VISUAL FUNCTION
AS AN
OBJECTIVE MEASURE
OF
ALCOHOL INTOXICATION

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Executive Summary

1. The aim in this project was to investigate the viability of using changes in visual system function as a measure of alcohol intoxication.

2. The best candidate measures of changes in visual function as a result of consuming alcohol were identified from the literature. These were aspects of motor and autonomic visual system function.

3. These abilities were measured with standard clinical tests in experiment 1. Young male students who defined themselves as social drinkers were randomly assigned to control and two alcohol groups (low and moderate BAL). They completed a baseline visual function test and 2 post-alcohol tests in the experimental session.

4. Results of experiment 1 showed significant changes to the functions of latent eye position, range of binocular movement and speed of fast voluntary eye movements. No significant change to induced pupil oscillations was demonstrated. Changes were more marked in the moderate BAL group than the low BAL group. There was no significant change in these abilities in the control group.

5. Experiment 2 was designed to investigate whether these aspects of visual function are susceptible to expectancy effects as performance tests have been found to be.

6. Subjects were randomly assigned to drinking or control status. 3 drinking groups were defined; a ‘told effect’ of alcohol on visual function group, a ‘told no effect’ group and a ‘told nothing’ group. Drinking subjects all consumed alcohol equivalent to 0.7 ml/kg body weight. The same series of tests as in experiment 1 was completed with the addition of the measurement of the motor function of involuntary inwards rotation of the eyes.

7. All alcohol groups demonstrated the significant changes in visual function found in experiment 1 in the moderate BAL group. Results also reached significance in the measurement of inwards rotation of the eyes. The control group again demonstrated no real change in the aspects of visual function examined, indicating the reliability of these measures.

8. Statistical testing indicated that there was no difference in the results of any of the manipulated expectancy groups indicating that these measures of visual function are not susceptible to expectancy effects.

9. These motor changes in visual function analysed in this study were due to a decrement in the neurological and muscular control of aspects of eye movement and eye muscle co-ordination. These changes interfere with the stability of the binocular and the fast eye movement control of the visual system.
10. Correlation of performance decrement with these documented changes to the visual system both as a result of alcohol and other drugs and drug combinations is needed.

11. Further research in this area would increase our knowledge of the functional implications of the observed alcohol-induced changes in visual function and also allow for the development of a reliable and objective test to determine the presence of drug-induced impairment of motor ability.
1 INTRODUCTION

1.1 BACKGROUND

Alcohol is well known for its ability to alter psychomotor co-ordination in a broadly dose-dependent manner. However, obtaining valid objective measures of performance under intoxication is extremely difficult. This is because alcohol intoxication is not a novel state for the subjects who are tested. Equally, at the dose-levels commonly employed, it is possible for some subjects to show a form of effortful tolerance in that they attempt to overcome the deleterious effects of alcohol on their performance, essentially by trying harder. The degree to which subjects are influenced by their expectancies concerning the likely effect of alcohol on their performance, and the extent to which they are inclined to attempt to overcome any deficits, often appears to have as large an impact on performance of traditional psychomotor tasks as the effect of the drug alone. This difficulty in obtaining valid objective measures has resulted in psychomotor incoordination being defined according to empirically established correlations between mean blood alcohol level (BAL) and mean performance decrements while driving. Being indirect, such an operational approach to measurement is inevitably subject to inaccuracies, both false negatives and false positives, in estimating the degree of individuals' actual or potential psychomotor incoordination. Although unsatisfactory in a purely scientific sense, there are no alternatives at present.

Although blood alcohol level is used in legislation to define inability to drive under the influence of alcohol, no standards for other drugs or combinations of drugs known to affect driving ability have been established. Objective measures of physiological systems (eg visual function) closely related to those abilities known to be adversely affected by alcohol and other drugs (eg psychomotor coordination), may well be preferable to the development of a multitude of separate biochemical assays, all of which would be based on correlational data. If the correlated physiological system does indeed correlate highly with actual performance deficits, then it may be more appropriate to measure that system than attempt to correlate performance deficits from biochemical measures.

It is hypothesized that a candidate for such an objective measure might be the fine motor and autonomic functions of the visual system which are known to be both susceptible to the effects of alcohol and other drugs, as well as largely involuntary and therefore theoretically not subject to expectancy effects on the part of the subject. The objectives of this research were to: (1) identify from existing literature best candidate measures of visual function; (2) establish dose-response curve for visual
system effects under alcohol; and (3) test the effect of subject expectancy on any alcohol-induced changes in visual function.

There are several potential benefits to road safety of demonstrating that changes in visual system functioning are not influenced by subject expectancy, and are correlated with psychomotor performance decrements under alcohol might include: the development of a model for identifying psychomotor deficits caused by other drugs; the possibility of using objective measures to define inability to drive safely under the influence of drugs, obviating the need for assays; and/or allow the possibility of pre-emptive identification and education of those young drivers who may be more susceptible to alcohol and drug effects. However, these possibilities lie in the future, and first some of the issues which bear upon the aims in this research must be considered.

1.2 THE VISUAL SYSTEM

The visual system comprises the eye as the receptor organ which allows the transmission of light to the retina, then via the visual pathway to be processed in the visual cortex. Two important subordinate systems, that is the binocular and eye movement systems allow the two eyes to function in a co-ordinated fashion and control the fine motor movement of the eyes.

The eye consists of three main layers. The outer coat of the eye consists of the sclera and cornea. The sclera is the opaque fibrous layer which protects the globe. The transparent cornea allows light rays to pass into the eye and be refracted. Its clarity is vital for optical integrity.

The middle vascular layer or uveal tract consists of the iris, ciliary body and choroid. The two muscles of the iris allow pupil constriction and dilation thus regulating light entering the eye. The ciliary muscle acts to regulate the mechanism of accommodation through control of the Zonule fibres which increase and reduce the convexity of the lens. The ciliary processes produce aqueous, a watery substance which maintains intraocular pressure and provides a nutritive function to the lens and cornea. The choroid is composed of several layers of blood vessels, the prime function being to nourish the outer layers of the retina.

The retina is the inner nerve fibre layer which houses the retinal receptors. The more sensitive rods function in low lighting levels (scotopic vision) and the more accurate cones function at higher lighting levels (photopic vision).

The transparent media consists of the aqueous, vitreous and the lens. The jelly like vitreous serves to support the outer coats. The biconvex lens changes shape to refract light entering the eye to allow clear focus of visual stimuli.

The stimulus for vision is light rays entering through the pupil and being refracted by the transparent media to stimulate the retinal receptors. This causes a series of chemical changes and impulses are generated by excited receptors and transmitted to the visual cortex in the occipital lobe of the brain via the visual pathway.
Vision is divided into three components: light sense, color sense and form sense. Visual acuity relates to form sense which discriminates the different parts of the visual image which is studied as resolving power and measured as the minimum angle of resolution. Vision charts are based on this principle of one minute of arc being the minimum resolvable angle so the area stimulated is equivalent to the diameter of a cone cell.

Visual field or peripheral vision is the ability to perceive the presence, motion or color of objects outside the direct line of vision. The further a stimulus is away from the central vision the more reduced the visual acuity of the stimulus will be due to the position of the retinal receptors.

Binocular Single Vision is the vision which is achieved by the co-ordinated use of both eyes so that images that arise separately in each eye are appreciated as a single mental impression in the visual cortex. Binocular vision acts as a locking device to align both eyes correctly, in a parallel position. The advantages of binocular single vision are depth perception, the judgement of distance and moving objects, and enlargement of the visual field.

The main components of binocular single vision are fusion and stereopsis. Fusion is the ability to see two similar images one formed on each retina and to blend them into one. This requires the sensory ability to perceive the similarity and the motor movement of the eyes to bring the images together. Stereopsis is the ability to perceive two slightly dissimilar images one formed on each retina and to blend them into one with the appreciation of depth. The degree of stereopsis is graded according to the amount of disparity of the images.

If there is a misalignment of the visual axes, and binocular single vision is unlocked a strabismus (squint) will result. This can be manifest (heterotropia) or latent (heterophoria). A small amount of heterophoria is considered to be normal and is controlled by the binocular single vision mechanism. Heterotropia occurring in an adult will cause double vision. Heterotropia untreated in a child will cause suppression or neglect of the deviating image and will result in some loss of vision in that eye.

Under normal conditions movement of one eye is associated with equal movement of the other eye. The 6 extra ocular muscles in each eye are responsible for movement. In conjugate movement the visual axes remain parallel with the eyes moving from central (primary position) into all other cardinal positions of gaze. In disgugate movement the eyes move out of parallel either inwards (convergent) position or outwards (divergent). The convergence reflex is part of the near reflex and is concerned with simultaneous movements of the two eyes so that they are able to be directed to objects at varying distances from the eyes. An extensive series of nerve centres and pathways are responsible for the control of ocular movement. (See Appendix A for a glossary of orthoptic terminology)
1.3 ALCOHOL INTOXICATION

1.3.1 Pharmacology

Alcohol is unusual among drugs in several aspects of its pharmacology. These features produce not only the wide variety of biological effects of alcohol but also several important methodological variables. It is a small molecule that penetrates cell membranes at the same rate as water, is absorbed and distributed without an active transport system, is completely water-soluble leading to distribution according to the water content of the various organs and tissues, and exhibits marked inter-individual variability in kinetics (Dubowski, 1985).

When administered orally, alcohol is rapidly absorbed into the circulation by diffusion across the gastric and intestinal mucosa. A major influence on alcohol metabolism is therefore the presence or absence of food in the stomach and intestine. An empty stomach gives rise to higher blood alcohol levels (BALs) slightly earlier in the BAL-time curve while a non-empty stomach leads to lower BALs slightly later in the curve (Sedman, Wilkinson, Sakmar and Wagner, 1976).

Once absorbed, alcohol distributes into the blood, the body's water compartment and into those organs which have a high water content, principally, liver, brain, kidneys and spleen (Erickson, 1975). The peak equilibrium concentration reached is a function of the body composition (Devgun and Dunbar, 1990). The time-course of blood alcohol concentration is often referred to as the Widmark curve (Wallgren and Barry, 1970) after the researcher who first described the pharmacokinetics of alcohol in the 1930s. The theoretical Widmark curve is presented in Figure 3.1. It should be noted that BAL does not stay constant as the time course of metabolism is relatively short, especially for low to moderate doses, so that opportunities to test any effects of alcohol at a particular BAL are fleeting. After absorption and distribution processes are completed, the BAL decreases at a constant rate (Jones, 1984). Elimination of alcohol through oxidation to acetaldehyde occurs predominantly in the liver, although other tissues such as the kidney, muscles, lungs, intestine and brain may metabolise small quantities (Erickson, 1975).

1.3.2 Measurement of Blood Alcohol Level (BAL)

As the major site of the effect of alcohol on behaviour is the brain, direct measurement of brain alcohol would be desirable. However, brain ethanol concentration is commonly inferred from blood ethanol concentration as there appears to be no blood-brain barrier to alcohol. Breathanalysis for ethanol is used as an index of blood alcohol concentration based on the principle that ethanol in the blood is represented proportionally by the ethanol in deep alveolar air (Erickson, 1975). The limitations of breath-analyzing to obtain estimates of BAL are that samples are invalid for the first 20 minutes after ingestion due to residual alcohol in the oral cavity, that they vary with operator training and subject compliance, and that they tend to always underestimate the true blood ethanol concentration (Jones, 1989).
1.3.3 Effect of Alcohol Intoxication

Being a small, readily absorbed molecule that easily crosses membranes, means that alcohol has the potential to, and does, affect a wide variety of physiological systems (Wallgren and Barry, 1970). This makes the question of the site of action of alcohol difficult to answer since there may well be a multiplicity of sites of different types of action at different times in the elimination curve.

