5.30pm, subjects were shown the testing apparatus and were informed of the nature of the experiment and the procedures, before giving written consent. Before signing the consent form subjects were advised that they would not be allowed to drive after session 2, when they were to receive alcohol, and that they were required to remain at the Centre on that evening until their BACs had returned to .05 mg/dl. A Drager Alcotest Breathalyser (Type 7110) was used to ensure a zero BAC before commencing the experiment. Subjects were asked to refrain from eating from 4:00pm because they would also have to do this for session 2 when they would be given alcohol. Subjects were weighed and randomly allocated to one of four dosage conditions before being administered the first three questionnaires: the Sleep Questionnaire, the Recent Drug and Alcohol Usage Questionnaire, and the AUDIT. Each subject was then individually administered the Last 30 Days of Drinking Questionnaire and the Lifetime Drinking History in a confidential interview.

Next, subjects were shown how the computerized behavioural tasks run. They were given a brief practice session in an attempt to bring them close to asymptotic performance before measurement commenced because there are notable practice effects associated with this type of behavioural task. Practice consisted of one full run through of the Divided Attention and Simple Reaction Time tasks, and a 10-minute shortened version of the Mackworth Clock.

Each subject was then fitted with an elasticised cap for recording the electroencephalograph (EEG). Although only one out of each pair of subjects had their EEG recorded - the other subject was also required to wear an elasticised cap to equate the conditions of testing for both subgroups. The full computerised test battery was then administered (all three tasks) to provide the sober baseline measure of performance. Once testing had been completed, subjects were given a meal and allowed to leave.

Session 2 (Acute Test)

Two days after baseline testing, subjects returned to the centre at approximately 7:00pm for testing under experimental conditions, according to their allocated group. After being breathalysed to ensure a zero BAC each subject was given the allocated dose of alcohol in two equal portions, each to be consumed steadily over fifteen minutes. Each aliquot consisted of pure alcohol (99.5%) diluted with orange juice in a ratio of 15% (v/v). The placebo dose, however, consisted of the same volume of orange juice as the .5 g/kg dose, with .5 ml pure alcohol floated on top just before serving.

The four dosage conditions, calculated for each subject according to bodyweight (kg) were: (i) 0 g alcohol/kg (placebo dose), (ii) .5 g alcohol/kg (3-4 standard drinks) (low dose), (iii) .75 g alcohol/kg (5-6 standard drinks) (medium dose), (iv) 1 g alcohol/kg (7-8 standard drinks) (high dose). These dosages were calculated to induce resultant BACs of approximately: 0.0, 0.05-0.07, 0.08-0.10, and 0.10-0.14 g/dl respectively. Subjects were instructed not to eat anything for at least three hours beforehand, in order to increase the likelihood of

comparable stomach contents and rapid absorption of the alcohol so that peak BAC would be reached during performance on the behavioural measures.

After drinking the allocated portions of alcohol, subjects waited ten minutes to allow some absorption of the alcohol before being breathalysed again. Results of all breathanalyses carried out after administration of the alcohol were not disclosed to subjects until completion of the experiment the next morning. It was ensured that mouths were rinsed with water prior to breathanalysis to prevent the possible inflation of the recording by residual alcohol in the saliva. During the alcohol administration period, the EEG cap was fitted and connected in preparation for recording.

Testing on the computerised battery began immediately after breathanalysis in order to provide the acute post-drug measure of performance. Following this 55 minute test session, subjects were once again breathalysed and provided with a meal and some quiet entertainment whilst waiting for their BACs to drop below .05 g/dl (the legal limit for driving a motor vehicle in the State of N.S.W.). Breathanalysis continued intermittently until a BAC below .05 g/dl was achieved (Note that the time taken for this is extremely variable, but that the subjects in the medium and high dose groups generally had to remain at the centre later than those in the placebo and low dose groups). Subjects were then driven home. They were picked up the next morning to facilitate their compliance with the requirement that they not drive an automobile until after the completion of testing the following morning.

Session 3 (Hangover Effects)

At approximately 7.30 am the following morning, subjects arrived at the Centre for the final testing session. After a breakfast of toast or cereal (no tea or coffee), subjects were breathalysed to ensure a zero BAC, fitted with the EEG equipment, and tested for the third time. On completion of the test battery, subjects were debriefed, thanked for their participation, and paid $50.

RESULTS

Analysis of Behavioural Data

Age, weight, measures of drinking history and sleep patterns were analysed for group differences by a multivariate analysis of variance. Separate analyses of covariance were carried out on the performance data from the various behavioural tests completed by subjects on sessions 2 (acute test) and session 3 (hangover test) with Dose (placebo, low, medium and high) as the between groups factor and performance on session 1 (baseline) as the covariate. This covariate analysis was used because individual differences in competence on these tasks can be great and can contribute disproportionately to the error variance in tests of statistical significance. Also, this was the analysis used in the Dauncey (1989) study which we have attempted to replicate and extend here.

Planned contrasts for linear trend were used to test the hypothesis that alcohol produces an acute dose-dependent decrement in performance on the Simple Reaction Time (regular) and Divided Attention tasks. A similar analysis was also used to test the hypothesis that a hangover effect of alcohol on simple reaction time performance is observed some 12 hours after alcohol consumption. When using the method of planned orthogonal polynomials decisionwise error rate was set at .05. Tests for higher order trends were *post hoc*, using the method of Scheffé. Analysis of covariance followed by *post hoc* trend analysis using the Scheffé method was also used to determine whether hangover effects of alcohol were observed in the divided attention and Mackworth Clock tasks. With the Scheffé method of *post hoc* trend analysis experimentwise error rate was controlled at the .05 level (Kirk, 1968).

Description of subjects

Tables 1 to 3 provide the overall and group means for age, weight, drinking history and sleep patterns of the subjects. The overall multivariate F-test for this analysis was statistically significant [$F(33,130)= 1.6$, $p<.05$]. Univariate F-

scores revealed that the groups differed significantly on number of years drinking alcohol [$F(3,54)=3.6$ $p<.05$] and hours slept the night before the final performance test [$F(3,54)=5.1$ $p<.01$]. On all other variables, the groups did not differ significantly from one another.

TABLE 1 - Mean age and weight for subjects in each dose group and overall.

Dose	Placebo $\bar{X}$ (±sd)	Low $\bar{X}$ (±sd)	Medium $\bar{X}$ (±sd)	High $\bar{X}$ (±sd)	Overall $\bar{X}$ (±sd)
Age (yrs)	26.0 (±8.9)	23.9 (±8.4)	23.6 (±4.3)	25.0 (±4.8)	24.6 (±6.6)
Weight (kg)	72.9 (±11.9)	72.5 (±8.5)	75.5 (±8.0)	75.4 (±7.5)	74.2 (±8.9)

TABLE 2 - Mean scores for each dose group and overall on the drinking history measures taken.

Dose	Placebo $\bar{X}$ (±sd)	Low $\bar{X}$ (±sd)	Medium $\bar{X}$ (±sd)	High $\bar{X}$ (±sd)	Overall $\bar{X}$ (±sd)
Weekly (drinks)	2.2 (±1.5)	2.1 (±2.0)	2.1 (±1.8)	2.3 (±2.2)	2.2 (±1.9)
Monthly (ml)	701 (±429)	922 (±638)	981 (±596)	960 (±672)	898 (±593)
Lifetime (l)	82.8 (±118.3)	34.6 (±36.0)	97.3 (±131.0)	62.9 (±51.2)	69.8 (±93.7)
Years drinking	7.6 (±6.1)	4.5 (±2.3)	7.3 (±4.6)	9.8 (±4.6)	7.4 (±4.9)

TABLE 3 - Mean scores on sleep questionnaires and mean number of hours reported slept for each dose group and overall.

Dose	Placebo $\bar{X}$ (±sd)	Low $\bar{X}$ (±sd)	Medium $\bar{X}$ (±sd)	High $\bar{X}$ (±sd)	Overall $\bar{X}$ (±sd)
Questionnaire	8.0 (±3.2)	10.6 (±3.6)	8.9 (±3.7)	8.4 (±3.5)	9.0 (±3.5)
Average sleep time	8.0 (±0.9)	8.0 (±1.0)	8.1 (±0.5)	7.6 (±0.9)	7.9 (±0.8)
Hours slept Night 1	7.7 (±0.9)	7.9 (±2.2)	8.6 (±1.8)	8.0 (±1.3)	8.1 (±1.6)
Hours slept Night 2	7.9 (±1.2)	7.8 (±1.8)	7.9 (±1.4)	7.8 (±1.2)	7.9 (±1.4)
Hours slept Night 3	6.8 (±0.9)	6.5 (±1.5)	7.0 (±0.9)	5.6 (±1.1)	6.5 (±1.2)

All subjects began each session with a BAC of 0.00 g/dl. Table 4 shows the BACs of subjects just before beginning the performance tasks on session 2 and immediately after completion of those tasks. It is apparent that all groups reached peak BAC during test performance.

TABLE 4. Mean BACs (mg/dl) before and after performance on the computerised tasks for each dose group.

	BAC g/dl $\bar{X}$ (±sd) Before	BAC g/dl $\bar{X}$ (±sd) After
Placebo	0	0
Low Dose	0.042 (±0.012)	0.032 (±0.011)
Medium Dose	0.065 (±0.012)	0.058 (±0.007)
High Dose	0.083 (±0.017)	0.082 (±0.009)

Performance measures with baseline performance as covariate

The initial analyses of performance during the acute (second) and hangover (third) sessions were planned to examine all polynomial trend relationships between dose and performance. Obviously, the trend of greatest interest was linear, that is, whether performance declined directly as a function of dose of ethanol. Using an α of .05 at 1 and 60 df, the critical F value for *a priori* contrasts is 4.0, and for *post hoc* contrasts, 8.3.

Simple Reaction Time (Regular)

Figure 1: Mean reaction time (ms) adjusted for baseline performance on session 1 for all subjects tested on the simple reaction time task when regular interstimulus intervals were used. Performance on sessions 2 and 3 is presented for each dose condition.

