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THE EFFECTS OF ALCOHOL ON THE OCULO-VESTIBULAR SYSTEM

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THE EFFECTS OF ALCOHOL ON THE OCULO-VESTIBULAR SYSTEM

CHAPTER I

INTRODUCTION

The Vestibular System

The non-auditory portion of the inner ear, composed of the saccule, the utricle, and three semicircular canals, is called the vestibular system. The saccule and the utricle are receptors for gravity and linear accelerations, and react to shearing forces (Trincker, 1962; Bekesy, 1966). Because of their structure, each canal positioned approximately at right angles to the others, the semicircular canals are capable of sensing angular acceleration about any plane. Although the normal or "adequate" stimulus for the semicircular canals is angular acceleration, they also react to changes in temperature induced under proper conditions (Howard and Templeton, 1966). Effective thermal changes can be produced by irrigating the external canal of the ear with water that is a few degrees above or below body temperature (a caloric irrigation). Rotatory or caloric stimulation, in addition to producing dizziness or sensations of motion, also results in a pattern of eye movements (composed of alternating fast and slow phases) called nystagmus. The gradual drift of the eyes away from center is referred to as the slow phase and is generally thought to reflect the degree of stimulation received by the semicircular canals

(Henriksson, 1955). The fast phase, a quick return of the eyes toward the midline, is initiated by a central mechanism and denotes direction of the response.

Alcohol and Vestibular Responses

Alcohol is well known as a depressant of central nervous system activity and its effects are evident in increases in simple reaction time (Carpenter, 1959) and decreases in motor skills (Asknes, 1954). Carpenter's (1963) review of studies concerning the effects of alcohol on psychological processes (reaction time, motor skills, driving skill and intellectual functions) includes a short discussion of the positional nystagmus which occurs as the result of alcohol intoxication. Positional alcohol nystagmus (PAN) is a sustained pattern of horizontal eye movement that occurs either while the subject is lying on his side or while the subject lies, in a supine position, with his head turned to the right or left side. This type of a nystagmic response, one that changed direction with changes in the position of the head, was referred to by Nylen (1950) as Type II positional nystagmus.

Flourens, in 1826, is considered to be the first to have studied the relationship between cerebellar processes and the degree of alcohol intoxication (1842). Several decades later, Joffroy and Serveaux (1897) found that one of the results of alcohol intoxication is the development of a spontaneous nystagmus. Since that time, various studies have shown that the direction of the nystagmus is dependent upon the position of the head and the time since consumption of alcohol. Aschan (1958), and Aschan et al. (1956) conducted extensive studies of PAN. In all of

these studies PAN, because of its changes in direction over time, has been divided into two phases (PAN I and PAN II). During the first phase, 20 min after alcohol ingestion and for 3-4 hours thereafter, the positional nystagmus beats in a downward direction; e.g., with the head in a left lateral position, nystagmus beats to the left; in the right lateral position, nystagmus beats to the right. Between the end of PAN I and the appearance of PAN II there is a quiescent period (no nystagmus) for approximately 1-2 hours. This period is followed by PAN II, where the nystagmus beats in an upward direction, e.g., with the head in the left lateral position, nystagmus beats to the right; in the right lateral position, nystagmus beats to the left. Whereas PAN II generally continues, on the average, up to 11 hours after consumption of alcohol, it may last for as long as 14 hours.

According to Aschan et al. (1956) the onset and duration of PAN I and the onset of PAN II are independent of the amount of alcohol consumed. The latter, however, does correlate with other measures of PAN I and PAN II; the amplitude of PAN I and PAN II is increased and the duration of PAN II is prolonged.

The experiences of the subject also bear some relationship to PAN I and PAN II. The amplitude of PAN I, according to Aschan et al. (1956), is related to the severity of the subjective symptoms (drunkenness). During PAN II, the intensity of the nystagmus is related to the intensity of the "hangover."

Several authors have sought to determine the pathogenesis of PAN by selective destruction or blockage of the semicircular canals or otoliths. Bilateral labyrinthine destruction results in an elimination of

PAN in rabbits (DeKleyn and Versteegh, 1930) and in cats (Nito et al., 1964; Money, Johnson, and Corlett, 1965; Suzuki et al., 1968). In the study by Nito et al. (1964), the otoliths were left intact but no PAN was observed. Aschan, Bergstedt, and Goldberg (1964) and Harris, Guedry, and Graybiel (1965) found that PAN did not occur in labyrinthine defective subjects following alcohol intoxication.

There are some data concerning the presence of PAN in animals or man when only one labyrinth is intact. DeKleyn and Versteegh (1930) found PAN I only when the heads of the rabbits were in one lateral position after removal of the opposite labyrinth. Contrary to this finding, Money, Johnson, and Corlett (1965) using cats, and Aschan, Bergstedt and Goldberg (1964) using humans, found PAN I and PAN II present while the head was in either lateral head position following unilateral destruction of one labyrinth. This lead Aschan et al. (1964) to conclude that PAN occurs as the result of both a peripheral (effect on the semicircular canals) and central influence of alcohol.

In addition to PAN, alcohol also produces nausea, vertigo, ataxia, and a disturbance of balance. There are, however, few quantitative studies of ataxia or disruptions of balance that occur during alcohol intoxication. Sunao (1961) used a slow bicycle riding test as a measure of labyrinth and equilibrium functioning. Following alcohol intoxication, the amount of decrement in performance was found to be related to the blood alcohol level (BAL) of the subject; the higher the BAL or the greater the degree of drunkenness, the larger the drop in performance. Using an ataxia battery, Fregly, Bergstedt and Graybiel (1965) found similar results. The amount of decrement in postural equilibrium was

significantly correlated with the BAL curve and the intensity of PAN I. Performance had returned to a level near normal by the time PAN II had appeared. Fregly and Graybiel (1968), using the same test battery, studied alcohol ataxia in labyrinthine defective subjects. Following alcohol intoxication the "...magnitude and duration of the intoxicating effects were found to be less than observed previously in normal persons (pg. 468)." Generalizing from these data, the authors feel that the semicircular canals may contribute to the disturbance of balance in intoxicated "normal" subjects.

Alcohol and Vestibular Responses to Rotation

Evidence concerning the effect of alcohol intoxication on ocular nystagmus induced by angular acceleration is contradictory. Barany (1911, 1912), one of the first to notice the effect of alcohol on the vestibular response to rotatory stimulation, reported that alcohol produced a reduction in the turning sensation but failed to produce any change in the duration of the eye movements that occur at the end of rotation. Data from a study by Manz (1939) contradict Barany's conclusions; alcohol ingestion prior to rotation resulted in a prolongation of the duration of post-rotatory nystagmus. A similar conclusion was reached by Taschen (1955a, 1955b). Taschen found that the duration was lengthened, amplitude was increased, and the nystagmus became coarser. Further support of the idea that alcohol heightens the nystagmic response is offered by analysis of the data of Schweitzer (1955) and of Schulte and Roth (1957).

Although Forster (1958), like his predecessors, used a stimulus of high angular velocity, he was the first investigator to separate the

acceleration from the deceleration. In all of the studies mentioned above, the stimulus involved high rates of angular velocity within a short rotation time (e.g., Manz used 10 rotations in 20 seconds; Taschen used either 5 rotations in 5 seconds, or 5 rotations in 10 seconds) with a brake deceleration in all cases. In these instances, the cupula is extremely deformed and there is a possibility that pre-rotatory nystagmus may interfere with post-rotatory nystagmus. This criticism was presented in Rauschke's (1958) discussion of contradictions among several of the studies. The difficulty may be overcome by using a longer period of rotation at constant velocity before initiating the deceleration.

Another significant difference between Forster's (1958) study and previous work is that he used electronystagmography (ENG) in observing the eye movements. With this technique, it is possible to obtain more precise information concerning the duration, frequency, and amplitude of the nystagmus than can be obtained by visual observation. Using the ENG approach, Forster found a suppression of all components (duration, frequency, and amplitude) of the nystagmic response to rotation following alcohol consumption.

Bochenek and Ormerod (1962) compared the effects of alcohol and various drugs on rotatory and caloric nystagmus. The amount of inhibition of rotatory nystagmus produced by alcohol was almost identical to that produced by some of the other drugs (an antihistamine, a barbiturate and a parasympatholyte). However, the inhibition produced by alcohol was more evident in the amplitude than in the duration or frequency of the nystagmus.

A decrease in nystagmus during alcohol intoxication was also

noted by Mizoi, Ishido, and Ohga (1962). In this study, the subjects were rotated 10 times in 20 seconds and then given a brake deceleration. While under the influence of alcohol, the duration, frequency, and amplitude of the rotatory nystagmus were all less than under sober conditions. The degree of suppression was related to the amount of alcohol consumed; the more alcohol ingested, the greater the decrease in nystagmus.

Ey (1963), although supporting Forster's (1958) conclusions, criticized Forster's study because the stimulus magnitudes were too far above threshold to provide a uniform nystagmic response. Ey noted that the threshold for rotatory nystagmus during alcohol intoxication was heightened, i.e., a stronger stimulus was required to produce nystagmus. During rotation, the pre-rotatory nystagmus became irregular or disappeared.

Further support of the depressive action of alcohol on rotatory nystagmus is provided by Di Guinta and Rosa (1968). These authors studied the quantitative and qualitative change in the nystagmic response to angular accelerations following the ingestion of alcohol. In all but one of five subjects, the response, following alcohol intoxication, was weaker and irregular.

In all of the more recent studies where the accelerations and decelerations were reproduced more accurately and where the ENG technique of recording was used, alcohol ingestion was shown to produce an inhibition of the vestibular response to rotatory stimulation. The difference between this finding and that of an enhanced response may also be attributed to the procedures involved. Under conditions of visual observation of nystagmus, the subject was rotated and decelerated in the light. After stopping in a lighted room the subject fixated visually on some object in

front of him; the nystagmus might last from 4-10 sec (Taschen, 1955). Following alcohol ingestion, the nystagmus duration, in most studies, was lengthened. This lengthening may have been due to the fact that the subject, under alcohol, was less able to fixate, and thereby the nystagmus would be observed for a longer time. Thus, the reported increase in response is not necessarily due to the effects of alcohol on the vestibular system, but due to the effects of alcohol on the ability of the subject to maintain visual fixation. Use of the ENG technique with the subject in total darkness obviates this problem, but may create a different problem involving arousal.

Alcohol and Vestibular Response to Calorization

There are very few data concerning the effect of alcohol on caloric nystagmus. Using a cold stimulus (21^{o}C), Manz (1939) found the duration of nystagmus, in most cases, was prolonged; in one case the duration, under alcohol, increased as much as 59 seconds. Schwab and Ey (1955) reported the duration of caloric nystagmus, following alcohol ingestion, as being irregular (shortened in most cases). However, there was a definite shortening of the latency of the first nystagmic beat. These data are supported by Rauschke (1958) who indicated that the onset of nystagmus was clearly earlier following alcohol consumption.

In a study mentioned previously, Bochenek and Ormerod (1962) compared the effect of alcohol and several drugs on the response to caloric irrigations. Alcohol was reported to be most effective in suppressing caloric nystagmus. However, the authors presented no data concerning the extent of the suppression. The difference between the results of their study and those of previous studies may be due to the fact that the

earlier investigations required visual fixation by the subjects, while Bochenek and Ormerod used the ENG technique in recording nystagmus in total darkness. Thus, the same factor (visual fixation) may form the basis for the contradictory findings in both rotation and caloric studies.

Alcohol and Optokinetic Nystagmus

Another way of producing nystagmus is through optokinetic stimulation; e.g., by passing a series of black and white stripes (or targets) before the subject's eyes. The data concerning the effects of alcohol on optokinetic nystagmus are more consistent than those concerning its effects on rotatory nystagmus. Scofield (1936), although using only mild doses of alcohol and a small number of subjects, indicated that there was a tendency for alcohol to disturb ocular adjustment; as the amount of alcohol increased there was an increase in the inadequacy of the pursuit movements during optokinetic stimulation. Krauland, Schuster, and Klein (1961) found that consumption of alcohol (BAL values from 0.36 to 2.75 per cent) produced a decrease in the frequency and amplitude of optokinetic nystagmus. The authors also noted a corresponding qualitative change (a disturbance of rhythm) in the appearance of the nystagmus. If the stimulus density (number of stripes per interval of time) is lowered, under normal conditions a modified after-nystagmus can be observed. According to Krauland, Schuster, and Klein (1961), a decrease in the presence of this after-nystagmus can be noted during the early stages of alcohol intoxication. Mizoi, Ishido, and Ohga (1962), reporting on the work of Yabuki, indicate that alcohol suppresses optokinetic nystagmus. Ey (1963) also reported a suppression of optokinetic responses

following alcohol intoxication; there was a decrease in the frequency of optokinetic nystagmus and a suppression of the optokinetic after-nystagmus.

In testing the optokinetic fusion limit, the speed at which the eyes are no longer able to follow a band of black and white stripes, Blomberg and Wassen (1962) found a small amount of alcohol to be sufficient to reduce the fusion limit. Under alcohol the eyes could not follow the stripes at lower rates of rotation. In all of the cited studies concerned with the effect of alcohol on optokinetic nystagmus, at all alcohol levels, a decrement in response was reported.

The Spiral Aftereffect

While watching a rotating Archimedes spiral, the spiral appears either to expand or contract (depending upon the direction of turn). After the spiral stops there is, for a short time, an apparent motion of the spiral (expansion or contraction) in a direction opposite to that perceived during the actual rotation. This illusory motion after the end of rotation is referred to as the spiral aftereffect (SAE).

In a recent study, Reason and Benson (1968) found a significant correlation between the duration of the spiral aftereffect and the duration of turning sensations following brake decelerations. In a follow-up study, Reason (1968) indicated that the SAE duration and the duration of the rotatory sensation bear the same logarithmic relationship to the "objective" stimulus. A correlation of the slope values for the two after-sensations was positive but statistically insignificant. Earlier work by Nilsson and Henriksson (1967) indicated a significant correlation between the duration of the oculogyral illusion (OGI) and the duration of

the spiral aftereffect. The OGI refers to the displacement of a small target light, in a dark room, during and after angular acceleration. The duration of the illusion is related to the duration of the sensation of rotation when no light is present. Because of this relationship, the data offer further support to the work of Reason and Benson. Reason (1968) theorized that this relationship reflects "...individual differences in the way stimulus energy is 'received' or transduced by the central nervous system (pg. 391)." If this correlation truly reflects an individual system of translating sensory information, alcohol might be expected to have an identical effect on the response. At the present time, there is no information concerning the effect of alcohol on the spiral aftereffect and only one study in which some information is provided concerning the effect of alcohol on the subjective experience during and after rotation. Barany (1912) found that alcohol ingestion depressed the duration of subjective turning following a brake deceleration. Thus, more information is needed concerning the effects of alcohol on turning sensations and on the duration of the SAE.

Influence of Arousal on Vestibular Responses

A neglected variable in studies of the effect of alcohol on the oculo-vestibular system is the alertness of the subject. Alcohol, because of its depressive action on the central nervous system, could be expected to lower the level of arousal of subjects, particularly if they are tested with eyes closed or in total darkness.

There is considerable data indicating that the vestibular response is susceptible to modification by changes in the psychological and physio-

logical state of the subject. Griffith (1920) found an enhanced nystagmic response from humans following a period of exercise or during a period of gastrointestinal upset. Reporting on results with a group of pigeons, Mowrer (1934) stated that "excited" (recently handled) pigeons, as opposed to unexcited birds, had nystagmus of longer duration. Wendt (1951) discussed the importance of an "environment directed orientation" as opposed to an "inward directed orientation" (i.e., an attention factor) in preventing nystagmus from becoming habituated in human subjects.

Most of the more recent studies have emphasized the usefulness of mental activity in controlling alertness during vestibular stimulation. In a series of articles (Collins, Crampton, and Posner, 1961; Collins, 1962, 1963, 1964; Collins and Poe, 1962), Collins has shown that mental activity can alter nystagmus. A nystagmus of greater amplitude and longer duration results from requiring the subject to work on a psychophysical task (estimation of time intervals) or a continuous arithmetic task, than that obtained during a relaxed or reverie state. Lachman, Aarons, and Erikson (1968) report an enhancement of spontaneous nystagmus (a clinical manifestation) by mental activity, with greater mental effort producing greater enhancement.

Present Study

The present study was designed to examine the effects of alcohol on the functioning of the vestibular system while attempting to control for some of the factors which may have produced inconsistencies in the earlier studies. Both Rauschke (1958) and Ey (1964) criticized several of the earlier studies on the basis of the stimulus used in producing

nystagmus; the deceleration stimuli were of far greater magnitude than that required to provide a "normal" vestibular response. A related problem concerns the lack of precision involved in the administration of the stimuli. Taschen (1955) required his subjects to rotate while standing upright. Under these conditions, where slight changes in head position or speed of rotation could alter the nystagmic response, there is no assurance that the intoxicated and sober subjects received identical stimuli. In this procedure, where a small number of rapid rotations was followed immediately by a brake deceleration, the authors also encountered the problem of overlap of the subjects' responses to the accelerations and decelerations. This problem may be overcome by allowing the subject to rotate at constant velocity for 2-3 minutes between the end of the acceleration and the initiation of the deceleration.

In all of the studies concerning the effects of alcohol on the responses to rotatory, caloric and optokinetic stimulation, only Blomberg and Wassen (1962) conducted a statistical analysis of their data. In cases where the differences were slight or where only a small number of subjects was used, it is difficult to determine whether or not the differences were statistically reliable.

Possibly the most significant problem with previous studies is the lack of control over the alertness of the subjects. The nystagmic response, according to recent work (Collins, 1962, 1963, 1964, 1965), is significantly affected by changes in the alertness of the subject. Alcohol, as a depressant, has a soporific effect on most individuals and would thereby be expected to decrease alertness and, concomitantly, reduce the nystagmic response. Under the conditions of previous studies,

where alertness was not controlled and a response reduction occurred following consumption of alcohol, it cannot be ascertained if the changes were due to alertness factors or to the direct effect of alcohol on the vestibular system. In the present study an attempt was made to maintain the alertness of the subjects through the use of a psychophysical task or mental arithmetic.

In all of the studies concerning the effects of alcohol on the vestibular system, only Barany (1911, 1912) mentioned any data concerning its effect on the subjective response. Although he did not specify the stimulus conditions, Barany (1912) found a decrease in the reported sensation of rotation following consumption of alcohol. Further investigation is needed, both to test Barany's results under specified conditions and to provide information concerning the effect of alcohol on the subjective response to caloric irrigations.

Another area where further investigation is needed concerns the relationship between the duration of turning sensations following rotation and the duration of the SAE. If these two measures truly represent an underlying mechanism for translating sensory information (Reason, 1968, 1969, Reason and Benson, 1968), alcohol should have an identical effect on the responses. However, if alcohol affects the vestibular system through peripheral means (direct action on the semicircular canals) it may result in a differential effect on the two responses; there may be a decrease in the subjective sensation to rotation, while there would be no effect on the duration of the SAE.

With reference to the previous data and the above discussion the following hypotheses were set forth.

1. A higher-amplitude, longer duration nystagmic response to caloric and rotatory stimulation will occur during the alert trials (MA or KP) than during the non-alert (REV) trials. This will be true under the sober and intoxicated conditions.
2. Alcohol will suppress the nystagmic eye movements resulting from rotatory, caloric, and optokinetic stimulation. All aspects of the nystagmic response will be affected; duration, frequency, displacement, and velocity. This hypothesis was formulated to agree with the later studies where the stimulus conditions were controlled more adequately.
3. Alcohol will also suppress the turning ("vertigo") sensations which occur as the result of the rotatory and caloric stimulation.
4. The degree of suppression of the nystagmic and subjective responses will be related to the amount of alcohol consumed.
5. The degree of suppression will be related to the time since consumption of the alcohol. In all of the alcohol groups the degree of response inhibition will be less on the Post II tests (four hours after ingestion) than on the Post I trials (one hour after ingestion).
6. There will be a significant correlation between the subjects' responses to the SAE and the duration of their sensations of turning following the brake decelerations.
7. The effect of alcohol on the duration of the SAE will be the same as its effect on the duration of the turning sensa-

tions; the duration of both will be shortened under alcohol intoxication.

CHAPTER II

EXPERIMENTAL METHOD

Subjects

Subjects were 60 male college students, ranging from 21 to 30 years of age, randomly placed in six groups of ten each. Three of the groups (a) received a series of rotatory stimulations and (b) reported on aftereffects caused by observation of a rotating spiral. The other three groups received (a) a series of caloric irrigations and (b) a series of ocular stimulations by a rotating optokinetic drum. Of the three groups, in each of the two experimental categories (rotating-spiral or caloric-optokinetic), one was designated as "high alcohol," one as "moderate alcohol," and the other served as a control group (no alcohol).

Apparatus

Rotation

The acceleratory stimuli were provided by a Stille-Werner RS-3 rotation device, located in a light-proof room. The RS-3 chair was modified by an enclosure (see Figure 1) which obviated any breeze cues to motion and was fitted with a head rest and bite-board to prevent unwanted head movements. The restraining device was adjusted to position each subject's head so that the horizontal canals were approximately in the plane of rotation.

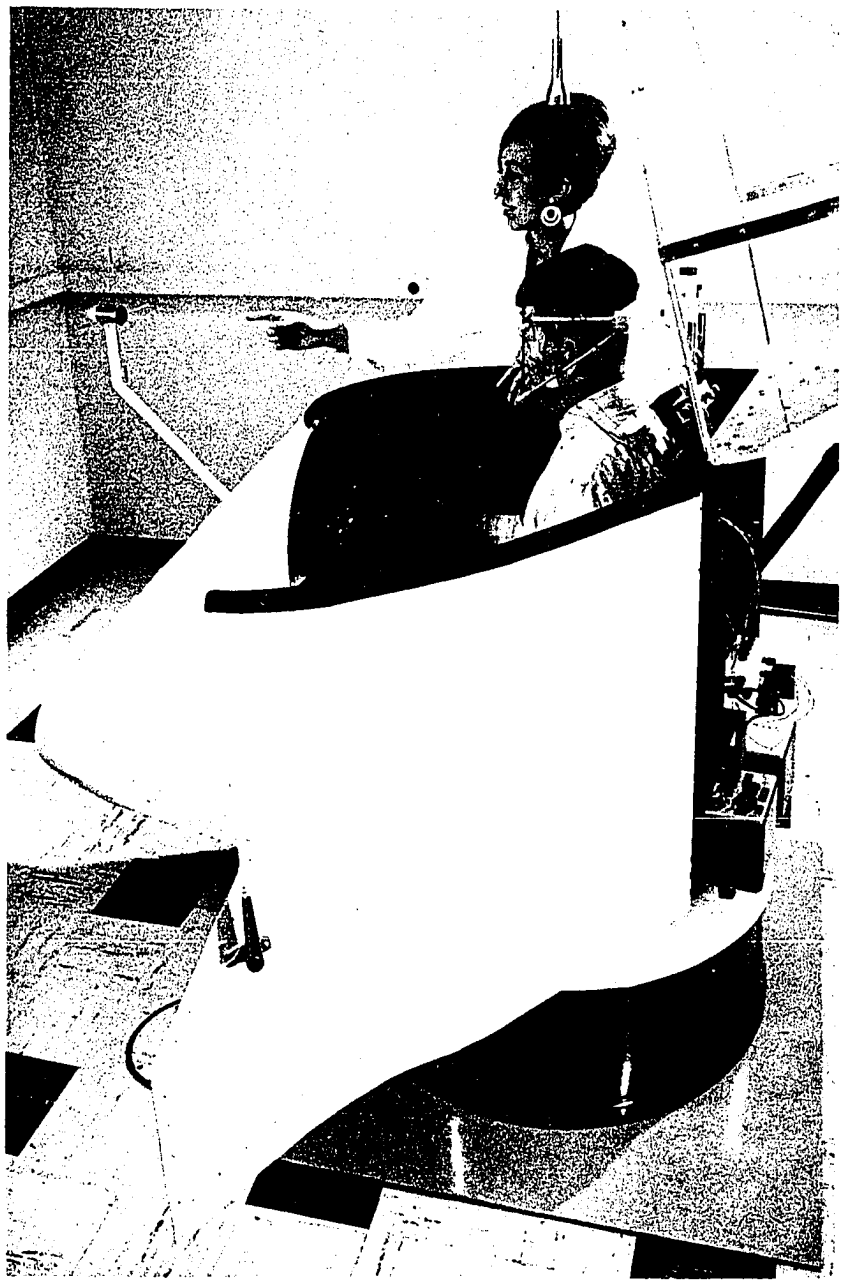

Figure 1. Modified Stille-Werner RS-3 chair used to administer the rotatory stimuli. The head rest and bite board were used to restrict the subjects' head movements and to keep the horizontal canals in the plane of rotation. The cross-shaped device at the front of the rotator contains the calibration lights. With the doors closed and the canopy down, all breeze cues are eliminated. A hole in the canopy permits voice communication with the control room via the microphone suspended above the subject's head.

Caloric

Two water baths equipped with Brownwill constant temperature circulators maintained water temperature at 30°C and 44°C (±0.01°C). Rubber tubing from the base of the water baths passed through solenoids and ended in plastic nozzles through which the caloric stimuli were administered. The solenoids were opened and closed by a foot switch through a Hunter electric timer, to provide a 30-second period of irrigation; the rate of water flow was 15cc per second.

The caloric irrigations were administered while the subject reclined in a supine position on an examining table (see Figure 2). The subject's head was anteverted 30° by means of a head rest which also served as a water receptacle (Collins, 1965).

Spiral Aftereffect

An eight-inch spiral, attached to a variable speed motor was located seven feet in front of the subject (see Figure 3). The duration of the rotation (15 seconds) was controlled by means of a Hunter timer. The speed of rotation was set at 80 rpm. A timer, activated by the depression of a microswitch, was used to measure the duration of the aftereffect.

Optokinetic Drum

Optokinetic nystagmus was elicited by rotation of a drum covered with alternating black (2-inch) and white (2½-inch) stripes (see Figure 4). The drum was 20 inches in diameter, 16 inches tall and located three feet in front of the subject. The drum speed was set at eight rpm. Stimulus duration (15 or 60 seconds) was timed by use of a stop watch.