The major site of action of alcohol is the central nervous system (CNS) (Wallgren and Barry, 1970) where clear effects are apparent in those structures that are involved in highly integrated functions such as the reticular activating system (Himwich and Callison, 1972). Starmer (1989) argued that, broadly, the effects of alcohol can be:

"conceptualised as involving alterations of the afferent input from the sense organs and/or changes in the CNS, which confer a potential for disruption of the analysis of sensory information and the control of intricate movement patterns."

Many types of tasks have been used to measure the effect of alcohol on mental performance. They include reaction time (Young, 1970), pursuit rotor and coding tasks (Linnoila, and Mattila, 1973; Linnoila, Erwing, Cleveland, Logue & Gnatry, 1978; Vogel-Sprott, 1979), vigilance (Vogel-Sprott, 1979), numerical reasoning and perceptual speed (Franks, Hensley, Starmer and Teo, 1976), tests of intelligence (Jones, 1974; Parker and Noble, 1977) and tests of variables which are associated with skills involved in driving (Chesher, Dauncey, Crawford and Horn, 1986; Clayton, 1980; Huntley and Centybear, 1974; Mitchell, 1985).
Despite methodological variation (i.e. dose and protocol) the common finding of these types of studies is that alcohol significantly and dose-dependently impairs performance although differences exist between tasks as to the extent of the impairment (Chesher et al, 1986; Moskowitz, Burns, and Williams, 1985; Pandina, 1982; Starmer, 1989; Wallgren and Barry, 1970).

1.3.4 Individual Differences

Individual differences in alcohol metabolism and elimination are pronounced. Williams, O’Neill and Dubowski (1983) showed that even in a carefully controlled drinking situation, individual variation in peak BAL is considerable. The data indicated that two individuals, one receiving double the alcohol dose of the other, could have similar peak BALs. Both the rate at which alcohol is consumed by the human subject and his previous history of alcohol usage influence alcohol metabolism and the BAL attained. Slower drinkers have been reported to have a slower absorption and faster elimination rate than fast drinkers (Jones and Vega, 1973; Moskowitz and Burns, 1976). There is obviously a large degree of interaction between these factors and the genetic differences in alcohol metabolism and effects demonstrated in twin studies (Martin, Perl, Oakeshott, Gibson, Starmer and Wilks, 1985; Jardine and Martin, 1984; Propping, 1977).

Dubowski (1985) pointed out that not all blood and breath alcohol curves follow the Widmark curve. Significantly large short-term fluctuations from the expected curve occur in some individuals leading to the extreme difficulty of establishing peak and metabolic phase without frequent measures of BAL. These findings suggest that tight experimental control over the effect of a standard dose of alcohol on the human subject is somewhat difficult to attain.

1.3.5 Dose versus BAL

The dose of alcohol administered does not necessarily correlate with the resulting BAL. Alcohol concentration, rate of consumption, the amount of alcohol, individual differences in metabolism and body composition all affect the eventual observed BAL. The importance of this is that it is not the administered dose that should constitute the levels of the independent variable (I.V.) in alcohol experiments, but rather the actual BAL achieved. The same dose administered to full or empty stomachs will lead to quite different BALs. Therefore, using dose to define levels of the I.V. is unlikely to validly assess the actual effect of alcohol.

1.3.6 Summary

There are several problematic areas in studying alcohol effects. The first is that there is a poor correspondence between the administered dose and the resulting BAL. A variety of factors can have an influence on the characteristics of the alcohol absorption and elimination curve. As well as these methodological influences on BAL, individual differences in metabolism of alcohol, and susceptibility to its effects, can be large and unpredictable. Although it has been shown to have differential
effects on some tasks according to whether BAL is rising or falling, such limb effects have not been demonstrated for the whole range of abilities that alcohol can affect, nor has it been shown that limb effects are independent of BAL.

A further issue which impinges on the experimental study of alcohol is the influence of prior exposure to alcohol. If subjects have experienced the sensations associated with alcohol intoxication, placebo conditions that last for longer than about thirty minutes would be almost impossible to achieve. This means that comparison of an experimental condition to a true placebo control condition over several hours is not possible due to the subjects' familiarity with the experience of intoxication. Therefore, treatment effects will inevitably be confounded with expectancy effects (see section 3.1).

Taken together, these difficulties mean that alcohol intoxication is not a precisely manipulable independent variable. It is inevitable that even with controlled administration, there will be marked heterogeneity in BAL profiles across subjects. The ramification of this for studies seeking to use levels of intoxication as the independent variable (IV) is that depending on the magnitude of the discrepancy between doses administered, there may be overlap between levels in terms of BAL achieved, which may adversely affect the conclusions drawn. A solution is to treat BAL rather than dose as the IV and therefore to determine levels of the IV after having administered a range of doses.

1.4 PREVIOUS RESEARCH ON ALCOHOL AND VISUAL FUNCTION

1.4.1 Introduction

The literature was examined to determine the changes to the visual system which have previously been documented when a person or driver consumes alcohol. The early literature in this area commenced around the late 1930's and early 1940's, where a variety of minor studies have sought to measure these changes. In these studies, copious quantities of 'quality rye and scotch' were consumed in the relaxed setting of a cocktail party. Subjects were tested for a variety of changes in their visual function, including vision, visual field and oculomotor balance over the course of the evening. In later studies authors attempt to examine more closely the earlier findings through more intensive investigations into the specific aspects of change in visual function. During the 1980's the work in part seems to repeat the early studies, with some supporting evidence but often conflicting results to the earlier findings. As would be expected the design and subject selection is more experimentally sound in these later studies and statistical testing has been applied to results.

To accurately review the literature in this area it is most appropriate to examine the changes to the various components of the visual system with an aim to deciding on the changes that have been most often and most clearly documented. Visual function has been separated into the components of visual function and oculomotor function when discussing the previous studies in this area.
1.4.2 Visual Function

Colson (1940) as a result of personal subjective observations of reduced peripheral field and problems of dark adaptation after a champagne binge measured changes in visual function. No statistical testing was applied to results, however subjects showed no change in near or distance visual acuity, visual fields, color vision or dark adaptation. Newman and Fletcher (1941) with subjects at variable levels of intoxication, and with poorly defined criteria of significance found at least seventy per cent of subjects with a decrease in visual acuity. The subjects tested were not subjectively aware of this decline. They report 85% impairment at higher blood alcohol levels (BAL) of 0.1 and above. Moskowitz, Sharma and Shapero (1972) in an extensive study of visual change with alcohol show no significant change in static visual acuity. Wilson and Mitchell (1983) in their study of 10 subjects found a significant reduction in distance visual acuity which is inconsistent with other studies. Later in 1990, Hill and Toffolon note no statistically significant loss of visual acuity found with increasing BAL. No conclusive impairment of static visual acuity has been obtained at low BALs (Perrine, 1973). Some impairment of vision is found at moderate BALs (Bretcher, Hartman & Leonard 1955; Mortimer 1963). The consensus of opinion is that static visual acuity is rather resistant to alcohol, even when low contrast stimuli have been used.

Only one experiment is known where dynamic visual acuity has been tested. Impairment of dynamic visual acuity is generally thought to be more important in driving. Honegger, Kampschulte & Klein (1970) used a visual tracking device in which a single letter was projected on a screen and then rotated in a circle at selected speeds. They showed that dynamic visual acuity is significantly reduced during the alcohol diffusion stage and begins to improve during the alcohol equilibrium stage. The greatest decrement was 30 mins after completing drinking with some subjects with BALs as low as 0.02. Dynamic visual acuity was already impaired before the subjects reported subjective feelings of alcohol intoxication.

One of the sensory effects to the visual system which has been attributed to alcohol and considered to present a driving hazard is decrement of the visual field or "tunnel vision". This implies that there is a reduction in the extent of the lateral visual field while the eyes are fixated on a point straight ahead. Even relatively high doses of alcohol do not appear to cause any appreciable reduction in the extent of the lateral field of vision in terms of sensory sensitivity as documented by Colson (1940), Peters (1942) and King (1943) who show no change to this function. In the study by Newman and Fletcher (1941) 16% of subjects showed some decrease in their peripheral fields of vision although the scale of measurement used here is difficult to identify. The more recent work of Hill and Toffolon (1990) suggest that the mean horizontal and vertical visual fields decreased with increasing BAL. The constriction of fields documented was most marked when the BALs were highest. This finding is contrary to other work.

Interest has moved to functional visual field examination tasks that require divided attention, that is time sharing of the fovea and an extra foveal task more or less simultaneously and/or the processing of information. Von Wright and Mikkonen (1970) found significant performance deficits at a BAL of approximately 0.05 when
tracking and visual recognition tasks were combined. Moskowitz and Sharma (1974) examined the detection of peripheral stimuli and autokinesis with subjects under the influences of alcohol and also marijuana. Alcohol was found to impair the central processing of peripheral visual information when processing of that information conflicts with processing of information from other sources. The deficit is in the ability to divide attention or "time share". Autokinesis refers to the phenomenon wherein a stationary point source of light viewed in the dark appears to move. The effect of alcohol is not significant to this function. It was concluded that the effect of alcohol on peripheral vision was a function of the information load on the central vision.

Dark adaptation in terms of detection of low contrast targets is impaired by BALs of 0.08 or higher. In the study by Moskowitz et al (1972) dark adaptation at the light levels administered in the study are not influenced significantly by alcohol. Miller, Pigion and Takahama (1986) confirm this result showing no systemic effect on dark adaptation. Reduction of glare resistance has been cited as a potential driving hazard although no studies provide strong evidence for a consistent influence of alcohol upon glare tolerance, resistance or recovery (Newman and Fletcher, 1941; Mortimer, 1963).

Critical flicker fusion refers to the transition point at which an intermittent rapidly flickering light source is first perceived as being continuous or fused. It is used as an index of the temporal resolution or acuity of the the visual system as well as an indicator of central nervous system function and condition. Literature offers differing conclusions concerning the effects of moderate doses of alcohol on critical flicker fusion. The results of the available studies generally indicate that the temporal acuity of the visual system is impaired by BALs of approximately 0.08% (Hill, Powell and Goodwin 1973; Carpenter, 1962) but below this level the findings are controversial. The concern here is that in the general population the range of normal results is not clearly defined.

The literature is inconsistent when studying the impact of alcohol on accommodation. This is the ability of the lens to change shape in order to focus on near objects. Hogan and Linfield (1983) show that there is a decrease of the stimulus accommodative convergence to accommodation (AC/A) ratio by 2 methods of assessment, producing a 22-24% reduction in the AC/A ratio. In this study the amplitude of accommodation is not affected at moderate ethanol levels. Miller Pigion and Martin (1985) show a tendency for accommodation to increase as subjects became intoxicated. This is the case for dark focus, near and far static accommodation. Hill and Toffolon (1990) show no change in the amplitude of accommodation as tested on the Royal Air Force (RAF) gauge.
1.4.3 Oculomotor Function

The movements of the eyes are the fastest and best controlled movements of which the body is capable. Even low doses of alcohol are capable of producing specific decrements in oculomotor function. The changes to saccadic eye movements which are the rapid conjugate shifts of gaze to follow a target will be discussed in the following section of the report when our results to the measurement of this function are revealed. However other aspects of eye movement function are important here.