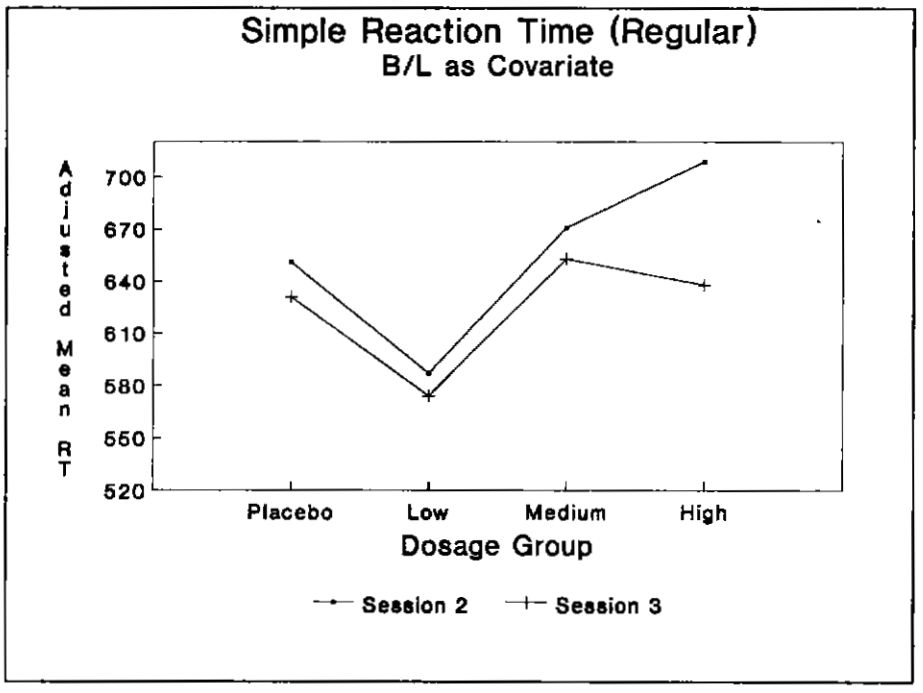

Figure 1 shows the results for sessions 2 and 3 from the simple reaction time task when a regular interstimulus interval was used. The results are presented as mean reaction times adjusted for baseline performance level. (The unadjusted mean scores for all performance data are presented in Tables 1A to 3A in Appendix 1.) The reaction times shown by groups given increasing doses of alcohol on session 2 was best described by a linear trend. *A priori* tests

revealed a statistically significant linear trend (F[1,60] = 4.5), and nonsignificant quadratic (F[1,60] = 3.1] and cubic (F[1,60] = 2.2) trends in the data. Thus, as dose of alcohol increased there was an increase in reaction time.

On session 3 (hangover test), the low dose of alcohol appeared to produce an improvement in reaction time on session 3 while the medium and high doses produced effects similar to that seen in the placebo dose group. The test for linear trend was nonsignificant (F[1,60] = 1.1), as were the *post hoc* tests for quadratic and cubic trend (All Fs < 6.0).

Simple Reaction Time (Irregular)

Figure 2: Mean reaction time (ms) adjusted for baseline performance on session 1 on the simple reaction time task when irregular interstimulus intervals were used. Performance on sessions 2 and 3 is presented for each dose condition.

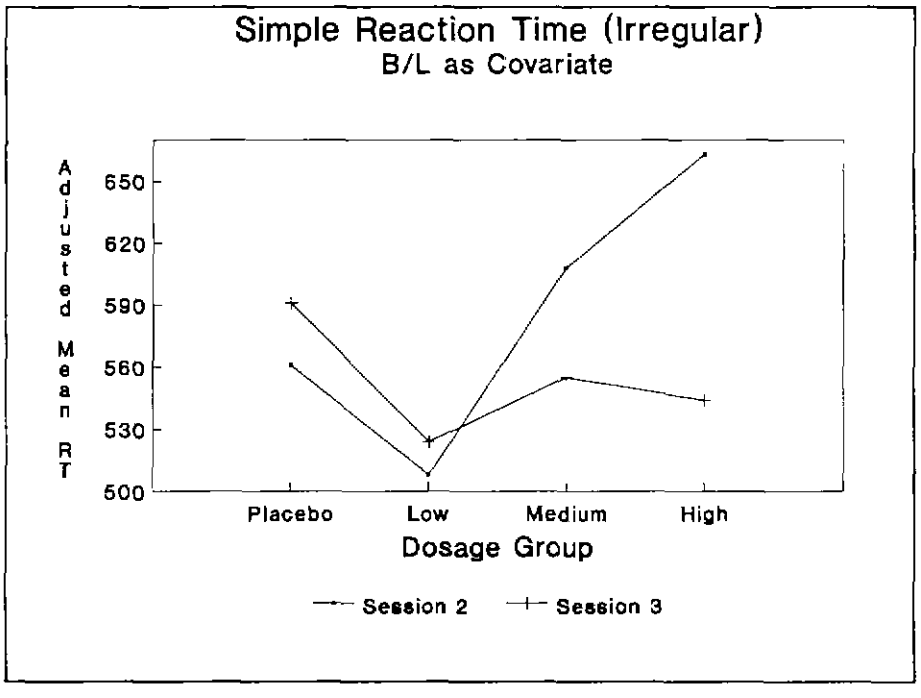

Figure 2 shows the adjusted means from the simple reaction time task when irregular interstimulus intervals were used on sessions 2 and 3. The pattern of reaction times shown on session 2 (acute test) approximated a linear trend increasing across dose. The test for linear trend across the increasing doses of

alcohol was marginally significant, just beyond the .05 α level (F[1,60] = 3.87). The tests for quadratic and cubic trend were both nonsignificant (All Fs < 2.0). There was no indication of a hangover effect of alcohol on performance on session 3. Tests for linear, quadratic and cubic trends were all statistically nonsignificant (All Fs < 1.0).

Divided Attention

Figure 3: Mean score adjusted for baseline performance on the divided attention task. Performance on sessions 2 and 3 is shown for each dose condition.

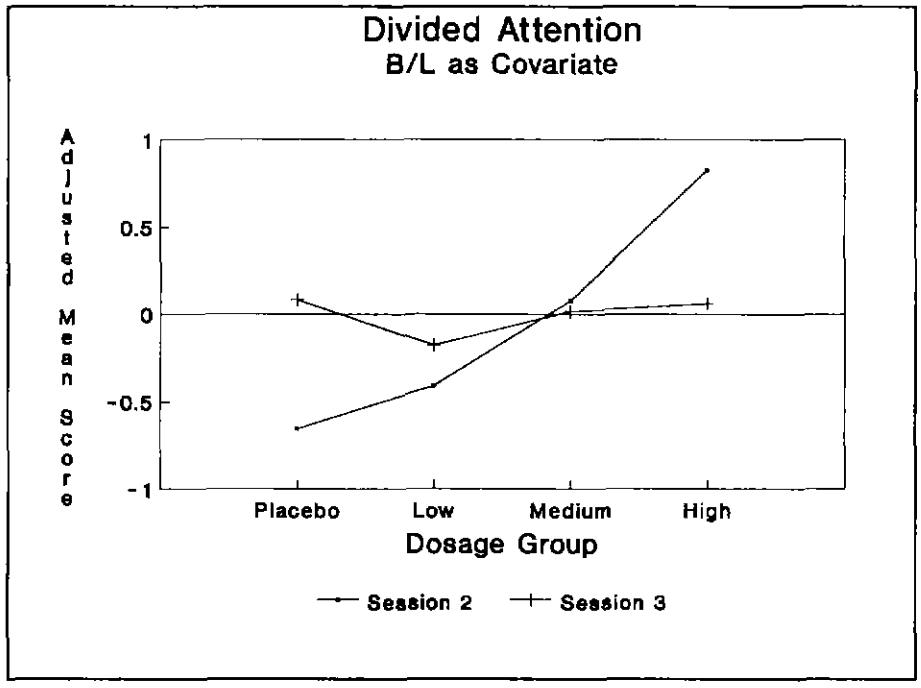

Figure 3 shows the results for sessions 2 and 3 for performance on the divided attention task. Performance on this task is reflected in a composite score comprising the terminal speed achieved during tracking and reaction time to respond to target stimuli presented in the peripheral visual field. The mean composite scores presented in the graph are adjusted for baseline performance. An acute dose-dependent impairment in performance was observed in this task on session 2. *A priori* polynomial contrasts revealed a significant linear trend (F[1,61] = 15.7) with the quadratic and cubic trends not reaching statistical significance (All Fs < 1.0). As the results from session 3 show, there was no

evidence of a hangover effect of alcohol on this task. With familywise error rate set at .05 and 3 and 61 df, the critical F value for *post hoc* tests for trend was 8.3. None of the tests for linear, quadratic or cubic trend was significant ($F < 1.0$).

Mackworth Clock - Misses

Figure 4: Mean number of misses adjusted for baseline performance on session 1 on the Mackworth clock task. Performance on sessions 2 and 3 is shown for each dose condition.

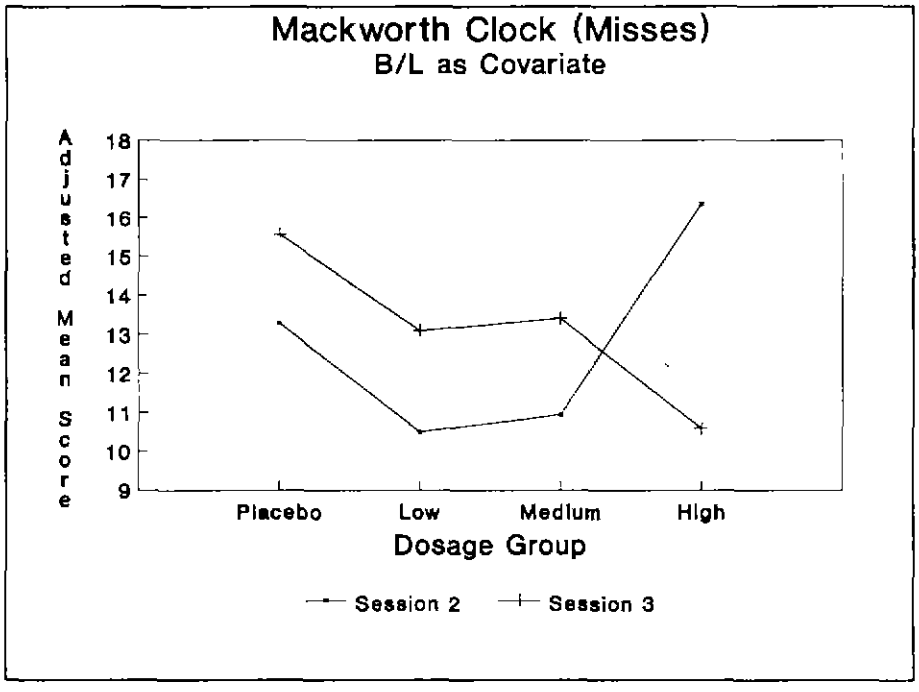

Figure 4 shows the adjusted mean scores for sessions 2 and 3 for the number of misses as a measure of performance on the Mackworth clock task. Although there appears to be a quadratic trend in the data on session 2 (acute test), this did not prove to be statistically significant. There was also no evidence of a hangover effect of alcohol on this task. With familywise error rate set at .05 and 1 and 59 df, the critical F value for *post hoc* tests for trend was 8.3. Analyses of trend revealed no statistically significant effects (All Fs < 3.5 on both sessions 2 and 3).

Mackworth clock - Reaction time

Figure 5: Mean adjusted reaction time (ms) on the Mackworth clock task for all dose groups on Sessions 2 and 3.