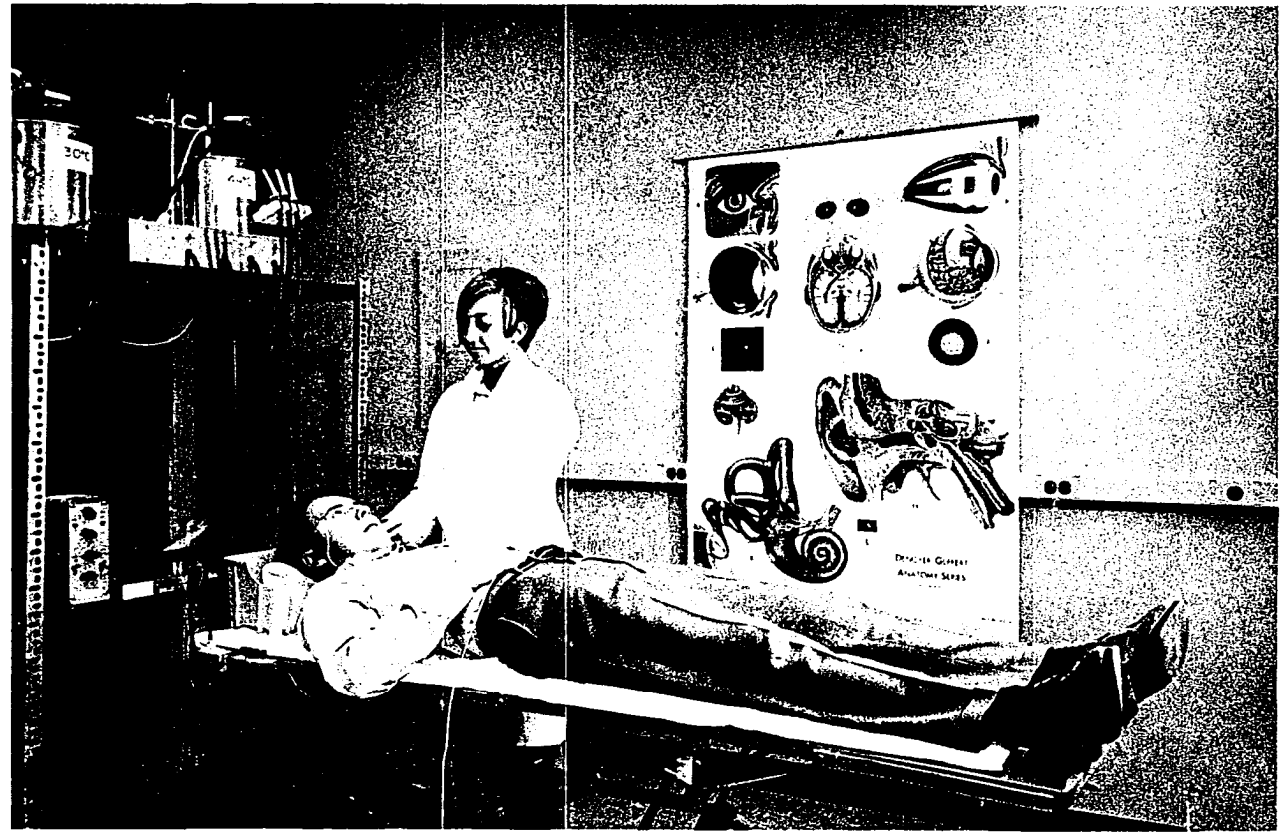

Figure 2. The experimental apparatus used to administer the caloric irrigations. The water passed from the water baths (30° or 44°C) through the solenoids, down the tubing, and through a plastic nozzle into the subject's external auditory canal. A foot switch activated the Hunter timer, which in turn opened and closed the solenoids in order to provide 30-sec irrigations. The device used to antevert the subject's head also served as a collection basin for the return flow of water from the subject's ear.

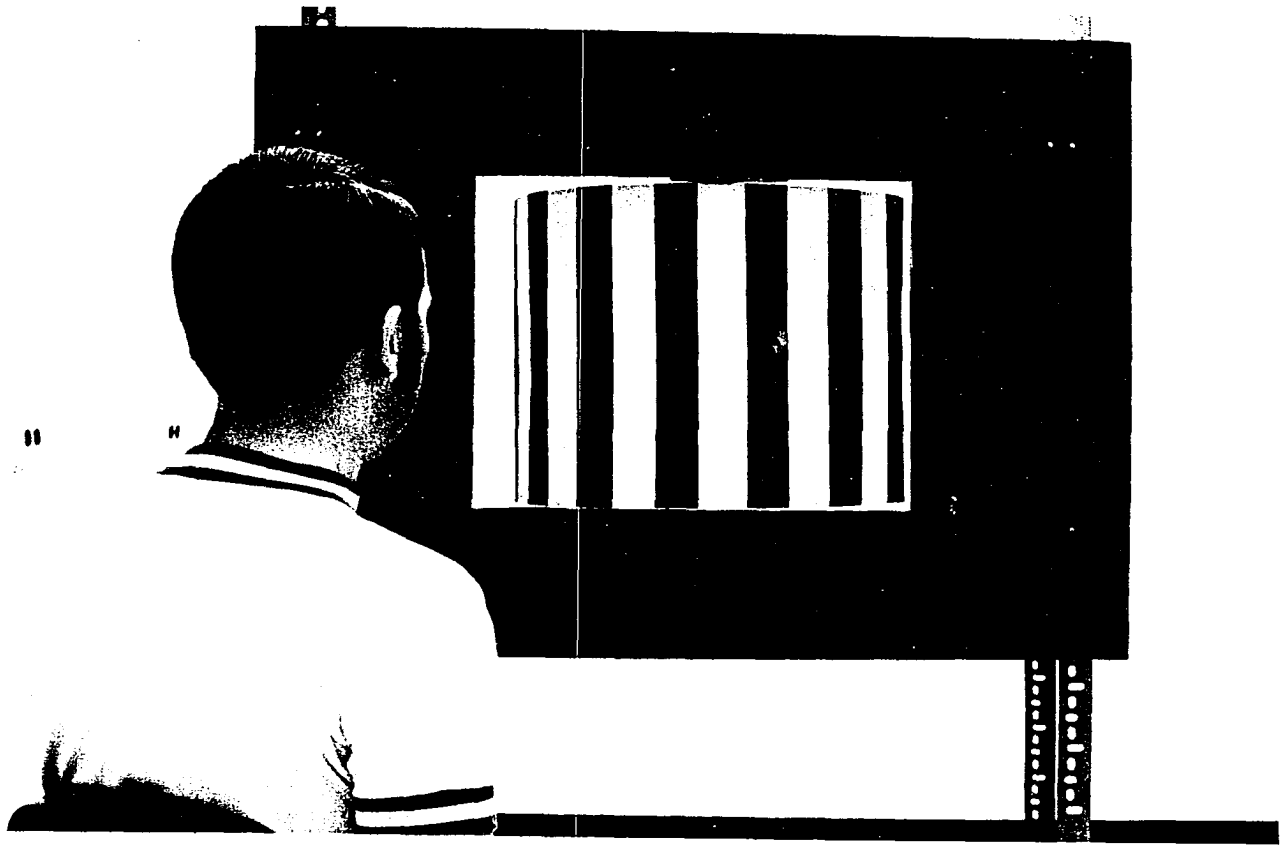

Figure 3. The optokinetic stimulator as it appeared to the subject.

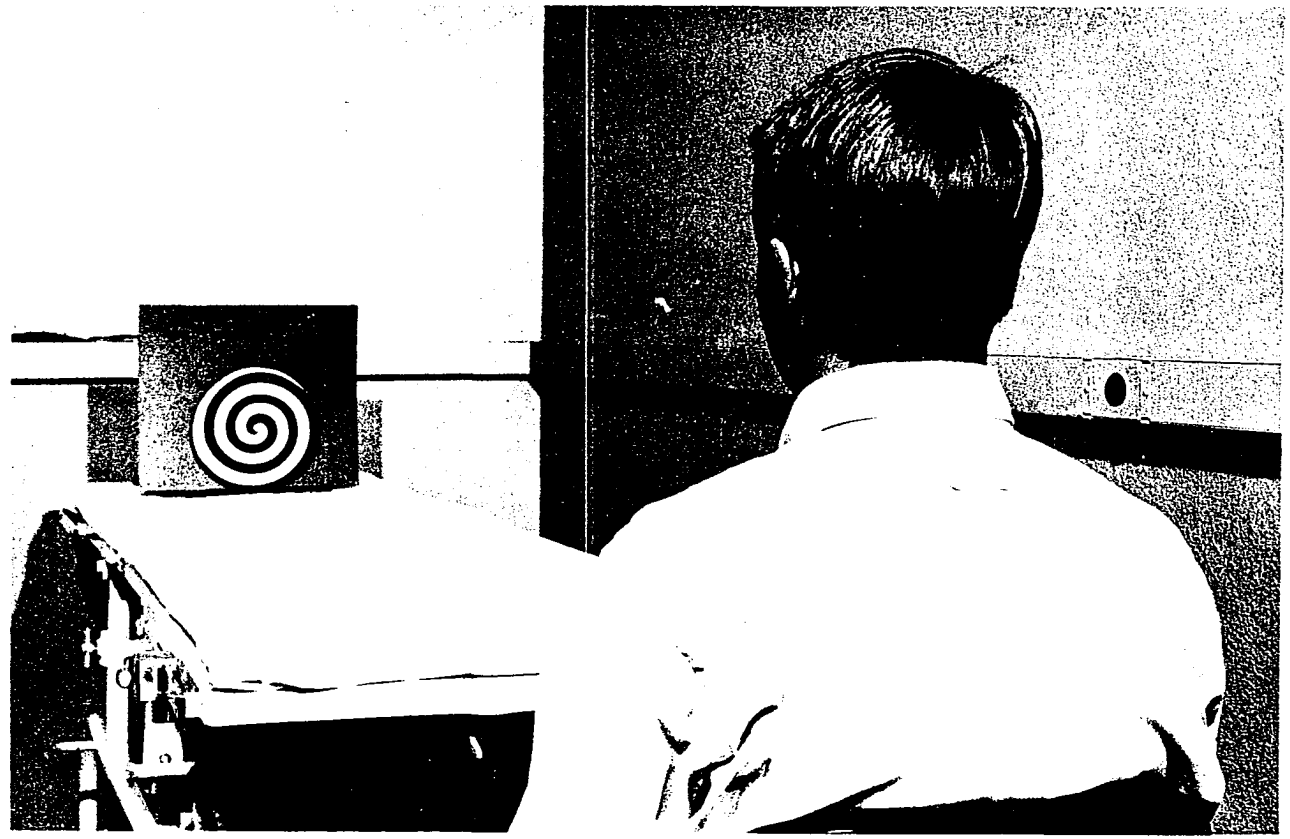

Figure 4. Experimental arrangement used in administering the spiral aftereffect trials.

Recording

Nystagmus

Silver disc electrodes were taped by the outer canthi of the eyes to record horizontal eye movements. An electrode on the forehead served as a ground. The electrodes picked up changes in the corneo-retinal potential and relayed them, through a terminal board (and thence through slip rings in the rotatory situation) to an Offner Type T electroencephalograph (rotation and optokinetic situation) or to an Offner Type Tc electroencephalograph (caloric situation). These signals were then amplified and recorded using a three-second time constant.

Eye movement calibrations were obtained by asking the subject to sweep his eyes between two black dots on a special card at a fixed distance (in the caloric and optokinetic situations), or between two flashing lights fixed to the rotator (see the cross-shaped device in Figure 1). The latter permitted calibration of the subject's eye movements in the dark, both prior to acceleration and during constant velocity prior to the deceleration. A switch, located inside the cabin, allowed the subject to turn the lights on and off. Each calibration tracing represented a fixed number of degrees of eye movement and was used to convert measures of nystagmus from the tracings into degrees of eye movement.

Procedure

Rotation and Spiral Aftereffect Groups

The thirty subjects in these three groups received identical Pre, Post I, and Post II trials. In order to acquaint the subjects with their

task during rotation, they received three practice trials. All three practice trials were counter-clockwise (CCW) rotations in total darkness. The first two practice trials involved an acceleration of $5^{o}/sec^{2}$ to a constant velocity of 60^{o}/sec, a period of two minutes at constant velocity, followed by a deceleration (at the rate of $5^{o}/sec^{2}$) to a stationary position. The third practice trial comprised an acceleration of $5^{o}/sec^{2}$ for 12 seconds, two minutes at constant velocity (60^{o}/sec), and a brake deceleration (approximately 1/2 second) to a standstill.

These practice trials were used to instruct the subjects in the technique of estimating rotational velocity from subjective sensations (Bekesy, 1955; Guedry and Lauver, 1961; Collins, 1964). During the acceleration of the first practice trial the subject signalled, by depression of a microswitch, the start and the end of his turning sensation. During the deceleration the subject was instructed to signal the start, the number of quarter turns he experienced (i.e., subjective movement through 90^{o} arcs), and the end of his turning sensation. On the second practice trial, the subject signalled the start, the quarter turns, and the end of his turning sensation resulting from both the acceleration and deceleration stimuli. Signals for the start, the quarter turns, and the end of the turning sensation were signalled only for the acceleration stimulus of practice trial 3; for the brake deceleration of this trial, the subject signalled only the start and end of his turning sensation, consistent with the technique of cupulometry (Van Edgmond, Groen and Jongkees, 1958).

These practice trials were followed by the experimental Pre trials. All three trials were clockwise (CW) rotations separated by

5-minute rest periods. The accelerations on the three Pre trials were $5^{o}/sec^{2}$ for 12 seconds (i.e., to a constant velocity of 60^{o}/sec). On all three trials, the subject rotated at constant velocity for three minutes. Following a calibration, the deceleration began. On trials 1 and 2, a brake deceleration was used ($120^{o}/sec^{2}$ for .5 seconds). The deceleration on trial 3 was a continuous deceleration at the rate of $5^{o}/sec^{2}$.

During the three Pre trials, the subjects performed different tasks. On one of the first two trials, the subject was instructed to work on a mental arithmetic (continuous division) problem (Collins, 1962; 1964). Different problems were given for the acceleration and deceleration periods, with the subject's answers recorded (approximately 1½ min after the end of the acceleration and the deceleration) to insure that the problems were viewed as an important part of the trial. Following his response, the subject was allowed to relax until alerted by the experimenter. On the other of the first two trials, the subject was instructed to signal his turning sensations as he had done on the third practice trial. Thus, the subject signalled the start, quarter turns, and end of his turning sensation for the acceleration stimulus, and the start and end of his turning sensation for the brake deceleration. After completion of his signals, the subject was told to remain alert and attentive and to call out and signal any other turning experience (secondary sensation). The order in which the subjects received these tasks was counterbalanced.

All of the subjects were given an identical task for the acceleration of trial 3. They were instructed to assume a "reverie" state, i.e.,

to relax, daydream, and not think about the rotation or anything of concern to them. Subjects were allowed to relax and daydream for 30 seconds before the acceleration started. The subject continued to relax at constant velocity until a few seconds before the calibration, at which time he was told to obtain eye movement calibrations and to prepare himself for deceleration. During the continuous deceleration, the room lights were turned on after the end of the deceleration for five subjects or during the deceleration for the remaining five subjects in each of the groups. In the former case, the lights were on for three seconds immediately after the chair stopped; in the latter, the lights were on for eight seconds at the start of the deceleration. After the lights went out, the subject was asked to signal any further turning sensation and to call out the direction in which he experienced motion. For those trials during which the lights were briefly turned on after the end of the deceleration, the subject signalled his turning sensation during deceleration (before the lights were turned on) and again after the brief period of light. Subjects were instructed to watch the walls of the room when the lights were on during the deceleration, and to fixate on special markers attached to the walls of the room while the subjects were at a standstill in illumination. The use of the room lights was designed to provide data regarding the interaction of visual and vestibular stimulation.

Post I and Post II rotation trials were identical to, and were conducted on the day following, the Pre rotation trials. Post I trials began 45 minutes after the consumption of alcohol while Post II trials began four hours after consumption.

Following the rotation trials, the subjects were taken to another room where they viewed a rotating spiral. They were told to fixate on the center of the spiral and to maintain fixation both during the rotation and after the rotation ceased. After watching the spiral for the first time, the subjects were asked to describe their observations including descriptions of the apparent contraction of the spiral during its rotation, and of the aftereffect (the apparent expansion of the spiral) after the end of the rotation. On the second practice trial, the subjects were instructed to signal the duration of the aftereffect by depressing a microswitch as the aftereffect commenced, and to continue depressing the microswitch until the aftereffect was no longer observed. The subjects were then given a series of four trials. During each trial, the spiral was rotated for 15 seconds at 80 rpm, with a three minute rest interval between trials.

Following these trials, the subject was tested for the presence of any positional nystagmus by having him assume a supine position on an examining table with his head slightly elevated. While in total darkness the subject's head was placed, for 30-second periods, in each of the following positions: upright, tilted to the right, upright, tilted to the left, and then upright; in each position, the subject was given a different mental arithmetic problem to maintain a state of alertness. With the exception of the practice trials, the same procedure was followed on the Post I and Post II trials.

Caloric and Optokinetic Groups

As was the case in the rotation groups, all of the subjects in the three caloric groups received identical Pre, Post I and Post II trials.

Prior to the start of the caloric trials the subjects were tested for positional nystagmus in the same manner as that used for the rotation groups.

The caloric irrigations were administered, in total darkness, with the subject in a supine position on an examining table, with his head elevated and anteverted 30°. Prior to each trial, the subject was given instructions designed either to enhance or reduce alertness. Under the alert condition, the subject was instructed to signal, by depression of a microswitch, the start, peak, and end of his sensation; he was also instructed to pay careful attention to his sensation so that he could describe it at the end of the trial. A few seconds after signalling the end of his sensation, the subject performed a reaction-time task, i.e., the subject reacted, as quickly as possible, by depressing a microswitch, to a series of 500 cps tones of 1/10 sec duration. The reaction time was used purely as an alerting technique (Collins, 1963; 1964). For the "reduced alertness" condition (reverie task), the subject was instructed to relax, ignore the environment, and daydream. The order in which the subjects received these tasks was counterbalanced. Trials were separated by 15-minute rest periods to insure a return to normal of the temperature within the ear.

The caloric trials consisted of two R_C irrigations (one Reverie trial and one Alert trial) and two L_W irrigations (one Reverie trial and one Alert trial). R_C and L_W refer to 30-second irrigations of the right ear with water at 30°C or of the left ear with water at 44°C; both drive nystagmus in the same direction. The order of presentation of the trials was counterbalanced.

At the end of his second caloric trial, the subject was asked to rate the two trials in terms of the intensity of the sensation. The trial with the stronger sensation was rated as 100%. The other three trials were rated in reference to this 100% trial. The subject was asked to remember the 100% trial because his ratings on the Post I and Post II trials would be made in reference to that trial. This rating technique has been used successfully in previous studies (Collins, 1965; Collins, Mertens, and Schroeder, 1969).

The order of presentation on the Post I and Post II trials was identical to the Pre trials. The Post I calorizations started approximately 45 minutes after consumption of alcohol while Post II trials started four hours after consumption of alcohol.

Following the caloric trials the subject was taken into an adjoining room and seated three feet from the optokinetic drum. He was told to fixate on the surface of the drum during its rotation and for 30 seconds after the rotation had ended. While watching the drum, the subject was instructed not to follow the stripes actively but to let his eyes follow naturally.

A practice trial (10 seconds of stimulation) was conducted to insure that the subject was watching correctly and to check the recording. The testing session consisted of four trials; of these two involved 60 seconds of optokinetic stimulation, and two involved 15-second stimuli. On two trials (one of 15 seconds and one of 60 seconds), the light was turned off at the end of the stimulus period. The subject's eye movements were recorded throughout the stimulus period and for a subsequent period of 30 seconds in darkness. On the other two trials (one of 15

seconds and one of 60 seconds) the drum was stopped at the end of the stimulus period (lights remained on). The subject's response was recorded during and for a few seconds following stimulation. To prevent fatigue, there was a two-minute rest period between trials. The order of presentation of the various trials was counterbalanced.

In all cases (Pre, Post I and Post II), the optokinetic trials started immediately following the last caloric irrigation.

Alcohol Ingestion and Blood Tests

Subjects in the moderate and high alcohol groups consumed, orally, a mixture of 100 proof vodka and orange juice. In the high alcohol groups, the mixture (approximately 700 cc) contained 2.5cc of vodka per kilogram of body weight. The moderate alcohol mixture (approximately 500 cc) contained 1.25cc of vodka per kilogram of body weight. The control group consumed approximately 400 cc of orange juice with a few drops of rum extract added to alter the taste somewhat. All subjects consumed their drinks in a 15-minute period.

Venous blood samples (three to five cc) were drawn a few minutes prior to drinking, then one-half hour, one hour, and four hours after consumption of the alcohol. The blood alcohol levels were determined by means of a gas chromatograph. Whereas all of the blood samples were analyzed for the alcohol groups, only the first (pre-alcohol) sample was analyzed for the control groups.

Scoring

The nystagmus was scored with respect to duration, frequency, slow phase output (number of degrees), and slow phase velocity (degrees/

sec) during peak slow phase activity. Duration refers to the time interval between the start of the stimulus and the last nystagmic beat. Frequency is the total number of nystagmic beats between the start of the stimulus and the end of the response or for a given time interval. Amount of slow phase eye movement was determined by measuring (in millimeters) the amplitude of each nystagmic beat from slow-phase peak to baseline. The number of millimeters was summed for all of the beats in each three-second interval (rotation and optokinetic trials) or five-second interval (calorizations). This sum was converted, using the calibrations, into degrees of slow phase eye movement. Using Bodin's formula (Bodin, 1966) the velocity of the eye movement was calculated from the average angle of the slow phase of the nystagmus during the five-second (caloric) or three-second (rotation) interval where the slow phase output was maximum.

CHAPTER III

RESULTS

Blood Alcohol Levels

The means and standard deviations for the blood alcohol levels are presented in Table 1; values for individual subjects are located in the appendix. Means for the high alcohol groups at the one hour testing session were nearly twice the average peak values reached by the moderate alcohol groups (90 vs. 52 mg% and 87 vs. 42 mg%). At the last testing sessions, four hours after the ingestion of alcohol, the moderate alcohol groups had very low blood alcohol levels (8 and 19 mg%) while the values for the high alcohol groups (65 mg%) were still above the levels reached by the moderate groups at either the 30-minute or one-hour testing sessions.

Rotation

Nystagmus

Tables 2 through 5 reflect the means and standard deviations for the total number of degrees of slow phase displacement, frequency, duration, and peak slow phase velocity of the nystagmic responses to the rotatory stimuli. Data from the three types of acceleration trials will be discussed separately from the deceleration data, since the latter involve different stimulus conditions.

TABLE 1

Means and Standard Deviations for the Blood Alcohol Levels (in mg. per 100 ml.). Each Value is Based on a Mean for 10 Subjects.

Group	Stimulus		Time Since the Ingestion of Alcohol		
			Thirty Minutes	One Hour	Four Hours
Moderate Alcohol	Caloric	M	41.6	38.4	8.7
		SD	16.7	11.2	5.0
	Rotation	M	44.6	52.1	18.6
		SD	14.1	10.9	3.8
High Alcohol	Caloric	M	70.9	86.9	64.8
		SD	23.6	15.3	10.1
	Rotation	M	64.2	90.0	64.7
		SD	19.7	18.0	10.1

TABLE 2

Means and Standard Deviations for the Slow Phase Nystagmus Displacement (in Degrees) Resulting from the Rotatory Stimuli. Each Group Was Comprised of Ten Subjects.

Group	Stimulus		Mental Arithmetic			Key Press			Reverie		
			Pre	Post I	Post II	Pre	Post I	Post II	Pre	Post I	Post II
Control	Acc	M	567.7	495.2	474.9	514.1	466.2	448.4	339.6	298.0	321.9
		SD	183.7	194.5	204.3	205.4	288.2	165.4	83.3	108.7	83.6
	Dec	M	668.5	759.8	597.0	523.2	492.3	475.3	Interrupted by visual stimulation		
		SD	354.2	442.1	231.1	285.8	347.5	192.9			
Moderate Alcohol	Acc	M	467.1	421.2	538.6	411.6	396.3	505.3	293.9	227.2	336.3
		SD	127.3	95.6	156.8	143.8	93.0	184.0	126.9	83.8	156.6
	Dec	M	443.8	380.8	475.5	359.2	347.5	421.9	Interrupted by visual stimulation		
		SD	157.6	141.3	195.9	146.2	157.6	169.6			
High Alcohol	Acc	M	582.7	307.9	431.7	493.4	315.1	412.9	377.9	184.1	256.7
		SD	313.6	133.7	207.9	211.9	178.2	173.4	168.6	119.2	110.7
	Dec	M	601.3	332.4	402.1	414.9	283.6	369.6	Interrupted by visual stimulation		
		SD	343.1	156.0	131.5	189.8	159.1	145.4			

TABLE 3

Means and Standard Deviations for the Number of Nystagmic Beats Resulting from the Rotatory Stimuli. Each Group Was Comprised of Ten Subjects.

Group	Stimulus		Mental Arithmetic			Key Press			Reverie		
			Pre	Post I	Post II	Pre	Post I	Post II	Pre	Post I	Post II
Control	Acc	M	76.6	79.6	77.2	72.1	78.1	75.7	67.7	61.8	67.2
		SD	19.7	21.5	22.6	21.1	21.3	16.8	16.9	23.6	18.3
	Dec	M	92.4	105.3	97.3	77.0	80.7	81.4	Interrupted by visual stimulation		
		SD	37.1	49.8	37.6	31.7	36.7	32.9			
Moderate Alcohol	Acc	M	76.9	56.4	74.3	71.4	55.2	72.3	64.3	46.3	61.0
		SD	30.6	15.6	22.2	27.9	15.5	20.5	28.6	12.8	20.3
	Dec	M	68.3	48.4	65.2	60.4	48.8	63.5	Interrupted by visual stimulation		
		SD	25.9	14.2	19.7	23.2	14.5	14.1			
High Alcohol	Acc	M	77.9	43.2	59.8	75.9	40.4	59.6	71.5	38.9	56.1
		SD	33.4	17.5	21.4	25.9	17.5	20.4	23.9	18.2	16.0
	Dec	M	80.8	48.8	60.0	68.7	42.8	55.7	Interrupted by visual stimulation		
		SD	31.9	18.0	19.5	23.7	18.3	17.5			

TABLE 4

Means and Standard Deviations for the Peak Velocity (Deg/Sec) of the Slow Phase Nystagmus Resulting from the Rotatory Stimuli. Each Group Was Comprised of Ten Subject.

Group	Stimulus		Mental Arithmetic			Key Press			Reverie		
			Pre	Post I	Post II	Pre	Post I	Post II	Pre	Post I	Post II
Control	Acc	M	37.3	36.3	37.5	41.2	35.0	40.6	29.8	25.4	31.9
		SD	7.4	12.4	11.8	12.1	11.6	8.6	9.0	9.6	7.0
	Dec	M	46.8	54.8	53.1	61.4	59.2	56.6	Interrupted by visual stimulation		
		SD	17.9	20.0	17.6	19.3	15.3	11.3			
Moderate Alcohol	Acc	M	29.4	32.4	36.8	32.1	32.3	35.1	25.3	26.0	31.7
		SD	11.8	8.0	13.7	10.1	9.7	9.5	8.4	12.4	14.4
	Dec	M	39.1	46.1	48.5	41.4	45.2	41.8	Interrupted by visual stimulation		
		SD	16.8	14.6	16.1	17.2	16.8	18.3			
High Alcohol	Acc	M	35.6	26.2	35.3	35.8	24.1	35.5	28.5	18.2	24.2
		SD	16.4	6.7	15.4	16.4	10.7	8.9	8.9	9.2	7.9
	Dec	M	48.6	43.6	43.7	46.0	39.0	54.6	Interrupted by visual stimulation		
		SD	25.4	11.2	19.3	14.5	21.4	14.1			

TABLE 5

Means and Standard Deviations for the Duration (in Seconds) of the Nystagmic Response Resulting from the Rotatory Stimuli. Each Group Was Comprised of Ten Subjects.