Colson (1940) found an obvious change in muscle balance showing an increasing esophoria at far distance in all subjects in his study with 2 subjects acquiring manifest strabismus with diplopia. He concluded that the diplopia that results from the alcohol is associated with a reduction, not an increase in convergence power and is dependent upon the development of esophoria too great to be overcome by the abduction power, although this remains unchanged. Powell (1938) also shows this tendency to esophoria in his study of 7 cases of intoxicated persons. Charnwood (1950) in a study of 4 subjects examined the change in vertical fusional ability after alcohol. He showed that alcohol reduces the subjects power of overcoming vertical prisms. Seedorff (1956) entertained his 7 subjects at a cocktail party after which he found that there was a tendency to esophoria after alcohol and deterioration of motor fusion reserves. The author also demonstrates the failing capacity for sensory fusional ability in his subjects although this change is not as marked. The decrease in oculomotor balance correlates with increasing alcohol levels. Newman and Fletcher (1941) find 72% of subjects showed change in eye co-ordination although it is unclear as to the actual change in this function. Overall there is a poor correlation between blood alcohol concentration and changes in visual function in this study. The authors suggest that there is a tendency for those with higher concentrations to show more change but this is far from true in every case and indicates the importance of recognizing the limitations of blood alcohol concentration as the sole criterion for intoxication.

Bretcher et al (1955) again show increasing esophoria at 6 metres and the development of exophoria at 33cm. Binocular fusion was retained at 82cm while it was decreased at 6m and 33cm. Fusion time became progressively longer with increasing alcohol intoxication. They argue that the strength of the fusion reflex is determined by the time that was necessary to elicit it. Power of voluntary convergence were also progressively impaired at higher alcohol levels. Vertical heterophoria is not significantly changed by alcohol intoxication. Moskowitz et al (1972) also demonstrate that there was no significant influence on vertical heterophoria. They demonstrated a mean change of 1.7 dioptres towards esophoria on distance heterophoria measurement. Vertical fusion and positive fusion were not significantly changed by alcohol however there was a decrease in abduction which seems to coincide with other studies.

In the study by McNamee, Piggins and Tong (1981) alcohol was found to produce a relatively esophoric shift without affecting vertical muscle balance as measured on a commercial vision screener. The BAL was 0.08% although changes are noted at BAL of 0.4%. Wilson and Mitchell (1983) in a study examining change at low BALs confirm the presence of esophoria at distance viewing and exophoria at near
viewing. Measurement of convergence near point was significantly decreased over time.

Hill and Toffolon (1990) also found that the amplitude of convergence tested on the RAF rule significantly decreased. Hogan and Linfield (1983) found that there is significant change in the near point of convergence with a mean decrease of 2cm. No change in positive fusional ability was found. The 10 subjects in this study were tested at moderate BAL levels of approximately 0.07, but all were ophthalmology students who were possibly already familiar with the measurement procedures. The results to measurement of heterophoria show a mean change at distance of 2.45 dioptries in the esophoric direction. There was a mean change of 1.15 dioptries towards exophoria at near, this was not tested but probably is not statistically significant. The measurement of negative fusional reserves is statistically significant with 75% of subjects showing a decrease in this ability. The measurement of positive fusional ability show no significant change which the author claims endorses the work of numerous other authors.

In the study by Miller et al (1986) intoxication produced a tendency toward increasing fusional (without accommodative stimulus) convergence for far viewing and increasing fusional divergence for near viewing. As is consistent with a number of studies already mentioned there was increased esophoria for far viewing and increased exophoria for near viewing. This is explained as a change in accommodative vergence. There is no systemic effect of dark vergence and the changes in this study were easier to demonstrate at a far viewing distance. Hogan and Gilmartin (1985) decided that a moderate dose of ethanol had no effect on tonic convergence or accommodation whilst producing the well documented changes in heterophoria, AC/A ratio, fusional ability and convergence near point. Again this study documents no change in the convergent fusional ability.

Defects in binocular co-ordination might be expected to cause impairment of depth perception. Wist et al (1967) reported that a BAL of 0.06% caused fixation disparity to increase significantly but stereoaucity was unaffected at this level. Hill and Toffolon (1990) confirmed this showing no significant change in stereoaucity in their subjects.

A number of miscellaneous findings are of interest in the literature regarding changes to ocular muscle balance. In the study by Wilson and Mitchell (1983) 2 changes have been found which have not been documented elsewhere. All subjects developed some inferior oblique overaction. They also find 2 subjects with unequal pupil reactions to light with one eye being more sluggish than the other, a finding also not documented elsewhere. Pitt and Lewis (1988) investigated pupil cycle time as a measure of autonomic nervous system function. They were interested to note change of this reflex activity with a variety of physiological variables. Pupil cycle time was measured at 10 minute intervals for 40 minutes after the ingestion of alcohol. Subjects showed a significantly shortened pupil cycle time 30 minutes after alcohol.
There are few reports in the literature regarding the specific influence of alcohol on the saccadic and smooth pursuit eye movement systems prior to 1974. Saccadic movements are the fine, fast motor movements of the eyes which allow accurate fixation from point to point. A typical saccade is initiated approximately 150 milliseconds after the target stimulus is activated and peak velocity is rapidly achieved. This speed can exceed 600 milliseconds. Normal subjects can miss the target by undershooting or overshooting which necessitates a secondary corrective saccade. Peak velocity increases with the amplitude of the movement reaching a maximum at saccades of approximately 30 degrees. These properties make saccades the fastest and best controlled movements of which the body is capable.

Wilkinson, Kime and Purnell (1974) examined the eye movements of 6 subjects who were tested for one hour after the consumption of 200ml of 25% alcohol. 30 degree saccades were measured. Results indicated that post alcohol the peak velocity was 81-84% of the pre alcohol result. These results match the results of Frank and Kdo (1970) well who observed a 24% reduction in peak velocity at BALs of .08 to .10. Wilkinson et al also found a change in the smooth pursuit system. This is the ability of the eyes to conjugately follow a moving target, keeping foveal fixation, in each eye. They found that after alcohol these pursuit movements became jerky as though the ability to follow was impaired requiring correction with catch-up small saccades. Dolls head movements were intact so they concluded that alcohol effects cerebral function earlier and to a greater extent than mid brain and brainstem function.

Lehiten, Lang, Jantti and Keskinen (1979) measured control and alcohol conditions in 16 subjects. The features of 20 degree saccades analysed were latency, mean and peak velocity and duration. They noted the most change 90 to 120 minutes post alcohol consumption with no significant change in latency, but a 9% decrease in in peak velocity and an 11% increase in duration of saccadic movements. The changes in peak velocity and duration correlated more closely with feelings of intoxication than with blood alcohol level. The range of BAL was .05% to .11% in this study. The decrease in peak velocity in this study is less than what is reported in other studies even though the amounts of alcohol were comparable. The differences found were explained in terms of technique of measurement, choice of subjects and inadequate control groups. Fatigue and reduced attention have also been shown to increase the duration of saccades and decrease the peak velocity but in this study the authors negatively correlated intoxication with tiredness and decrease of subjective eye movement control.

Katoh (1988) again showed the slowing effect of alcohol on voluntary eye movements. 6 subjects consumed 1.0 ml/kg of alcohol. Results show a decrease latency of between 8 and 17% and decreased velocity of between 17 and 25%. Heller and Lucke (1987) examined a variety of amplitude saccades in their study involving 6 subjects with BAL of 0.08. They show no effect of alcohol on saccades of less than 12 degrees and just a 7% change in larger amplitude saccades. This is the first finding indicating no change at some amplitudes although saccades of smaller amplitudes have not been commonly reported in the literature. It is commonly agreed that the larger the amplitude of the saccade becomes the less accurate it becomes. Lancaster (1941) argued that 99% of saccades are less than 15 degrees which may contribute to their relative accuracy.
Blomberg and Wassen (1962) examined optokinetic ability of 21 subjects by increasing the angular speed of optical stimulation until the eyes could no longer follow the black and white bands on the optokinetic stimulus. BALs of between 0.08 and 0.16 showed a significant decrease in this ability. This response is similar to the action of some barbiturates on the optokinetic response. The authors do not imply the exact site of action of alcohol on the central nervous system.

A number of studies have documented the induced nystagmus resulting from alcohol ingestion. This is repetitious oscillations of the eyes when the subject is placed in the horizontal position and is referred to as positional alcohol nystagmus. Howells (1956) examined this nystagmus produced by alcohol. In all subjects nystagmus was produced after a variable period of time. 50 ml of absolute alcohol for a person of average weight seemed to induce this physical sign. In the study by Wilson and Mitchell (1983) 2 of the 10 subjects studied, developed end point nystagmus. All subjects displayed nystagmus as a result of the action of ethanol on the vestibular organ and cerebellum in the study by Seedorff (1956).

It has been suggested that oculomotor function is of more importance than visual acuity in the ability to drive safely. From the literature it can be seen that the functions most consistently influenced by the ingestion of alcohol over a range of blood alcohol levels are particular components of ocular motor control of the eyes. There is some evidence for changes in visual abilities and changes to the visual field but these findings are not consistent or seem to occur only at the higher BALs. The other concern here is that studies into visual field and information processing are still in an early stage of development. The incidence of nystagmus after alcohol ingestion is well known and in fact forms the basis of a roadside sobriety testing device which has been piloted in a number of states in America.

The oculomotor system endeavours to maintain comfortable binocular co-ordination and binocular single vision. Numerous authors demonstrate the tendency for esophoria to develop at distance viewing and the tendency towards exophoria at near viewing. There is no change to vertical heterophoria but there is a commonly associated decrease in the negative motor fusional reserves and some decrease in the ability to converge at near range. The change to some components of saccadic eye movements, namely peak velocity and latency although variable are consistently reported. In view of these well documented oculomotor changes it appears that aspects of oculomotor function are most likely to show strong effect of alcohol.
2

THE EFFECT OF ALCOHOL ON VISUAL FUNCTION

2.1 INTRODUCTION

It is clear from the literature that some sensory, fine motor and autonomic functions of the visual system are susceptible to the influence of alcohol. There were two aims in this study. The first was to confirm the findings in literature that alcohol adversely affects visual function. The second was to test for this effect using a more rigorous and systematic methodology than that used in previous studies.

Although explicitly discussed in the preceding section, there was no attempt to correlate deficits in visual function and decrements in psychomotor performance. There were two reasons for not including a psychomotor task, one logistical and the other conceptual. The conceptual reason was that if traditional measures of psychomotor performance under alcohol are subject to expectancy and practice effects, and are therefore poor estimates of the degree of incoordination, then the correlation between oculomotor function and these traditional measures would be poor if oculomotor measures were good estimators of incoordination, and very poor if they were also subject to expectancy and practice effects. The logistical reason was that because of the dynamics of alcohol absorption and elimination, there is only a short period of time during which a set of tests can be conducted so as to have been conducted at approximately the same BAL. The relationship between psychomotor performance and alcohol-induced changes in visual function is best explored in subsequent research.