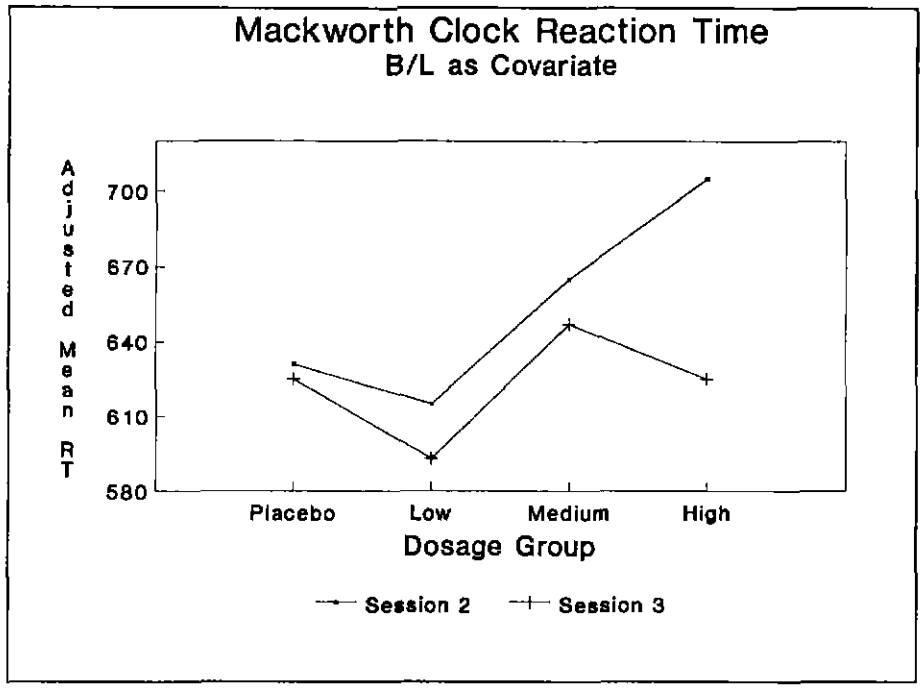

Figure 5 shows the adjusted mean reaction times for the four dosage groups on the acute and hangover test sessions. A linear trend indicating increasing reaction time with dose of ethanol is apparent on the acute, but not the hangover, session. The obtained F for this trend was ($F[1,57] = 8.03$). While this did not achieve statistical significance as a *post hoc* contrast, the strength of the relationship supports the analysis of this measure carried out in Part 2. There was no evidence of a hangover effect on this measure (all $Fs < 4.0$).

Self-Ratings of Mood and Intoxication

Separate multivariate analyses of variance were carried out to test for differences in ratings due to alcohol dosage condition on each of the three test sessions. Only the analysis of the ratings made after drinking on session 2 showed a significant effect of dose (Multivariate $F[30,147]=2.12$, $p<.003$). Univariate ANOVA results revealed that significant dose effects were observed in the three ratings of intoxication: "RIGHT NOW, how drunk do you feel?"

(F[3,59]=11.6, p<.001), "Would you drive a car now?" (F[3,59]=3.0, p<.05) and "How well could you drive now?" (F[3,59]=4.4, p<.009) The mean ratings for each of these questions at the 4 doses tested are shown in Table 5.

Table 5. Mean (±sd) self-ratings on three indicators of intoxication made after performing the psychomotor tasks on session 2 in the four alcohol dose conditions.

	Placebo $\bar{X}$ (±sd)	Low $\bar{X}$ (±sd)	Medium $\bar{X}$ (±sd)	High $\bar{X}$ (±sd)
"Drunk"	1.7 (±1.4)	3.5 (±1.6)	4.3 (±2.0)	5.3 (±2.1)
"Willingness to drive"	5.0 (±3.2)	3.0 (±2.7)	2.1 (±2.5)	2.5 (±3.0)
"Driving competence"	6.1 (±2.5)	4.9 (±2.4)	3.7 (±2.7)	3.0 (±2.8)

Ratings of drunkenness increased across dosage conditions while ratings of willingness to drive and driving competence declined.

Performance measures with additional covariates

In addition to the linear dose response relationship found for simple reaction time (regular and irregular) at the 3 hour session by Dauncey (1989), an interaction with drinking history was noted for this relationship. Heavier drinkers tended to show the effect to a greater extent. In the preliminary report, the number of years of drinking (Years Drinking) that each subject reported was used as a proxy measure for ethanol tolerance. There were two reasons for this. First, it was assumed that the longer a subject had been drinking, the more likely that a stable drinking pattern had emerged, and that the subject would be accustomed to the amount of ethanol currently consumed. Second, it was noted that the groups differed significantly on this variable, and it was considered necessary to test for the possible effects of this on performance.

Since that time, we have obtained information on the calculation of the original quantity/frequency classification used by Dauncey (1989), and have reproduced the classifications for the present sample. Since the same questions were used for both studies, the classifications are the same. The classifications represent approximately the average daily consumptions shown in Table 6.

Table 6. Quantity/frequency codings used by Dauncey (1989) and their relation to average daily consumption.

Coding (Dauncey, 1989)	Approximate daily ethanol (g)	Interpretation
A	-	*Non-drinker
B	<= 23	light
C	<= 46	light to moderate
D	<= 80	moderate
E	> 80	heavy

*There were no non-drinkers in either study.

The Years Drinking measure was tested for significance as a covariate with all of the performance measures, as there was a significant difference on this measure. It was considered necessary to examine any possible effects that this difference may have had on performance in both the acute and hangover sessions. The Quantity/Frequency measure has only been tested with the Simple Reaction Time task, as there were no significant between group differences on the Quantity/Frequency measure, and its importance to the analysis of the behavioural measures is principally in relation to the replication of the Dauncey (1989) results.

The critical value of F used in the following analyses was 8.3 (α = .05, df = 1,60), as these contrasts were defined *post hoc*.

Simple reaction time (regular)

There was a significant difference in the adjusted means for the simple reaction time (regular) task across dosage groups in the second (acute) test session. Reaction time increased as a linear function of increasing dosage. Neither of the other two polynomial contrasts (quadratic and cubic) were significant in the acute session. The Years Drinking measure, when introduced as a covariate, reduced the effect of the linear (acute) trend to a level below statistical significance. The Quantity/Frequency measure showed no relationship to this effects.

Simple reaction time (irregular)

None of the polynomial contrasts tested for simple reaction time (irregular) were found to explain a significant amount of the variance of scores in either the second (acute) or third (hangover) session. A marginal trend toward a linear relationship of dose to reaction time on the acute session was noted. When the Years Drinking measure was added as a covariate, this linear relationship became much stronger ($F[1,60] = 8.2$), although not exceeding the critical value of F previously defined for *post hoc* contrasts. Again, the Quantity/Frequency measure was unrelated to differences in reaction time.

Divided attention

A highly significant linear relationship of dose to performance was found for the composite score on the Divided Attention task during session 2. No significant relationship of dose to performance was seen during session 3. The addition of the Years Drinking measure as a covariate had no effect on the results from the acute or hangover test sessions.

Mackworth clock

No significant dose response relationships were found for the number of missed targets in either session. Addition of the Years Drinking measure as a covariate did not alter this outcome.

It is clear from the analysis of this Quantity/Frequency measure that the "Years Drinking" measure initially used was inappropriate to test for the relationship between current alcohol intake and strength of hangover effect found by Dauncey. The relationship found between the "Years Drinking" measure and the cubic trend in means on the simple reaction time task in the hangover session remains unexplained. However, this is certainly related to the overall superiority in performance of the low dose group.

DISCUSSION

Both simple reaction time (regular and irregular) and the divided attention tasks showed linearly decreasing trends in performance as a function of the dose of alcohol consumed on session 2. In the simple reaction time tasks, the linear trend consisted of a slight improvement in reaction time in the low dose group relative to the placebo group, and increasing decrements in reaction time in the medium and high dose groups relative to the placebo control group. This pattern of results only partially replicates that seen by Chesher *et al.* (1986) and Dauncey (1989) in which a simple linearly decreasing trend in performance was observed across the four dose groups used. In their study, the placebo group showed the best level of performance.

On session 2, the acute effects test, the results from the divided attention task followed a simple linear trend indicative of performance becoming progressively poorer as the dose of alcohol was increased. This finding has been reported by other researchers (Lemon, Mascord & Starmer, submitted for publication) and serves to demonstrate that the doses of alcohol used in the present experiment were sufficient to impair performance on a relatively complex task. Although alcohol acutely impaired performance on this task, there was no indication of a impairing effect due to hangover when subjects were re-tested on session 3. If anything, the group given placebo appeared to perform worse on session 3 than on session 2.

Another study has found that alcohol produced a dose-dependent increase in the number of misses on this version of the Mackworth clock task in an acute effects test (Chesher, Lemon, Gomel & Murphy, 1989). However, the present study used a relatively short inter-target interval for this test to provide sufficient data for the EEG analysis. Two of the reaction time tests (SRT regular and irregular) were adversely affected in the acute session by alcohol. The Mackworth clock showed a similar trend, but did not reach statistical significance. Neither of the Mackworth clock performance measures were affected by hangover effects of alcohol when subjects were tested on this task again 12 hours later (session 3).

There was no evidence of an impairing effect of alcohol on any of the behavioural tasks used in this study when subjects were tested 12 hours after consumption had ended. In the study by Dauncey (1989), a residual effect of alcohol had been observed on the simple reaction time tasks (both regular and irregular interstimulus intervals). It should be noted, however, that there were a number of procedural differences between the two studies. In the Dauncey study, practice, baseline, acute and residual effects of alcohol were all tested within a 10-12 hour period. The residual effects of alcohol were observed 3 hours after the acute test. In the present study, baseline, acute and hangover effects of alcohol were tested on different days. The hangover effect of alcohol was examined after the subjects had had an opportunity to sleep for between 6 and 12 hours. The opportunity to have a night's rest may have helped to ameliorate any effects of alcohol hangover. Additionally, most of the subjects in the Dauncey study had also smoked marijuana, as that study was principally concerned with the interaction of the two drugs.

One aspect of the procedure of experimentation on hangover has varied considerably across studies. In Dauncey (1989) and the present study, a measured dose of ethanol was consumed within a relatively short period (20 minutes). Other studies (e.g. Laurell and Törnros - cited in Franck, 1983) have used extended drinking periods of up to several hours, and allowed subjects to

regulate the amount of their drinking. In these studies, subjects would typically have much more gradual rises in BAC, and remain at or near their peak BAC for longer. Since the reported effects of ethanol hangover show considerable individual variation (Harburg *et al.*, 1981), it is plausible that hangover effects are sensitive to the rate and duration of ethanol consumption. While it is essential that these parameters be controlled in a study examining the time-dependent effects of hangover, these factors should be taken into account when attempting to compare the outcomes of different studies.

In summary, the results of the present study demonstrate that although the doses of alcohol used were sufficient to produce acute decrements in performance on a divided attention and simple reaction time tasks, they did not result in decrements in performance on these or other tasks when subjects were retested 12 hours after consumption had ended. The failure to demonstrate a long-lasting effect of alcohol on these laboratory tasks in no way precludes the existence of a subjective "hangover" effect commonly reported by drinkers, nor that performance on other tasks is affected by hangover. It is possible that higher doses of alcohol or different patterns of alcohol consumption are required to produce these effects reliably. In the previous study by Chesher *et al.* (1986) and Dauncey (1989), in which subsidiary analyses revealed a significant residual effect of alcohol, the time since cessation of consumption was much shorter (only 3 hours) and the subjects may have been more fatigued as they had been repeatedly tested over a single day. A night's sleep may have helped to offset these negative aftereffects of alcohol consumption in our subjects. Perhaps if more sensitive behavioural tests were employed or higher doses of alcohol were used, long-lasting effects of alcohol might become apparent at intervals greater than 3 hours after consumption. Reanalysis of the Simple Reaction Time (regular) results with the quantity/frequency measure used by Dauncey has revealed no indication of a hangover effect or interaction with consumption as found in that study.