Group	Stimulus		Mental Arithmetic			Key Press			Reverie		
			Pre	Post I	Post II	Pre	Post I	Post II	Pre	Post I	Post II
Control	Acc	M	53.4	52.8	51.0	51.0	52.2	48.2	45.8	44.4	43.8
		SD	8.6	8.4	11.4	9.8	13.9	8.2	7.2	7.8	3.0
	Dec	M	59.1	62.2	57.6	52.2	55.1	48.6	Interrupted by visual stimulation		
		SD	19.2	27.6	21.9	24.2	23.3	19.0			
Moderate Alcohol	Acc	M	61.1	49.1	56.4	55.6	45.1	52.0	45.4	35.0	42.6
		SD	13.6	7.6	11.0	11.5	8.8	8.3	9.8	8.4	8.1
	Dec	M	54.5	40.2	46.8	47.3	36.2	41.6	Interrupted by visual stimulation		
		SD	13.0	7.1	7.1	5.9	5.3	8.5			
High Alcohol	Acc	M	58.4	39.1	43.9	53.2	36.4	43.3	46.6	30.4	38.5
		SD	10.7	7.2	7.0	9.6	8.3	6.5	5.5	12.2	10.6
	Dec	M	53.1	37.6	39.0	44.8	30.1	35.4	Interrupted by visual stimulation		
		SD	13.2	13.7	8.7	8.0	12.5	9.2			

Acceleration Stimuli.

The various measures of the responses to the acceleration stimuli were subjected to separate analyses of variance. The "trial" factor was significant (.05 - .001 levels) for all analyses (Table 6). Several t tests were then conducted in order to determine if the significant effects were due to the presence of alcohol, or if the Pre-Post I-Post II variations occurred equally across all three groups.

Control Group. Both the mean slow phase displacement and velocity measures evidenced slight Pre to Post I declines across the MA, KP, and Rev trials; 9-13% in displacement and 3-15% in velocity. Pre to Post I changes in duration varied from a 3% decline for the Rev stimulus to a 2% increase for the KP stimulus. Similar changes were evident in the frequency of the nystagmus; a 9% reduction to an 8% increase. Results of the paired t tests (Table 7) indicate that the slow phase displacement for the MA acceleration and peak velocity for the KP acceleration were significantly lower (.05 and .01 levels respectively) for the Post I trials. None of the other Pre-Post I changes were significant.

While the mean Post II values for peak velocity were above the Post I values, most of the other measures of the nystagmic responses evidenced slight Post I to Post II declines. However, only the t test (.01 level) for the Pre-Post II change in slow phase displacement for the MA acceleration stimulus was significant (Table 7).

With the exception of slight Pre to Post II response declines, there was, in general, little change in the nystagmic responses of the control group. The changes which did occur probably can be attributed to habituation.

TABLE 6

Results of the Analyses of Variance for the Various Measures of the Nystagmic Responses Resulting from the Rotatory Stimuli.

Source	F			
	Slow Phase Displacement	Frequency	Duration	Peak Velocity
Groups (G)	0.62	1.85	2.98	1.40
Instructions (I)	32.12***	6.67**	36.02***	15.94***
I x G	0.06	0.46	1.48	0.56
Trials (Tr)	15.98***	28.14***	33.30***	8.44***
Tr x G	7.15**	10.21***	9.97***	2.61*
I x Tr	0.72	0.35	0.50	0.45
I x Tr x G	0.56	0.58	0.22	0.74

* $p < .05$
** $p < .01$
*** $p < .001$

TABLE 7

Results of the Paired t Tests for the Pre vs. Post I and Pre vs. Post II Comparisons of the Nystagmic Responses of the Control Group to the Rotatory Stimuli.

		Condition				
		Mental Arithmetic		Key Press		Reverie
Measure	Comparison	Acc	Dec	Acc	Dec	Acc
Displacement	Pre vs. Post I	3.45**	1.07	1.16	0.92	0.98
	Pre vs. Post II	3.94**	1.15	2.06	0.92	0.68
Frequency	Pre vs. Post I	0.81	2.03	2.12	0.83	0.85
	Pre vs. Post II	0.15	0.79	0.66	1.27	0.13
Duration	Pre vs. Post I	0.26	0.78	0.64	0.82	0.44
	Pre vs. Post II	0.62	0.48	1.34	1.17	0.88
Velocity	Pre vs. Post I	0.32	1.83	2.60*	0.64	1.21
	Pre vs. Post II	0.05	1.79	0.24	1.36	0.71

* $p < .05$
** $p < .01$

Moderate Alcohol Group. Mean peak velocity was the only measure of the nystagmic response of the moderate alcohol group to show a Post I increase (1-10%) from its Pre level. Although the mean Post I slow phase displacement values were all below the Pre levels, only the decline (23%) for the Rev accelerations was larger than comparable declines evidenced by the control group. None of the above changes, according to t tests, was significant (Table 8). Mean values for duration of the nystagmic responses for the Post I trials were 19-23% below their Pre levels, while frequency was down 23-28%. With the exception of the decline in frequency for the Rev acceleration, all of the declines in frequency and duration were significant (.05 - .01 levels).

The Post II measures of slow phase displacement and peak velocity were always above the Pre levels. Although mean duration values for the Post II trials were all above the Post I levels, they were still 6-8% below the Pre levels. The frequency means for the Post II trials ranged from 1% above to 5% below the Pre levels. A significant increase (.05 level) in the peak velocity of the nystagmus for the KP acceleration was the only statistically significant Pre-Post II change.

High Alcohol Group. All Post I means for the acceleration stimuli were below their Pre levels. These declines, across the MA, KP, and Rev trials, were: 45-47% in frequency, 36-51% in slow phase displacement, 32-35% in duration, and 26-36% in velocity. The Post I responses for all measures were significantly below (.05 - .001 levels) the Pre responses (Table 9).

Means for the Post II trials were always above the Post I levels; however, some were still significantly lower than the Pre values (Table

TABLE 8

Results of the Paired t Tests for the Pre vs. Post I and Pre vs. Post II Comparisons of the Nystagmic Responses of the Moderate Alcohol Group to the Rotatory Stimuli.

		Condition				
		Mental Arithmetic		Key Press		Reverie
Measure	Comparison	Acc	Dec	Acc	Dec	Acc
Displacement	Pre vs. Post I	1.12	1.28	0.31	0.27	1.80
	Pre vs. Post II	1.60	0.57	1.70	1.35	0.89
Frequency	Pre vs. Post I	3.40**	4.12**	2.75*	1.91	2.11
	Pre vs. Post II	0.44	0.75	0.16	0.57	0.45
Duration	Pre vs. Post I	2.79*	3.69**	3.96**	4.89**	3.57**
	Pre vs. Post II	0.94	1.93	1.04	1.90	1.01
Velocity	Pre vs. Post I	0.07	0.93	0.76	2.33*	0.21
	Pre vs. Post II	0.90	0.08	2.71*	3.18*	1.78

* $p < .05$
** $p < .01$
*** $p < .001$

TABLE 9

Results of the Paired t Tests for the Pre vs. Post I and Pre vs. Post II Comparisons of the Nystagmic Responses of the High Alcohol Group to the Rotatory Stimuli.

		Condition				
		Mental Arithmetic		Key Press		Reverie
Measure	Comparison	Acc	Dec	Acc	Dec	Acc
Displacement	Pre vs. Post I	3.50**	2.96*	3.97**	3.00*	4.97***
	Pre vs. Post II	2.04	2.43*	1.74	1.38	3.34**
Frequency	Pre vs. Post I	5.48***	4.91***	7.45***	4.99***	5.08***
	Pre vs. Post II	3.46**	4.02**	3.09	2.89*	2.75*
Duration	Pre vs. Post I	6.60***	2.53	5.18***	3.17*	5.03***
	Pre vs. Post II	4.37**	6.42***	3.32**	2.62*	3.31**
Velocity	Pre vs. Post I	2.37*	0.94	2.65*	1.08	3.39**
	Pre vs. Post II	0.09	0.77	0.07	2.52*	1.74

* $p < .05$
** $p < .01$
*** $p < .001$

9). Post II mean frequency and duration values all remained significantly depressed (.05 - .001 levels). None of the Pre to Post II differences in velocity was significant and the only significant Pre to Post II difference (.01 level) for slow phase displacement was for the response to the Rev acceleration.

Overview. These data from the alcohol groups point to the depressive influence of alcohol on the nystagmic responses to angular accelerations; these changes are evident in the duration, frequency and slow phase displacement scores for the average response curves (Figures 5 to 10). The change in the response from the Pre to the Post I testing session is also evident in the nystagmic tracings of a subject (DM) presented in Figure 11.

Alertness. The effects of variation in task (alertness) on responses resulting from the acceleration stimuli were also examined (see Figure 11). Nystagmic output, for all measures, was greater for the KP and MA stimuli than for the Rev condition. Data from the analyses of variance (Table 6) indicate that the "task" factor was significant (.05 - .001 levels) for all measures. These changes in the response as the result of variations in instructions designed to influence the alertness of the subjects are also evident in the average curves for frequency and slow phase displacement (Figures 5 through 10). Peak slow phase values for the KP and MA conditions (alert) were around 22°/sec (65° per 3-sec interval) while the peak for the Rev condition (non-alert) was about 17°/sec (50° per 3-sec interval). The longer durations for the KP and MA conditions, as opposed to those for the Rev condition, are also evident in those figures as are the effects of alertness on the magnitude

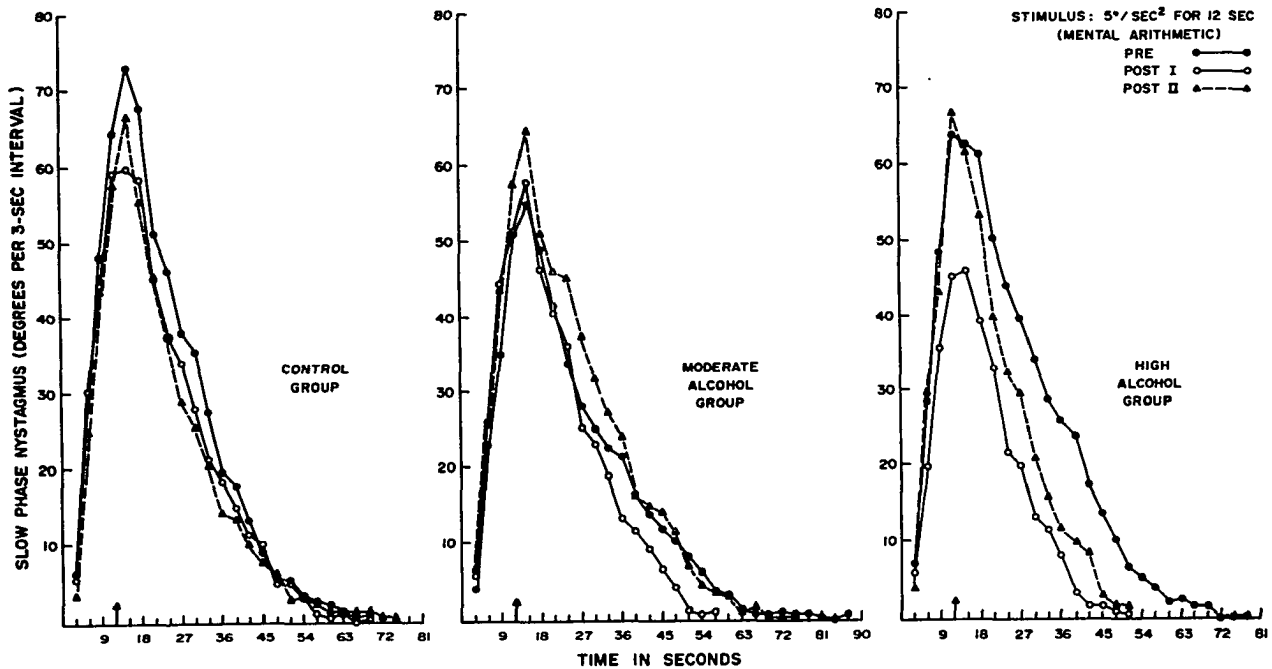

Figure 5. Response data for the average number of degrees of slow phase eye movement resulting from the angular accelerations ($5°/sec^2$ for 12 sec), under the Mental Arithmetic condition. Pre refers to the response recorded prior to the ingestion of alcohol, while the Post I and Post II data were obtained 45 min and four hours after ingestion. The arrow on the abscissa indicates the end of the stimulus. The values are plotted in 3-sec intervals; each point is a mean for 10 subjects.

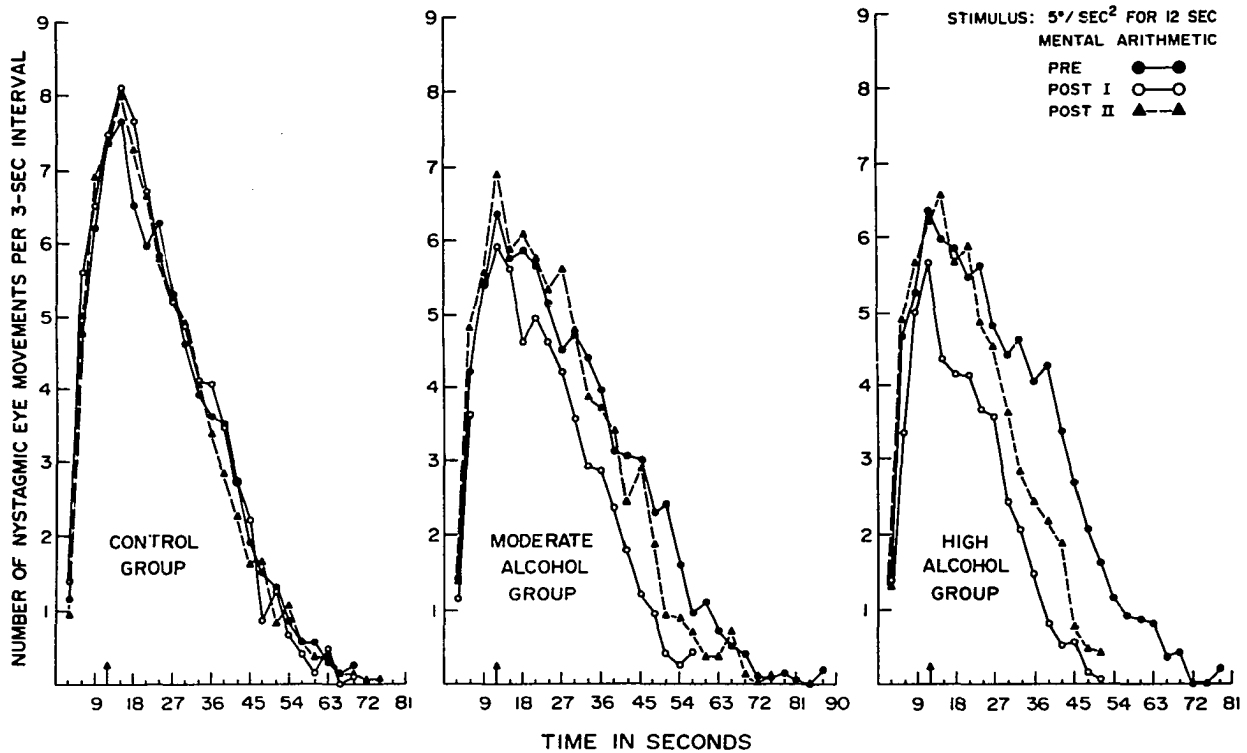

Figure 6. Response data for the average number of nystagmic eye movements resulting from the angular accelerations ($5^{o}/sec^{2}$ for 12 sec), under the Mental Arithmetic condition. Symbols and markings are identical to those used in Figure 5.

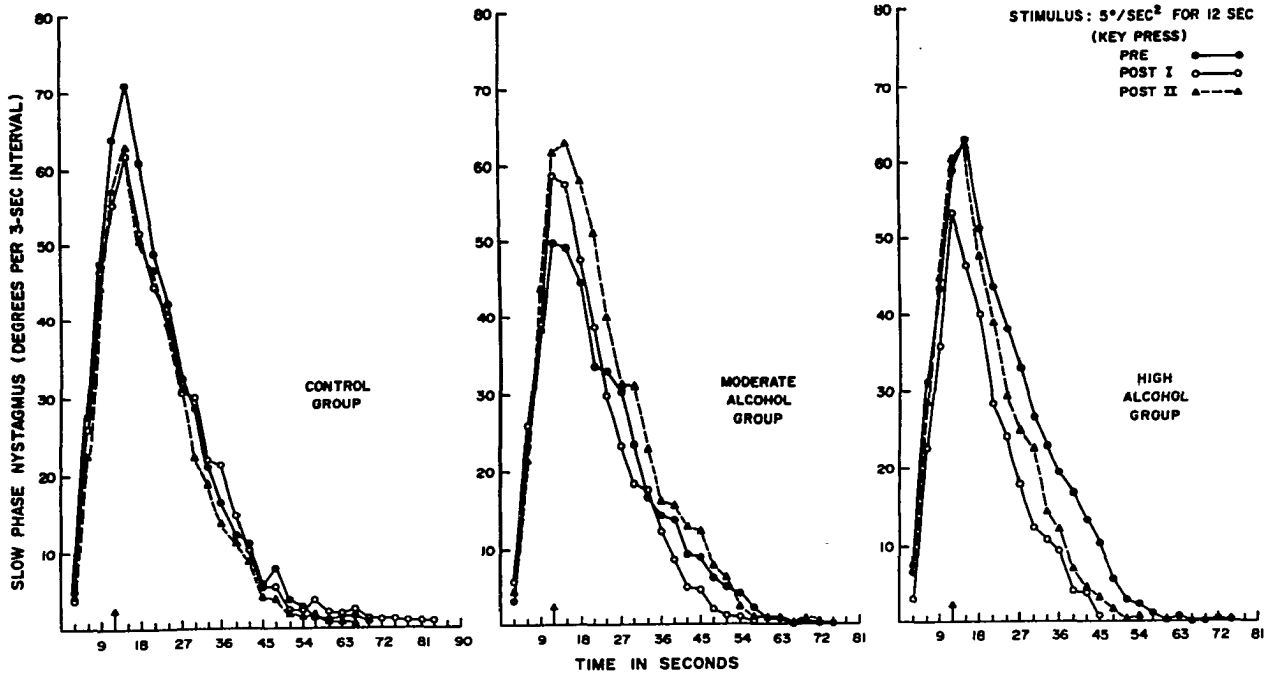

Figure 7. Response data for the average number of degrees of slow phase eye movement resulting from the angular accelerations ($5^{o}/sec^{2}$ for 12 sec), under the Key Press condition. Symbols and markings are identical to those used in Figure 5.

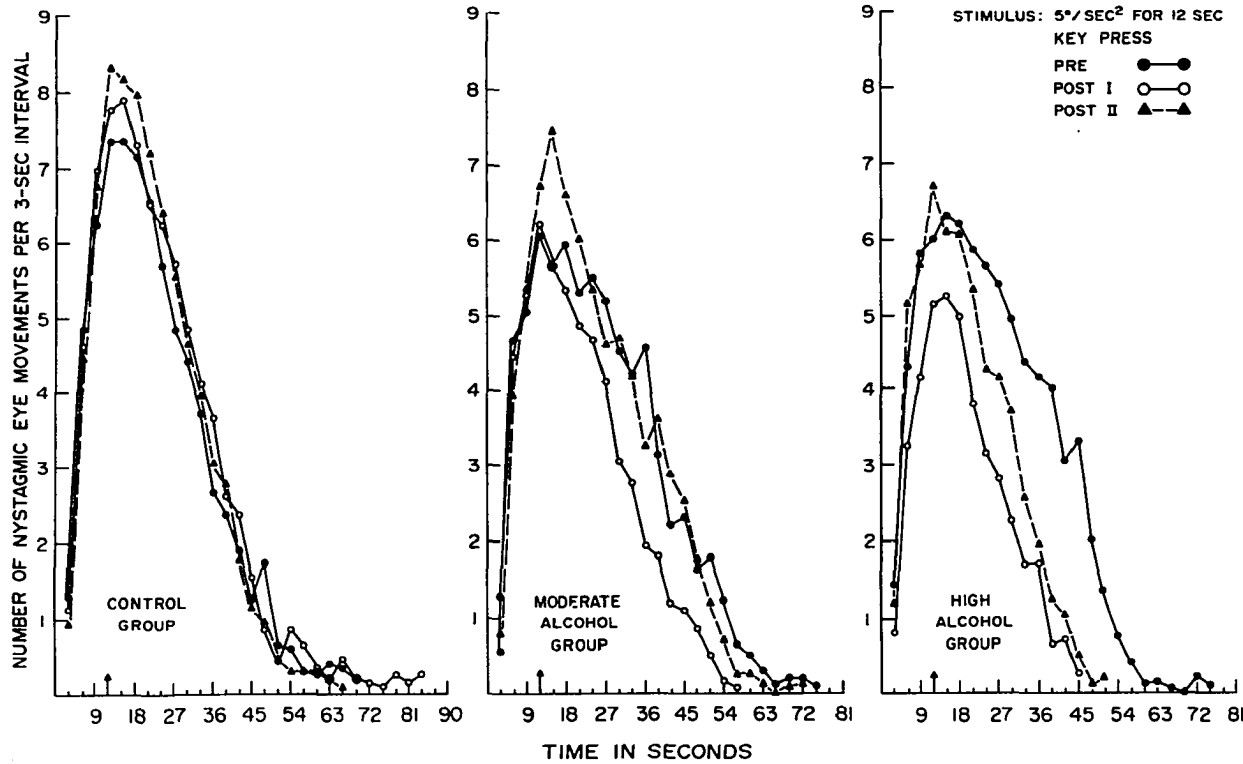

Figure 8. Response data for the average number of nystagmic eye movements resulting from the angular accelerations ($5^{o}/sec^{2}$ for 12 sec), under the Key Press condition. Symbols and markings are identical to those used in Figure 5.

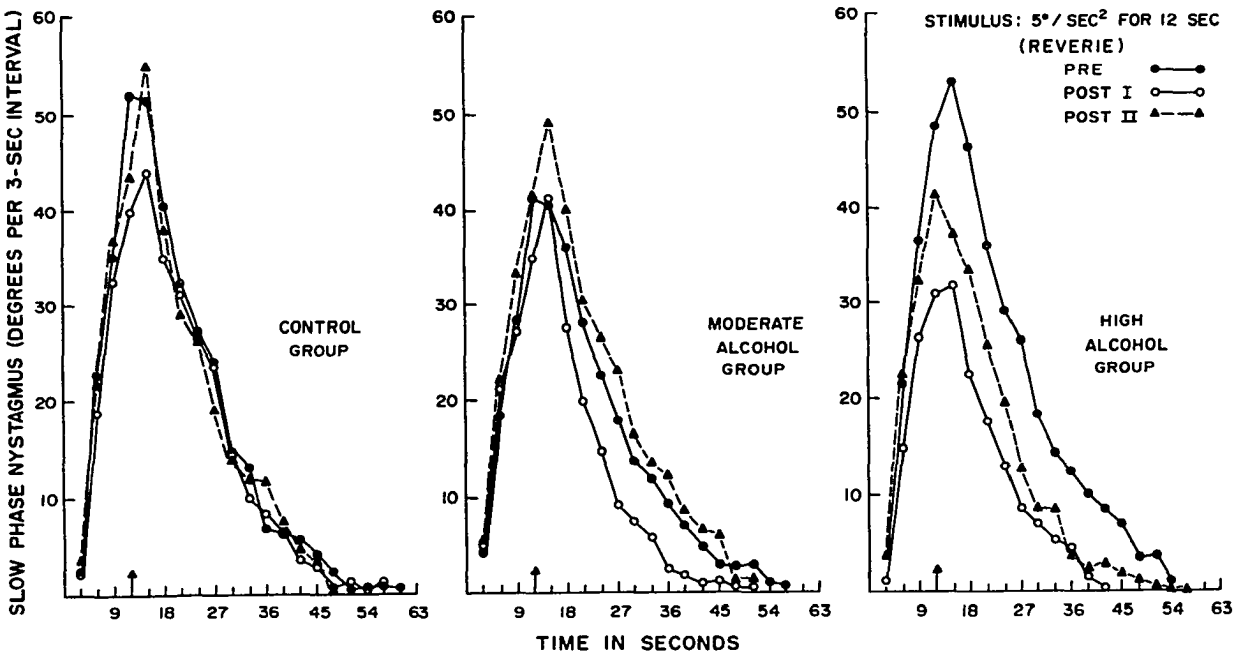

Figure 9. Response data for the average number of degrees of slow phase eye movement resulting from the angular accelerations ($5^{o}/sec^{2}$ for 12 sec), under the Reverie (relaxed) condition. Symbols and markings are identical to those used in Figure 5.

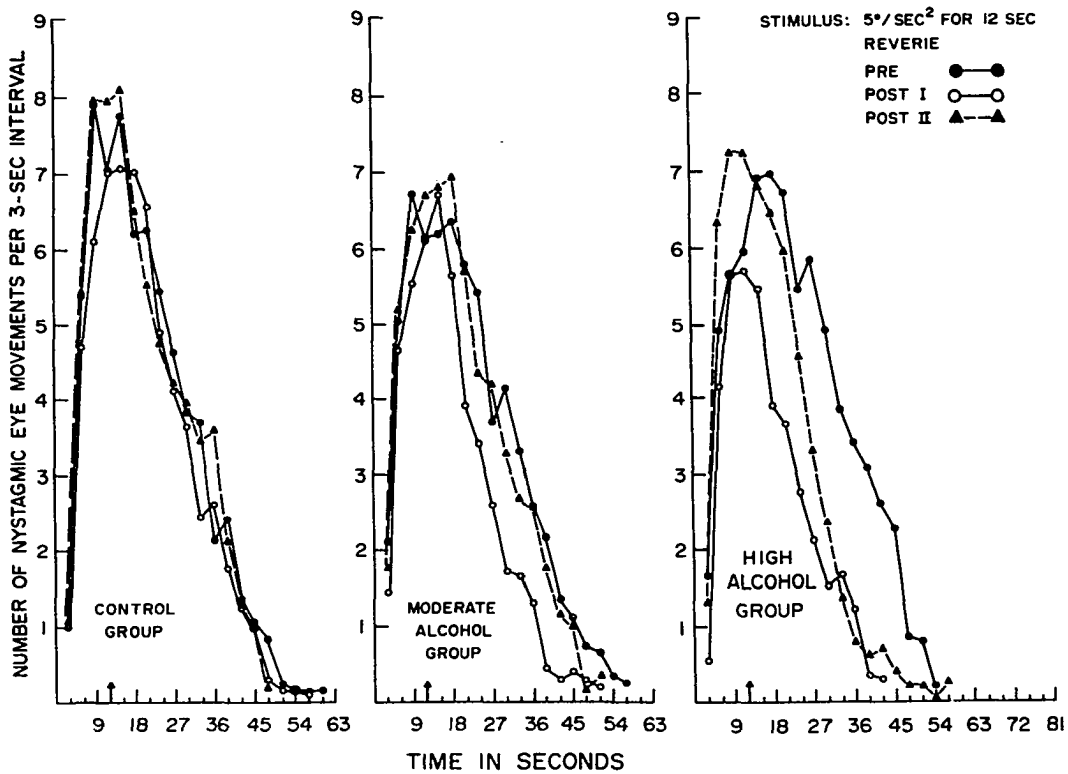

Figure 10. Response data for the average number of nystagmic eye movements resulting from the angular accelerations ($5^{o}/sec^{2}$ for 12 sec), under the Reverie (relaxed) condition. Symbols and markings are identical to those used in Figure 5.