It was expected in this experiment that all measures of visual function would be affected by alcohol and that the effect of alcohol would be as a decrement in performance abilities. It was also expected that the effect of alcohol would be dose dependent in the sense that the effect would be proportional to the BAL. A difficulty in demonstrating the latter outcome is that there is a less than perfect correlation between dose and BAL. This means that in group experiments where groups are defined according to administered dose, and with the level of individual differences in alcohol metabolism, there is a risk that a not too great a difference between doses, will lead to little difference between recorded BALs. In order to avoid this problem, this experiment used groups defined according to average recorded BAL. In this way it was possible to minimise heterogeneity between groups with respect to BAL and thus be able to explore the effect of actual alcohol 'dose'.
2.2 METHOD

2.2.1 Subjects

Subjects were male university students who volunteered in response to campus advertisements. A total of 44 subjects participated in the experiment. The subjects tested were aged between 18 and 30 years, \((M = 21.8, SD = 3.8)\). Body weight ranged from 55 to 95kg \((M = 72.7, SD = 9.2)\). All subjects reported themselves to be social drinkers (see Appendix B for Drinking History Questionnaire, and Appendix C for a summary of the subject's responses). Subjects were informed of the aim of the experiment when they attended an initial interview. This interview was designed to establish whether their vision was normal, and to explain the testing procedure and the requirements of the study.

The criteria to determine normal visual function were:

- no known history of ocular or visual pathology
- visual acuity of 6/9 or better with or without refractive correction and no more than one Snellen line difference between 2 eyes
- no manifest squint at either 1/3 metre or 6 metres
- full eye movement excursions
- binocular single vision as demonstrated by a result of at least 120 secs of arc on the TNO test for stereopsis

2.2.2 Design

Subjects were assigned to one of three experimental groups. One group was a no-drug control and received only some water. The other groups received one of two body-weight related doses of alcohol estimated to induce low to moderate levels of BAL during the course of the experiment.

Two independent variables were investigated in this experiment: alcohol condition (three levels: zero, low and moderate BAL) and test (three levels). Measures of visual function for each subject were taken before drinking (Test 1), while BAL was rising (Test 2) and when BAL was falling (Test 3). With alcohol condition a between-subjects factor and test a within-subjects factor, this experiment utilised a two-way repeated measures design with one repeated measure.

2.2.3 Instrumentation

Standard clinical testing procedures were used in this study. The following test were performed in a laboratory with subdued lighting:
**Heterophoria (latent deviation of the visual axes)**

This was measured with a hand held Maddox Rod at 6 metres, then 1/3 metre. The Maddox rod is placed over the subjects right eye allowing fixation of a spot light with the left eye. A prism bar or single prisms where appropriate with the base in the direction required to move the line to the centre of the spot light were introduced on top of the Maddox Rod until the subject indicated that the red vertical line image produced by the rod intersected the centre of the spot light. Subjects were asked not to focus on the target but to relax their vision when completing this testing procedure. If subjects complained of peripheral reflection the position of the rod was checked to be flat across the bony orbit of the subjects right eye.

**Positive and negative fusion range**

This function was examined on the Clement Clarke synoptophore, an instrument designed to measure the 3 grades of binocular single vision, that is simultaneous perception, fusion and stereopsis. Objective angle of heterophoria present was first determined with paramacular simultaneous perception slides. Subjects were asked to place the 'lion on the cage' by moving the left tube of the synoptophore with there left hand in the required direction. The lion stimulus was placed in front of the left eye and the cage stimulus in front of the right eye.

The negative range of fusion was assessed first with fusion slides. Using the 'cat' slides the locked mechanism of the instrument was rotated slowly in an outward direction in front of both eyes and the subject was asked to decide when the single image had split into 2 definite images. That is, the subject was asked to indicate as soon as there was a split and space between the two cats.

Positive vergence was then tested with the locked mechanism rotated in an inwards direction until the subject noted the split of one image into two. Here the subjects response was checked by the investigator noting the objective break of fusion by the loss of binocular convergence. If the subject was not adequately responding to the task required, three dioptre sphere concave lenses were introduced to the viewing barrels of the synoptophore in an effort to stimulate convergence power. 3 assessments of both negative and positive fusion amplitude were measured and recorded. The mean of the 3 results was calculated to complete statistical analysis.

**Edge light pupil cycle time**

This was measured on an Inami slit lamp binocular microscope. Subjects were asked to fixate on a 3 cm square placed at the line of sight on a blank wall at a 2 metre distance. A rectangular beam of light 5mm length and 0.6 width with maximum light intensity from the illumination system angled at 90 degrees was projected and focused on the lower limbal margin. The subject sat comfortably with their head pressed firmly against the forehead rest to ensure a static position. The light was raised into the lower pupil until induced pupil cycle response commenced. 20 full cycles were timed with a Lorantz digital stop watch which records to 2 decimal places. The result was recorded and rounded to 1 decimal place. The procedure was repeated 5 times.
Horizontal saccadic eye movements
The Intelligent Eye Monitor System comprises an infrared illumination device, video camera and tracking system. It is linked to IBM computer which generates eye movement sequences and records and stores the data obtained. Subjects were positioned with chin and forehead restraint to minimise head movement. The test stimulus comprised a horizontal bar of red light emitting diodes (LEDs) at a distance of 2 metres. The testing procedure was explained to the subject with particular emphasis on the need to resist anticipating the position of the next LED in the test sequence. Subjects were asked to fixate the light then change fixation when the next stimulus in the test sequence was introduced. After practice with a random sequence generated by the computer, 3 sequences each of 10 eye movements of 20 degree horizontal saccades were measured and recorded. Right eye was measured for a centre/left, centre/right and centre/ left and right combined measurement sequence. The sequences were introduced in a previously determined random order for each subject. Results were stored for later analysis.

2.2.4 Alcohol and Breathanalysis
Subjects were requested not to consume alcohol the night before the experiment and to fast for five hours prior to reporting to the laboratory. Subjects were also requested to abstain from coffee or tea after 9.00 am.

The low dose group received 0.4 ml of ethanol per kilogram body-weight (kg bw), made up in the following manner: 1.0ml/kg bw of 40 proof vodka. The moderate dose group received 0.8 ml of ethanol per kilogram body-weight (kg bw), made up in the following manner: 2.0ml/kg bw of 40 proof vodka. Each dose was administered mixed in a 1:4 ratio with chilled orange juice. The total volume of alcohol and orange juice was divided into 4 equal drinks which the subjects were instructed to consume evenly over a 20 minute period. At the end of the drinking period subjects thoroughly rinsed their mouths to remove any residual alcohol.

Analysis of BAL was performed using a breathalyzer (Lion Laboratories Alcometer SD2). Subjects were prevented from seeing the digital readout and remained ignorant of both their BAL and the exact amount of alcohol consumed until debriefing at the end of the experiment.

2.2.5 Procedure
The testing sessions were conducted in the clinical and research areas of the Division of Orthoptics, Lincoln School of Health Sciences LaTrobe University. This first experiment was conducted on the Carlton Campus. The testing session commenced at 2pm and concluded at approximately 5.30pm. Three subjects were tested during each session. A timeline of the testing session is presented in Figure 2. On arrival subjects gave informed consent (see Appendix D) and drew marbles from a bag to allow random assignment into the 3 groups, ie control, low dose and moderate dose. Allocation was known to the subjects. Drinking subjects were not told the amount of alcohol they be would be consuming or whether they were low or high dose subjects.
Weight was measured on digital scales and recorded. Any subject questions were answered by the investigator. Subjects were questioned as to whether they had complied with the instructions which had been outlined in the initial interview.

The order of measurement was as follows:

- heterophoria.
- positive and negative fusion range.
- edge light pupil cycle time.
- horizontal saccadic eye movements.

This series of measurements was completed 3 times over the course of the afternoon, constituting a baseline (Test 1), and 2 examinations post alcohol (Test 2 and Test 3). Subjects had a 35-40 minute break between each test. Each test took 20-25 minutes.

A baseline BAL was measured prior to drinking to determine freedom from alcohol and to allow subjects familiarity with the testing device. BAL was then measured before and after each series of measurements (BAL tests 1-4) and prior to debriefing. The break of 35-40 minutes between each testing series was spent in a designated room where subjects were able to chat, study, listen to music or read. A relaxed environment was encouraged. Control subjects followed the same procedure without the requirements of alcohol ingestion or further breathalyser measurements once their BAL had been revealed to be 0.0.

At the end of the session all subjects were given feedback regarding the results of their ocular tests and drinking subjects told about the blood alcohol levels attained over the course of the session. Subjects were offered $20 for their participation and, if required, offered a taxi to their homes. Any further discussion regarding the experiment or experimental aims was completed and subjects were thanked for their co-operation and participation.

2.2.6 Data Analysis

Because of the imperfect correlation between dose and BAL discussed above [see section 1.3.5] low and moderate BAL groups were defined prior to data analysis on the basis of the highest BAL recorded. A BAL of 0.04 was the cutoff for the two groups. This resulted in groups sizes of 12 (control), 17 (low BAL) and 16 (moderate BAL).

In order to minimise the influence of baseline differences between groups on interpretation of results (through erroneous interactions), scores on each of the dependent variables was expressed as an absolute difference from the value obtained on Test 1. This was done assuming that each dependent variable is measured on an interval scale, and thus amount of change throughout the range of values measured represents the same amount of change in the underlying variable.

The appropriate analysis for this experimental design is a two-way repeated measures ANOVA with one repeated measure. For each of the saccadic variables...
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The appropriate analysis for this experimental design is a two-way repeated measures ANOVA with one repeated measure. For each of the saccadic variables
there was a third within-subjects independent variable, version (adduction and abduction). The appropriate analysis for this experimental design for this variable was therefore a 3-way with 2 repeated measures ANOVA. Working with change scores tends to produce group main effects at the expense of interactions. In the present study it was expected that evidence for an effect of alcohol on visual function would be apparent in a significant group main effect. A group x test interaction, either alone or in combination with a group main effect, would also be indicative of an alcohol effect. It was not expected that there would be a test main effect when using change scores as these tend to reduce the size of any time effect. Rejection of statistical null hypotheses was set at $p \leq .05$.

Figure 2. Procedure timeline for experiment 1.
2.3 RESULTS AND DISCUSSION

2.3.1 Blood Alcohol Level

The mean blood alcohol level (BAL) at each of the four main breathanalysis test times is presented in Figure 3. The mean BAL during T2 was 0.027% for the low BAL group and 0.047% for the moderate BAL group. The mean BAL during T3 was 0.023% for the low BAL group and 0.055% for the moderate BAL group.

![BAL Chart](image)

Figure 3. Mean Blood alcohol level (BAL) for each group at the four breath-analysis measurement times.

2.3.2 Heterophoria

The mean change in measures of distance and near heterophoria are shown in Figures 4 and 5 respectively. It can be seen that for both measures, the degree of change was related to BAL with the greatest effect being found in the moderate BAL group. In both cases the control group showed little change. Both a group main effect \([F(2,42) = 6.55, p = .0033]\) and interaction \([F(2,42) = 3.97, p = .0264]\) were significant in heterophoria-near. For heterophoria-distance, only the group x test interaction was significant \([F(2,42) = 3.36, p = .0444]\).
Figure 4. Change from Test 1 on Tests 2 and 3 for each group on measures of Heterophoria-distance.

Figure 5. Change from Test 1 on Tests 2 and 3 for each group on measures of Heterophoria-near.
The findings regarding the change in near and distance heterophoria measurement are generally consistent with those reported in the literature. The low dose group showed a mean change of 0.5 prism dioptres towards esophoria and the moderate group a mean change of 1 prism dioptre. There was a very slight change in the control group but the tendency here is in the opposite direction and towards exophoria indicating the tendency of distance heterophoria to remain stable on repeated measures or to become less esophoric. This indicates that the measurement of the latent position of the visual axes is relatively static. There was obviously an effect on this function due to the ingestion of alcohol with the relative position of the visual axes assuming a more convergent position. These findings are significantly different from the results of the control group. A control group is a feature that is lacking in the majority of studies which have investigated the influence of alcohol on the visual system.