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APPENDIX 1

Tables 6 - 8 show the mean scores and standard deviations for the performance tasks on sessions 1, 2 and 3. The Simple Reaction Times (SRT regular and SRT irregular) are in milliseconds. Performance on the Divided Attention task is reported as a computed standardised score which reflects the asymptotic tracking speed achieved and the correct reaction time to peripheral targets. Mackworth clock performance is reported as the number of missed targets.

TABLE 1A - Performance measures for session 1.

	Placebo $\bar{X}$ (±sd)	Low $\bar{X}$ (±sd)	Medium $\bar{X}$ (±sd)	High $\bar{X}$ (±sd)	Overall $\bar{X}$ (±sd)
SRT regular	669 (±163)	691 (±113)	622 (±72)	655 (±89)	657 (±112)
SRT irregular	590 (±175)	531 (±102)	498 (±72)	535 (±140)	537 (±129)
Mackworth clock RT	649 (±83)	636 (±84)	609 (±79)	668 (±86)	640 (±84)
Divided attention	0.28 (±1.93)	-0.48 (±1.41)	-0.11 (±1.12)	0.28 (±1.45)	0.03 (±1.48)
Mackworth clock: Misses	13.7 (±8.85)	11.9 (±10.4)	7.0 (±5.12)	10.7 (±8.61)	10.7 (±8.56)

TABLE 2A - Performance measures for session 2.

	Placebo $\bar{X}$ (±sd)	Low $\bar{X}$ (±sd)	Medium $\bar{X}$ (±sd)	High $\bar{X}$ (±sd)	Overall $\bar{X}$ (±sd)
SRT regular	658 (±136)	609 (±108)	649 (±79)	708 (±123)	660 (±136)
SRT irregular	586 (±164)	505 (±111)	589 (±233)	663 (±228)	590 (±199)
Mackworth clock RT	639 (±126)	611 (±99)	636 (±87)	731 (±143)	656 (±122)
Divided attention	-0.50 (±1.26)	-0.75 (±1.79)	-0.01 (±1.37)	0.99 (±1.47)	0 (±1.61)
Mackworth clock: Misses	15.3 (±10.5)	11.47 (±12.5)	9.24 (±7.28)	15.6 (±13.8)	12.9 (±11.4)

TABLE 3A - Performance measures for session 3.

	Placebo $\bar{X}$ (±sd)	Low $\bar{X}$ (±sd)	Medium $\bar{X}$ (±sd)	High $\bar{X}$ (±sd)	Overall $\bar{X}$ (±sd)
SRT regular	635 (±99)	589 (±71)	638 (±80)	637 (±128)	627 (±99)
SRT irregular	613 (±186)	521 (±141)	539 (±146)	543 (±94)	553 (±144)
Mackworth clock RT	633 (±115)	589 (±91)	619 (±112)	651 (±100)	624 (±105)
Divided attention	0.26 (±1.63)	-0.48 (±1.52)	-0.08 (±1.23)	0.24 (±1.65)	0 (±1.51)
Mackworth clock: Misses	17.4 (±11.7)	13.8 (±11.2)	11.2 (±6.46)	10.6 (±5.84)	13.0 (±9.13)

PART 2

THE ACUTE AND HANGOVER EFFECTS OF ETHANOL ON EVENT-RELATED POTENTIALS

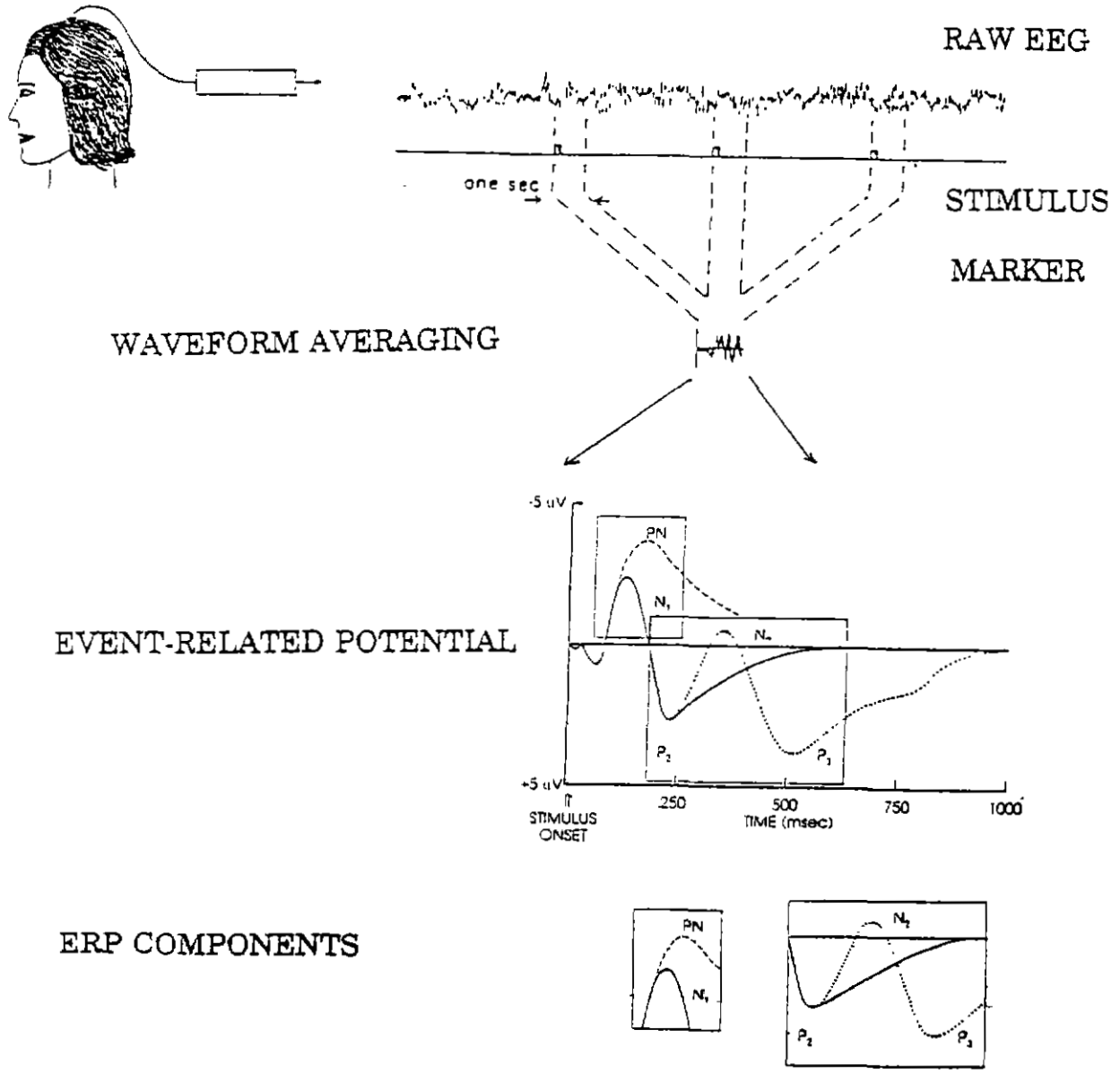
INTRODUCTION

The event-related potential (ERP)

The event-related potential, or ERP, is a recording of the electrical activity of the brain in response to an event or stimulus. The electroencephalograph (EEG) is recorded from electrodes placed on the surface of the scalp while subjects are presented with stimuli. Portions of the EEG that are time-locked to the presentation of the stimulus are then extracted and averaged. Averaging over a large number of stimuli cancels out the background electrical activity which is not time-locked to stimulus presentation, leading to the development of a waveform that contains components which are directly related to the processing of the stimulus. The resultant ERP consists of a series of peaks and troughs, labelled according to their polarity (negative (N) or positive (P)) and the ordinal position within a waveform (eg P3, to indicate the third positive deflection in the waveform) or the peak latency (eg P300, to indicate a positive deflection occurring 300 milliseconds after the presentation of a stimulus). The early components in the ERP (<80 msec), or exogenous components, vary as a function of the stimulus characteristics, and are relatively insensitive to information processing demands. The later components are typically labelled the endogenous components. These components are less sensitive to the physical characteristics of the stimuli, but do vary as a function of the information processing required (see Hillyard and Kutas, 1983 for a general review). The procedure is illustrated in Figure 1.

One paradigm frequently employed within the literature on the effects of alcohol on components of the ERP is the 'oddball' paradigm. In the oddball paradigm, infrequently occurring target stimuli are embedded within a series of repetitive 'non-target' stimuli. Subjects are usually required to count or respond with a button-press to the infrequently occurring target stimuli. The ERPs elicited by the infrequently occurring target stimuli typically elicit a negativity at approximately 200 msec (N2), and a later parietally distributed positivity (P3).

Figure 1. Schematic representation of an event-related potential (ERP). The horizontal axis in this figure represents time, with stimulus onset indicated by the vertical bar. Following the presentation of stimuli, a series of peaks and troughs can be identified in the waveform. The peaks are typically labelled to indicate whether they are positive or negative deflections in the waveform, and to indicate the latency at which they occur. Positivity in amplitude with respect to the baseline, is typically plotted as a downward deflection.



The peaks may consist of overlapping components, for example, within the auditory modality, the exogenous N1 component may be enhanced by a superimposed, longer duration 'processing negativity' reflecting selective attention to the relevant characteristics of the stimuli (Naatanen and Michie, 1979). When the difficulty of discrimination between stimuli is enhanced, the amplitude of the processing negativity to the unattended stimuli is larger and its duration longer (Alho, 1987; Alho, Sams, Paavilainen & Naatanen, 1986; Alho, Tottola, Reinikainen, Sams & Naatanen, 1987). The characteristics of processing negativity have typically been investigated by comparing stimuli possessing attributes which match the target stimulus, but which do not require an overt response. Therefore, the differential processing reflecting selective attention can be monitored without the confounding motor potentials, decision-making and response-related factors which occur following a target stimulus. The components elicited during visual selective attention appear to be more complex than those elicited during the auditory modality. The effects of selective attention on visual evoked potentials have been examined when stimuli vary on a variety of features including location, colour, orientation and spatial frequency (see Harter and Aine, 1984). Okita, Wijers, Mulder and Mulder (1985) reported a biphasic attentional effect, with selection on the basis of location and orientation resulting in an enhanced negativity occurring at approximately 200 msec, followed by an enhanced positivity occurring at approximately 500 msec.