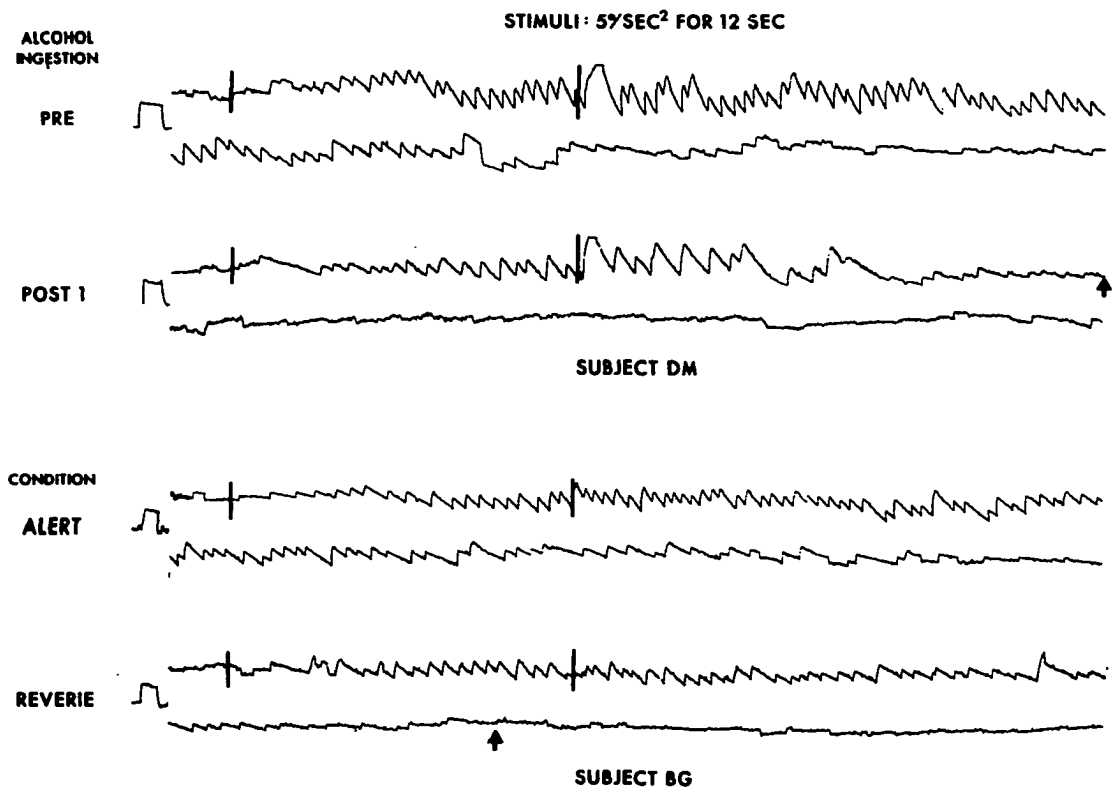

Figure 11. A portion of the nystagmic tracings, for two subjects, resulting from a 12-sec acceleration of $5^{o}/sec^{2}$. Calibrations (15^{o} of eye movement) appear before each trial. The vertical bars demarcate the stimulus period and the arrows indicate the end of the primary nystagmic response. The tracing for subject DM reveals the suppressive effect of the high alcohol dose on the nystagmic response (Mental Arithmetic condition). All aspects of the Post I nystagmic response are suppressed (slow phase displacement, frequency, peak velocity and duration of the nystagmus). The tracings for subject BG in the lower half of the figure (both taken from Pre trials) reflect the influence of alerting instructions (Key Press) on the nystagmic response. All measures of the nystagmic response during the Alert condition are enhanced in comparison with the Reverie condition.

of the Pre-Post I response declines. In most instances the decline was less under the alert than under the non-alert condition. An example of this is the Pre-Post I change in frequency for the control group; under the KP and MA conditions, the responses evidenced 8 and 4% increases, while under the Rev condition the response declined 9%.

Brake Deceleration Data.

Means and standard deviations for the deceleration stimuli appear in Tables 2 through 5. Average frequency and slow phase response curves for these stimuli are in Figures 12 through 15. Results of the paired t tests for the Pre-Post I and Pre-Post II comparisons appear in Tables 7 through 9.

Although the deceleration stimuli were of much higher magnitude and much shorter duration than the acceleration stimuli, the responses of the control, moderate alcohol, and high alcohol groups were very similar to those noted for the acceleration stimuli. The control group evidenced little Pre-Post I-Post II changes for any of the response measures. The Post I trials for the moderate alcohol group were, in some instances, significantly below the Pre levels (.05-.01 levels); however, at the four-hour testing session (Post II), no significant differences from the Pre levels were evident. As was true for the acceleration stimuli, the high alcohol group exhibited the largest Pre-Post I decline in the response measures for the deceleration stimulus. With the exception of the peak nystagmus velocity measure, all of the Post I measures were significantly below (.05-.001 levels) their Pre values (Table 9). Mean Post II measures of the nystagmic responses were always above the Post I level for the high alcohol group. However, for

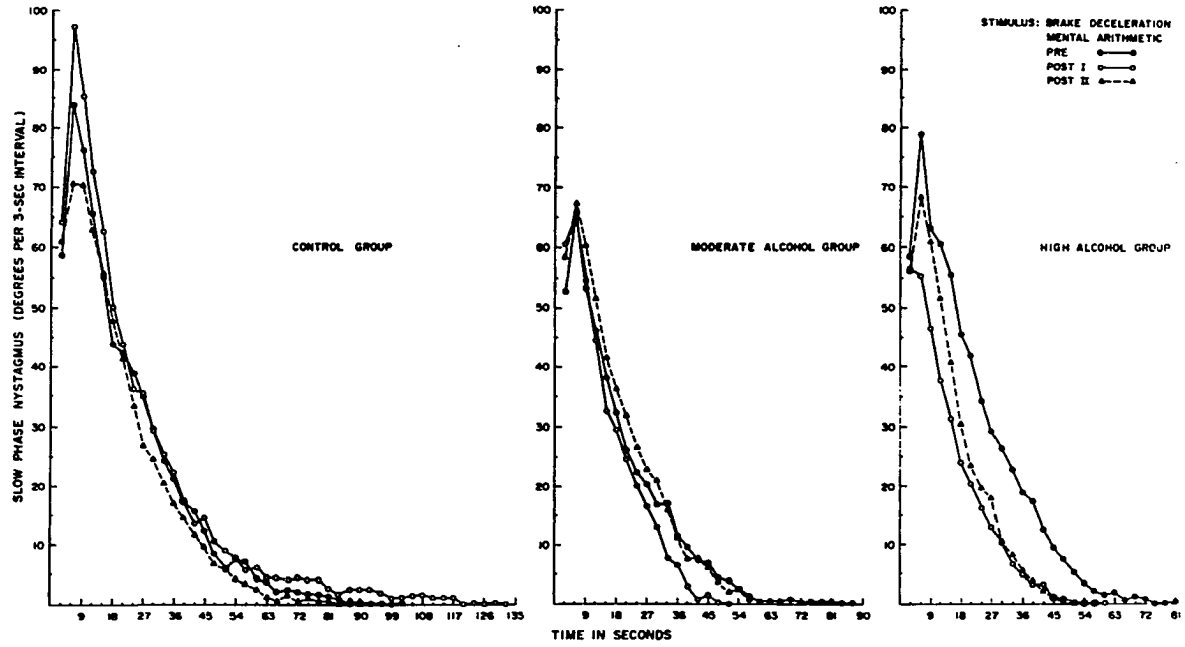

Figure 12. Response data for the average number of degrees of slow phase nystagmus resulting from the brake decelerations, under the Mental Arithmetic condition. Since the deceleration (from 60^{o}/sec) was very rapid (approximately 0.5 sec), the values are plotted from the start of the deceleration. Symbols and markings are identical to those used in Figure 5.

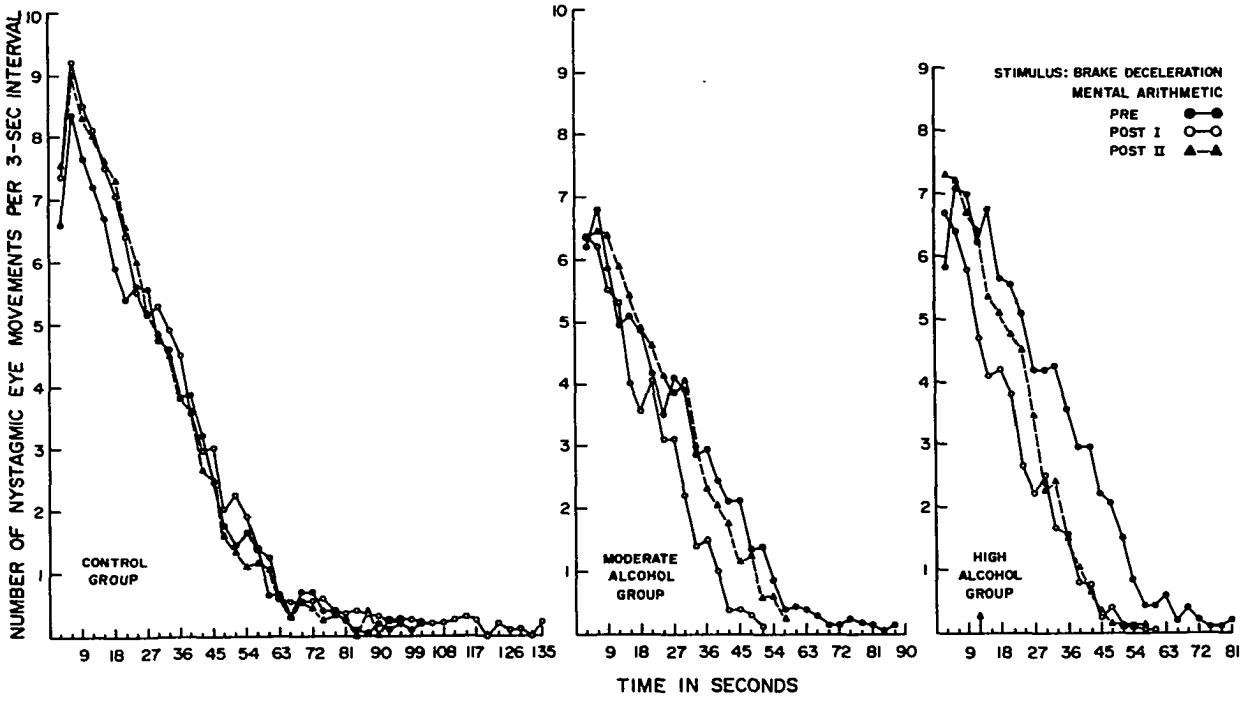

Figure 13. Response data for the average number of nystagmic eye movements resulting from the brake decelerations under the Mental Arithmetic condition. Symbols and markings are identical to those used in Figure 5.

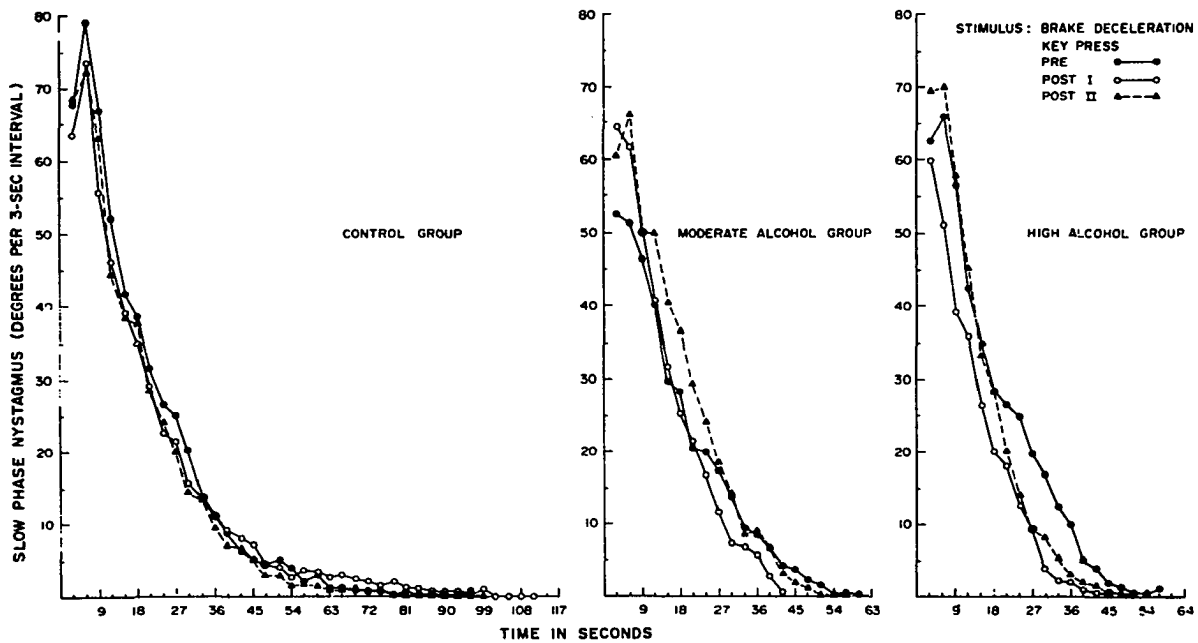

Figure 14. Response data for the average number of degrees of slow phase nystagmus resulting from the brake decelerations under the Key Press condition. Symbols and markings are identical to those used in Figure 5.

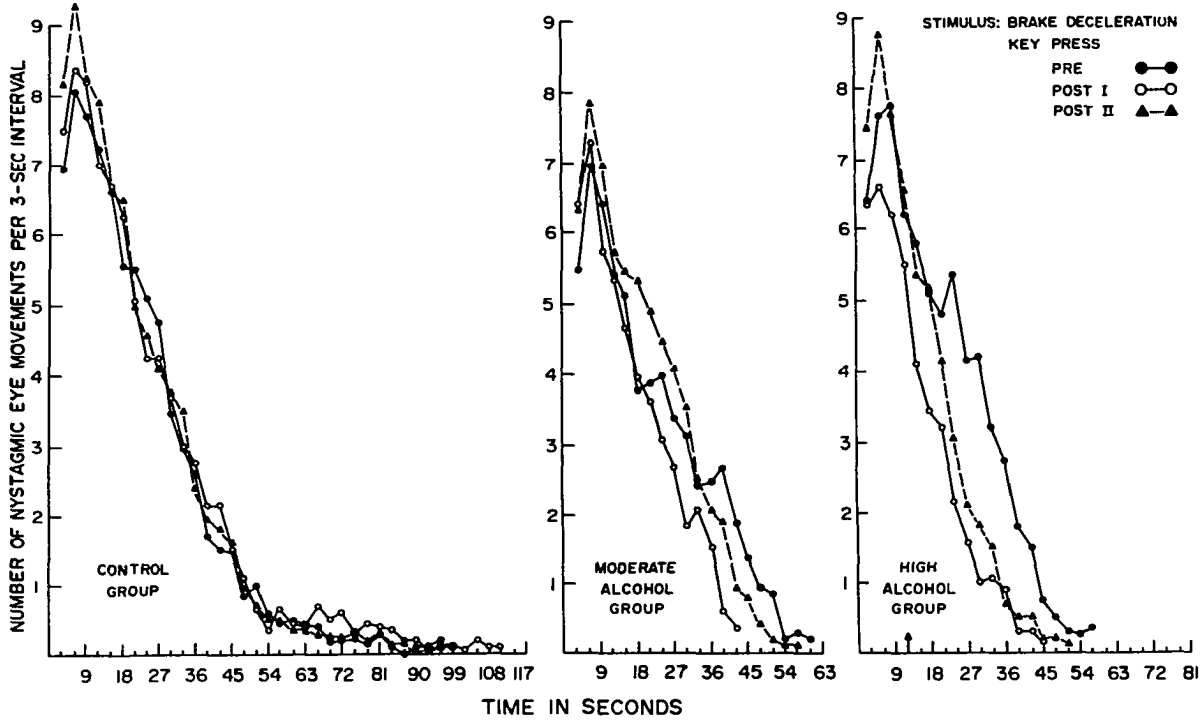

Figure 15. Response data for the average number of nystagmic eye movements resulting from the brake decelerations under the Key Press condition. Symbols and markings are identical to those used in Figure 5.

some of the measures the responses were still significantly below their initial levels (Table 9).

Light Deceleration Data.

Means and standard deviations for the slow phase displacement and frequency of the primary and secondary nystagmus resulting from the prolonged (light) decelerations are presented in Tables 10 and 11. Means and standard deviations for the duration of the primary nystagmus resulting from the prolonged light decelerations are presented in Table 12; for comparative purposes duration values for a comparable stimulus (a dark acceleration) are also presented. Since each group was divided into a 3-sec light and an 8-sec light sub-group, each mean represents the average response for five subjects. Average response curves for the slow phase displacement and frequency of the combined primary and secondary responses are presented in Figures 16 through 19. Due to the small number of subjects in each of the groups these results should be interpreted with caution.

Control Group. Post I mean values for both the 3-sec light and the 8-sec light subgroups of the control subjects were lower (4 and 16% respectively) than the Pre values for slow phase displacement of the primary nystagmus resulting from the light decelerations. In contrast to this, the Post I mean frequency of the primary nystagmus was higher than the Pre level for both of the control light groups. Values for the displacement and frequency measures were very near the Pre level for the Post II trials.

Moderate Alcohol Group. Although the Pre to Post I declines in frequency and slow phase displacement for the 8-sec light subjects of the

TABLE 10

Means and Standard Deviations for the Slow Phase Displacement of Primary and Secondary Nystagmus (in Degrees) Resulting from the 12-sec Deceleration of $5^{\circ}/sec^{2}$. The Room Lights Were on for Either 3-sec After the Decel or for 8-sec at the Start of the Deceleration.

Group	Light Interval		Nystagmus					
			Primary			Secondary		
			Pre	Post I	Post II	Pre	Post I	Post II
Control	3 sec	M	276.7	251.3	265.4	54.6	54.2	67.5
		SD	46.2	57.1	42.2	39.1	41.9	65.5
	8 sec	M	245.6	235.6	246.3	128.9	81.5	79.8
		SD	289.0	205.9	193.0	141.3	72.1	83.7
Moderate Alcohol	3 sec	M	221.4	282.2	241.1	58.2	73.9	71.5
		SD	34.6	97.7	69.8	55.8	36.0	46.9
	8 sec	M	156.7	138.3	156.7	48.4	60.1	46.2
		SD	93.7	64.1	89.0	42.2	51.7	39.1
High Alcohol	3 sec	M	258.5	231.5	228.2	44.0	53.9	42.0
		SD	162.9	130.2	120.4	40.3	28.3	28.5
	8 sec	M	201.6	156.0	243.2	51.4	26.1	80.1
		SD	119.4	76.3	88.6	33.0	32.6	23.8

TABLE 11

Means and Standard Deviations for the Number of Primary and Secondary Nystagmic Eye Movements Resulting from the 12 sec Deceleration of $5^{o}/sec^{2}$. The Room Lights Were on for Either 3-sec After the Decel or for 8-sec at the Start of the Deceleration.

Group	Light Interval		Nystagmus					
			Primary			Secondary		
			Pre	Post I	Post II	Pre	Post I	Post II
Control	3 sec	M	46.6	50.0	52.6	14.6	15.4	15.4
		SD	16.1	23.2	24.2	8.8	9.1	9.4
	8 sec	M	37.2	44.3	36.6	23.0	16.9	22.5
		SD	29.1	21.9	19.0	15.1	10.2	14.8
Moderate Alcohol	3 sec	M	46.4	55.5	47.0	14.8	18.0	19.1
		SD	8.4	9.8	15.4	11.1	7.6	10.8
	8 sec	M	32.4	24.1	30.0	15.2	17.2	12.3
		SD	20.0	11.6	17.2	13.4	13.0	9.2
High Alcohol	3 sec	M	46.0	43.2	47.1	13.0	16.4	14.0
		SD	17.7	15.1	18.5	10.9	5.8	8.4
	8 sec	M	39.1	27.7	41.4	16.8	11.4	22.2
		SD	21.3	12.9	15.4	10.6	11.8	4.7

TABLE 12

Means and Standard Deviations for the Duration (in Seconds) of the Primary Nystagmus Resulting from the Decelerations During Which (8 sec) or Following Which (3 sec) Room Lights Were on. For Purposes of Comparison, Similar Data Are Presented for the Acceleration in Total Darkness.

			Deceleration			Acceleration		
Group	Interval		Pre	Post I	Post II	Pre	Post I	Post II
Control	3 sec	M	39.0	39.6	42.6	48.3	47.6	42.7
		SD	8.2	14.8	12.3	4.6	7.0	3.7
	8 sec	M	38.4	42.0	42.0	53.6	56.5	53.6
		SD	27.8	14.2	15.2	13.3	18.1	8.1
Moderate Alcohol	3 sec	M	46.8	44.4	40.2	60.7	44.1	51.9
		SD	8.6	6.2	3.4	10.7	9.1	4.4
	8 sec	M	40.8	33.0	30.0	53.8	46.0	52.0
		SD	9.4	7.4	9.5	7.9	9.4	11.7
High Alcohol	3 sec	M	41.4	37.2	38.4	53.6	41.8	45.0
		SD	6.5	8.9	10.3	4.1	3.1	6.4
	8 sec	M	37.8	31.8	40.2	52.8	30.9	41.6
		SD	13.2	4.6	5.8	13.9	8.3	6.8

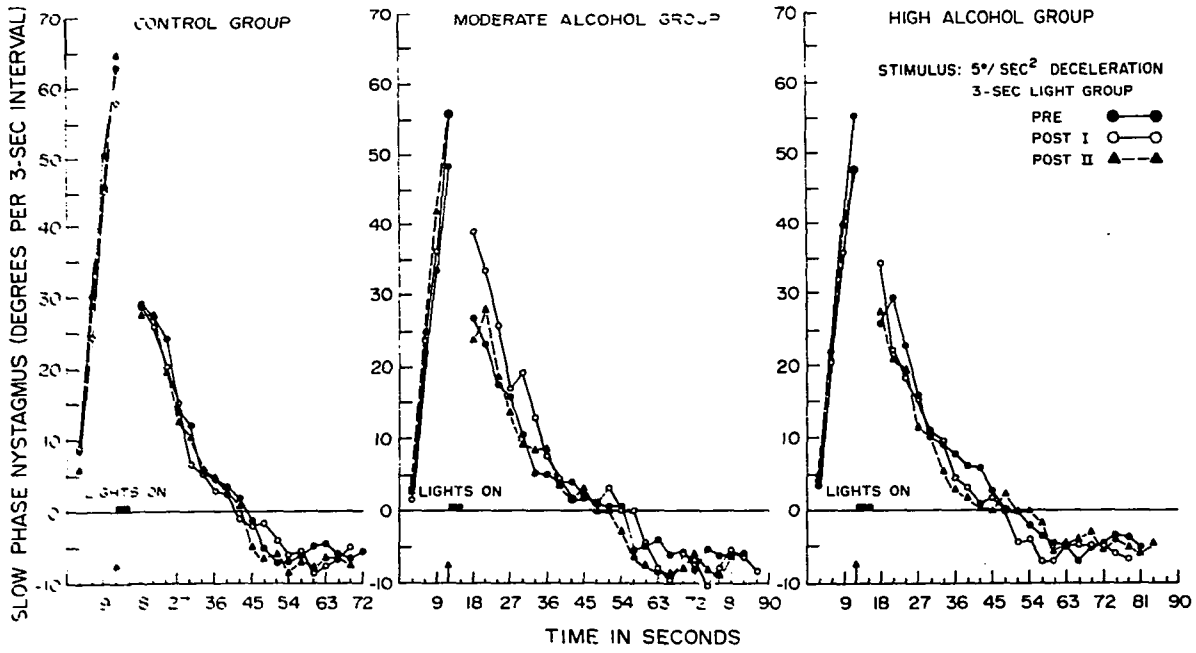

Figure 16. Response data for the average number of degrees of slow phase eye movement resulting from the 12-sec deceleration of $5^{o}/sec^{2}$. Immediately following the end of the deceleration, the room lights were turned on for 3-sec, during which time the subject fixated on a target. The short bar above the zero line represents the period of room illumination while the arrow indicates the end of the stimulus. Points plotted above the zero line represent the primary response, while those below represent the secondary response. Pre refers to the response obtained prior to the ingestion of alcohol, while Post I and Post II refer, respectively, to data obtained 45 min and four hours after ingestion. The values are plotted in 3-sec intervals; each point represents the average response of five subjects.

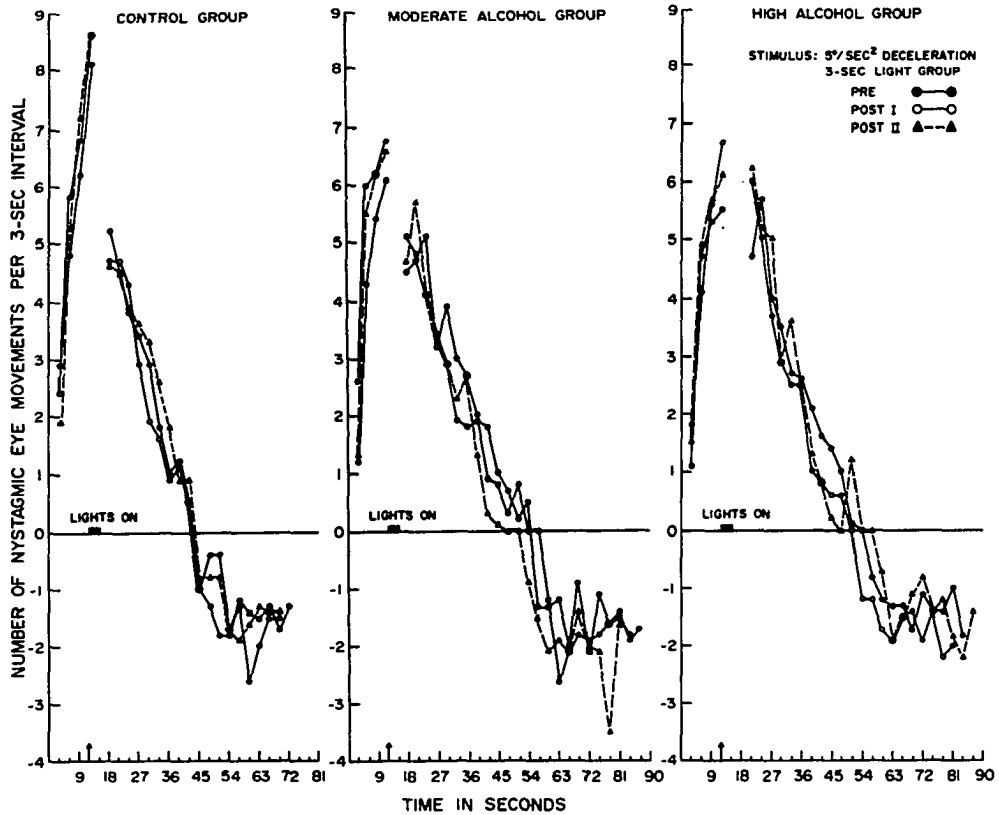

Figure 17. Response data for the average number of nystagmic eye movements resulting from the 12-sec deceleration of $5^{o}/sec^{2}$. Following deceleration, room lights were on for 3-sec, during which time the subject fixated on a target. Symbols and markings are identical to those used in Figure 16.