The results of near heterophoria change over the course of the session are of greater magnitude than the distance measurement. Our findings are somewhat contrary here as the greater change has been found by other authors to be at distance measurement. In this study there was a mean change of 1.3 prism dioptres towards exophoria in the low group and a total mean change of 2.1 prism dioptres in the moderate group. The control group move slightly towards esophoria and then towards exophoria over the course of the testing session. This is not a significant finding and indicates the reliability of this measurement in the control group. The movement towards exophoria was marked in the moderate group again showing a greater change with a larger amount of alcohol demonstrating an increasing effect with increasing BAL. The results of this study indicated that the effect at near distance with the finding of increasing exophoria would indicate that this function was more influenced by alcohol than the measurement of distance heterophoria. The latent position of the visual axes for near viewing is more critical than for distance viewing in the process of focusing with greater demands being made on the visual system at close distance. Other functions of the visual system influence this latent position at near viewing, namely convergence and accommodation. An investigation of the extent to which these functions are influenced by alcohol requires investigation and description, as we have endeavoured to do in the next experiment.

Powell (1938), Wilson and Mitchell (1988) explained these changes in heterophoria position as a decrease in muscle tone and muscular control. They suggested that there may be depression of the cortical centres which control muscular activity resulting in inco-ordination of extrinsic and intrinsic ocular muscles through the depression of the reticular activation centre and the parapontine reticular formation. Some of Colsons' (1940) subjects who were taken to higher levels of intoxication in fact have a breakdown of their latent deviation into a manifest deviation resulting in the inducement of a double (diplopic) image. This occurrence is anecdotally also known to be a finding of the very intoxicated person. A symptom which becomes disturbing is there is an inability of the person to suppress the induced double (diplopic) image. This was not a finding in this study which is due to the lower levels of intoxication measured. It is likely that we would see these changes also at higher levels because the tendency toward the breakdown of this function is already present at the BALs administered. Colson (1940) hypothesized that this finding is associated with increasing convergence power which causes an esophoria which is too great to
be overcome by the power of abduction which remains unchanged. Charnwood (1950) explained this change as decreasing muscular power which inhibits the ability to overcome the resulting heterophoria. He also indicated that diplopia is induced which could not be suppressed by the cortical processing centres. He stated that the presence of exophoria at near is due to a toxic irritative effect of alcohol on the sixth cranial nerve which can be likened to the the early stages of anaesthesia where this effect induces a divergent strabismus. Miller (1986) explained the increasing exophoria as being related to changes in accommodative vergence.

The results of this study indicated that there was a decrease of neuromuscular ability under the influence of alcohol, with the changing position of the visual axes and an obvious increase in the amount of heterophoria present. Our subjects displayed an increasing exophoria at near and an increasing esophoria at distance. This occurs because of the decrement in muscular ability of the medial recti at near range and the lateral recti at distance range. This refutes the statement by Colson that there is an increase in convergence power and hence tendency towards esophoria at far distance.

2.3.3 Fusion

The mean change in measures of divergent and convergent fusion are shown in Figures 6 and 7 respectively. It can be seen that for both measures, the degree of change was related to BAL with the greatest effect being found in the moderate BAL group. In both cases the control group showed little change. Only main effects for group were significant in each measure \[ F(2,42) = 3.49, p = .0396; F(2,42) = 13.35, p < .0001. \]

![Diagram](image_url)

**Figure 6.** Change from Test 1 on Tests 2 and 3 for each group on measures of Fusion-divergence.
Changes in fusion range showed a tendency for greater change at the moderate BAL level. In the measurement of divergent range there was a mean decrease of 1 degree in the moderate BAL group and a very small shift of 0.25 degrees in both the low and control groups. The change in the control group can be explained as a slight fatigue of this function on repeated measure. It would appear again that small doses of alcohol did not severely impede this function but at the increasing BAL the change was more obvious. When measuring the convergent or positive range there was a mean decrease of 1.7 degrees in the low group over time and a decrease of 4.75 degrees in the moderate group. Bretcher et al (1955) argued that the decrease in fusion power is due to impairment of neuromuscular co-ordination by alcohol which at the same time affects the position of the visual axes and diminishes the power of voluntary convergence. They suggested that this function may be controlled by separate divergence and convergence centres and the removal of these mechanisms by alcohol causes the eyes to assume an intermediate or neutral position. Miller et al (1986) also showed that there is some regression of the system towards the tonic position which may be as a result of direct muscle tone or just an artefact of alcohol decreasing the fusion range with the tonic position being somewhere in this range. The overall effect here is for the eyes to be aligned at an intermediate resting position. Some authors found no change in the positive (convergent) range but a decrease in the negative (divergent) ability supporting the theory of changed muscle tonus and a biasing of the system towards a differing zero position of the eyes and hence a differing intermediate resting position. The resting position of the eyes at which alcohol does not cause heterophoria to change is about 60 cm. Our results indicated an overall decrement of the system with changes in both directions of the
fusion range supporting the authors who explained an alternative neuromuscular influence of alcohol on the motor control of the eyes.

2.3.4 Pupil cycle time

The mean change in each group for the measure of pupil cycle time is shown in Figures 8. It can be seen that there is little difference between the groups. No effects were statistically significant.

There is a variety of opinion as to the effect of alcohol on the autonomic functions of the ocular system. Some authors set out to examine the changes to accommodation, these studies investigated different components of this complex mechanism and the results have been varied. Lewis and Pitt (1988) measured pupil cycle time as an objective measurement of autonomic nervous system function. In this very small study of 6 subjects a significant quickening in the timing of pupil oscillations is reported. This study did not control for BAL. In our study which compared both control and a range of BALs the results showed no significant change in timing both over time and over groups. This would indicate at the levels of alcohol measured this autonomic nervous system function is not influenced by small to moderate amounts of alcohol. Further studies at higher BALs would have to be conducted to confirm this for all levels of alcohol consumption. It is suggested that at higher levels more specific change would be seen as we know that alcohol has impact on other facets of autonomic nervous system function.

![Figure 8](image-url)

**Figure 8.** Change from Test 1 on Tests 2 and 3 for each group on measures of mean pupil cycle time.
2.3.5 Horizontal Saccades.

The 3-way ANOVA for latency showed no significant effects, whereas that for peak velocity revealed a significant group main effect \( F(2,37) = 3.91, p = .0289 \) and a significant main effect for test \( F(1,37) = 10.01, p = .0031 \). Figures 9 and 10 shows these effects.

**Figure 9.** Main effect of group for the change in peak velocity of horizontal saccades from Test 1.

**Figure 10.** Main effect of test for the change in peak velocity of horizontal saccades from Test 1.
At the alcohol levels tested there is no significant change in the latency of 20 degree horizontal saccadic eye movements either between the groups or over the course of the testing session. The data showed very little change in all of the groups. There is a significant difference between the groups on peak velocity of 20 degree horizontal abduction and adduction saccades. Over tests, the control group showed a slight decrease (1.2%) possibly due to fatigue, the low BAL group showed a slight increase (2.3%) while the moderate BAL group showed a marked reduction (8.6%) indicating a slowing of peak velocity compared to their pre alcohol performance.

It is interesting to note that the low BAL group with concentrations of blood alcohol of less than 0.04 do not suffer the decrease in peak velocity seen in the higher BAL group. In fact this group if anything slightly increase the peak velocity result which may be indicative of a tendency towards a biphasic effect where an improvement of performance is initially seen but then results recede either over time or with increasing BAL concentrations. A cautious response to these results however would indicate that the saccadic system of eye movement is not significantly effected at the low concentrations administered to these subjects. Other reports in the literature do not examine the influence of alcohol at such low doses as those administered in this group of subjects. Significant change is seen at levels greater than 0.05 in other studies.

Our findings in the moderate BAL group correlate well with other studies in the area who find a percentage reduction of between 7 and 25%. Our moderate BAL subjects show a mean decrease slowing of 9% in peak velocity. This is the first known study where assessment of horizontal saccadic movement has been examined with the Intelligent Eye Movement System which has just recently been developed as a non invasive technique of eye movement assessment. Other studies utilize electroocuIarography and purpose built systems in this assessment which may in part contribute to the range in results found by a number of authors.

From the literature it appears that the most used measure of drug effects has been peak saccade velocity. The literature also suggests that latency as a measure of drug effects is inconsistent. We support those authors who do not find a significant increase in latency duration as a result of alcohol consumption. Jantii et al (1979) report a decreasing latency duration just 15 minutes after alcohol consumption indicating a biphasic effect. The variation in this result can also be attributed to the difficulties experienced in accurately measuring this component of saccadic eye movement and the well known individual variations in this result. Reaction times have been shown to vary from session to session (Jantii et al, 1979).

Wilkinson et al (1974) explained the changes in peak velocity found in their study as alcohol effecting cerebral function earlier and to greater extent than mid brain and brain stem function. They are concerned that this imperfect ability to accurately and quickly fixate periperal stimuli must contribute to the other known sensory, motor and co ordinate performance decrements experienced by the intoxicated subject. We would endorse the comments regarding slowing of eye movements as an added barrier to the ability to drive safely in view of our findings.
Katoh (1988) reported a slowing of latency of 8 to 17% and impaired saccadic velocity of between 17 and 25%. Task complexity in this study affected latency but not the velocity results. Jantii et al. (1979) report a mean saccade velocity and peak velocity decrease of 9%, a finding very similar to our study and generally a lesser percentage than other studies. This can be explained by a lack of adequate control groups and the lack of naive subjects in other studies and as previously mentioned the variety in method of assessment of saccades. The authors suggest that the possible site of location for the influence seen is the mesencephalic and pontine reticular formation where the frontomesencephalic saccade controlling and occipitomesencephalic pursuit controlling pathways meet. They suggest that alcohol, sedatives and fatigue may all operate at the same brainstem level.

Baloh, Sharma Moskowitz and Griffith (1979) with their subjects at BALs of between 0.05 and 0.10 report more of an impact on saccades than pursuit with latency increased by 32% and peak velocity decreased by 12%. The results are greater in larger amplitude saccades. Drugs effect the para pontine reticular formation and hence the supranuclear control of saccadic and smooth pursuit eye movements. They hypothesize that alcohol decreases transmission through the pathways in the parapontine region.

Our findings both for the measurement of horizontal saccades and the measurement of other components of oculomotor control would support this idea of the influence being neurological rather than muscular, with the site of action of alcohol being at brainstem level.

2.3.6 Summary

Oculomotor aspects of visual function, that is measurement of heterophoria at near and distance, measurement of positive and negative fusion range and the peak velocity of horizontal saccades demonstrated a significant change as a result of consuming alcohol over the course of the testing session. This is more marked in the moderate BAL group than the low BAL group. Results of pupil cycle time assessment indicate no influence of alcohol at the range of levels measured. The control group results to all aspects of oculomotor visual function assessed remained static over the course of the testing session.
3

ALCOHOL EXPECTANCY EFFECTS
AND VISUAL FUNCTION

3.1 INTRODUCTION

A major problem in studying the effects of an acute dose of alcohol is that since subjects are always experienced drinkers, they have strong expectations regarding the way alcohol affects them (Laberg and Lörberg, 1989; Leigh, 1989; Marlatt and Rohsenhow, 1980). This awareness of possible effects caused by familiarity with alcohol creates a problem for control in experiments on acute effects of alcohol that is largely absent in studies of other less common drugs. The usual double blind design approach is appropriate for those studies where it is possible for the experimenter to subtly influence the performance of subjects. Although it successfully controls for experimenter bias, this design does not provide an adequate control for subject expectancies. Since the subject expects to receive the real drug in both conditions, if expectancy plays a major role in determining the response, there is no way to assess the effect of the drug alone.