The amplitude of the parietally distributed P3 component elicited by task-relevant novel target stimuli is proportional to the attentional resources allocated to the task (Israel, Chesney, Wickens and Donchin, 1980; see Donchin, 1979, 1981; Pritchard, 1981 for review). The peak latency of the P3 component varies considerably, depending on the characteristics of the task, and can occur between approximately 300-700 msec after presentation of the target stimulus. The latency of the P3 component is related to stimulus evaluation time, independent of response selection and execution processes (Kutas, McCarthy & Donchin, 1977; McCarthy & Donchin, 1981).

One of the major advantages of the ERP is that it provides a non-invasive technique for evaluating stages of information processing with high temporal resolution (in the order of milliseconds) which may not be observable with overt performance measures alone, for example, reaction time.

Acute Effects of Alcohol on ERPs

A number of studies have investigated the effects of acute administration of alcohol on the amplitude of the exogenous components (eg Coger, Dymond, Serafetinides, Lowenstam and Pearson, 1976; Lewis, Dustman and Beck, 1970; Wagman, Allen, Funderburk and Upright, 1978) and endogenous components of the ERP (eg Campbell and Lowick, 1987; Elmasian, Neville, Woods, Schuckit and Bloom, 1982; Krein, Overton, Young, Spreier and Yolton, 1987; Pfefferbaum, Horvath, Roth, Clifford and Kopell, 1980; Rohrbaugh, Stapleton, Parasuraman, Zubovic, Frowein, Varner, Adinoff, Lane, Eckardt and Linnoila, 1987; Roth, Tinklenberg and Kopell, 1977; see Porjesz and Begleiter, 1985 for review). However, relatively few studies have examined the nature of the task used in the elicitation of the ERPs, or administered a variety of doses of alcohol to examine dose-related effects.

Most studies investigating the effects of alcohol on the endogenous components have recorded ERPs during target detection tasks (visual and auditory), assessing the effect of ethanol on the amplitude and latency of the P3 component. The amplitude of the P3 component to target stimuli was reduced in two studies (Rohrbaugh et al, 1987; Elmasian et al, 1982) but failed to reach statistical significance in other studies (Krein et al, 1987; Pfefferbaum et al, 1980; Campbell and Lowick, 1987), although the results were in the expected direction. These results suggest that the acute administration of ethanol may reduce the allocation of attentional resources to the task.

Rohrbaugh et al (1987) report a monotonic decrease in P3 amplitude over the four doses administered (breath alcohol concentrations (BAC) approximately 0.0, 0.03, 0.07 and 0.09mg% at time of testing). Elmasian et al (1982) reported

an interaction between the acute effect of ethanol and family history of alcohol related problems on P3 amplitude, reporting a larger decrease in P3 amplitude following ingestion of alcohol for subjects with a positive family history. It is therefore possible that other confounding factors (eg predisposition) may interact with the acute effects of alcohol on the amplitude of the P3 component.

In contrast to the effects on the amplitude of the P3 component, the acute administration of alcohol appears to consistently lead to a delay in the latency of the P3 component to task relevant target stimuli. The five studies cited above reported a significant delay in P3 latency to targets following acute administration of ethanol. Rohrbaugh et al (1987) reported a significant dose-related increase in P3 latency over the four dosage groups investigated. Krein et al (1987) reported that the latency was not significantly delayed at BACs of approximately 0.06%, but was significantly delayed at BACs of 0.14%. The measures were obtained during the ascending limb of the BAC curve for a single dose of alcohol in this study. Pfefferbaum et al (1980) reported significant delays in P3 latency at low (BAC .035%) and high (BAC .08%) doses. These results suggest that the time required to evaluate a novel stimulus is consistently delayed following ingestion of alcohol.

Most investigators (see Mitchell, 1985) agree that the major effect of ethanol on driving-related skills is with infrequently occurring events. As Rohrbaugh et al (1987) discuss, the acute administration of alcohol appears to affect performance on complex laboratory tasks to a greater degree than on simple overlearned tasks. However, they note that as driving represents a highly overlearned skill, it is somewhat anomalous that ethanol is consistently implicated in road accidents. They suggest that temporary lapses in sustained attention or vigilance may be responsible for this apparently anomalous result. Therefore, investigating the effects of alcohol on performance and ERP components during tasks requiring sustained attention or vigilance may provide further information on the nature of the relationship between alcohol and road and traffic safety.

Indeed, performance on vigilance tasks does seem to be impaired by ethanol in some studies. Erwin, Wiener, Linnoila and Truscott (1978) report a statistically significant decrease in the number of correctly detected targets and an increase in reaction time to a visual vigilance task requiring detection of spatially displaced stimuli. These decrements were only significant in the high dose group (BAC 0.102%) and not in the low or medium dose group (BAC 0.036% and 0.070%). Rohrbaugh et al (1987) reported a linear dose-related reduction in the number of correctly detected targets and a dose-related increase in reaction time during a visual vigilance task. This study also reported a steeper decline in performance over time for the higher doses of alcohol. However, other studies have reported no effect of alcohol on auditory vigilance task performance (eg Pearson and Neal, 1970; Talland, 1966). These discrepancies may be related to the modality of stimulus presentation and to the type of vigilance task employed. Gustafson (1986a and b) suggested that delays in simple reaction time tasks may be apparent at lower doses of alcohol for visually presented stimuli than for auditorily presented stimuli. Parasuraman (1979) has suggested that performance on vigilance tasks reflects two independent processes. With high event-rate/successive discrimination tasks, performance decrements are related to perceptual sensitivity, whereas with low event-rate/simultaneous discrimination tasks, performance decrements may reflect changes in response criterion, rather than perceptual sensitivity. Classification of the type of vigilance task is therefore important in studies assessing the effects of alcohol on stimulus evaluation and response parameters. The tasks employed by Erwin et al (1978) and Rohrbaugh et al (1987) were both high event-rate/successive discrimination vigilance tasks.

Although many studies have investigated the acute effects of alcohol on ERPs, few have studied the 'hangover' effect when the BAC has returned to zero following administration of alcohol. Begleiter, Porjesz and Yerre-Grubstein (1974) recorded somatosensory evoked responses (SEPs) in patients attending a treatment program for alcohol-related problems. They administered a high dose of alcohol daily (3.2g/kg) for four days and monitored evoked responses on

the 'morning after' (commencing 10 hours after ingestion). They reported an increase in the amplitude of the recovery function of the SEP components on the morning after alcohol consumption. This amplitude increase continued over the four days of alcohol ingestion, and was significantly correlated with overt withdrawal symptomatology (Begleiter, Gross and Porjesz, 1973). Other studies have also reported an increase in the amplitude of the visual evoked responses during withdrawal in severely dependent problem drinkers (Coger et al, 1976; Wagman et al, 1978). The increase in evoked potential responses has been interpreted as a CNS rebound effect, although the effects of alcohol were confounded with the effects of medication (Antabuse) in these studies. Animal studies have suggested that the duration of the CNS hyperexcitability is related to the duration of exposure to alcohol (see Porjesz and Begleiter, 1985). The hangover effects of alcohol on the information processing components of the ERP have not been investigated.

The present study was designed to investigate the acute and hangover effects of three doses of alcohol (0.5g/kg, 0.75g/kg and 1.0g/kg) and a placebo condition on the endogenous components of the ERP elicited during a high event-rate/successive discrimination vigilance task. This task would enable evaluation of the time taken to evaluate a stimulus independent of response selection factors (P3 latency), and the allocation of attentional resources to the task (N2 and P3 amplitude) following administration of varying doses of alcohol.

METHOD

Subjects

Thirty-two of the subjects participated in the ERP recording phase of the experiment. The selection criteria are described in Part 1. The mean age of this sub-group was 26.1 years (s=7.0).

Procedure

The procedure is described in detail in Part 1 of the accompanying report.

Behavioural Task

The vigilance task utilised for the elicitation of the ERPs was the Mackworth Clock (Mackworth, 1948). In this task, 24 dots are continuously displayed in a circle on a visual display (Apple IIe). A moving rectangle flashes briefly (100 msec duration) on each dot, circling clockwise. Occasionally, the rectangle skips a dot, and the subject must respond by pressing a button when a dot is skipped. This stimulus is designated the infrequent target stimulus in the analysis of the event-related potential data. All other stimuli are referred to as the frequent stimuli. The interstimulus interval (ISI) between successive flashes was 500ms. The task continues for a duration of 40 minutes, with 60 targets randomly presented in a total of 2400 stimuli. This task satisfies the criteria of a high event-rate/successive discrimination task as the stimuli are presented at a rate of two per second, and the target discrimination is based on a comparison of the location of the current stimulus with the location of the previously presented stimulus.

Event-related potential recording

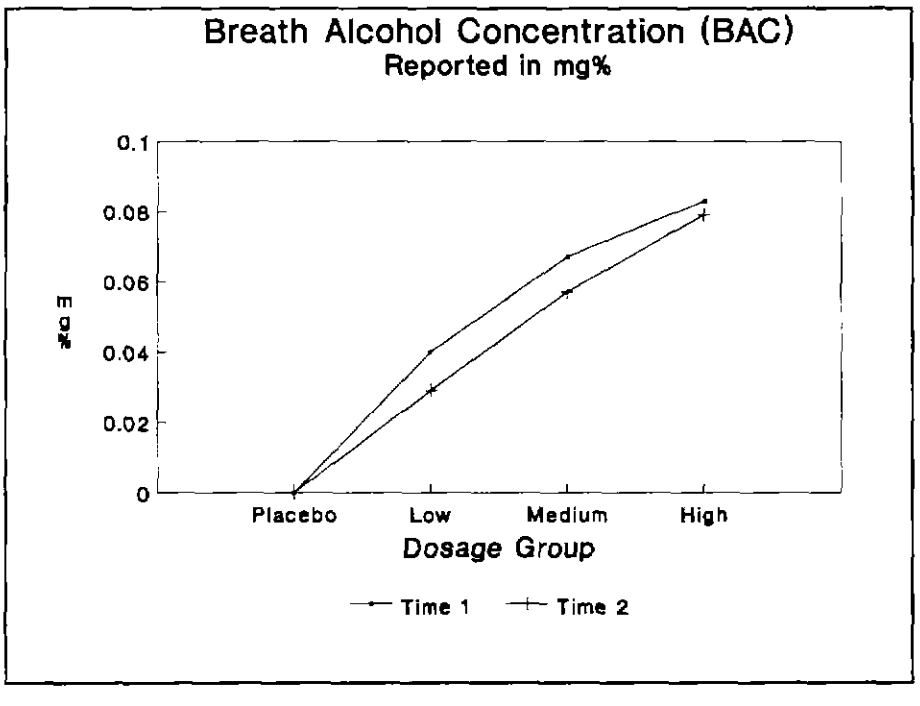
The electroencephalograph (EEG) was recorded at Fz, Cz and Pz electrode sites (frontal, central and parietal midline sites) using an electrode cap (Electro-Cap International) and referenced to the tip of the nose. Vertical and horizontal eye movement were monitored with electrodes placed 2cm below and on the outer canthus of the left eye. The data were amplified using Neomedix NT114-A amplifiers with lower and upper frequency cutoffs of 0.016 and 50 Hz respectively (3dB down). The data were digitised at a rate of 8 ms per channel and stored on computer disk for subsequent averaging. Data were averaged separately for the frequent and infrequent target stimuli, over a 1300ms epoch commencing 32ms prior to stimulus onset. Trials containing excessive eye movement or artefact were excluded from the averages. The data from three subjects were excluded from the analyses as there was excessive contamination from eye movement. Data from 29 subjects were therefore analysed, seven subjects in the placebo, low and medium dose groups and eight subjects in the high dose group.