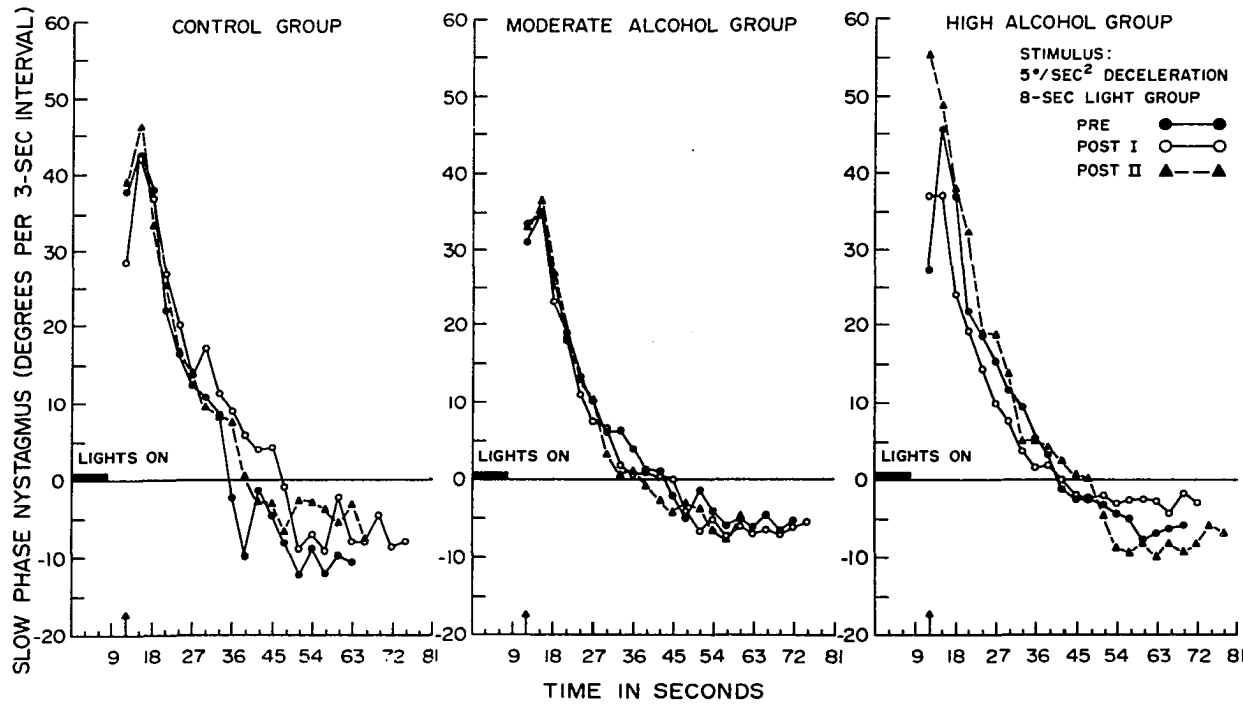

Figure 18. Response data for the average number of degrees of slow phase eye movement resulting from the 12-sec deceleration of $5^{o}/sec^{2}$. The room lights were turned on at the start of the deceleration and remained on for 8-sec. Symbols and markings are identical to those used in Figure 16.

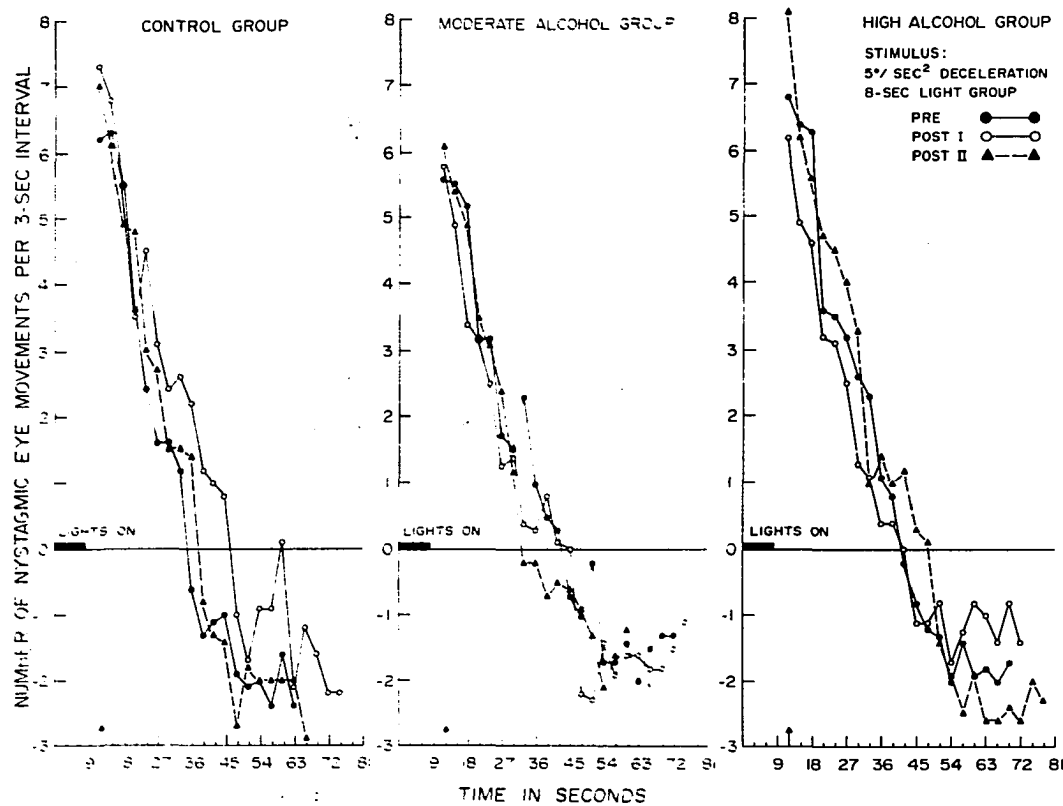

Figure 19. Response data for the average number of nystagmic eye movements resulting from the 12-sec deceleration of $5^{o}/sec^{2}$. Room lights were turned on at the start of the deceleration and remained on for 8-sec. Symbols and markings are identical to those used in Figure 16.

moderate alcohol group were considerable (12 and 26%), the 3-sec light group evidenced a large Pre to Post I increase for both measures (28 and 20%). Once again the Post II values were nearly the same or above the Pre levels for both of the response measures.

High Alcohol Group. Pre to Post I declines in mean frequency and slow phase displacement of the nystagmic responses were evident for both subgroups of the high alcohol subjects. The declines in slow phase displacement were 10% and 23%, while the frequency declined 6% and 29% for the 3-sec light and 8-sec light subgroups, respectively. With the exception of a 12% Pre-to-Post II decline in displacement for the 3-sec light group, all of the other Post II values were above the Pre levels.

Secondary Nystagmus. The Pre to Post I to Post II changes in the secondary nystagmic responses were, in most instances, the same as those reflected in the primary nystagmic responses. Thus, a Pre to Post I decline in the primary nystagmus was accompanied by a similar decline in the secondary nystagmus.

Overview. The effectiveness (the extent to which the primary response was suppressed) of the brief intervals of visual fixation did appear to be influenced by the ingestion of alcohol. The average slow phase displacement for all subgroups of the alcohol subjects during the first 3-sec (dark) interval after the lights were off was always above the Pre level for the Post I responses (see Figures 16 through 19). The Pre and Post I values of the control group, for the same 3-sec interval, were nearly identical. Similar differences were noted for most of the Pre to Post II comparisons. These differences between the control and alcohol groups were not as evident in the frequency of the nystagmus.

Changes in the primary nystagmic response, as the result of the visual fixation, are evident in comparisons of the frequency and slow phase displacement response curves for the deceleration with the brief intervals of light (Figures 16 through 19) with the response curves obtained for the dark accelerations (Figures 5 through 10). (The nystagmus from the accelerations and decelerations are in the opposite direction, but the stimuli are comparable.) Although primary nystagmus returns after the brief intervals of light, the magnitude of both the slow phase displacement and frequency of the response had diminished; the reductions in slow phase displacement appear to be greater than those of frequency.

The opportunity for visual fixation also produced a stronger secondary nystagmus than that obtained during trials in total darkness. The secondary responses for the acceleration (dark) stimuli were not scored because they were, in general, too infrequent and too weak to measure accurately. This difference in the amount of secondary nystagmus for the two stimuli is evident in the tracings presented in Figure 20.

The influence of the brief periods of visual fixation was also evident in comparisons of the duration of the primary nystagmus for the Light decelerations and the Dark accelerations (the stimulus rates were comparable). Means and standard deviations for the duration of the primary response for the two Light conditions are presented in Table 12. The duration of the primary nystagmus was in most cases longer for the Dark accelerations than for the decelerations with the brief intervals of light. The Post I trials for the 3-sec sub-group of the moderate alcohol subjects and the 8-sec sub-group of the high alcohol

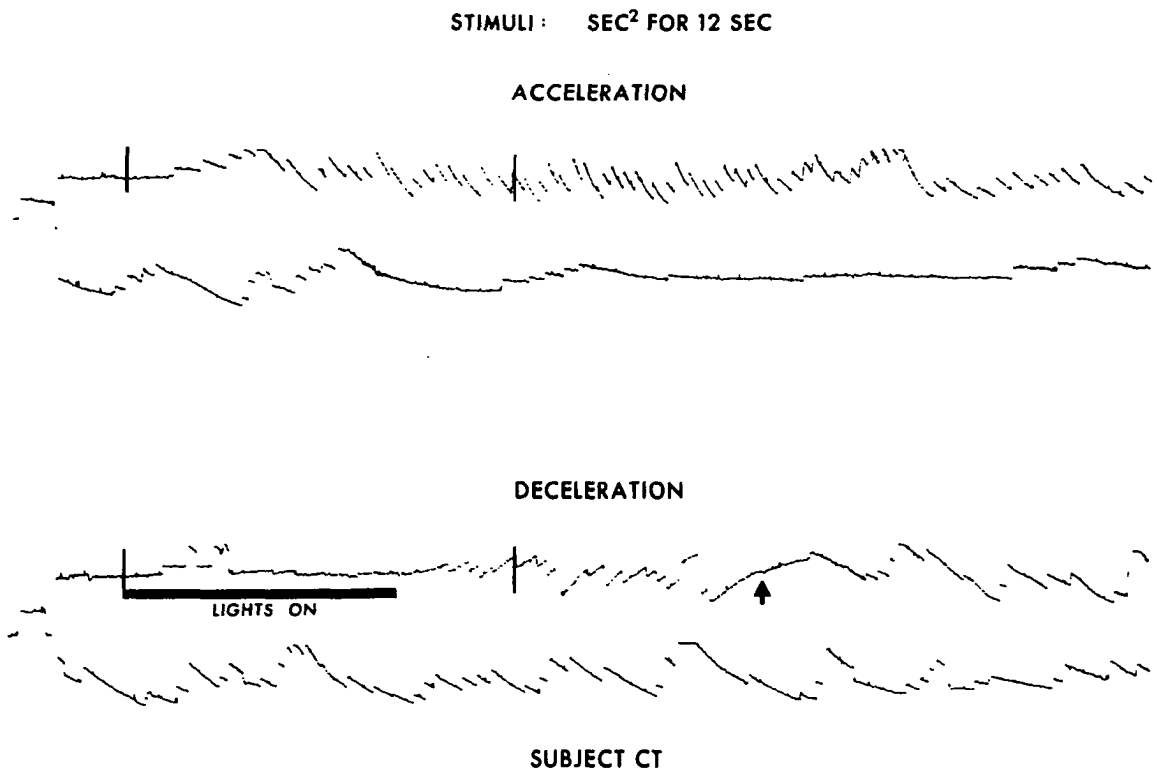

Figure 20. A portion of the nystagmic tracings for a subject's response to the dark acceleration and to the deceleration with the 8-sec period of illumination. The stimuli were comparable: a 12-sec acceleration and a 12-sec deceleration each at the rate of $5^{o}/sec^{2}$. Although the nystagmic responses are in opposte directions the response characteristics should be similar. Differences in primary or secondary nystagmus may be attributed to the effects of visual fixation during the interval of light. The dark horizontal bar indicates the period of room illumination, while the vertical bars demarcate the stimulus interval. Calibrations (15^{o} of eye movement) appear before each of the trials. The arrow indicates the point at which the primary nystagmus ends. The effects of visual fixation are evident in the shortened primary nystagmus and the enhanced secondary nystagmus depicted in the lower set of tracings.

subjects were the only instances where the duration of the primary nystagmus was longer for the light conditions (see Table 12). Visual fixation, following alcohol ingestion, was apparently less effective in suppressing the primary nystagmic response to rotatory stimulation. The degree to which the duration of the primary nystagmus was shortened was dependent upon the duration of the light period; the deceleration with the 8-sec light period resulted in shorter nystagmus durations than did the deceleration with the 3-sec light period.

Subjective Responses

Dark Acceleration and Deceleration Data.

Means and standard deviations for the subjects' estimations of turning velocity for the KP acceleration stimuli are presented in Table 13. Average response curves for the same data are presented in Figure 21.

While the control group evidenced little change (1% decline) in mean total subjective displacement for the Post I trial, the declines for the alcohol groups were larger; 8% for the moderate group and 15% for the high group. The mean Post II levels for the control and high alcohol groups were lower (7% and 17%, respectively) than the Post I levels. The moderate alcohol group mean increased 4% from the Post I to the Post II trials. Although there was a trend for the Pre-Post I declines of the alcohol groups to be larger than the decline for the control group, an analysis of variance revealed no significant differences (see Table 14).

Table 15 presents the mean and standard deviations for the duration of the subjects' turning sensations for the KP acceleration and

TABLE 13

Means and Standard Deviations for the Total Subjective Displacement (in Degrees) for the KP Acceleration (Total Darkness).

Group		Trial		
		Pre	Post I	Post II
Control	M	1368	1350	1269
	SD	703.3	468.6	477.1
Moderate	M	1323	1224	1287
	SD	511.0	728.9	578.7
High	M	1107	945	918
	SD	395.8	360.6	257.4

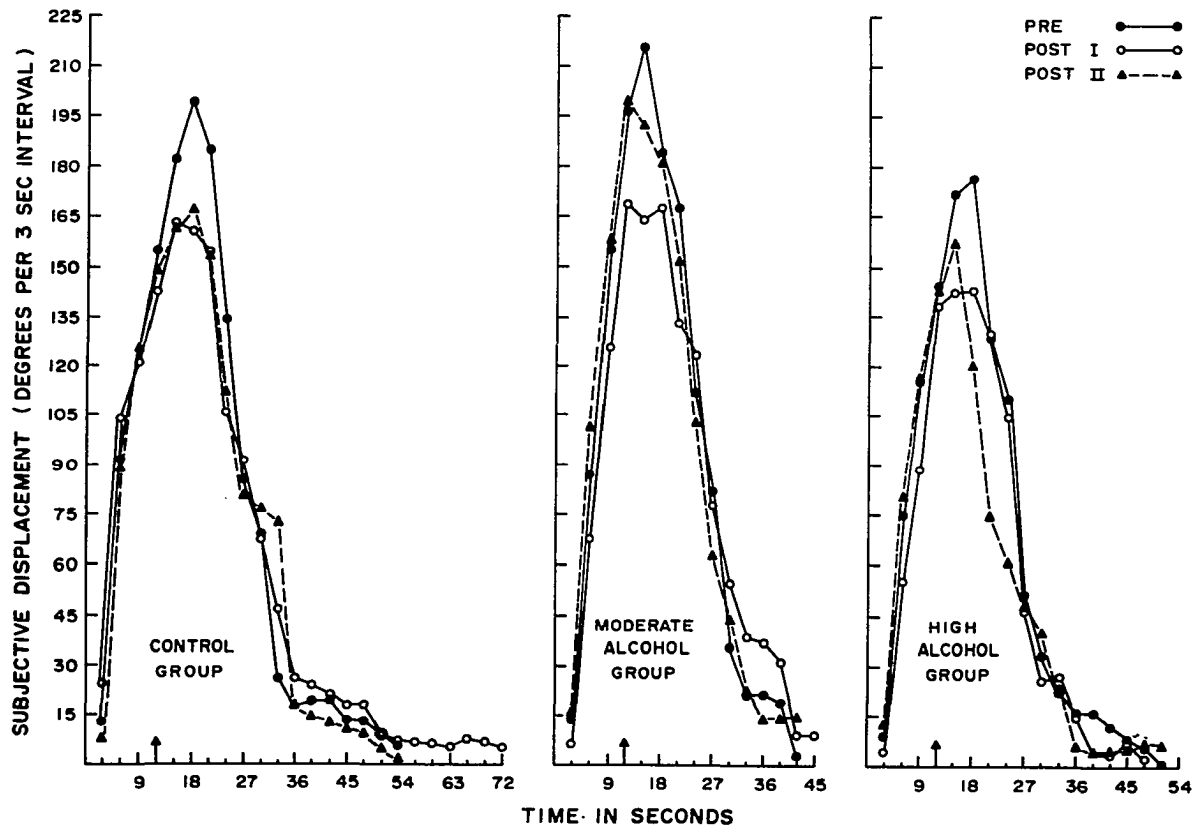

Figure 21. Response data for the average number of degrees of subjective displacement resulting from the accelerations ($5^{o}/sec^{2}$ for 12 sec). Pre refers to the response recorded prior to the ingestion of alcohol, while the Post I and Post II data were obtained, respectively, 45 min and four hours after ingestion. The arrow on the abscissa indicates the end of the stimulus. The values are plotted in 3-sec intervals; each point is a mean for 10 subjects.

TABLE 14

Results of the Analyses of Variance for the Displacement (in Degrees) and Duration (in Seconds) of the Subjective Reactions During the KP Rotation Stimuli.

		Subjective Displacement (Accelerations)		Duration (Accelerations)		Duration (Decelerations)	
Source	df	Mean Squares	F	Mean Squares	F	Mean Squares	F
Groups (G)	2	1002.33	1.54	45.61	1.54	33.99	1.05
Subj./within Groups (S/G)	27	649.41		59.37		32.40	
Trials (T)	2	102.69	1.33	3.32	1.15	14.69	3.88*
T x G	4	27.36	0.35	3.45	1.20	5.33	1.41
T x S/G	54	77.10		2.87		3.79	

* $p < .05$

TABLE 15

Means and Standard Deviations for the Duration (in Seconds) of the Subjective Turning Sensations Resulting from the KP Acceleration and Deceleration. Each Group Was Comprised of Ten Subjects.

		Pre		Post I		Post II	
Group		Acc	Dec	Acc	Dec	Acc	Dec
Control	M	30.8	31.5	36.6	32.1	32.2	31.7
	SD	9.5	11.5	16.3	11.9	13.0	19.6
Moderate Alcohol	M	26.6	28.5	25.6	23.3	25.4	25.1
	SD	10.2	9.4	8.2	9.7	7.7	7.7
High Alcohol	M	28.0	31.1	27.7	23.0	26.2	24.6
	SD	10.3	8.8	8.4	9.4	10.8	14.6

deceleration stimuli. Post I mean duration values were above their Pre mean levels (18% for the acceleration and 2% for the deceleration) for the control group, but were below these levels for the two alcohol groups. With respect to the latter, the degree to which the duration was shortened appeared to be related to the amount of alcohol ingested: i.e., 1% and 18% declines (acceleration and deceleration, respectively) for the moderate alcohol group, and 3% and 26% declines for the high alcohol group.

Separate analyses of variance were conducted for the duration measures. None of the differences for the acceleration durations were significant. However, the "trials" factor was significant for the deceleration stimuli data (Table 14); subsequent t tests indicated significant differences (.05 and .01 levels) in the Pre-Post I declines for both alcohol groups (Table 16). At the four-hour testing session none of the Pre-Post II differences was statistically significant (Table 16).

Light Deceleration Data.

The data (see appendix) from the subjective estimation of velocity during the light decelerations are plotted in Figures 22 and 23. The variability in the response curves was due to the small number (five) of subjects in each of the groups.

The reported sensations for the 3-sec light groups, prior to the onset of the light, were similar to those obtained in total darkness (compare with the acceleration data in Figure 21). In contrast to the strong primary sensation (CCW turning) experienced by all of the subjects prior to the (3-sec) light interval, only a few subjects reported any primary sensation following cessation of the light, and these sensations

TABLE 16

Results of the Paired t Tests for the Duration of the Rotatory Sensation Resulting from the KP Deceleration.

Group	Pre vs. Post I	Pre vs. Post II
Control	0.21	0.04
Moderate Alcohol	2.66*	1.62
High Alcohol	4.11**	1.97

* $p < .05$
** $p < .01$

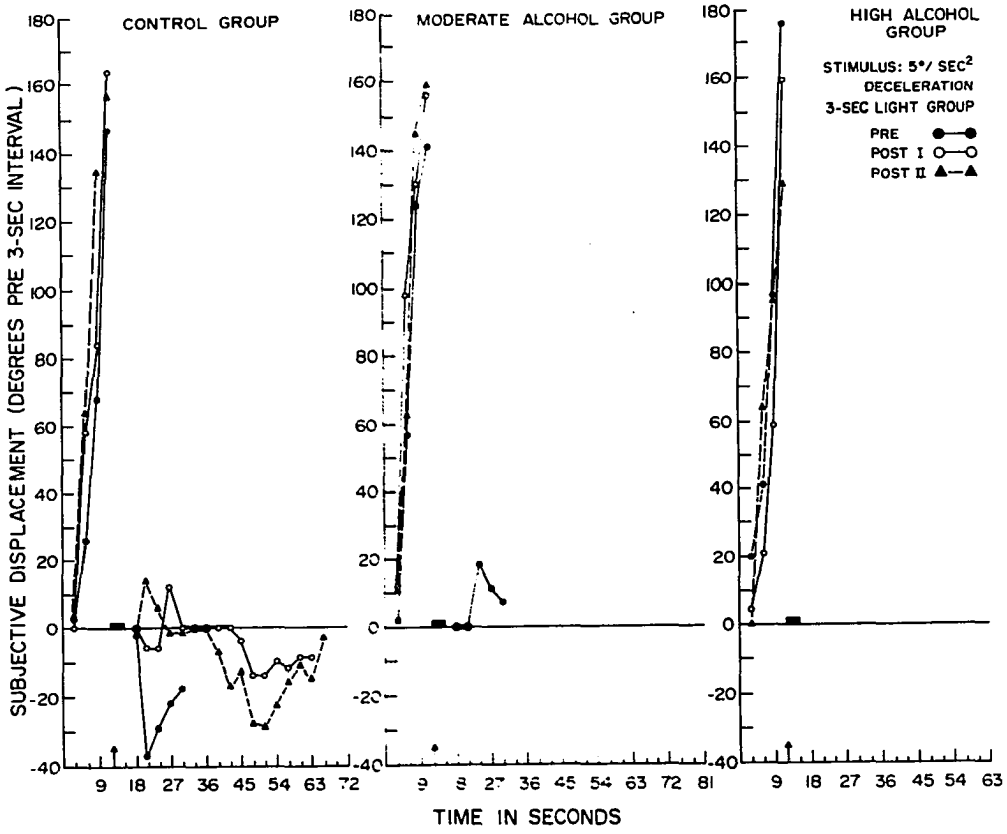

Figure 22. Response data for the average number of degrees of subjective displacement resulting from the 12-sec deceleration of $5^{o}/sec^{2}$. Immediately following the end of the deceleration, the room lights were turned on for 3-sec, during which time the subject fixated on a target. The short bar above the zero line represents the end of the stimulus. Points plotted above the zero line represent sensations in the primary direction, while those below represent sensations in the secondary direction. Pre refers to the response obtained prior to the ingestion of alcohol while Post I and Post II refer to data obtained, respectively, 45 min and four hours after ingestion. The values are plotted in 3-sec intervals; each point represents the average response of five subjects.

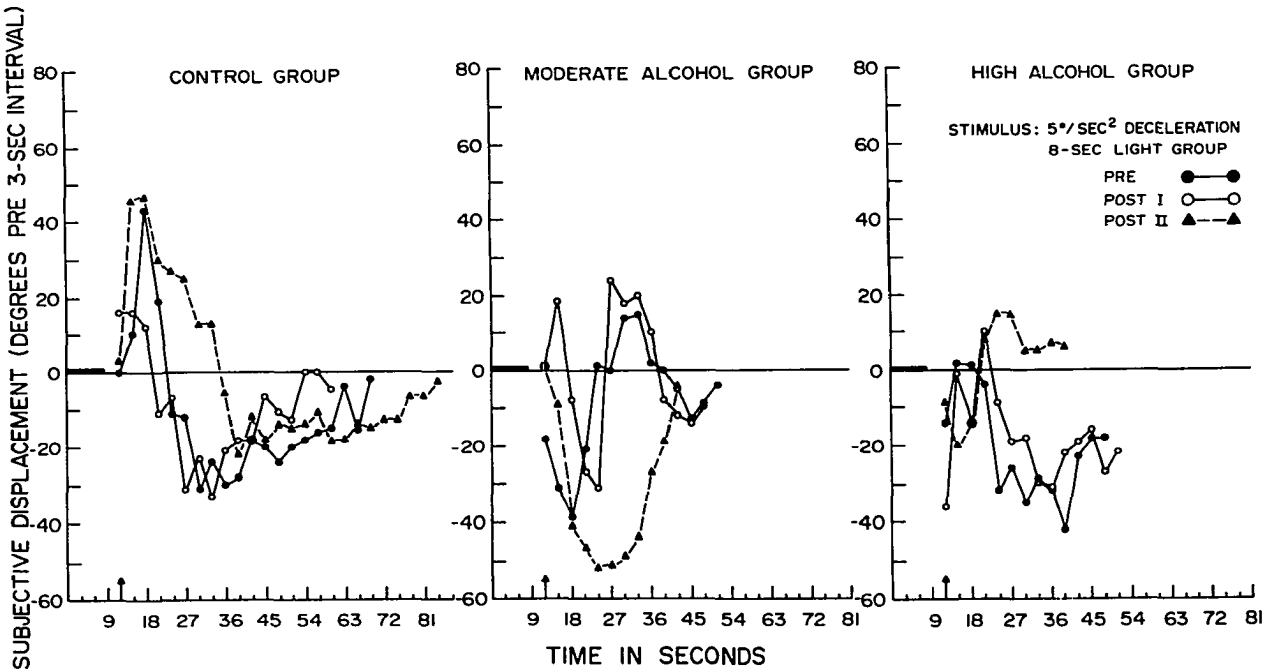

Figure 23. Response data for the average number of degrees of subjective displacement resulting from the 12-sec deceleration of $5°/sec^2$. The room lights were turned on at the start of the deceleration and remained on for 8-sec. Symbols and markings are identical to those used in Figure 22.

were very weak. Although most of the subjects failed to experience any turning following cessation of the light, several subjects in the control group reported relatively strong secondary (CW) sensations (see Figure 22).