The solution Marlatt, Demming and Reid (1973) developed was to factorially combine expectancy and alcohol conditions. This approach, termed 'balanced placebo', gives a four group design: expect alcohol/receive alcohol, expect alcohol/receive placebo, expect placebo/receive placebo and expect placebo/receive alcohol (Rohsenhow and Marlatt, 1981). A test of the utility of the balanced placebo design in studies of psychomotor skills under alcohol (Vuchinich and Sobell, 1978) found that subjects' beliefs regarding the presence of alcohol in the consumed drink exerted a strong influence over performance. The results indicated that the belief about the presence of alcohol only influences performance in the absence of alcohol. This suggests that for psychomotor tasks, the physiological effects of alcohol are greater than possible expectancy effects.

Although an improvement on the traditional double blind approach, the balanced placebo design does have certain disadvantages. The most obvious problem is that it requires twice the number of subjects to achieve a powerful test. This could cause difficulties if volunteers are not readily available. A possible solution would be to use the expectancy condition only for the placebo drug condition since Vuchinich and Sobell (1978) reported that expectancies did not influence performance under alcohol intoxication. This would produce a three group design requiring fewer subjects.
Another problem is that Marlatt et al (1973) and Vuchinich and Sobell (1978) used the balanced placebo design over short periods. It is likely that as time passes during an experiment long enough to test during both absorption and elimination phases, subjects in the expect alcohol/receive placebo condition would become aware that they had not in actual fact received alcohol. This is because the sensations associated with alcohol intoxication are so well known that it is unlikely that the belief that alcohol had been consumed would continue to determine the subjects’ responses in the absence of the familiar physiological sensation. Changing beliefs about having consumed alcohol halfway through the experiment could detrimentally affect the results (if only from annoyance at the experimenter's perfidy) and negate the advantage of using the balanced placebo design (Bradlyn and Young, 1983).

Beyond this problem, lies another difficulty in using a balanced placebo design in studying the effect of alcohol on visual function. If expectancy for alcohol effects is being controlled in a study of psychomotor ability when intoxicated by manipulating information only about the dose administered, then it must be being assumed that the subjects have some knowledge of the effect of the alcohol on the task being tested. If the subjects do not know the effect of alcohol on the task, because the task is novel and outside the experience of any subject, then the use of expectancy control based on dose would indicate that it is possible that the subjects can bring about the internal state associated with alcohol intoxication and thereby cause performance on an unknown but alcohol sensitive task to alter in the direction of an alcohol effect. In the present case, it is unlikely that any subjects would be aware of the subtle and essentially personally undetectable changes that can occur in visual function when intoxicated. As such there is little to be gained by manipulating expectancy merely via dosage information. It would be of greater advantage to explicitly manipulate expectancies about the effect of alcohol on the dependent variables.

There were two aims in this experiment. The first was to test the hypothesis that measures of visual function are not affected by subject expectancies about the effect of alcohol on visual function. The second aim was to confirm the results obtained in the previous experiment which indicated a clear dose-dependent effect of alcohol.

3.2 Method

3.2.1 Subjects

Subjects were male university students who volunteered in response to campus advertisements. A total of 52 subjects participated in the experiment. The subjects tested were aged between 18 and 28 years, $\bar{M} = 20.2$, $SD = 2.7$. Body weight ranged from 55 to 82 kg ($\bar{M} = 69.3$, $SD = 5.5$). All subjects reported themselves to be social drinkers (see Appendix B for Drinking History Questionnaire, and Appendix C for a summary of the subject's responses). Subjects were informed of the aim of the experiment when they attended an initial interview. This interview was designed to establish whether their vision was normal, and to explain the testing procedure and the requirements of the study. The criteria to determine normal visual function were as in the previous experiment.
3.2.2 Design

Subjects were assigned in equal numbers to one of four experimental groups. One group was a no-drug control who were told nothing about the effect of alcohol on visual function. The other three groups all received the same dose of alcohol but differed in what information they received from the experimenter about the likely effect of alcohol on their visual functions. One group was told that there would definitely be an effect (T-Effect), one that there would definitely not be an effect (T-No effect) and the fourth group was told nothing at all (T-Nothing). This last group was the alcohol control condition to assess the effect of expectancies. The no-drug control group was included for comparison with the alcohol/expectancy conditions and to confirm the findings of the previous experiment.

Two independent variables were investigated in this experiment: alcohol/expectancy condition (four levels: control, T-E, T-NE, T-N) and test (three levels). Measures of visual function for each subject were taken before drinking (Test 1), while BAL was rising (Test 2) and when BAL was falling (Test 3). With alcohol/expectancy condition a between-subjects factor and test a within-subjects factor, this experiment utilised a two-way repeated measures design with one repeated measure.

3.2.3 Instrumentation

Heterophoria

In this experiment measurements were made using the same protocol as that used in the previous experiment.

Positive fusional vergence

In this experiment the procedure was identical except that when measuring positive fusional vergence in experiment 2 the subject was asked to also indicate the 'blur point' of fusion, that is when the stimulus appeared to blur prior to splitting into 2 images. This result was recorded if it could be identified by the subject. Some subjects were not aware of this loss of accommodative vergence and hence this result could not be determined for all subjects.

Edge light pupil cycle time

The fixation target in this experiment was placed at 3 metre distance along the line of sight on a blank wall. (the different distance was as a result of the variation in testing room configuration).

Convergence Near Point

This was measured on the Royal Air Force (RAF) guage with a single line fixation stimulus. The instrument was rested on the cheek bone of the subject and angled so that the eyes are in a slightly depressed position during the examination. The target was moved in slowly until the objective break of convergence near point was noted by the loss of binocular response in one or both eyes. Subjects were encouraged to
maintain a single image for as far as possible. The measurement was repeated 3 times. The mean of the 3 results was calculated to complete statistical analysis.

**Horizontal Saccadic Eye Movements**

In this experiment, the procedure differed slightly from that in the previous experiment in that only a centre left and centre right sequence was measured and recorded for each subject's right eye. These sequences were repeated until the predicted validity of the results was gained.

### 3.2.4 Alcohol and Breathanalysis

All subjects were requested not to consume alcohol the night before the experiment and to fast for five hours prior to reporting to the laboratory. Subjects were also requested to abstain from coffee or tea after 9.00 am.

Those subjects in the 3 alcohol conditions received 0.7 ml of ethanol per kilogram body-weight (kg bw), made up in the following manner: 1.75ml/kg bw of 40 proof vodka. Each dose was administered mixed in a 1:3 ratio with chilled lemon mineral water. The total volume of alcohol and orange juice was divided into 4 equal drinks which the subjects were instructed to consume evenly over a 15 minute period. At the end of the drinking period subjects thoroughly rinsed their mouths to remove any residual alcohol.

Analysis of BAL was performed using a breathalyzer (Lion Laboratories Alcometer SD2). A baseline BAL was measured prior to drinking to determine freedom from alcohol and allow subjects familiarity with the testing device. Subjects were prevented from seeing the digital readout and remained ignorant of both their BAL and the exact amount of alcohol consumed until debriefing at the end of the experiment.

### 3.2.5 Procedure

This experiment was conducted on weekday afternoons in the clinical and research areas of the Division of Orthoptics, Lincoln School of Health Sciences, La Trobe University on the Bundoora campus. The testing session commenced at 1.30 pm and concluded at approximately 5 pm.

Four subjects were tested during each session. On arrival, subjects were randomly assigned to alcohol or control conditions. For those allocated to an alcohol condition, it had been previously decided prior to the testing session whether those in that day's session would be a told effect (TE), told no effect (TNE) or told nothing (TN) group. All drinkers in each session were of like group to avoid confusion if the subjects happened to discuss the experiment during the testing breaks. Subjects were told whether they were control or drinking subjects. Drinks were poured in the room while subjects were present but subjects were not aware of the actual amount of alcohol they were consuming.
The order of measurement was as follows:

- Measurement of heterophoria
- Measurement of negative range and positive fusional vergence including the determination of the 'blur' and 'break' points.
- Measurement of edge light pupil cycle time
- Measurement of horizontal saccadic eye movements
- Measurement of convergence near point

This series of measurements was completed 3 times over the course of the afternoon, constituting a baseline (T1), and 2 examinations post alcohol (T2 and T3) Subjects had a 30-35 minute break between each examination. In this experiment the total testing time for each series was 25-30 mins, slightly longer than previously with the addition of the examination of convergence near point and positive vergence blur point. The first examination formed the baseline assessment for all drinking subjects. A timeline of the testing session is presented in Figure 9.

There was a break of 30-35 minutes between each testing series which subjects spent in a designated room where they were able to chat, study, or read. A relaxed environment was encouraged. Control subjects followed the same procedure without the requirements of alcohol ingestion or further breathalyser measurements once their BAL had been revealed to be 0.0

3.2.6 Data Analysis

Data analysis followed the approach adopted in the previous experiment. In order to facilitate comparisons with the previous experiment, scores on each of the dependent variables was expressed as an absolute difference from the value obtained on Test 1.

The appropriate analysis for this experimental design for all variables except saccades is a two-way repeated measures ANOVA with one repeated measure. For each of the saccadic variables there was a third within-subjects independent variable, version (adduction and abduction). The appropriate analysis for this experimental design for this variable was therefore a 3-way with 2 repeated measures ANOVA. In this study it was expected that evidence for an effect of expectancy on visual function would be apparent in a significant group main effect when comparing the three expectancy conditions. A group x test interaction, either alone or in combination with a group main effect, would also be indicative of an expectancy effect. An effect of alcohol on visual function would be apparent in a significant main effect for group when comparing all four conditions, in the absence of a group main effect across the three expectancy conditions. The presence or absence of a test main effect was of little interest in this experiment. Rejection of statistical null hypotheses was set at $p \leq .05$. 

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Figure 11. Procedure timeline for experiment 2.
3.3 RESULTS AND DISCUSSION

3.3.1 Blood Alcohol Level

The mean blood alcohol level (BAL) at each of the four main breath analysis test times is presented in Figure 12. The mean BAL for the three alcohol conditions T-E, T-NE and T-N during T2 was 0.068%, 0.069%, and 0.075% respectively. The mean BAL for the three alcohol conditions T-E, T-NE and T-N during T3 was 0.0045%, 0.058%, and 0.0615% respectively.

![Figure 12. Mean blood alcohol level (BAL) for each alcohol group at the four breath-analysis measurement times.](image)

3.3.2 Heterophoria

The mean change in measures of distance and near heterophoria for all groups are shown in Figures 13 and 14 respectively. It can be seen that for both measures, the degree of change was greater in the alcohol conditions than the control group. Two-way analysis of test x expectancy showed no significant effects for either distance or near. This indicates that expectancy did not affect measures of heterophoria.