RESULTS

Breath Alcohol Concentration

The mean breath alcohol concentrations at two time periods (10 minutes after ingestion, 70 minutes after ingestion) are presented for the four dosage conditions in Figure 2. These data were analysed with a repeated measures analysis of variance with group (placebo, low, medium, high) as a between subjects factor. The four doses differed significantly on both readings (Time 1, $F=68.4$, $p<.001$; Time 2, $F=87.1$, $p<.001$).

Figure 2. The mean BAC reported in mg%, for each of the four dosage groups at Time 1 (10 minutes after ingestion of ethanol) and Time 2 (70 minutes after ingestion of ethanol.



Behavioural Results

The performance measures analysed were number of misses and reaction time (RT). The behavioural data for the three sessions (means and standard deviations) for each of the dosage groups is presented in Appendix 1. The data were analysed separately for the acute and hangover effects of ethanol. Planned contrasts for linear trend were carried out to evaluate the hypothesis that alcohol produced dose-related changes on the behavioural and ERP measures both acutely and on the following morning, using a decision-wise error rate of 0.05. The less conservative analysis for linear trend was adopted for these analyses due to the smaller number of subjects included in this part of the project. On the basis of previous research, it was predicted that dose-related linear trends would be observed with the dosage levels employed in the present study. However, higher order trends (quadratic and cubic) were evaluated with *post-hoc* comparisons using the method of Scheffé. The critical F-value for *post-hoc* comparisons was 8.85.

The mean level of performance (number of misses) for the acute and 'hangover' sessions (adjusted for baseline performance) is presented graphically in Figure 3. The mean reaction time for the acute and hangover sessions (adjusted for baseline scores) is presented in Figure 4.

There were no statistically significant linear or higher-order dose-related trends on the number of misses, either immediately after ingestion of alcohol or on the following morning (all Fs < 1.5)

There was a statistically significant linear dose-related increase in reaction time following acute administration of alcohol (F=7.19, p=.0125). The higher-order trends failed to reach statistical significance for the acute effects of alcohol on reaction time (all Fs < 1.0). There were no significant linear or higher-order trends on reaction time during the hangover session (all Fs < 1.0).

Figure 3. Mean number of misses on the Mackworth Clock for each of four dosage groups (placebo, low, medium and high).

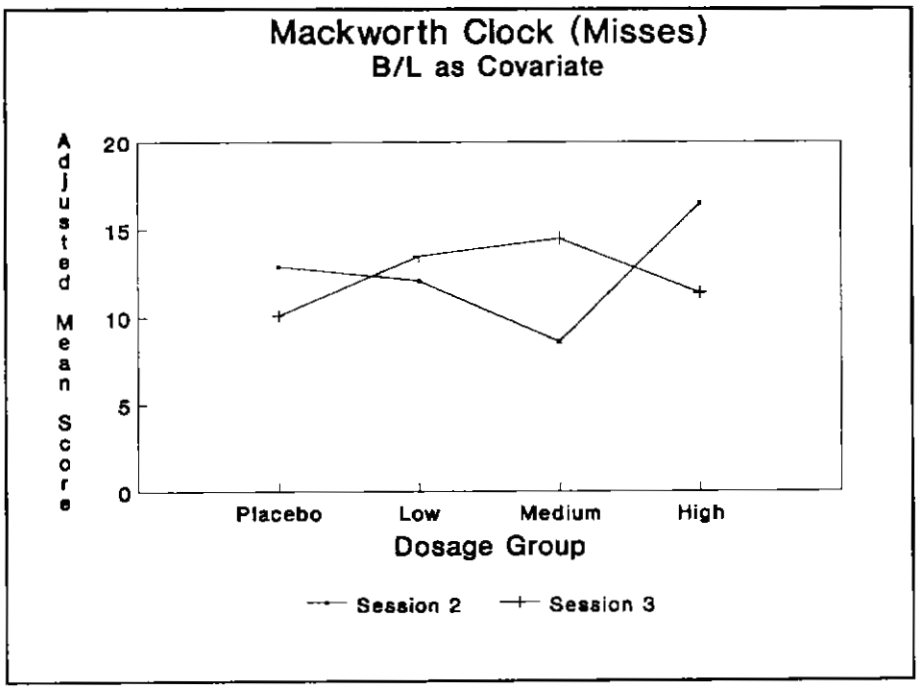
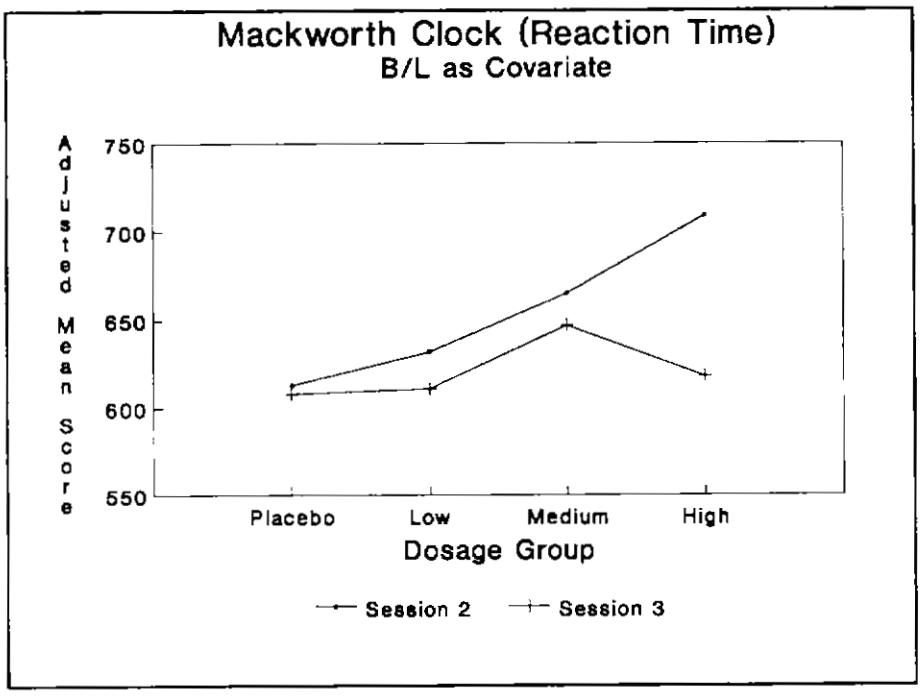


Figure 4. Mean reaction time for the four dosage groups (time in milliseconds).



Event-related potential results

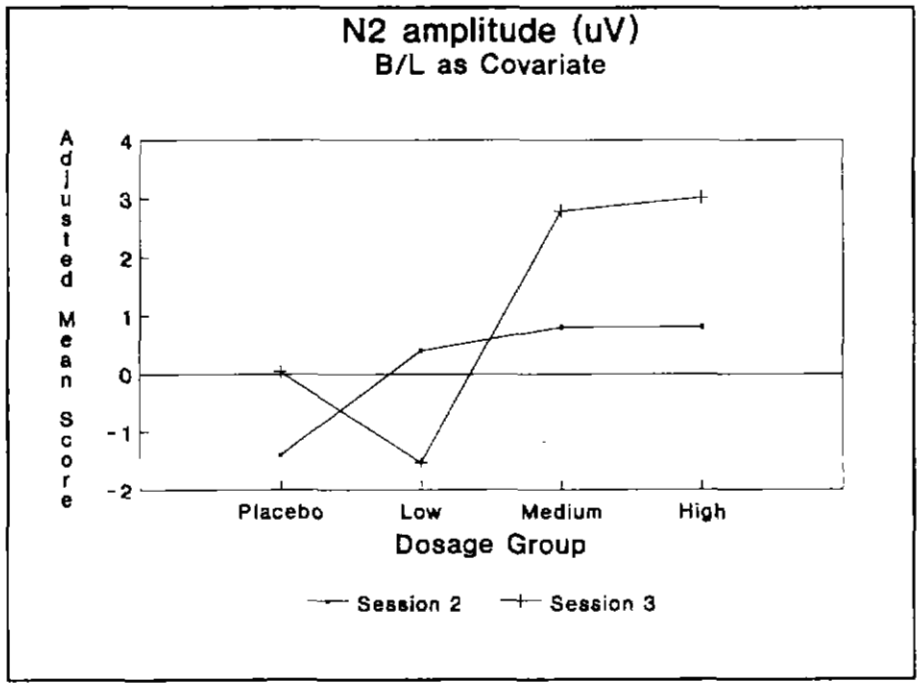
Difference waveforms were created by subtracting the ERPs elicited by the frequent stimuli from the ERPs elicited by the target stimuli. Therefore, the ERPs displayed reflect differences in information processing components rather than differences in the exogenous components, as the stimulus characteristics were identical for the frequent and infrequent stimuli.

The grand mean waveforms analysed and reported were for the parietal electrode site (Pz). The grand mean waveforms comparing the baseline and acute sessions for the four dosage groups separately are presented in Appendix 2a. The grand mean waveforms comparing the baseline and hangover sessions for the four dosage groups separately are presented in Appendix 2b. For comparison of dose-related effects, the grand mean waveforms comparing the four dosage groups during the baseline, acute and hangover sessions separately are also presented in Appendix 2c.

The target stimuli elicited a negative deflection (N2) at approximately 200ms, followed by a large positive deflection (P3) occurring at approximately 500ms. The mean amplitude over the 150-350ms epoch was measured for the amplitude of the N2 deflection and the mean amplitude over the 350-750ms epoch was measured for the amplitude of the P3 component. The latency of the most positive peak occurring within the 350-750ms window was measured for the peak latency of the P3 component.