The subjects in the groups exposed to the 8-sec light interval during the decelerations (Figure 23), evidenced considerably stronger secondary (CW) sensations, than the 3-sec light groups. Although the secondary turning experience appeared to be stronger than the primary (CCW) sensation, several subjects failed to experience any turning at all following the end of the light interval.

Alcohol ingestion failed to produce any noticeable differential effect in the rotatory sensations for any of the light groups, as affected by the brief periods of room illumination.

Spiral Aftereffect

Mean SAE duration values and standard deviations are presented in Table 17. There were no striking Pre-Post I-Post II changes in the responses of the three groups. Although slight Pre to Post I declines in the duration of the aftereffect were obtained from the alcohol groups, an analysis of variance revealed no statistically significant differences (see Table 18).

In order to provide further testing of Reason's theory (Reason 1968, 1969), that the duration of the sensation resulting from angular stimulation is related to the duration of the spiral aftereffect, product-moment correlations were computed for the two measures. Correlations between the duration of the SAE and the duration of the rotatory sensa-

TABLE 17

Means and Standard Deviations for the Duration (in Seconds) of the Spiral Aftereffect (SAE). Each Group Was Comprised of Ten Subjects.

Group		Trial		
		Pre	Post I	Post II
Control	M	12.7	13.0	12.8
	SD	6.4	6.3	7.1
Moderate	M	15.3	13.0	14.1
	SD	7.2	5.4	6.1
High	M	14.2	13.8	15.1
	SD	10.1	11.9	16.1

TABLE 18

Results of the Analysis of Variance for the Duration Scores Resulting From the Spiral Aftereffect.

Source	df	MS	F
Groups	2	21.46	0.09
Subjects/Groups	27	228.25	
Trials	2	6.13	0.56
Groups x Trials	4	6.18	0.56
Trials x Subjects/Groups	54	11.03	

TABLE 19

Product Moment Correlations for the Duration of the Spiral Aftereffect and the Duration of the Rotatory Sensations for the 12-sec Accelerations and the Decelerations.

	Acceleration Durations			Deceleration Durations		
Group	Pre	Post I	Post II	Pre	Post I	Post II
Control	.26	.03	.25	.10	-.13	-.15
Moderate Alcohol	.27	.25	.71*	.04	.18	.45
High Alcohol	.81**	.89**	.93*	.61*	.81**	.72**

* $p < .05$
** $p < .01$

tion for the brake decelerations were low and were significant only for the high alcohol group (Table 19). Correlations for the duration of the SAE and the duration of the rotatory sensation resulting from the 12-sec acceleration stimuli were all higher than those noted above, but only the three correlations for the high alcohol group and that for the Post II trial of the moderate alcohol group were significant (Table 19).

Caloric Irrigations

Nystagmus

Means and standard deviations for the total number of degrees of slow phase displacement, frequency, duration, and peak velocity of the slow phase of the eye movements obtained during the caloric irrigation trials are presented in Tables 20 through 23. Separate analyses of variance, across all groups, were conducted for each of the above measures (Table 24). F ratios for the "trials" factor were significant (.05 - .001 levels) for all measures, and a significant (.05 level) "trials x group" interaction was obtained for the frequency data. Other significant findings will be discussed in later portions of the text. In order to provide further information concerning the variations in the responses for the Pre, Post I, and Post II trials, separate paired t tests were conducted. These results will be included in the discussion concerning the responses of each of the groups considered separately (Tables 25 through 27).

Control Group. Relatively large Pre-Post I reductions (15-27%) in the mean total slow phase eye displacement for the control group were evident for the four caloric trials. Smaller declines were noted for the other measures; a 2% increase to a 5% drop in frequency, a 4-14%

TABLE 20

Means and Standard Deviations for the Slow Phase Nystagmus Displacement (in Degrees) Resulting from the Caloric Irrigations. Each Group Was Comprised of Ten Subjects.

Group	Condition		44°C to the left ear (Lw) Pre	Post I	Post II	30°C to the right ear (Rc) Pre	Post I	Post II
Control	Task	M	1338.1	1048.3	1087.0	1387.5	1180.7	1129.8
		SD	581.2	368.9	577.4	602.6	409.4	497.1
	Rev	M	1285.9	1018.4	909.2	1295.0	948.2	1023.5
		SD	601.1	473.6	331.7	796.3	382.5	453.2
Moderate Alcohol	Task	M	1419.4	1175.2	1136.6	1467.3	1157.9	1166.3
		SD	623.5	710.2	596.1	668.1	416.0	624.3
	Rev	M	1192.4	1071.8	1050.2	1319.3	828.2	1078.2
		SD	666.0	650.5	588.6	415.8	360.6	650.5
High Alcohol	Task	M	1481.7	795.0	1352.0	1485.4	967.5	1496.8
		SD	785.6	473.5	783.6	668.6	493.0	546.5
	Tev	M	1041.8	650.8	976.8	1144.2	614.0	1039.2
		SD	828.3	505.4	608.3	564.2	426.7	616.6

TABLE 21

Means and Standard Deviations for the Number of Nystagmic Beats Resulting from the Caloric Irrigations. Each Group Was Comprised of Ten Subjects.

Group	Condition		44°C to the left ear (Lw) Pre	Post I	Post II	30°C to the right ear (Rc) Pre	Post I	Post II
Control	Task	M	228.5	228.2	216.2	234.4	237.9	228.2
		SD	55.2	70.4	37.2	38.0	41.3	37.7
	Rev	M	223.4	212.6	223.5	241.0	224.9	241.1
		SD	38.3	57.5	50.4	38.9	40.4	44.6
Moderate Alcohol	Task	M	242.1	202.4	215.1	250.7	202.6	215.4
		SD	102.9	77.6	98.1	80.1	60.2	87.4
	Rev	M	220.9	193.5	196.1	255.2	170.9	201.3
		SD	110.9	83.6	83.9	87.4	69.6	91.2
High Alcohol	Task	M	197.3	145.4	174.5	214.9	130.5	189.5
		SD	86.4	111.0	70.0	63.8	60.5	52.0
	Tev	M	177.5	126.3	185.1	189.3	115.2	172.9
		SD	87.6	107.4	96.5	50.1	77.0	83.4

TABLE 22

Means and Standard Deviations for the Peak Velocity (Deg/Sec) of the Slow Phase Nystagmus Resulting From the Caloric irrigations. Each Group Was Comprised of Ten Subjects.

Group	Condition		44°C to the left ear (Lw) Pre	Post I	Post II	30°C to the right ear (Rc) Pre	Post I	Post II
Control	Task	M	19.2	17.8	16.8	18.3	16.9	15.8
		SD	7.8	5.6	6.1	6.5	5.5	6.7
	Rev	M	20.1	18.4	17.3	16.6	14.8	16.3
		SD	8.5	6.9	6.5	8.4	5.9	5.1
Moderate Alcohol	Task	M	23.4	22.1	20.5	21.7	20.4	21.2
		SD	13.3	12.6	10.0	10.6	9.0	14.7
	Rev	M	19.6	23.2	21.8	20.4	17.2	19.0
		SD	13.4	14.3	9.6	8.2	6.8	9.5
High Alcohol	Task	M	24.8	17.4	24.4	20.6	17.2	22.1
		SD	9.5	8.5	10.2	6.8	7.1	8.5
	Rev	M	21.8	16.5	21.4	19.2	15.4	18.4
		SD	19.5	8.3	10.0	7.0	7.7	10.0

TABLE 23

Means and Standard Deviations for the Duration (in Seconds) of the Nystagmus Resulting From the Caloric Irrigations. Each Group Was Comprised of Ten Subjects.

Group	Condition		44°C to the left ear (Lw) Pre	44°C to the left ear (Lw) Post I	44°C to the left ear (Lw) Post II	30°C to the right ear (Rc) Pre	30°C to the right ear (Rc) Post I	30°C to the right ear (Rc) Post II
Control	Task	M	163.3	157.2	150.8	182.3	166.8	161.4
		SD	36.0	20.9	25.9	48.3	23.5	38.4
	Rev	M	153.4	131.8	138.0	169.8	148.4	157.2
		SD	29.9	21.3	19.2	30.9	24.2	27.0
Moderate Alcohol	Task	M	187.1	149.5	161.2	185.3	156.4	166.4
		SD	54.1	29.1	38.6	29.2	12.1	24.1
	Rev	M	185.8	137.4	145.6	186.8	130.6	156.1
		SD	52.0	41.4	44.6	35.4	35.6	30.5
High Alcohol	Task	M	162.5	108.1	135.6	171.0	128.7	149.2
		SD	25.6	48.4	21.7	51.1	30.6	34.4
	Rev	M	145.0	100.5	127.1	161.0	116.6	118.6
		SD	25.9	38.3	17.3	30.0	38.7	50.9

TABLE 24

Results of the Analyses of Variance for the Various Measures of the Nystagmic Responses Resulting from the Caloric Irrigations.

	F				
	Slow Phase Displacement	Frequency	Duration	Peak Velocity	Subjective Intensity
Group (G)	0.11	2.49	3.43*	0.60	0.59
Temp (Te)	0.30	0.97	2.45	2.96	8.83**
Te x G	0.10	0.32	0.32	0.02	1.05
Instructions (I)	41.52***	6.98*	6.23*	6.67*	0.05
I x G	4.19*	1.38	0.39	1.45	1.20
Trials (Tr)	16.33***	17.10***	30.96***	4.02*	21.67***
Tr x G	2.25	4.32*	0.79	2.45	1.19
Te x I	0.68	0.00	0.77	1.12	0.03
Te x I x G	0.07	1.04	0.15	0.28	1.11
Te x Tr	0.44	1.52	0.46	0.11	6.81**
Te x Tr x G	0.56	0.37	0.60	0.74	0.81
I x Tr	0.08	1.99	2.34	0.24	0.82
I x Tr x G	0.87	0.95	0.96	0.55	0.90
I x Tr x Te	2.82	0.86	0.26	0.63	2.69
I x Tr x Te x G	0.13	1.02	1.65	0.45	0.66

* $p < .05$
** $p < .01$
*** $p < .001$

TABLE 25

Results of the Paired t Tests for the Pre vs. Post I and Pre vs. Post II Comparisons of the Nystagmic Responses of the Control Group to the Caloric Irrigations.

		44°C to the left ear (Lw)		30°C to the right ear (Rc)	
Measure	Comparison	Task	Reverie	Task	Reverie
Displacement	Pre vs. Post I	1.86	1.88	1.24	1.49
	Pre vs. Post II	1.49	2.63*	2.19	1.21
Frequency	Pre vs. Post I	0.04	0.90	0.33	1.11
	Pre vs. Post II	1.21	0.01	0.59	0.01
Duration	Pre vs. Post I	0.72	2.84*	1.56	2.42*
	Pre vs. Post II	1.28	2.11	3.72**	1.51
Velocity	Pre vs. Post I	0.68	0.88	0.67	0.72
	Pre vs. Post II	0.96	1.58	1.66	0.10

* $p < .05$
** $p < .01$

TABLE 26

Results of the Paired t Tests for the Pre vs. Post I and Pre vs. Post II Comparisons of the Nystagmic Responses of the Moderate Alcohol Group to the Caloric Irrigations.

		44°C to the left ear (Lw)		30°C to the right ear (Rc)	
Measure	Comparison	Task	Reverie	Task	Reverie
Displacement	Pre vs. Post I	1.42	0.76	2.39*	7.99***
	Pre vs. Post II	2.31*	0.89	2.33*	1.91
Frequency	Pre vs. Post I	2.97*	1.02	6.13***	4.12**
	Pre vs. Post II	2.95*	0.93	1.89	2.84*
Duration	Pre vs. Post I	3.04*	4.14**	4.61**	4.28**
	Pre vs. Post II	1.85	3.21**	5.60***	2.83*
Velocity	Pre vs. Post I	0.54	1.37	0.40	3.07*
	Pre vs. Post II	1.61	0.59	0.13	0.71

* $p < .05$
** $p < .01$
*** $p < .001$

TABLE 27

Results of the Paired t Tests for the Pre vs. Post I and Pre vs. Post II Comparisons of the Nystagmic Responses of the High Alcohol Group to the Caloric Irrigations.

		44°C to the left ear (Lw)		30°C to the right ear (Rc)	
Measure	Comparison	Task	Reverie	Task	Reverie
Displacement	Pre vs. Post I	3.56**	1.99	4.69**	3.93**
	Pre vs. Post II	1.04	0.51	0.08	0.62
Frequency	Pre vs. Post I	1.90	1.66	7.16***	4.37**
	Pre vs. Post II	1.69	0.72	2.29*	0.76
Duration	Pre vs. Post I	4.07**	5.77***	4.38**	6.64***
	Pre vs. Post II	3.28**	2.41*	1.59	2.14
Velocity	Pre vs. Post I	2.38*	1.01	2.09	2.03
	Pre vs. Post II	0.18	0.09	0.78	0.35

* $p < .05$
** $p < .01$
*** $p < .001$

reduction in duration, and a 7-11% decline in peak velocity. According to the t tests (Table 25), none of the frequency, slow phase displacement, or peak velocity measures for the Post I trials was significantly different from the Pre values. Of the Post I declines in duration, only the values for the Rc/Rev and Lw/Rev were significantly different (.05 level) from the Pre values.

The responses for the Post II trials, for all measures, were very near the Pre levels. With the exception of a significantly shorter duration for the Rc/Task trial (.01 level) and a significantly lower slow phase eye displacement response for the Lw/Rev trial (.05 level), no other significant Pre-Post II differences were found.

Moderate Alcohol Group. Although there was a 19% increase in peak velocity for the Post I Lw/Rev trial, the PostI mean responses for the other measures and trials were lower than the Pre levels. The declines in the mean values for the other measures, across all of the caloric trials, were: 10-37% in total slow phase displacement, 12-33% in frequency, 16-30% in duration, and 6-16% in velocity. The durations were significantly lower (.05 - .01 levels) for all of the Post I trials. With the exception of no significant changes in frequency and slow phase displacement for the Lw/Rev trials, all other Post I trials yielded significantly lower outputs of frequency and slow phase displacement (.05 - .001 levels). Only the Rc/Rev trial evidenced a significant Post I decline (.05 level) in peak velocity.

Most of the mean responses for the Post II trials showed recovery from the Post I levels, but some of the Pre-Post II differences were still statistically significant (Table 26). With the exception of the

Lw/Task trial, all of the Post II durations for the other trials, were still significantly shorter (.01 - .001 levels) than the Pre levels. Significant differences were also evident in the frequency (Lw/Task and Rc/Rev) and slow phase displacement (Lw/Task and Rc/Rev) measures (.05 level). Although there was some response recovery for the Post II trials, the percentage changes were still greater than those of the control group. None of the Pre-Post II differences in peak velocity was significant.

High Alcohol Group. Differences in the Post I and Post II response levels, from the Pre levels, were much more evident for the high alcohol group. The decline in the means, across all of the caloric trials, for the different measures ranged from 35-46% in displacement, 26-39% in frequency, 25-33% in duration, and 16-30% in peak velocity. In agreement with the data from the moderate alcohol group, the reduction for the Post I trials were, in general, less evident for the Lw trials, although for this group, the Pre-Post I response declines in duration were significant (.01 - .001 levels) for both Lw trials. The only other significant declines in the response for the Lw trials were in the slow phase displacement and peak velocity for the Lw/Task trial (.05 and .01 levels). While there was no significant change in the peak velocity for the Rc trials, all other measures were significantly lower (.01 - .001 levels) on the Post I Rc trials.

The Post II means, for all measures and all trials, were above their respective Post I levels. All of the Post II mean values for frequency, total slow phase displacement, and peak velocity were within 12% of their Pre levels. The Post II duration values in general, evidenced less recovery than the other measures. The mean Post II

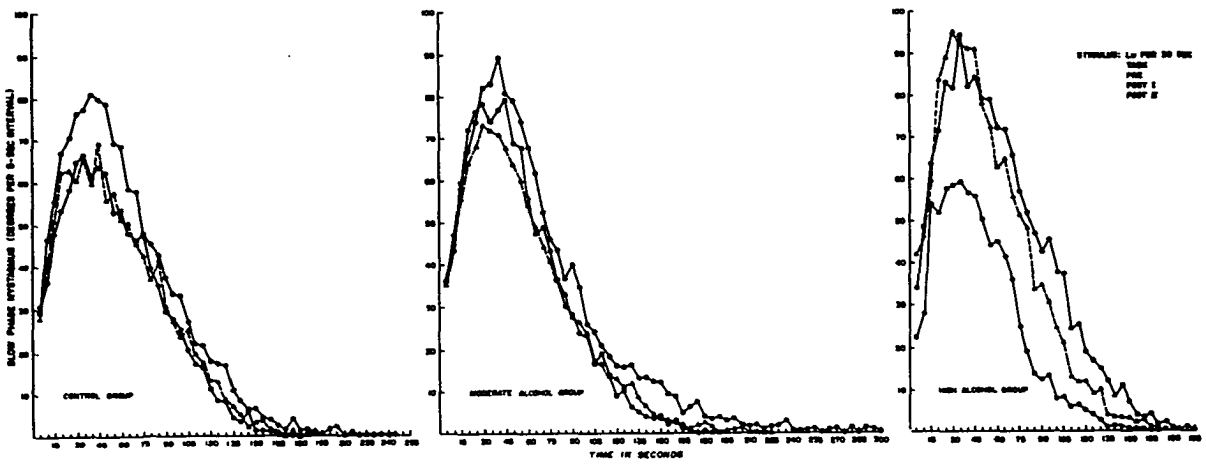

Figure 24. Response data for the average number of degrees of slow phase eye movement, resulting from the Lw caloric irrigations under the Task condition. Pre refers to the response recorded prior to the ingestion of alcohol, while the Post I and Post II data were obtained, respectively, 45 min and four hours after ingestion. The stimulation, in each case, was a 30-sec unilateral irrigation. The values are plotted in 5-sec intervals beginning immediately after the end of the irrigation; each point is a mean for 10 subjects

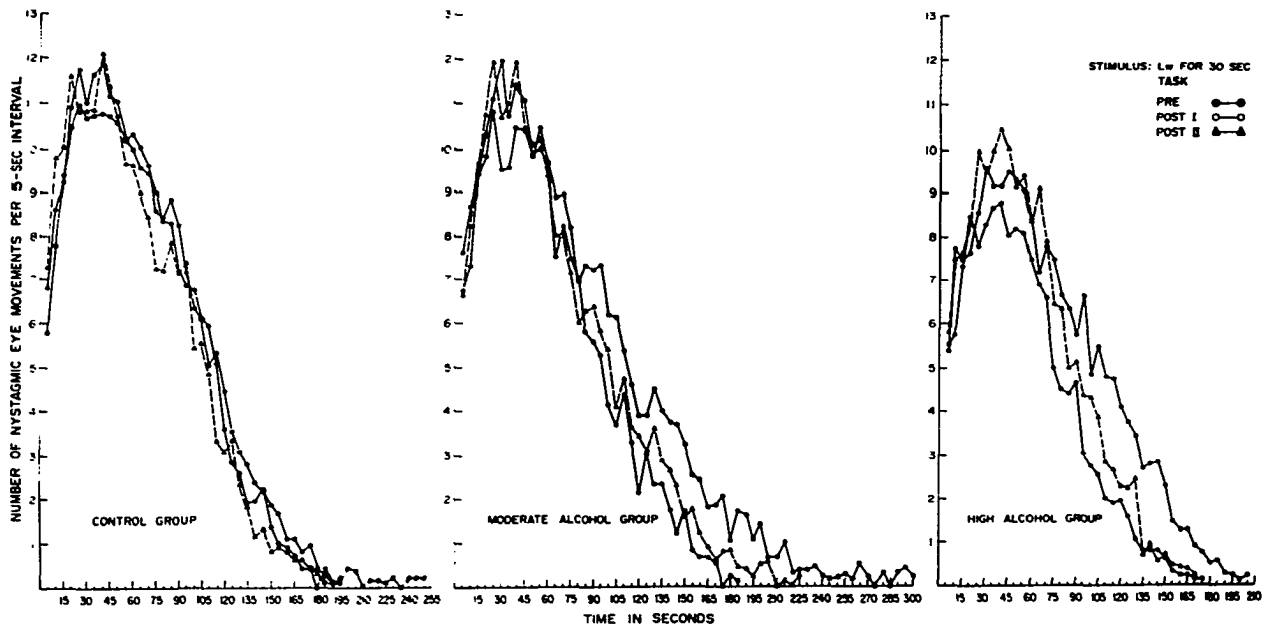

Figure 25. Response data for the average number of nystagmic eye movements resulting from the Lw caloric irrigations under the Task condition. Symbols and markings are identical to those used in Figure 24.

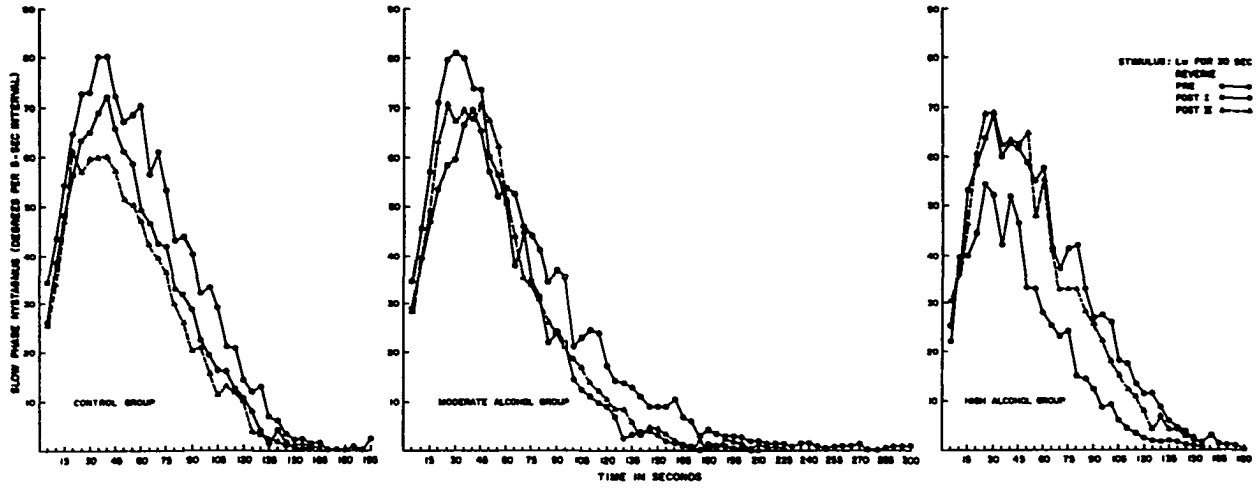

Figure 26. Response data for the average number of degrees of slow phase eye movement, resulting from the Lw caloric irrigations under the Reverie condition. Symbols and markings are identical to those used in Figure 24.

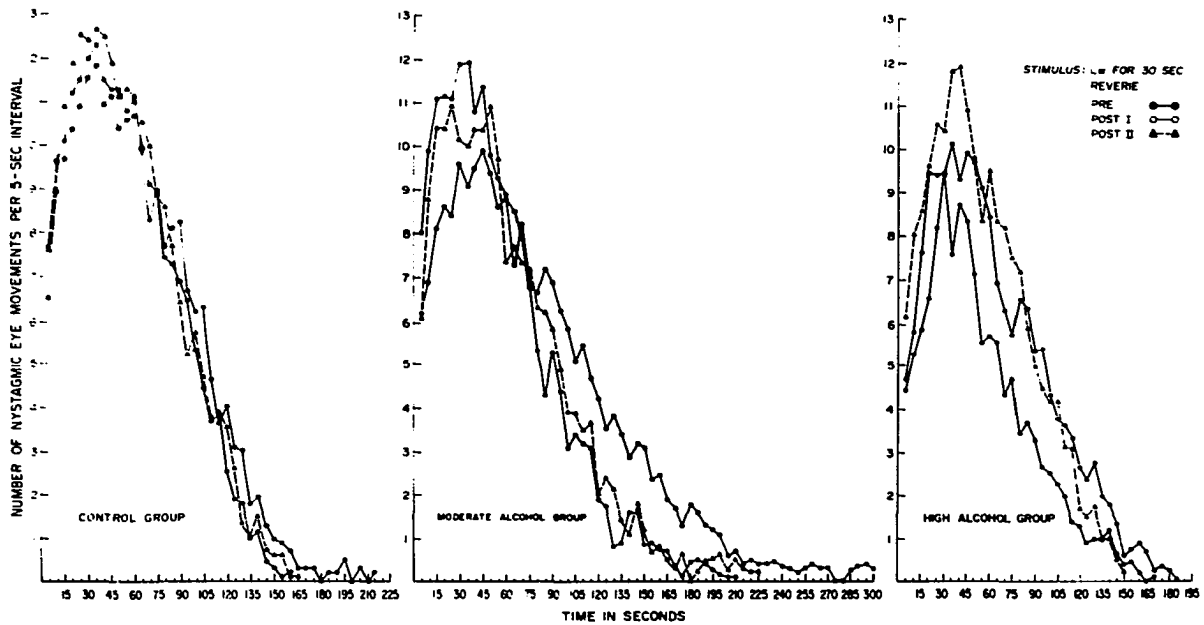

Figure 27. Response data for the average number of nystagmic eye movements, resulting from the Lw caloric irrigations under the Reverie condition. Symbols and markings are identical to those used in Figure 24.

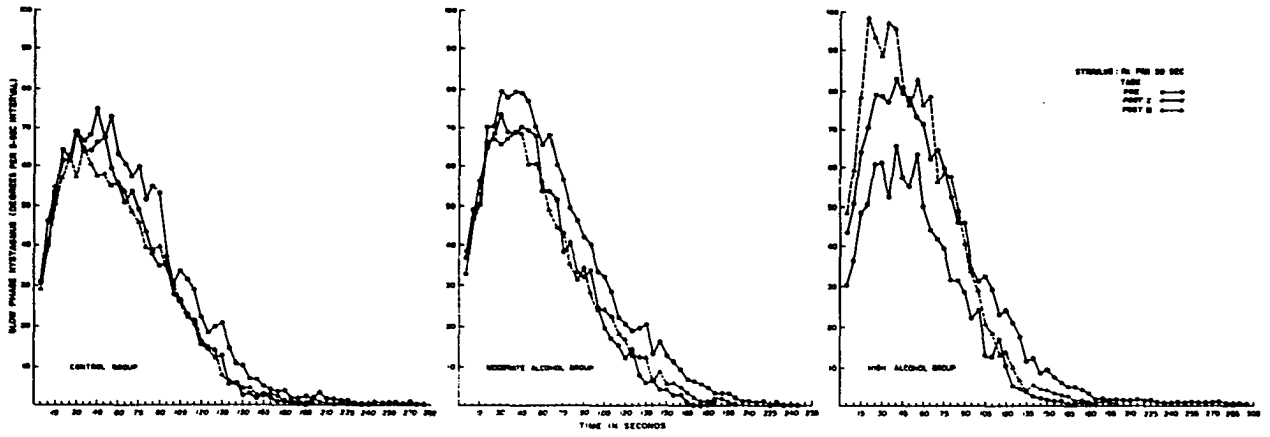

Figure 28. Response data for the average number of degrees of slow phase eye movement, resulting from the Rc caloric irrigations under the Task condition. Symbols and markings are identical to those used in Figure 24.