Two-way analysis of test x alcohol showed significant main effects for group on distance [F(3,52) = 4.2, p = .0097] and on near [F(3,52) = 3.93, p = .0278], as well as significant main effects for test on distance [F(1,52) = 5.15, p = .0274] and near [F(1,52) = 23.264, p = .0001]. These results indicate that alcohol affected measures of heterophoria more than did the control condition.
The results of the measurement of near and distance heterophoria in both the control and drinking groups are very similar to the changes found in experiment 1. The control group's distance measurement showed a very slight decrease in esophoria at test 2 and a slight increase in esophoria at test 3 while the drinking groups show a mean change over time with increasing of esophoria of 1.1 prism dioptres. Near measurement in the control group again showed a very slight tendency towards decreasing exophoria. Drinking groups showed a mean change over time of increasing exophoria of 1.6 prism dioptres. There is no significant difference between the drinking groups who all display these relative changes, nor is there any real difference between the results of experiment 1 indicating the repeatability of the testing procedures and the significance of the influence of alcohol on these functions.

Figure 13. Change from Test 1 on Tests 2 and 3 for each group on measures of Heterophoria-distance.
Figure 14. Change from Test 1 on Tests 2 and 3 for each group on measures of Heterophoria-near.

3.3.3 Fusion and Fusional Vergence

The mean change in fusion-divergence for all groups is shown in Figure 15. It can be seen that the degree of change was greater in the alcohol conditions than the control group. Two-way analysis of test x expectancy showed no significant effects. This indicates that expectancy did not affect measures of fusion-divergence.

Two-way analysis of test x alcohol showed a significant main effect for group \( F(3,52) = 4.01, p = .0121 \), as well as a significant main effects for test on distance \( F(1,52) = 5.22, p = .0265 \). These results indicate that alcohol affected measures of divergent fusion more than did the control condition.

The mean change in measures of blur and break-point convergent fusion for all groups are shown in Figures 16 and 17 respectively. It can be seen that for both measures, the degree of change was greater in the alcohol conditions than the control group. Two-way analysis of test x expectancy showed no significant effects for either blur or break-point. This indicates that expectancy did not affect measures of these measures of convergent fusion.

Two-way analysis of test x alcohol showed significant main effects for group on blur-point \( F(3,52) = 5.79, p = .0016 \) and on break-point fusion \( F(3,52) = 3.747, p = .0164 \). These results indicate that alcohol affected measures of convergent fusion more than did the control condition.
Measurement of negative fusion range again replicates the results of experiment 1 with a very slight decrease in this ability over time in the control group and a significant mean change over time of 0.8 degrees in the drinking groups. There was no statistical difference between drinking groups indicating the absence of any expectancy effect on the part of the subjects when undertaking the measurement of this function.

The measurement of positive fusional vergence was separated into the 'blur' and 'break' point in an effort to separate the components of this mechanism into accommodative convergence and fusional convergence. Results indicated that the blur point of the control group slightly increased over time while all drinking groups showed a decrease over time with a mean decrease of 1.6 degrees. Break point results showed very slight improvement in this function over time while drinking groups showed a mean decrease of 3.1 degrees at test 2 and 2.8 degrees at test 3. This break point result confirms the significant decrement in this function as demonstrated in experiment 1. Again there is no significant difference between the drinking groups indicating the absence of an expectancy effect when measuring this function. These results indicated that there was an influence of alcohol on both of these components of the vergence system. As accommodation is so closely linked to convergence, we could hypothesize that the function of accommodation is also affected by alcohol. This has not been extensively documented elsewhere in the literature.

![Figure 15](image.png)

Figure 15. Change from Test 1 on Tests 2 and 3 for each group on measures of Fusion-divergence.
Figure 16. Change from Test 1 on Tests 2 and 3 for each group on measures of Fusion-convergence-blur point.

Figure 17. Change from Test 1 on Tests 2 and 3 for each group on measures of Fusion-convergence-break point.
3.3.5 Convergence Near Point

The mean change in convergence near point for all groups is shown in Figure 18. It can be seen that again the degree of change was greater in the alcohol conditions than the control group. Two-way analysis of test x expectancy showed no significant effects. This indicates that expectancy did not affect measures of convergence near point. Two-way analysis of test x alcohol showed a significant main effect for group [F(3,52) = 3.075, p = .0355]. These results indicate that alcohol markedly affected measures of convergence near point compared to the control condition.

The control group showed a very small mean increase at test 2 and then a very small mean decrease at test 3. These results are not significant. All drinking subjects show a significant change over time with no difference between the groups indicating the absence of expectancy effect when measuring this function. Test 2 shows a mean decrease of 2.25 cm for drinking subjects and test 3 a mean decrease of 2.05 cm. There is a large standard deviation in the results to this function indicating marked variance in these results. It appeared that some individuals have a marked change in response to this function while others do not show these large decrements. Wilson and Mitchell (1983) related the reduction in convergence near point to a reduction in accommodative vergence and an associated reduction of muscle tonus, but claim that fusional vergence should still be intact. This is contrary to our findings which suggested a definite impact on both positive and negative fusion range. Wilson and Mitchell (1990) argued that accommodation and convergence may be controlled by differing pathways and that these pathways may differ in their sensitivity to alcohol. This change in convergence near point ability can be explained by the decrease in general neuromuscular co-ordination and is certainly related to the other changes seen at near of increasing exophoria and decreasing positive fusion and accommodative vergence.

3.3.6 Pupil Cycle Time

The mean change in summed pupil cycle time near point for all groups is shown in Figure 19. It can be seen that there is little consistent difference between the groups and the differences are small. Two-way analysis of test x alcohol showed no significant effects. This finding indicates that as in the previous experiment, alcohol had no effect of pupil cycle time and was therefore unlikely to be subject to an expectancy effect. Two-way analysis of test x expectancy showed no significant effects.

Investigation of pupil cycle time in this experiment showed no significant change in this function either over time or in any of the drinking or control groups. This confirms the findings of experiment 1 indicating that this autonomic nervous system function is not influenced by alcohol at the moderate BALs tested.
Figure 18. Change from Test 1 on Tests 2 and 3 for each group on measures of Convergence Near Point.

Figure 19. Change from Test 1 on Tests 2 and 3 for each group on measures of total pupil cycle time.
3.3.7 Horizontal Saccades.

The 3-way ANOVA for latency showed no significant effects, whereas that for peak velocity revealed a significant group main effect \( [F(3,48) = 4.106, p = .0113] \). There was no significant difference between the three alcohol groups \( [F(2,36) = 0.948, p = .3969] \). Figure 20 shows the group main effect.

![Figure 20](image)

**Figure 20.** Main effect of group for the change in peak velocity of horizontal saccades from Test 1.

The latency duration analysis again shows no statistically significant change either between the groups or over the time of the testing session. All groups in experiment 2 showed a very slight increase in the latency duration from test 1 through to test 3. This result confirms the result found in experiment 1.

As discussed in the first study latency is not thought to be the most reliable measure of drug effects on the nervous system. Our results again are consistent with the previous research which did not find significant change in latency duration. Baloh et al (1979) reported significant change in latency although this was more marked in saccades of a larger amplitude than 20 degrees.

The peak velocity results are similar to those of experiment 1 with the alcohol groups showing a marked decrease. The control group again shows only a very slight decrease in mean peak velocity over tests. This result is consistent with a fatigue effect which has been documented in the literature. Comparison of the three alcohol groups, showed that there were no differences in the decline of their peak velocity.
measures (7 - 10%) as a result of consuming alcohol. This indicated the absence of an expectance effect when measuring this dependent variable. This decrease in peak velocity is consistent with results of the moderate BAL group in experiment 1 which showed a decline of 8.6% of abduction and adduction saccadic movements.

The subjects in study 2 had a slightly higher mean maximum BAL of 0.07 which is 0.015 higher than the mean maximum of drinking subjects in experiment 1. The only other change in this second study was a procedural one where subjects were asked to repeat the sequence of saccadic movement until the examiner believed that the subject was complying exactly with directions and the numerical result was acceptable for its predicted validity. The procedure was changed because these changes were deemed appropriate after the data collection phase of experiment 1 was completed as this was the first time the equipment had been used as a research tool. As the method in experiment 2 was more refined, the results obtained are likely to be more robust. Both studies however, have indicated the same statistically significant results. Alcohol subjects at a BAL of 0.04 therefore can have a marked slowing of horizontal saccadic eye movements. This measure is not influenced by an expectance effect.

3.3.8 Summary

The results obtained in this experiment replicated the findings of the influence of alcohol on oculomotor and autonomic aspects of visual function found in experiment 1. Significant change in the measurement of heterophoria, negative fusion range and peak saccadic velocity is found at moderate BALs. Positive fusional vergence and convergence near point were also significantly effected. There was no statistically significant difference between the results of the three alcohol groups indicating the absence of subject expectance effect in the measurement of these oculomotor aspects of visual function.
Both experiments in this study have clearly documented specific changes to aspects of the oculomotor function of the visual system. This in part allows binocular single vision and the control of eye movement in a co-ordinated manner. Latent position of the eyes is clearly influenced by alcohol with the development or increase in measurement of esophoria at distance range, a feature which does not exist without the influence of alcohol for subjects examined. Similarly, development or increase in measurement of exophoria is found at near range, a finding again induced by the intoxicated state of the subject. Fusion range and fusional vergence, the ability to move the eyes together over a range of distances to maintain a single, focused image is also detrimentally affected by the consumption of alcohol. Convergence, a component of the near reflex which allows focusing at very close range recedes significantly, especially in some individuals.

As stated, the abilities which have been examined combine to control the binocular status of the eye movement system allowing the eyes to co-ordinate in a stable manner. The results found indicate that alcohol interferes with components of this system which will at the least interfere with the integral balance of the system. This is manifested as a decrease of one or more of the components of the binocular system and hence an overall decrement of the system is seen due to the generalized impact of alcohol on neuromuscular control.

The study has also analysed some of the changes to the saccadic eye movement system as a result of consuming alcohol. These are the fine, fast, motor movements of the eyes which allow accurate fixation from point to point. Our study indicates that there may be a trend for the reaction time of fast eye movements to be delayed and there is a marked slowing in the speed of these movements. These findings regarding the slowing of eye movement and the control of the binocular system must contribute to the general imperfect performance of sensory, motor and co-ordinative tasks undertaken by the intoxicated subject. This has particular significance for the ability to drive safely.

These measurements of binocular control and the saccadic eye movement system cannot be influenced by the expectancy of the subject as shown by the results and experimental design implemented in the second stage of this study. These motor measures of the visual system may potentially be a more objective measure of performance decrement of the intoxicated person. Our results therefore fulfil the aims of this study.
Further research into the question of visual function measure as a method of measuring alcohol intoxication can involve a number of steps. First, there is a necessity to correlate these changes in motor aspects of visual function with changes in psychomotor performance. This is in order to verify the assumption that the oculomotor and other motor systems are equally affected by alcohol, and that therefore changes in visual function can be reliably used as markers for changes in general psychomotor performance that are associated with impaired driving ability. Second, the functional implications of the observed degradation of the binocular and saccadic systems in relation to driving ability need to be investigated. This is because even if oculomotor performance is not correlated with general psychomotor control, the findings of this and other research indicate that there are changes in these systems at BALs close to the legal limit. Therefore the possibility exists that alcohol induced changes in these systems might have a deleterious effect in marginal conditions.