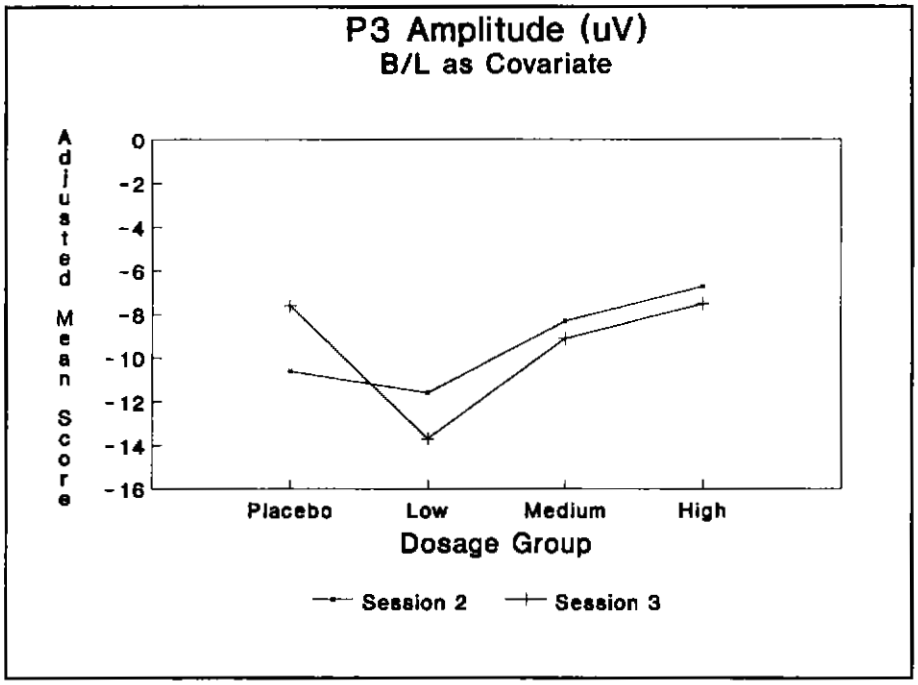
The mean amplitude and latency measures (adjusted for baseline performance) are displayed graphically in Figures 7 to 9. The mean values (and standard deviations) are presented in Appendix 1.

Figure 7. Mean amplitude measures at the Pz electrode site over the 150-350ms epoch (N2 amplitude).



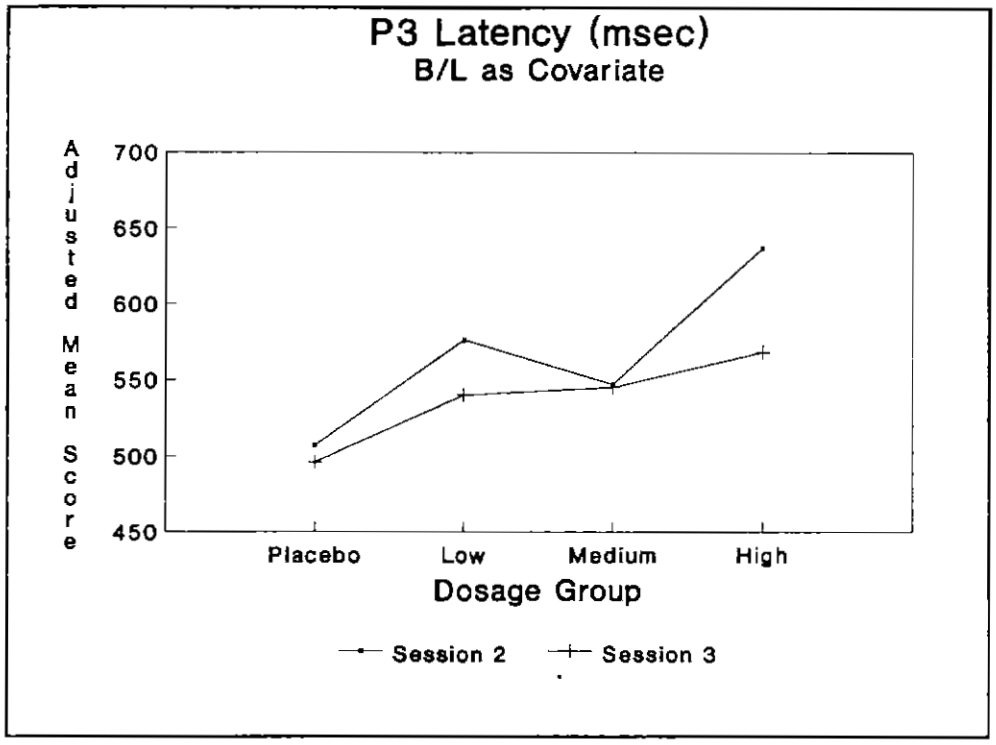
There were no significant linear or higher-order trends for the acute effect of ethanol on N2 amplitude (linear trend, F=1.28, p=.27; higher-order trends all Fs < 1.0). However, there was a significant linear dose-related increase in the amplitude of the N2 component on the morning after ingestion of alcohol (linear trend, F=7.94, p=.009). The higher-order trends failed to reach statistical significance (quadratic trend, F = 0.60; cubic trend, F=4.49).

Figure 8. Mean P3 amplitude measures at the Pz electrode site.



There were no significant linear dose-related effects of ethanol on the amplitude of the P3 component after acute administration (linear trend, $F=1.70$, $p=.20$) or on the following morning (linear trend, $F=0.18$, $p=.67$). Higher-order trends also failed to reach statistical significance (all $Fs < 2.0$).

Figure 9. Mean P3 peak latency measured at the Pz electrode site.



Acutely, ethanol led to a statistically significant dose-related linear increase in the peak latency of the P3 component (F=10.1, p=.004). The P3 latency effects were not statistically significant on the following morning (linear trend, F=2.74, p=.11). The higher-order trends also failed to reach statistical significance (acute effects: quadratic trend, F=0.18; cubic trend, F=3.41; hangover effects: quadratic trend, F=0.02; cubic trend, F=0.89).

As subjects were required to remain at the laboratory until their BAC dropped below .05mg% on the night when ethanol was administered, subjects allocated to the higher doses had to stay later than subjects from the lower doses, and it is possible that differences in the number of hours slept on the preceding night may account for the increased N2 amplitude observed on the following morning

in the present study. To investigate this hypothesis, the dose-related effects were examined after including the number of hours slept on the night before the hangover session. The sleep questionnaire data was not completed by two subjects, and was therefore coded as missing data. The number of hours slept on the night after ingestion of alcohol was not significantly related to the N2 amplitude ($F=2.90$, $p=.10$), and the dose-related linear trend describing the N2 amplitude increase on the following morning remained marginally significant after controlling for baseline performance and the number of hours slept on the preceding night ($F=3.99$, $p=.0579$).

All analyses were carried out fitting years of drinking and typical quantity per occasion as covariates. Similar results were obtained for the dose-related effects in these analyses. Typical quantity per occasion was marginally related to the amplitude of the P3 component following acute administration of ethanol ($F=3.65$, $p=.07$) and the dose-related linear trend on the amplitude of the P3 component after fitting years drinking and typical quantity consumed per occasion was also marginally significant ($F=3.29$, $p=.08$). Number of years drinking was marginally related to the latency of the P3 component following acute administration of alcohol ($F=4.34$, $p=.05$).

DISCUSSION

The results from the present experiment replicate a number of studies demonstrating a latency delay in the P3 component elicited by infrequent task-relevant stimuli following ingestion of ethanol. The increase in reaction time to target stimuli paralleled the increase in P3 latency. These results suggest that the time taken to evaluate a novel task-relevant stimulus is affected by ethanol, and that the event-related potential provides a particularly sensitive index of stimulus evaluation processes. This study also replicates the findings reported by Rohrbaugh et al (1987) of a statistically significant dose related-increase in reaction time. Pfefferbaum et al (1980) reported P3 latency delays

following ethanol administration, but with no corresponding increase in RT. However, the task employed in their study was an auditory target detection paradigm, with easily discriminable tones (detecting infrequent tones of either 400Hz or 1600Hz from the frequent 800Hz tones). These results suggest that the effects of alcohol on reaction time may be different during visual and auditory vigilance tasks and the relationship between response parameters (as indexed by reaction time) and stimulus evaluation processes (as indexed by the latency of the P3 component) will vary as a function of the type of vigilance task employed. As noted earlier in this report, the distinction between high event-rate/successive discrimination and low event-rate/simultaneous discrimination tasks may also influence the relationship between stimulus evaluation and response time (Parasuraman, 1979). In studies assessing the effects of alcohol on driving related skills, it would therefore be particularly important to utilise visual vigilance tasks rather than auditory vigilance tasks.

In the present study, there was no significant reduction in the amplitude of the P3 component elicited by the target stimuli. This result is consistent with a number of studies cited (Krein et al, 1987; Pfefferbaum et al, 1980; Campbell and Lowick, 1987). The acute effects of ethanol on the amplitude of the P3 component may interact with other factors (eg drinking history and predisposition), and thus exhibit greater variability.

There was no evidence of a residual dose-related effect on the components of the ERP which were acutely affected by alcohol in the present study. Therefore, stimulus evaluation time, as reflected in the peak latency of the P3 component, was not significantly delayed 12 hours after ingestion of alcohol.

However, there was a significant dose-related linear increase in the amplitude of the N2 component on the morning after ingestion of alcohol. Rohrbaugh et al (1987) report an increase in the amplitude of the N2 component following administration of ethanol compared to placebo, although the main effect of dose

failed to reach statistical significance in their study ($p < .12$). They suggested that this increase may be related to the drowsiness induced by ethanol. It is possible that the dose-related increase in the amplitude of the N2 component observed in the present study is related to subjective symptoms of hangover such as fatigue. Methodological differences between the procedure employed by Rohrbaugh et al (1987) and the procedure employed in the present study may explain the effect occurring during an acute session in the Rohrbaugh study, and in the hangover session in the present study. In the Rohrbaugh study, subjects were given an initial loading dose of ethanol (consumed over a 30 minute interval), and then were given maintenance doses at 30 minute intervals to ensure the BAC level remained constant for the duration of the testing session. The testing session lasted approximately 2 1/2 hours. In the present study, subjects were given an initial dose of ethanol (consumed over a 20 minute interval) and were then tested over a period of approximately one hour. Therefore, any acute effects of alcohol-induced drowsiness would be more likely to be manifest in the longer testing session. It is tempting to speculate that the N2 amplitude increase is related to the reported symptoms of hangover and hence may be more clearly observed on the morning after alcohol ingestion than when under the influence of alcohol.

The observed effect is unlikely to be related to an enhancement of the exogenous components of the ERP, or CNS hyperexcitability, as it was apparent in the difference waveforms analysed. These waveforms were created by subtracting the ERPs for the frequent stimuli from the ERPs elicited by the infrequent stimuli, and the stimulus characteristics were identical for both stimulus types.

One possible interpretation of the increased N2 amplitude is that this reflects enhanced processing negativity on the morning after ingestion of alcohol. Within the auditory modality, the amplitude of processing negativity to unattended stimuli increases when the discrimination between stimuli is made more difficult. Therefore, this result suggests that the ability to discriminate

relevant stimuli may be more difficult on the morning after ingestion of ethanol.

An alternative explanation of the enhanced negativity over the 150-350ms epoch is that the onset of the P3 component may be delayed. However, this interpretation is unlikely as the peak latency of the P3 component was not significantly delayed in the hangover session. Rohrbaugh et al also report a dissociation between experimental variables affecting the amplitude of the N2 component and the latency of the P3 component, suggesting that the effect on the N2 component was not simply due to latency effects on P3.

It is important to note though, that performance deficits were not apparent in the data from the present experiment. Also, the amplitude of the N2 component is confounded in the present experiment with the later decision-making and response components of the ERP. It would therefore be desirable to investigate this result in a visual selective attention paradigm where the effects of selective attention and target detection can be separated.