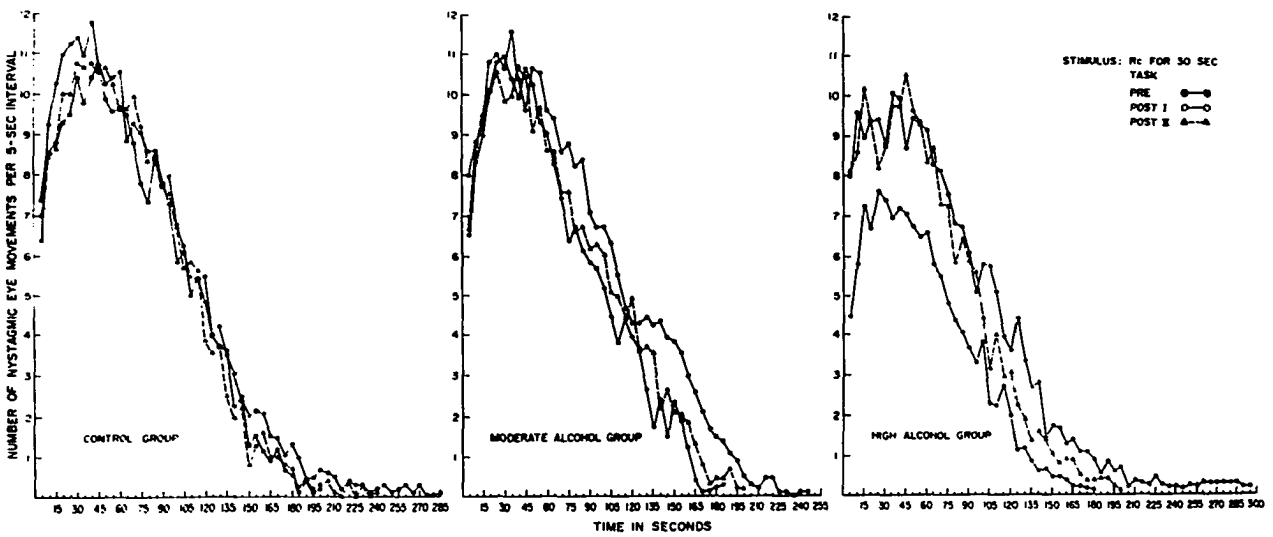

Figure 29. Response data for the average number of nystagmic eye movements, resulting from the Rc caloric irrigations under the Task condition. Symbols and markings are identical to those used in Figure 24.

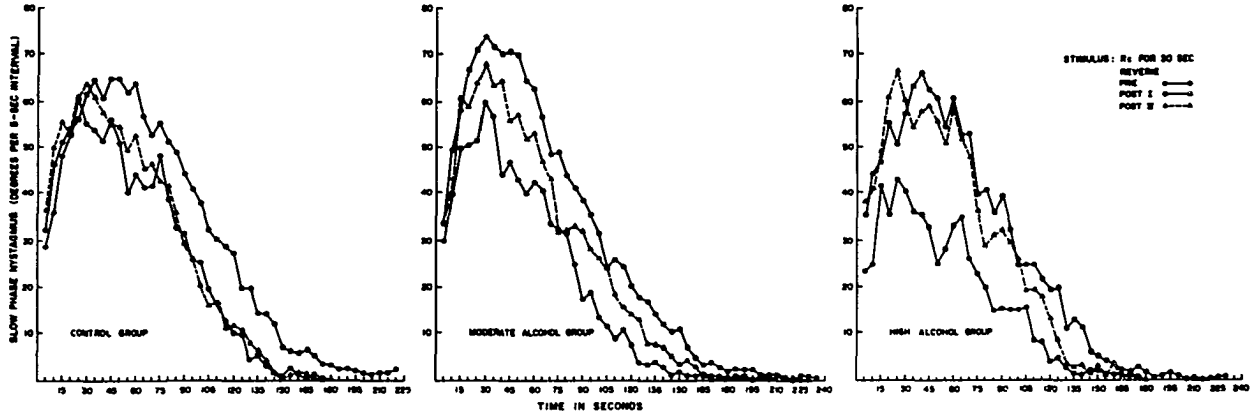

Figure 30. Response data for the average number of degrees of slow phase eye movement, resulting from the Rc caloric irrigations under the Reverie condition. Symbols and markings are identical to those used in Figure 24.

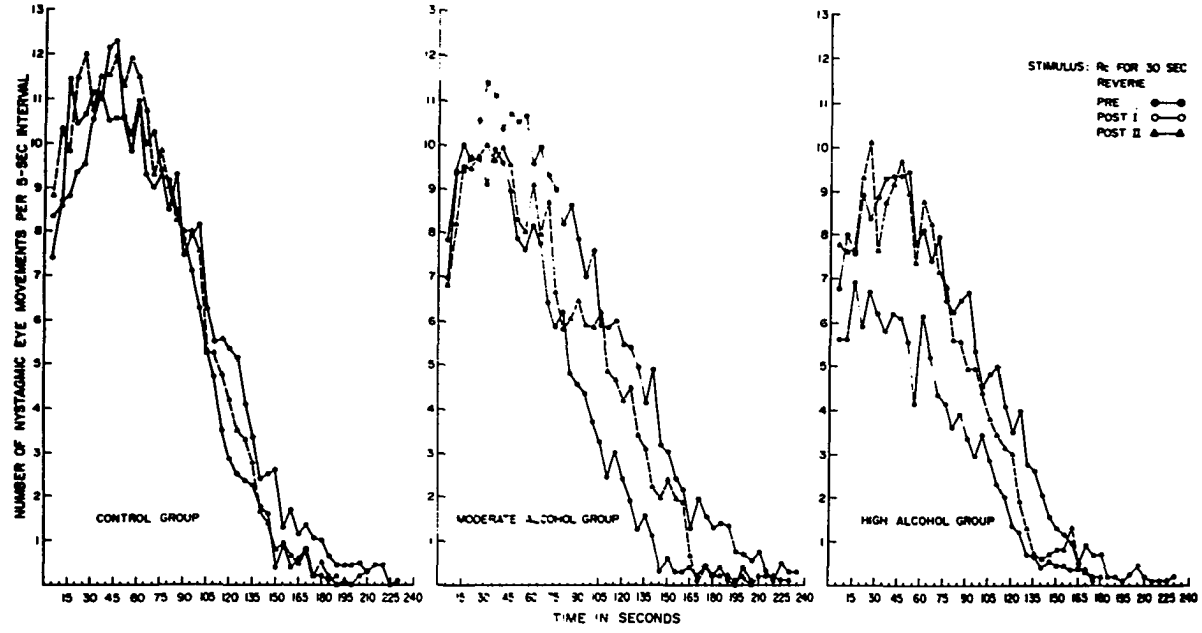

Figure 31. Response data for the average number of nystagmic eye movements, resulting from the Rc caloric irrigations under the Reverie condition. Symbols and markings are identical to those used in Figure 24.

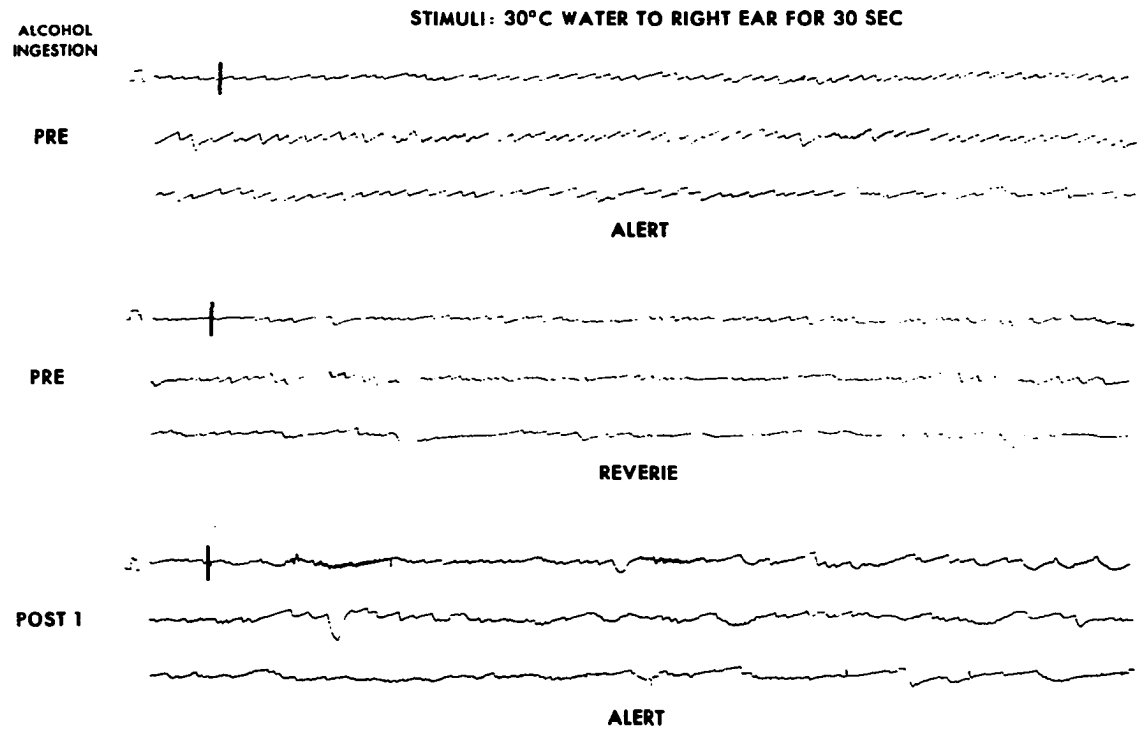

Figure 32. A portion of the nystagmic tracings of responses by subject TW to a 30°C unilateral irrigation to the right ear. The calibrations, appearing before each of the trials, represent 20° of eye movement. The vertical bars indicate the end of the stimulus. Comparison of nystagmus under the Alert and Reverie conditions prior to alcohol ingestion indicates an enhanced response under the Alert condition. A comparison of the Alert responses for the Pre and the Post I (45 min after alcohol ingestion) conditions reveals the suppressive influence of alcohol; nystagmus is much weaker following alcohol ingestion and resembles more closely the response during Reverie prior to alcohol ingestion than it does the pre-drinking Alert response.

duration values for the Lw trials were still significantly (.05 and .01 levels) below their Pre levels. The only other significant Pre-Post II decline was for Rc/Task frequency (.05 level). No significant differences were found for the slow phase displacement and velocity measures. Overall Post II response recovery for the high alcohol group appeared to be greater than that of the moderate alcohol group. This was true even though the percentage of decline from the Pre to the Post I trials was much greater for the high alcohol group.

Alertness. The nystagmic responses from the caloric irrigations were also analyzed with respect to the effect of variations in the alertness of the subject. Results from the analyses of variance indicated a significant effect of instructions on all measures of the nystagmic response. Although the differences were not as clear-cut for the velocity and frequency measures, for all other measures the means for the Task trials were above the Rev means. The differences are also evident in the average response curves for frequency and slow phase measures (Figures 24 through 31). Under the alert condition, the response was of higher amplitude, higher frequency, and longer duration. These alertness effects and the effects of alcohol on the nystagmic responses to caloric irrigations are evident in the representative tracings presented in Figure 32.

Subjective Ratings

Intensity. Means and standard deviations for the subjects' ratings of the intensity of their "vertigo" sensations for the caloric irrigations are presented in Table 28. An analysis of variance, across all groups, revealed significant F ratios (.01 - .001 levels) for temper-

TABLE 28

Means and Standard Deviations for the Intensity of the Caloric Irrigations as Rated by the Subjects. Each Group Was Comprised of Ten Subjects.

Group	Condition		44°C to the left ear (Lw) Pre	Post I	Post II	30°C to the right ear (Rc) Pre	Post I	Post II
Control	Task	M	73.8	55.0	40.5	68.5	38.5	32.5
		SD	29.4	26.1	25.1	26.5	34.8	27.2
	Rev	M	76.0	54.5	44.5	75.0	33.5	24.1
		SD	33.7	26.6	27.6	26.2	20.2	22.0
Moderate Alcohol	Task	M	85.5	79.5	35.5	66.7	32.0	16.0
		SD	53.5	82.8	36.5	33.0	17.2	17.1
	Rev	M	73.8	106.3	46.5	71.5	33.8	20.0
		SD	44.5	80.9	35.4	38.7	30.0	24.9
High Alcohol	Task	M	81.5	90.5	40.0	73.8	53.5	37.4
		SD	35.0	54.1	29.5	32.0	31.4	27.0
	Rev	M	63.3	87.8	36.0	69.1	52.8	41.5
		SD	36.3	58.0	35.0	31.2	45.0	34.2

ature, trials, and the "temperature by trials" interaction (Table 24). In order to determine if the changes in intensity were the same for all groups, paired t tests were conducted (Table 29).

The control group evidenced a steady decline in the mean intensity ratings from the Pre to the Post II trials. Although the declines in intensity for the Lw trials neared significance, only the declines for the Rc Post I trials were significantly different (.05 - .01 levels) from the Pre ratings (Table 29). All of the Post II intensity ratings were significantly below (.05 - .001 levels) those of the Pre test.

Similar Pre-Post I-Post II declines in the intensity ratings were noted for both alcohol groups. With the exception of a nonsignificant decline in intensity for the Post I Rc/Rev trials for the high alcohol group, all of the Rc intensity ratings for both alcohol groups were significantly below (.05 - .001 levels) the Pre levels (Table 29). The mean Post I Lw intensity ratings made by the moderate and high alcohol groups, with the exception of a slight drop in ratings for the Lw/Task trial by the moderate group, were all above their Pre levels. This fact, that the Post I Rc intensity ratings evidenced a significant decline, while the Lw ratings were, in general, above their Pre levels account for the significant "trials by task" interaction in the analysis of variance (Table 24). Although the Pre-Post II comparison for the Lw/Task trial for the moderate group failed to evidence a statistically significant reduction, all of the other declines were significant (.05 - .001 levels).

Variations in the instructions to the subjects regarding alertness produced little change in their intensity ratings. Results of the

TABLE 29

Results of the Paired t Tests for the Intensity Ratings Resulting From the Caloric Irrigations.

Group	Comparison	44°C to the left ear (Lw)		30°C to the right ear (Rc)	
		Task	Reverie	Task	Reverie
Control	Pre vs. Post I	1.79	2.20	2.27*	3.96***
	Pre vs. Post II	2.78*	2.79*	6.30***	4.69**
Moderate Alcohol	Pre vs. Post I	.23	1.14	3.89**	3.54**
	Pre vs. Post II	3.06*	1.37	5.11***	5.03***
High Alcohol	Pre vs. Post I	.63	1.42	3.60**	1.27
	Pre vs. Post II	3.11*	2.73*	4.21**	3.29**

* $p < .05$
** $p < .01$
*** $p < .001$

analysis of variance (Table 24) indicated that the effect of variations in instructions (alertness) did not produce any significant change in the intensity of the "vertigo" reports.

Duration of "vertigo." The means and standard deviations for the duration of the "vertigo" sensations for the caloric trials are presented in Table 30. An analysis of variance for these data revealed a significant effect for trials (Table 31).

The mean durations of the "vertigo" sensations for the Post I and Post II trials of the control group were all lower than the Pre levels. The duration for both the Post I and Post II trials were more clearly shortened for the Rc trials (22 and 42%) than for the Lw trials (5 and 25%). Comparisons of these differences by paired t tests (Table 32) indicated that the Post I and Post II durations for the Rc trials were significantly shorter (.01 level) than the Pre durations, while no significant changes occurred in the durations for any of the Lw trials.

Although most of the Pre-Post I declines in mean duration, for the alcohol groups, were larger than those of the control group only the change in response for the Rc trial of the high alcohol group was significant (.05 level). All of the Pre-Post II declines in mean duration were significant (.05 and .01 levels).

Overview. The duration and intensity measures of the response to the caloric irrigations, when compared to the objective (nystagmic) measures, appear to be less susceptible to variations in the alertness of the subject. The steady Pre to Post I to Post II decline in the subjective measures may indicate that these variables are also more susceptible to the effects of repeated stimulation (i.e., to habituation).

TABLE 30

Means and Standard Deviations for the Duration (in Seconds) of the Subject's "Vertigo" Sensations Resulting from the Caloric Irrigations under the Task Condition.

		44°C to the left ear (Lw)			30°C to the right ear (Rc)		
Group		Pre	Post I	Post II	Pre	Post I	Post II
Control	M	74.0	70.1	55.2	84.7	66.1	49.4
	SD	22.4	19.3	16.7	23.3	21.4	22.9
Moderate Alcohol	M	85.0	73.7	46.5	62.1	62.3	38.1
	SD	29.5	20.8	30.1	38.3	28.3	27.1
High Alcohol	M	76.4	67.8	53.1	84.7	58.7	55.3
	SD	31.2	20.1	24.8	35.8	29.6	26.2

TABLE 31

Results of the Analysis of Variance for the Durations of the Subjects' "Vertigo" Sensations Resulting from the Caloric Irrigations

Source	df	Mean Squares	F
Groups (G)	2	510.27	0.24
Sub/Group (S/G)	27	2,121.81	
Temperature (Te)	1	900.48	1.13
Te x G	2	1,068.07	1.34
Te x S/G	27	800.00	
Trial (Tr)	2	12,069.52	31.71***
Tr x G	4	371.74	0.98
Tr x S/G	54	380.64	
Tr x Te	2	178.38	0.64
Tr x Te x G	4	454.89	1.64
Tr x Te x S/G	54	277.92	

*** $p < .001$

TABLE 32

Results of the Paired t Tests for the Durations of the Subjects' "Vertigo" Sensations Resulting From the Caloric Irrigations Under the Task Condition.

	44°C to the left ear (Lw)		30°C to the right ear (Rc)	
Group	Pre vs. Post I	Pre vs. Post II	Pre vs. Post I	Pre vs. Post II
Control	0.54	2.16	4.15**	4.44**
Moderate Alcohol	2.04	3.60**	0.02	2.61*
High Alcohol	1.06	2.73*	3.24*	3.21*

* $p < .05$
** $p < .01$

Optokinetic Stimulus

Tables 33 and 34 reflect the means and standard deviations for the total slow phase displacement and frequency measures of the nystagmic responses for the optokinetic trials. Results of separate analyses of variance for each group appear in Table 35. These means, standard deviations, and analyses are based on the responses made during the stimulus period. Data concerning the afternystagmus (i.e., the response after termination of the stimulus) will be presented in a separate section.

Control Group. A comparison of the means (Tables 33 and 34) or of the average response curves (Figures 33 through 37) indicate little variation across trials in either the slow phase displacement or frequency measures of the nystagmic responses for the control group. The Post I and Post II mean slow phase displacement and frequency responses were all within $\pm$ 6% of their respective Pre values. Separate analyses of variance for each of the four stimulus conditions revealed no significant effects (Table 35).

Moderate Alcohol Group. All of the Post I responses of the moderate alcohol group were below their Pre levels (Tables 33 and 34). The degree of Pre to Post I response decline was similar for both measures of nystagmus, 12-22% in total slow phase displacement and 9-15% in frequency. According to the analyses of variance (Table 35), only the Pre-Post I-Post II changes in slow phase displacement for the 60-seconds LON (lights remained on after the stimulus) and the 60-seconds LOFF (lights were turned off immediately after stimulus termination) trials were significant. According to paired t tests (Table 36), the Pre-Post I differences for both of the 60-second trials, were signifi-

TABLE 33

Means and Standard Deviations for the Slow Phase Nystagmus Displacement (in Degrees) Resulting From the Optokinetic Stimuli. Each Group Was Comprised of Ten Subjects.

Group	Condition		15 sec Pre	15 sec Post I	15 sec Post II	60 sec Pre	60 sec Post I	60 sec Post II
Control	Lights On	M	165.1	155.8	158.0	616.7	618.0	604.4
		SD	25.4	29.5	32.4	126.0	78.3	105.5
	Lights Off	M	160.9	163.4	161.3	641.4	641.8	610.4
		SD	15.9	27.1	23.9	79.1	103.6	99.0
Moderate Alcohol	Lights On	M	157.3	138.9	150.3	637.1	518.4	551.4
		SD	15.7	36.6	36.4	75.5	114.4	117.2
	Lights Off	M	151.8	136.2	148.5	620.6	483.0	553.2
		SD	21.0	34.4	28.4	88.3	119.8	132.9
High Alcohol	Lights Off	M	141.5	94.6	138.8	540.7	344.6	505.6
		SD	26.9	43.5	34.7	82.8	226.9	154.0
	Lights Off	M	154.6	92.1	129.7	580.6	343.2	511.4
		SD	23.3	58.5	28.4	145.5	260.0	140.7

TABLE 34

Means and Standard Deviations for the Number of Nystagmic Beats Resulting From the Optokinetic Stimuli.

Each Group Was Comprised of Ten Subjects.

Group	Condition		15 sec Pre	15 sec Post I	15 sec Post II	60 sec Pre	60 sec Post I	60 sec Post II
Control	Lights On	M	39.4	41.0	39.6	148.0	147.4	151.9
		SD	5.6	6.2	7.8	26.9	18.4	20.5
	Lights Off	M	42.1	41.1	42.2	154.2	158.4	159.9
		SD	4.9	5.9	5.7	14.5	16.4	16.8
Moderate Alcohol	Lights On	M	38.2	33.1	33.7	144.4	124.1	135.1
		SD	7.6	9.3	10.0	34.5	27.2	33.4
	Lights Off	M	36.4	33.0	34.0	136.2	115.3	126.5
		SD	8.1	8.7	8.1	31.1	26.0	30.8
High Alcohol	Lights On	M	35.6	24.7	35.8	141.4	81.7	130.5
		SD	8.0	6.5	6.1	31.3	46.7	22.6
	Lights Off	M	37.9	23.6	34.8	140.2	73.5	124.3
		SD	5.4	11.0	7.4	20.7	55.9	27.3

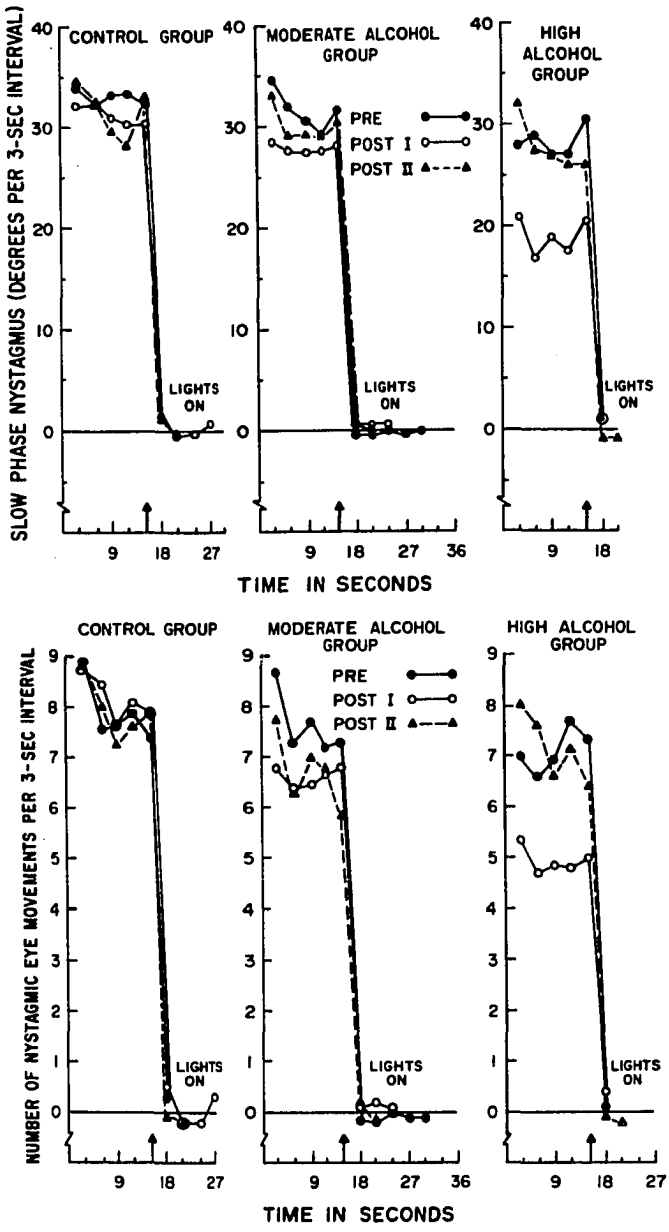

Figure 33. Response data for the average number of degrees of slow phase eye movement (upper half) and average number of nystagmic eye movements (lower half) resulting from the 15-sec optokinetic trials. Pre refers to the response obtained prior to the ingestion of alcohol, while Post I and Post II refer, respectively, to the data obtained 45 min and four hours after ingestion. "Lights on" indicates that the room lights remained on both during and following the stimulus period. The values are plotted in 3-sec intervals; each point is a mean of 10 subjects.

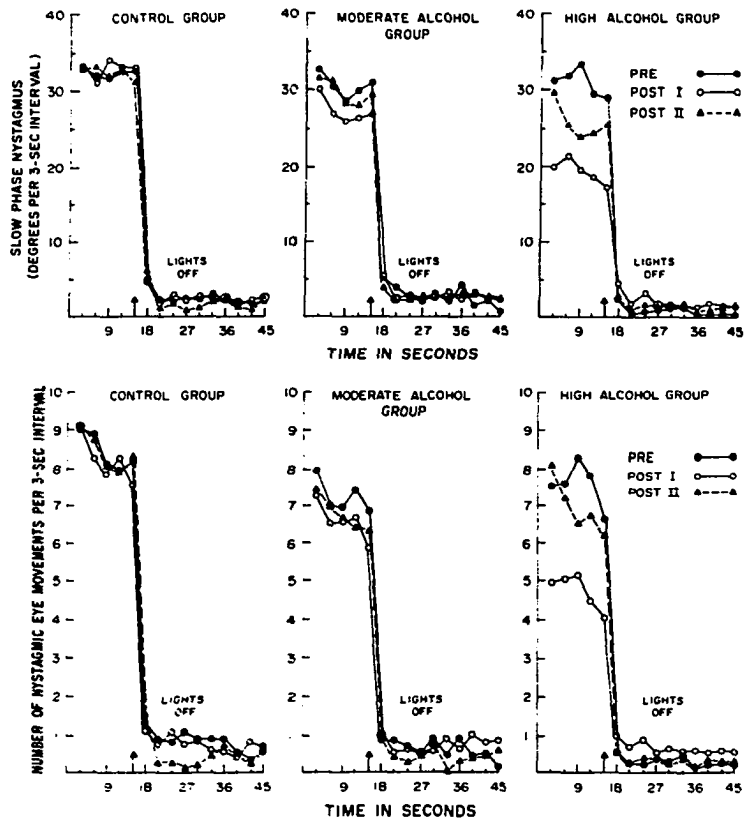

Figure 34. Response data for the average number of degrees of slow phase eye movement (upper half) and average number of nystagmic eye movements (lower half) resulting from the 15-sec optokinetic trials. "Lights off" indicates that the room lights were turned off immediately after the end of the stimulus. Other symbols and markings are identical to those used in Figure 33.