It is important to remember that the focus of this research was not to provide an alternative to the current practice of using biochemical assay (i.e. breath analysis) for the measurement and definition of alcohol-induced incoordination. The issue of legally defining inability to drive has been addressed. However the issue of measuring alcohol and drug interactions, as well as the myriad of other drugs which can have an effect on driving has not been solved. This research was intended to explore the utility of using changes observed in the oculomotor system under alcohol intoxication as a model for the analysis of the impact of other drugs and drug combinations on the visual system. If a relationship between changes in the oculomotor and other motor systems is demonstrated, the next stage would involve extending this research to investigate these other drugs. The ultimate aim would be to develop, or appropriate, a clinical test of visual function that could be used to reliably and objectively indicate the presence of drug-induced impairment of motor ability.
REFERENCES


APPENDIX A

Glossary of Orthoptic Terminology

Abduction  Rotation of one eye horizontally towards the temple and away from the midline

Accommodation  The adjustment of the eye for seeing at different distances, accomplished by the changing shape of the lens through action of the ciliary muscle, thus focussing a clear image on the retina.

Binocular single vision  The ability to use both eyes in a co ordinated manner to obtain single vision with depth perception in normal seeing conditions.

Congugate  Movements of the two eyes in the same direction

Convergence  A disgugate movement in which the visual axes are directed nasally

Dioptre  A unit of measurement of the power of a lens. A lens which has a focal length of one metre has a refractive power of on dioptre.

Diplopia  Double vision caused by misalignment of the visual axes.

Disgugate  Movement of the eyes in which the visual axes do not remain parallel but move in opposite directions. these are known as vergences and consist of convergence, divergence and vertical vergence.

Divergence  A disgugate movement of the eyes where the visual axes are directed temporally or outwards

Dolls Head  Brisk head turning used to elicit congugate eye movement in the opposite direction.

Esophoria  A latent convergent deviation where there is a tendency of the eyes to deviate inwards.

Exophoria  A latent divergent deviation where there is a tendency of the eyes to deviate outwards.

Fusion  The mental ability to blend 2 similar images one formed in each eye and perceive them as one.
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<th>Term</th>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Fusion Range</strong></td>
<td>The range or distance of convergence and divergence over which fusion can be maintained.</td>
</tr>
<tr>
<td><strong>Extrinsic ocular muscles</strong></td>
<td>The muscles which rotate the eyeball of which there are 6 in each eye.</td>
</tr>
<tr>
<td><strong>Heterophoria</strong></td>
<td>A latent deviation of the visual axes which are held in a parallel position by the fusion mechanism when both eyes are open. On dissociation of one eye from the other the eyes will tend to drift in a particular direction, convergent (inwards) or divergent (outwards), elevated or depressed.</td>
</tr>
<tr>
<td><strong>Heterotropia</strong></td>
<td>A manifest deviation of the visual axes away from the parallel position. The squinting eye will be deviated in a convergent, divergent, elevated or depressed position.</td>
</tr>
<tr>
<td><strong>Nystagmus</strong></td>
<td>A disturbance of ocular posture involving involuntary repeated oscillations of one or both of the eyes in one or a variety of positions of gaze.</td>
</tr>
<tr>
<td><strong>Optokinetic nystagmus</strong></td>
<td>Nystagmus induced by lines which move across the field of vision.</td>
</tr>
<tr>
<td><strong>Prism dioptre</strong></td>
<td>A unit of measurement of the strength of a prism. A prism of one dioptre strength displaces the image of the object through 1 cm. for each metre of distance between object and prism.</td>
</tr>
<tr>
<td><strong>Refraction</strong></td>
<td>Deviation in the course of light rays entering the eye in passing from one transparent medium to another of differing density or shape.</td>
</tr>
<tr>
<td><strong>Refractive error</strong></td>
<td>This is the inability of the eyes dioptric apparatus (cornea and lens) to form a sharply focussed image on the retina.</td>
</tr>
<tr>
<td><strong>Saccade</strong></td>
<td>Fast voluntary eye movement to allow accurate fixation from point to point.</td>
</tr>
<tr>
<td><strong>Smooth pursuit</strong></td>
<td>Smooth conjugate eye movement to follow a moving target.</td>
</tr>
<tr>
<td><strong>Stereopsis</strong></td>
<td>Disparity of the retinal images resulting in depth perception.</td>
</tr>
<tr>
<td><strong>Supression</strong></td>
<td>A condition in which an object perceived in one eye is mentally ignored or neglected. It is a method of overcoming double vision.</td>
</tr>
</tbody>
</table>
APPENDIX B

DRINKING HISTORY QUESTIONNAIRE

Name: ____________________________________________________________

Address: _________________________________________________________

Phone Number: ____________________________

Please circle the applicable response:

1. Do you smoke? YES NO

2. Alcoholic beverage most often used: BEER WINE SPIRITS

3. On how many days of the week, on average, would you drink?
   1 2 3 4 5 6 ALL

4. How many drinks do you have, on average when you drink?
   1 2 3 4 5 6 7 8 9 10 >10

5. When you drink, how fast do you drink? (i.e. drinks per hour?)
   1 2 3 4 5 6 7 8 9 10 >10

6. How many times in the last month have you been very drunk?
   0 1 2 3 4 5 6 >6

7. How strong an effect do you feel alcohol has on you when you drink?
   None Slight Moderate Strong Extreme

8. What effect do you feel alcohol has on your abilities?
   Marked Improvement Slight Improvement No effect Slight Impairment Marked Impairment

9. For how many years have you been drinking?
   1 2 3 4 5 6 7 8 9 10 >10
APPENDIX C

SUMMARY OF THE SUBJECT'S RESPONSES TO DRINKING HISTORY QUESTIONNAIRE IN EACH EXPERIMENT

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Control</th>
<th>Low BAL</th>
<th>Mod BAL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Days do you drink?</td>
<td>1.917</td>
<td>1.62</td>
<td>2.41</td>
</tr>
<tr>
<td>Drinks per hour?</td>
<td>5.42</td>
<td>3.2</td>
<td>4.94</td>
</tr>
<tr>
<td>How fast?</td>
<td>3.08</td>
<td>1.93</td>
<td>2.94</td>
</tr>
<tr>
<td>How often drunk?</td>
<td>1.25</td>
<td>1.60</td>
<td>1.71</td>
</tr>
<tr>
<td>Alcohol effect?</td>
<td>2.92</td>
<td>.78</td>
<td>3.17</td>
</tr>
<tr>
<td>Abilities and alcohol?</td>
<td>4.33</td>
<td>1.07</td>
<td>4.00</td>
</tr>
<tr>
<td>Years drinking?</td>
<td>4.83</td>
<td>3.00</td>
<td>5.88</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Control</th>
<th>T-Effect</th>
<th>T-No effect</th>
<th>T-Nothing</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Days do you drink?</td>
<td>2.0</td>
<td>.9</td>
<td>3.6</td>
<td>1.5</td>
</tr>
<tr>
<td>Drinks per hour?</td>
<td>4.9</td>
<td>2.6</td>
<td>6.1</td>
<td>1.9</td>
</tr>
<tr>
<td>How fast?</td>
<td>2.7</td>
<td>.8</td>
<td>3.6</td>
<td>.8</td>
</tr>
<tr>
<td>How often drunk?</td>
<td>2.8</td>
<td>2.4</td>
<td>3.3</td>
<td>2.3</td>
</tr>
<tr>
<td>Alcohol effect?</td>
<td>3.1</td>
<td>.7</td>
<td>3.1</td>
<td>.6</td>
</tr>
<tr>
<td>Abilities and alcohol?</td>
<td>4.4</td>
<td>1</td>
<td>3.9</td>
<td>1.5</td>
</tr>
<tr>
<td>Years drinking?</td>
<td>4.9</td>
<td>2.9</td>
<td>5.3</td>
<td>2.6</td>
</tr>
</tbody>
</table>
APPENDIX D

INFORMATION

Visual Function as an Objective Measure of Alcohol and Drug Effects on Driving

This study aims to compare some aspects of your vision before and after having a small measured dose of alcohol, and comparing these results with measures of psychomotor coordination.

In order for you to be included in this study you must have a history of mild to moderate social drinking, as well as vision which is 'normal' against a set of defined criteria and would be acceptable for all visual purposes. Drinking history will be assessed with a questionnaire. Establishing normal visual function will involve asking you about any eye or vision problems you may have experienced and any treatment you have had. You will then be asked to complete a small number of standard tests of visual function in order to establish your suitability to be included. It is quite acceptable for you to wear glasses or contact lenses. The examiner will inform you of the results of these tests, none of which are invasive, painful or cause discomfort.

Prior to the experimental session you will have abstained from drinking alcohol or taking prescribed or unprescribed drugs for 24 hrs. On the day of the experimental session you are requested to abstain from eating or drinking other than water after 9.00am. Before this you may have a light breakfast of tea or coffee, cereals and/or toast.

The measurements to be included in the study will be carried out to assess the function of your pupils, your eye movements and the focusing of your eyes. You will then be asked to drink either an alcoholic beverage or a placebo beverage and after a period of time, to repeat the experimental measures. At several points during the session your blood alcohol level (BAL) will be measured with a breathalyser. Your maximum BAL will be about 0.05%. You will only be able to leave the laboratory when your BAL is below this level. The session will last for 3-4 hours. At the end of the session you will be informed about your performance, offered $20 for your participation and if required, provided with a taxi to your home.

PROBATIONARY LICENCE HOLDERS ARE ADVISED THAT THEY WILL HAVE A POSITIVE BLOOD ALCOHOL LEVEL FOR SEVERAL HOURS AFTER THE SESSION AND THEREFORE SHOULD NOT DRIVE.

Your participation in this project will be of benefit to us in our understanding of the effects of alcohol on the visual system, and subsequently to the community in the effects of alcohol on driving ability. The benefits to you as a participant are that you will have an assessment of your visual function as well as receive breathalyser feedback on the state of mild alcohol intoxication experienced. Any questions regarding this project can be directed to Cathy Devereux in the Division of Orthoptics, Lincoln School of Health Sciences on Tel. 479 1807 or 479 1920.

YOU ARE FREE TO WITHDRAW FROM THIS STUDY AT ANY TIME

You are booked to participate in this experiment in Room 422 in the Health Sciences Building

Please contact us if you cannot keep this appointment. Thanks.
Visual Function as an Objective Measure of Alcohol Intoxication.

Author(s)
Devereux, Catherine J.
Story, Ian H.
Pitt, Alison

Performing Organisation (Name and Address)
Division of Orthoptics
Lincoln School of Health Sciences
La Trobe University

Sponsor
Federal Office of Road Safety
GPO Box 594
CANBERRA ACT 2601

Abstract
The aim of this project was to investigate the viability of using changes in visual system function as a measure of alcohol intoxication. Results indicated that latent eye position, range of binocular movement and fast voluntary eye movements of young male subjects were significantly influenced by alcohol in a dose dependent manner. These measures of visual function appear not to be susceptible to expectancy effects in the way that performance measures of alcohol intoxication have been found to be. These findings suggest the possibility of oculomotor function as a potential measure of alcohol and drug effects on psychomotor performance.

Keywords
objective measure, drugs, alcohol, motor, impairment

NOTES:
(1) FORS Research reports are disseminated in the interests of information exchange.
(2) The views expressed are those of the author/s and do not necessarily represent those of the Commonwealth Government.
(3) The Federal Office of Road Safety publishes four series of research reports:
   (a) reports generated as a result of research done within the FORS are published in the OR series.
   (b) reports of research conducted by other organisations on behalf of the FORS are published in the CR series.
   (c) reports based on analyses of FORS' statistical data bases are published in the SR series.
   (d) minor reports of research conducted by other organisations on behalf of FORS are published in the MR series.