In summary, these results suggest that the recording of event-related potentials provides a non-invasive procedure for investigating aspects of information processing impairments which could affect the ability to drive safely. The time taken to evaluate an infrequently occurring target stimulus in a sustained attention or vigilance task was delayed following ingestion of alcohol, and delays in reaction time paralleled the increase in stimulus evaluation time. It was suggested that the modality of stimulus presentation may be an important factor in understanding conflicting research results investigating the effect of alcohol on reaction time. There were no apparent long-lasting or residual dose-related effects on stimulus evaluation time, as indexed by the latency of the P3 component, or on response parameters, as indexed by reaction time, on the morning after ingestion of alcohol.

However, there was evidence in the analysis of the event-related potential data

that on the following morning it may be more difficult to selectively attend to a location in space. It is tempting to speculate that these ERP differences may be related to the reported symptoms of fatigue which are commonly associated with the hangover phenomenon, and which may also affect the ability to drive safely. Although the ERP indices were statistically significant on the morning after alcohol ingestion, there were no corresponding performance decrements in the present study. Investigation of these effects in a paradigm designed to separate the effects of selective attention from those of target detection, as in the visual selective attention paradigm employed by Okita et al (1985) may clarify the present finding.

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APPENDIX 1

Tables 1 to 3 report the mean scores and standard deviations from the performance tasks and ERP measures on Sessions 1, 2 and 3. Mackworth clock performance is reported as the number of misses and reaction time (in milliseconds). The ERP amplitude measures are reported as microvolts, and latency measures are reported in milliseconds.

TABLE 1A - Performance and ERP measures for Session 1.

	Placebo $\bar{X}$ (±sd)	Low $\bar{X}$ (±sd)	Medium $\bar{X}$ (±sd)	High $\bar{X}$ (±sd)	Overall $\bar{X}$ (±sd)
Misses	13.4 (±11.4)	10.1 (±8.7)	5.4 (±4.0)	8.1 (±5.1)	9.2 (±7.9)
Reaction Time	659 (±71)	640 (±100)	599 (±84)	650 (±89)	637 (±85)
N2 Amplitude	1.8 (±3.8)	-1.54 (±6.8)	3.3 (±1.3)	1.5 (±5.0)	1.3 (±4.8)
P3 Amplitude	-9.7 (±8.8)	-14.9 (±13.0)	-10.6 (±6.5)	-10.9 (±7.6)	-11.5 (±9.0)
P3 Latency	536 (±89)	570 (±78)	539 (±68)	534 (±94)	544 (±80)

TABLE 2A - Performance and ERP measures for Session 2.

	Placebo $\bar{X}$ (±sd)	Low $\bar{X}$ (±sd)	Medium $\bar{X}$ (±sd)	High $\bar{X}$ (±sd)	Overall $\bar{X}$ (±sd)
Misses	14.3 (±11.2)	12.4 (±15.1)	7.3 (±5.5)	16.1 (±12.2)	12.7 (±11.4)
Reaction Time	640 (±100)	636 (±130)	619 (±81)	725 (±176)	657 (±130)
N2 Amplitude	-1.2 (±3.5)	-0.8 (±4.1)	1.7 (±3.6)	0.9 (±5.7)	0.2 (±4.3)
P3 Amplitude	-9.9 (±4.7)	-12.8 (±8.4)	-8.0 (±7.1)	-6.5 (±10.0)	-9.2 (±7.8)
P3 Latency	503 (±88)	586 (±80)	545 (±37)	632 (±84)	569 (±87)

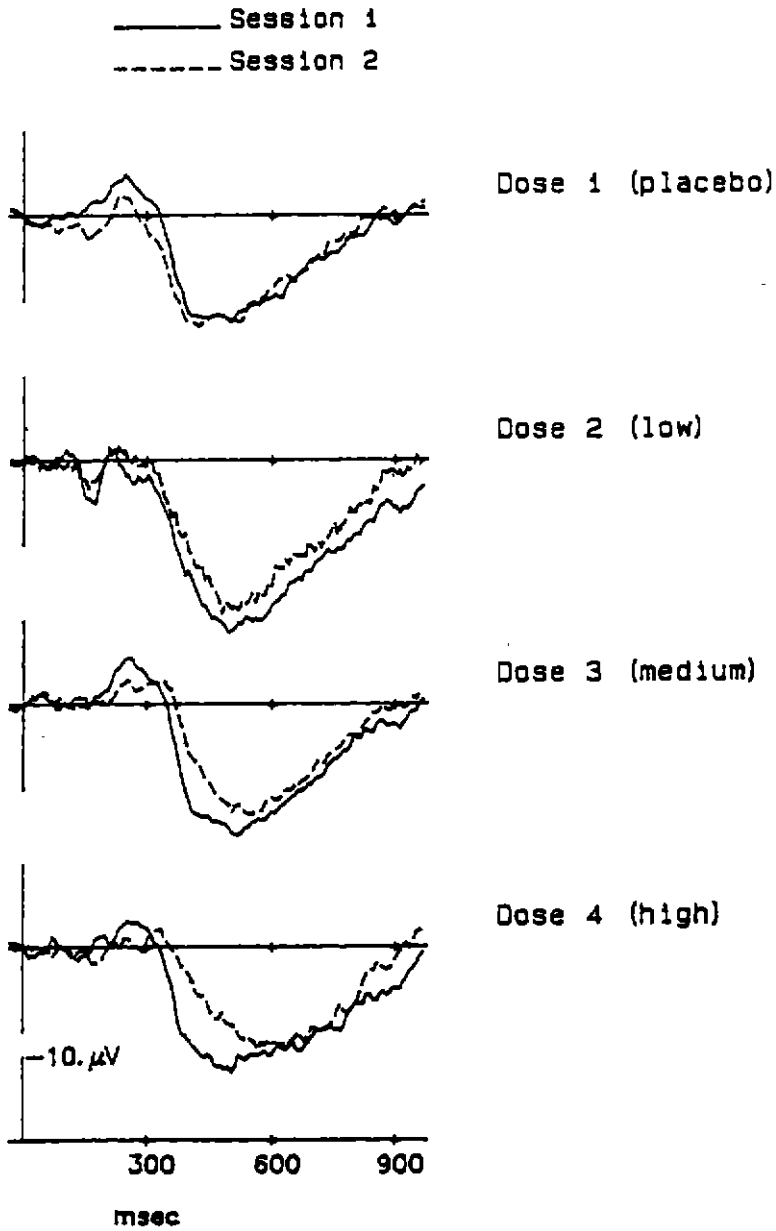
TABLE 3A - Performance and ERP measures for Session 3.

	Placebo $\bar{X}$ (±sd)	Low $\bar{X}$ (±sd)	Medium $\bar{X}$ (±sd)	High $\bar{X}$ (±sd)	Overall $\bar{X}$ (±sd)
Misses	13.9 (±10.8)	14.3 (±12.8)	11.0 (±6.5)	10.4 (±8.7)	12.3 (±9.6)
Reaction Time	630 (±100)	614 (±109)	610 (±75)	631 (±119)	622 (±98)
N2 Amplitude	0.1 (±2.3)	-2.0 (±3.2)	3.1 (±2.2)	3.1 (±2.9)	1.1 (±3.4)
P3 Amplitude	-6.9 (±6.1)	-14.8 (±8.7)	-8.8 (±9.8)	-7.3 (±5.8)	-9.4 (±8.0)
P3 Latency	504 (±74)	538 (±44)	541 (±69)	568 (±115)	539 (±80)

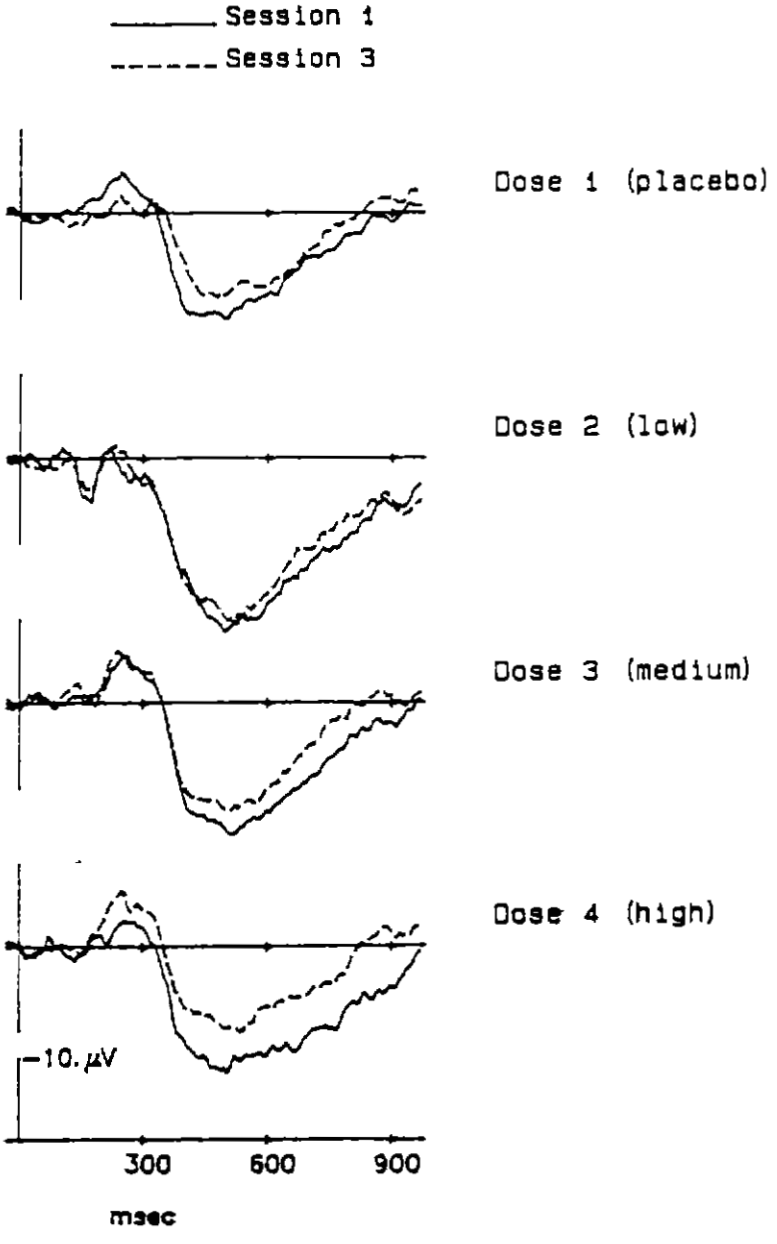
APPENDIX 2

Grand mean difference waveforms (target - frequent stimuli) at Pz electrode site for the four dosage groups (placebo, low, medium, high).

a) Grand mean difference waveforms superimposing the ERPs elicited during session 1 (baseline) and session 2 (acute).



b) Grand mean difference waveforms superimposing the ERPs elicited during session 1 (baseline) and session 3 (hangover).



c) Grand mean waveforms superimposing the four dosage groups (placebo, low, medium, high) for session 1 (baseline), session 2 (acute) and session 3 (hangover).