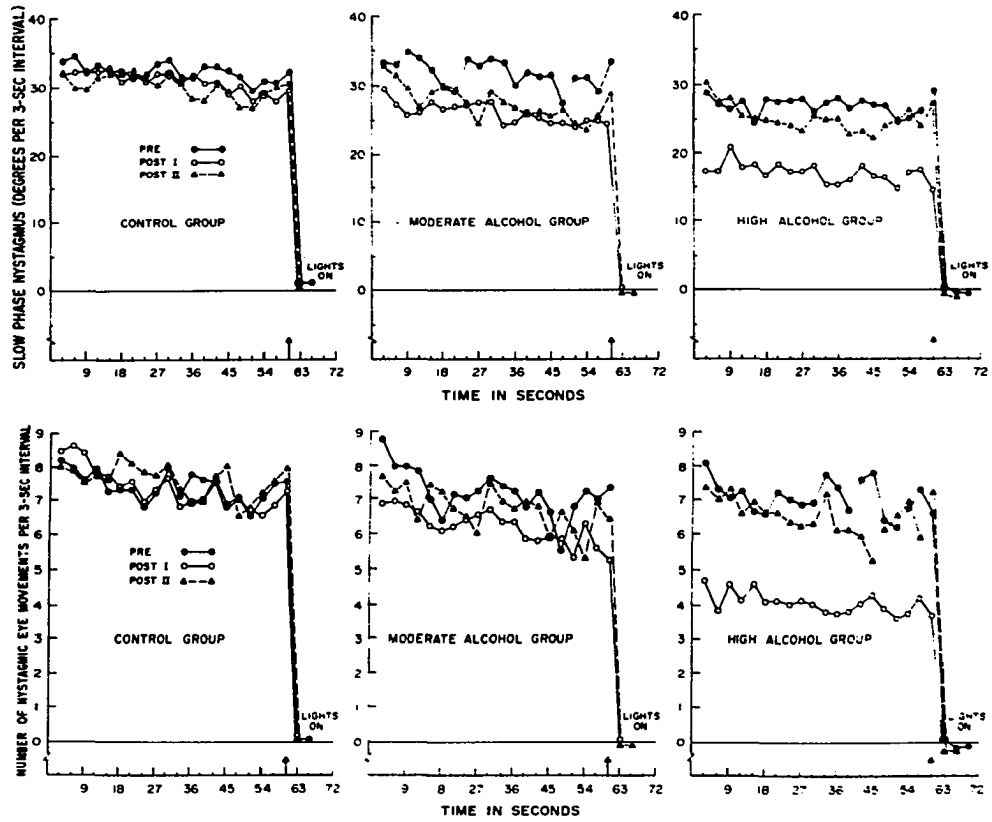

Figure 35. Response data for the average number of degrees of slow phase eye movement (upper half) and average number of nystagmic eye movements (lower half) resulting from the 60-sec (lights on) optokinetic trials. Symbols and markings are identical to those used in Figure 33.

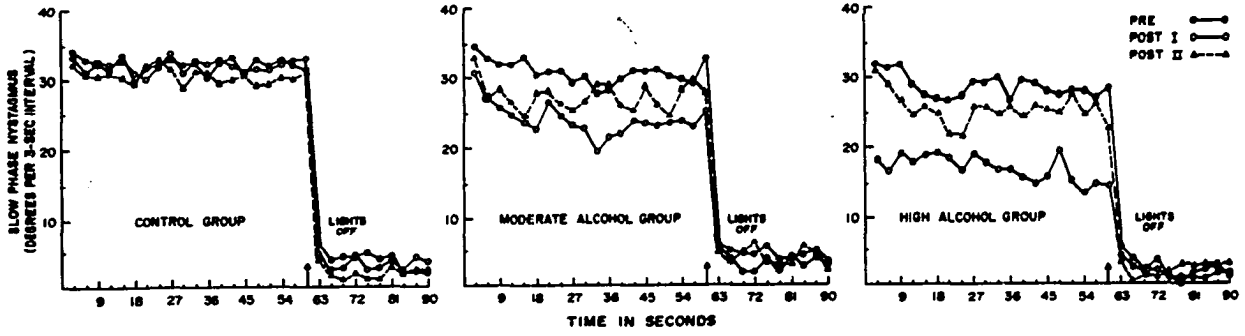

Figure 36. Response data for the average number of degrees of slow phase eye movement resulting from the 60-sec (lights off) optokinetic trials. Symbols and markings are identical to those used in Figure 33.

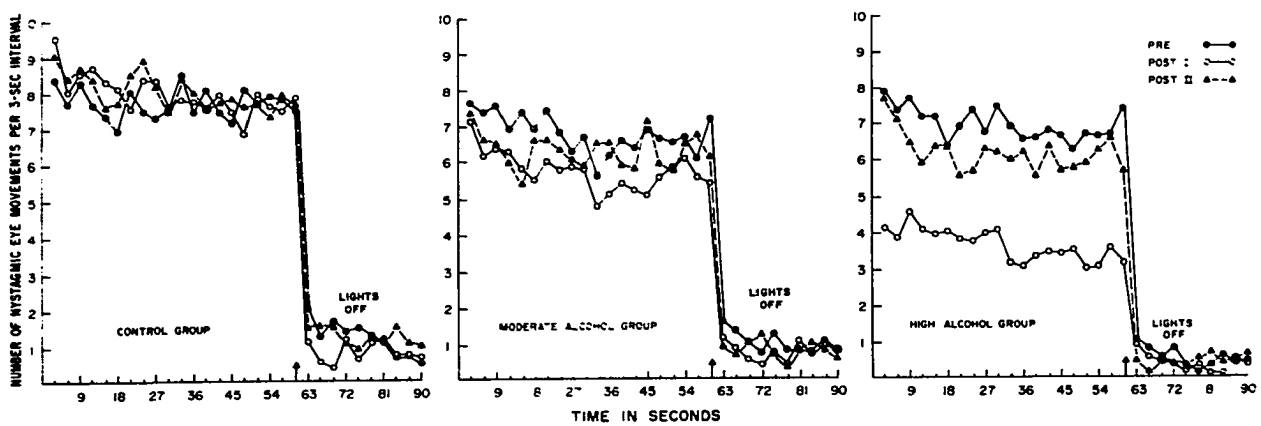

Figure 37. Response data for the average number of nystagmic eye movements resulting from the 60-sec (lights off) optokinetic trials. Symbols and markings are identical to those used in Figure 33.

TABLE 35

Results of the Analyses of Variance for the Number of Degrees of Slow Phase Nystagmus and Number of Nystagmic Eye Movements Resulting from Optokinetic Stimulation. Stimuli Were of 15 or of 60 Seconds Duration.

		Lights On		Lights Off	
Measure	Group	15-sec	60-sec	15-sec	60-sec
Slow Phase Displacement	Control	0.38	0.05	0.03	0.36
	Moderate Alcohol	0.88	3.46*	0.84	3.57
	High Alcohol	5.45**	4.00*	5.26*	4.12*
Frequency	Control	0.17	0.12	0.12	0.30
	Moderate Alcohol	0.95	1.02	0.44	1.17
	High Alcohol	7.29**	8.27**	8.27**	8.46**

* $p < .05$
** $p < .001$

TABLE 36

Results of the Paired t Tests for the Pre vs. Post I and Pre vs. Post II Comparisons of Nystagmic Responses to the Optokinetic Stimuli.

Measure	Group	Comparison	15 second stimulus		60 second stimulus	
			Lights On	Lights Off	Lights On	Lights Off
Slow Phase Displacement	Control	Pre vs. Post I	0.97	0.50	0.05	0.02
		Pre vs. Post II	1.22	0.08	0.31	1.99
	Moderate Alcohol	Pre vs. Post I	2.23	1.72	4.32**	5.80***
		Pre vs. Post II	0.78	0.51	2.52*	2.01
	High Alcohol	Pre vs. Post I	3.10*	3.67**	3.14*	3.65**
		Pre vs. Post II	0.22	3.15*	0.88	1.58
Frequency	Control	Pre vs. Post I	1.11	0.84	0.11	1.11
		Pre vs. Post II	0.09	0.18	0.80	1.78
	Moderate Alcohol	Pre vs. Post I	2.18	1.68	2.17	2.86*
		Pre vs. Post II	1.31	1.99	1.02	1.22
	High Alcohol	Pre vs. Post I	3.28**	3.67**	4.06**	3.60**
		Pre vs. Post II	0.11	2.05	1.70	2.61*

* $p < .05$
** $p < .01$
*** $p < .001$

cant (.01 and .001 levels), but for the Pre-Post II comparisons, only the 60-second LON trial evidenced a significant decline (.05 level) in slow phase displacement.

High Alcohol Group. The depressive influence of alcohol was most evident in the means (Tables 33 and 34) and average response curves (Figures 33 through 37) for the high alcohol group. As was true of the moderate alcohol group, the degree of Pre-Post I change in the two measures of the nystagmic responses was nearly identical; 31-48% declines in frequency and 33-41% declines in slow phase displacement. The F ratios for the "trial" factor were significant for both measures and for all trials (Table 35). All of the Pre-Post I response declines were significant (.05 - .01 levels) according to paired t tests (Table 36).

Responses for the Post II trials evidenced some recovery from the Post I levels. Only two of the Pre-Post I comparisons were significantly different; the 11% decline in frequency for the 60-second LOFF trial (.05 level), and the 16% decline in the slow phase displacement for the 15-second LOFF trial (.05 level).

Afternystagmus. Following the end of the optokinetic stimulus period, the subject's responses were recorded for 30 seconds in the light (LON condition) or for 30 seconds in the dark (LOFF condition). Examination of the average response curves (Figures 33 through 37) reveals almost no nystagmic response, after the end of the stimulus, for the LON condition. Only one or two subjects responded with a beat or two of primary or secondary nystagmus. Due to this lack of response, no reliable differences were noted for the three groups.

Under the LOFF condition, at least one-half of the subjects dis-

TABLE 37

Means and Standard Deviations for Slow Phase Displacement (in Degrees) and Number of Nystagmic Beats of Afternystagmus Resulting From the Lights Off Optokinetic Stimuli.

			15-sec Lights Off			60-sec Lights Off		
Measure	Group		Pre	Post I	Post II	Pre	Post I	Post II
Slow Phase Displacement	Control	M	25.0	25.6	20.3	39.2	34.6	21.1
		SD	26.4	40.7	36.0	34.6	58.6	54.3
	Moderate Alcohol	M	27.7	29.9	28.8	46.8	33.0	42.0
		SD	34.5	25.8	30.7	39.4	29.2	42.4
	High Alcohol	M	9.1	19.9	15.1	24.4	18.4	23.4
		SD	12.8	28.0	26.1	32.7	26.2	34.5
Frequency	Control	M	8.3	7.6	4.9	12.3	8.4	13.1
		SD	8.2	12.7	10.5	9.7	12.6	14.9
	Moderate Alcohol	M	6.6	7.5	4.8	10.6	8.2	8.6
		SD	8.8	6.2	11.7	7.4	6.5	9.3
	High Alcohol	M	3.0	6.7	3.3	5.8	4.1	5.1
		SD	4.4	8.5	5.6	6.8	5.7	7.6

played a primary afternystagmus. A 30-second period of the average afternystagmus following the end of the stimulus is plotted in Figures 35, 36, and 37. Means and standard deviations for the total slow phase displacement and frequency of the after-response appear in Table 37. The Pre-Post I responses for the three groups were nearly identical. All three groups evidenced a decline in slow phase displacement and frequency of the afternystagmus for the 60-second Post I trials, while the responses for the 15-second trials during Post I were all slightly above their Pre values. The Post II data were, in general, of somewhat lower magnitude than the Post I values. Once again there were no noticeable differences among groups. Alcohol ingestion failed to produce any observable changes in the afternystagmus resulting from the optokinetic stimulation.

CHAPTER IV

DISCUSSION

Alertness

Attempts to control the subject's alertness are especially important in studies involving repeated vestibular stimulation, or where Pre- versus Post-treatment comparisons are made. The subject, during the first testing session, is typically alert and anxious because he is unfamiliar with the experimental (or clinical) situation. Once he has adapted to the situation and learned what is required of him, he tends to relax.

Numerous studies by Collins (e.g., Collins, 1962, 1963, 1964) and others (e.g., Mowrer, 1934; Wendt, 1951) have indicated that variations in alertness produce changes in the nystagmic responses to caloric and rotatory stimulation. These studies have indicated that the nystagmic response under alert conditions, as opposed to relaxed conditions, will be of higher amplitude and of longer duration. Moreover, alertness can be maintained by appropriate instructions.

Data from the present study indicate that all measures of the nystagmic response are susceptible to instruction-produced variations in alertness. The overall analyses of variance (Tables 6 and 24) indicate, for both the rotatory and caloric stimuli, that instructions

regarding different degrees of alertness produced significant changes in the amplitude, duration, frequency, and peak slow phase velocity of the eye movements. However, the effect was much more pronounced in the amplitude and duration measures.

If alertness were uncontrolled, a large Pre to Post decline in responses could be obtained which might be attributable to changes in the level of the subject's alertness rather than to the effect of any experimental variable. This would appear to be particularly true when testing the effects of depressants on the vestibular system. A depressant, depending on the dosage level, may produce large changes in the subject's level of alertness. If alertness is not maintained, through the use of various tasks, changes in the responses may be due entirely to changes in alertness rather than to the effects of the drug on the vestibular system per se. The significant Pre to Post I decline in the duration of the nystagmic response, from the caloric irrigations, for the Reverie (relaxed) condition (Table 25) is evidence of the magnitude of change in the response which may occur if alertness is not controlled.

Subjective Vestibular Reactions

Rotatory Stimuli. Barany (1911), the only previous author to report any information concerning the effects of alcohol intoxication on the sensations resulting from rotatory stimulation, noted that alcohol inhibited turning experiences. This inhibition was attributed to the influence of alcohol on the cerebrum.

Results from the present study provide only partial support for Barany's conclusions. While both alcohol groups, when compared to the control group, evidenced larger Pre to Post I declines in the duration

and the subjective velocity of the rotatory sensations, only the duration measures for the brake decelerations were significantly below their Pre levels. This slight inhibitory effect of alcohol on the rotatory sensation was much less evident than the effect of alcohol on the various measures of the nystagmic response to the rotatory stimuli. Since Barany (1912) failed to describe his experimental conditions, there is no obvious explanation for the differences among these sets of data.

Caloric Stimuli. There was no evidence of any inhibitory effect of alcohol on the "vertigo" sensations resulting from the caloric irrigations. However, there was some evidence of a possible differential effect of alcohol on the intensity ratings for the Rc and Lw stimuli. All of the groups evidenced significant Pre to Post I declines in the intensity of the sensations resulting from the Rc irrigations. Although the control group showed a similar decline for the Lw irrigations, both alcohol groups rated the Post I Lw irrigations as being the same, or more intense, than the Pre irrigations. This difference was not evident in the duration measures, where all groups reported shorter durations for the Post I Rc irrigations, while the declines for the Lw irrigations were smaller.

Since the differential effect was evident only in the intensity ratings, and not in the duration of vertigo, nor in the nystagmic measures, the subjects may have been responding to a different aspect of the experimental situation during the Post I trials. Rather than reacting to the intensity of the resultant "vertigo" sensations, the subjects may have been reacting to their perception of the intensity of the water temperature. It is possible that the alcohol, because of the

changes it induces in the vascular system, altered the subject's perception of the heat of the Lw irrigations. Changes in the perceived intensity of the temperature of the Lw irrigations may thus be responsible for the difference in the Post I ratings for the Lw as compared to the Rc irrigations. Further research is needed to determine if this difference is consistent rather than an artifact.

Whereas the alcohol groups evidenced some response recovery for most of the measures of the nystagmic responses in the Post II sessions, none was evident for either the intensity ratings or the duration of the subjective sensations. The continual decline from the Pre to Post II trials is probably due to habituation of the response. Since all of the caloric irrigations produced a left-beating nystagmus, there was probably a sufficient number of trials to result in a significant, directionally-specific response reduction (habituation).

Nystagmus

Optokinetic Stimuli. Results of the optokinetic trials agree with the results obtained in previous studies by Scofield (1936), Krauland, Schuster and Klein (1961), Mizoi, Ishido, and Ohga (1962), Ey (1963), and Bloomberg and Wassen (1962). Acute alcohol intoxication suppresses the nystagmic response to optokinetic stimuli. These data also indicate that the degree of suppression was related to the amount of alcohol ingested; the high alcohol group evidenced larger Pre to Post I declines than the moderate alcohol group. The response was nearly at its normal level at the Post II testing session.

Comparison of the afternystagmus during the light trials revealed

no significant differences between the alcohol and non-alcohol conditions.

These data provide little information concerning the locus of effect of alcohol. Alcohol ingestion may result either in a form of paralysis of the eye muscles or it may interfere (centrally) with the ability of the subject to maintain his fixation. The role of visual fixation in optokinetic nystagmus has been discussed by several authors (ter Braak 1936; Rademaker and ter Braak 1948; Honrubia, et. al., 1968) who have indicated that optokinetic nystagmus is weaker under conditions where the subject maintains his fixation on a point in front of, or behind, the drum. An optimal optokinetic nystagmus occurs when the subject fixates on the surface of the stimulator.

The effects of alcohol on optokinetic nystagmus appear to be similar to those produced by drugs which act as depressants of central nervous system functioning. According to Bergman, et al., (1952) and Bender and O'Brien (1946), a barbiturate (sodium amytal) will also suppress the nystagmus resulting from optokinetic stimulation. Other types of research are required to determine the locus of effect of alcohol and to ascertain whether the elimination of optokinetic nystagmus is common to all drugs which depress CNS functioning.

Rotatory Stimuli. Alcohol ingestion produced a significant suppression of all aspects of the nystagmic response to rotatory stimulation. The degree of suppression was related to the amount of alcohol consumed; the high alcohol group evidenced larger Pre to Post I declines in the various response measures than did the moderate alcohol group.

Nearly all measures of the nystagmic responses evidenced con-

siderable recovery at the Post II testing sessions (four hours after ingestion). As the alcohol level in the blood was decreasing, there was a corresponding increase in the intensity of the nystagmic response.

These data support the conclusions reached in earlier reports by Forster (1958), Bochenek and Ormerod (1962), Mizoi, Ishido, and Ohga (1962), Ey (1963), and Di Guinta and Rosa (1968). Studies by Manz (1939), Taschen (1955a, 1955b), Schweitzer (1955), and Schulte and Roth (1957) still represent conflicting viewpoints since these authors reported that alcohol enhanced the nystagmic response following alcohol intoxication.

Caloric Stimuli. The conclusions reached following analysis of the caloric data are in agreement with those presented concerning rotatory stimulations. Alcohol intoxication produced a significant suppression of all aspects of the nystagmic response. As was true for the rotatory stimuli, the degree of suppression varied with the amount of alcohol consumed and there was considerable response recovery evident at the Post II testing session.

These data support the results of previous investigations by Bochenek and Ormerod (1962). Earlier studies by Manz (1939), Schwab and Ey (1955) and Rauschke (1962) are not supported, since these authors concluded that alcohol ingestion either prolonged the duration of the nystagmus or shortened the latency of the response.

Resolution of Conflicting Findings Among Studies

Under conditions of the present study, where the nystagmus was recorded in total darkness, alcohol ingestion suppressed the nystagmic

response to caloric and rotatory stimulation. In an attempt to explain the discrepancy between this investigation and those which indicate that alcohol enhanced vestibular responses, the literature was reviewed with respect to the procedures under which the studies were conducted. Although the precise experimental conditions were not mentioned in all cases, the data appear to fall into separate categories on the basis of whether the nystagmus was observed visually or whether it was recorded in total darkness using the ENG technique. An enhanced nystagmus was reported following alcohol ingestion whenever ocular fixation by the subject was allowed while the experimenter visually observed the eye movements, and a suppressed nystagmus was reported under conditions where the nystagmic response was recorded in the absence of visual stimulation.

In order to test the effect of alcohol on visual fixation during a vestibular response, several subjects underwent rotatory and caloric stimulation while fixating in the light. Eye movements were recorded before and 45 minutes after alcohol ingestion. Representative tracings of two subjects' nystagmic responses to the rotatory or caloric stimuli appear in Figures 38 and 39.

The rotatory condition began with an acceleration ($5^{o}/sec^{2}$ for 12 sec) while the subject fixated on a small, dimly illuminated target with the room otherwise in total darkness. The target was secured to the rotator and moved with it. Following two min at constant velocity (10 RPM) the subject received a brake deceleration; the room lights were turned on immediately after the end of the deceleration. Prior to the ingestion of alcohol the subject was able, through visual fixation, to suppress markedly the nystagmic response. Following alcohol ingestion,

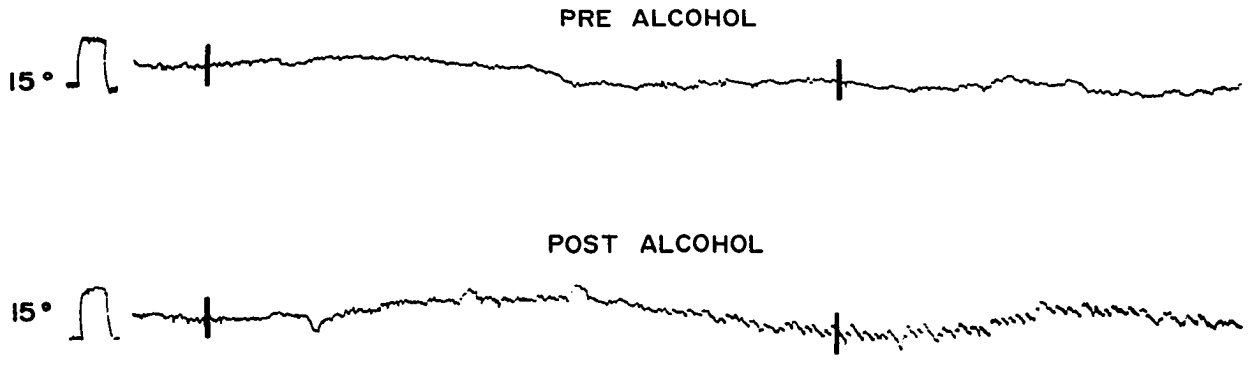

Figure 38. A portion of the tracings of the nystagmic response of a subject to a 12-sec acceleration of $5^{o}/sec^{2}$ during visual fixation of a dimly lit target-light. The vertical bars indicate the beginning and end of the stimulus. The calibration, representing 15^{o} of eye movement, appears before each trial. The subject's response was recorded before and 45 min after the ingestion of 2 cc of 100 proof vodka per kg of body weight. The effect of alcohol on the visual fixation system is evident in the relatively strong nystagmic response for the Post condition. Visual fixation, while under alcohol intoxication, is much less effective in suppressing the nystagmic response to vestibular stimulation.

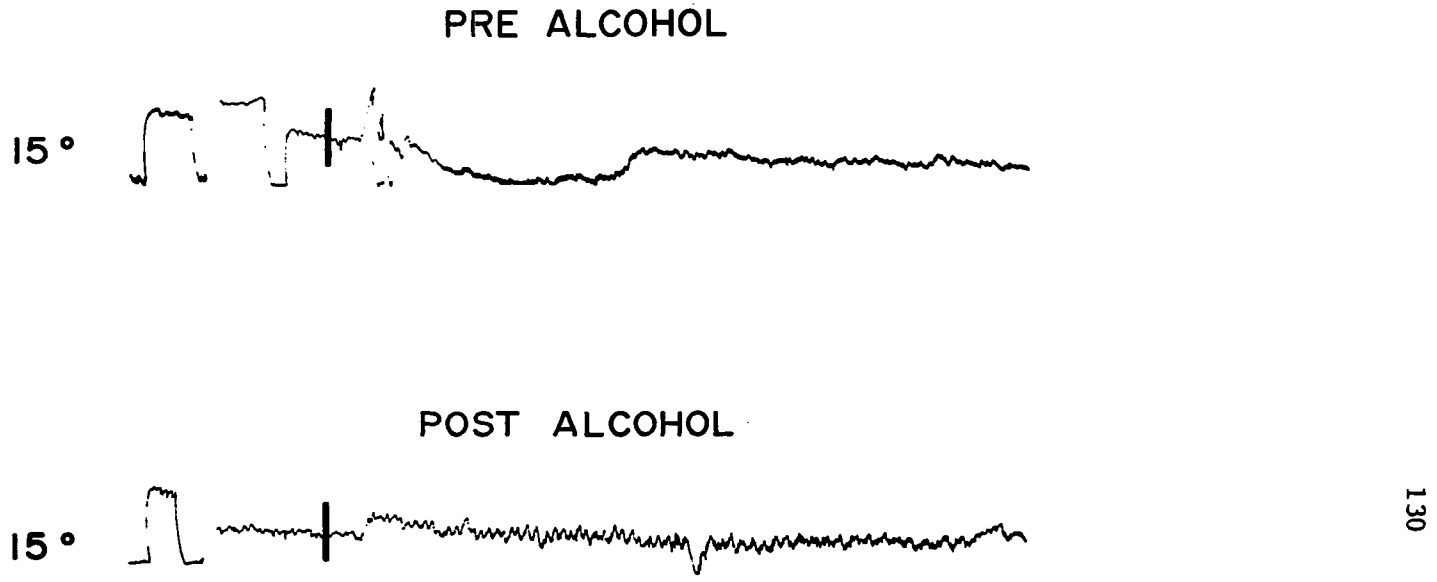

Figure 39. A portion of the tracings of the nystagmic response of a subject to a brake deceleration, following two min of constant velocity at 60°/sec. The vertical bar indicates the start of the deceleration and the point where the room lights were turned on. The subject was instructed to fixate on the target-light as soon as the room lights came on. A calibration representing 15° of eye movement appears before each trial. Changes in the response under alcohol are similar to those noted in Figure 38.

a high frequency nystagmus was obtained. A similar effect was noted when the subject was administered caloric irrigations before and after alcohol ingestion (Figure 40).

These data suggest that alcohol interferes with the ability of the individual to maintain ocular fixation during vestibular eye movements. Under normal conditions the subject was able to suppress the nystagmic response to a mild vestibular stimulus. However, because of the depressive influence of alcohol, the subject's ability to still-fixate was weakened, thereby permitting the nystagmic response to manifest itself. These differences between the alcohol and non-alcohol conditions were also evident in the introspective reports made by the subjects. They noted much more difficulty in attempting to fixate on the target during the alcohol session than during the non-alcohol session.

Thus, the apparently conflicting results in previous studies can be resolved by analysis of differences in experimental techniques. Under conditions of total darkness, alcohol ingestion has a suppressive effect on the vestibular response. What had been reported in earlier studies as an enhancing effect of alcohol can now be attributed to the depressive effect of alcohol on the ability of the individual to fixate visually. This interference with the visual fixation center renders the subject less able to inhibit the vestibular response. These data are supported by Jatho (1965), who reports that alcohol intoxication results in inadequate recovery movements of the eye when the individual is required to shake his head. Jatho (1965) relates this alcohol-induced change to the inability of the intoxicated driver to pick out different cars, signs,

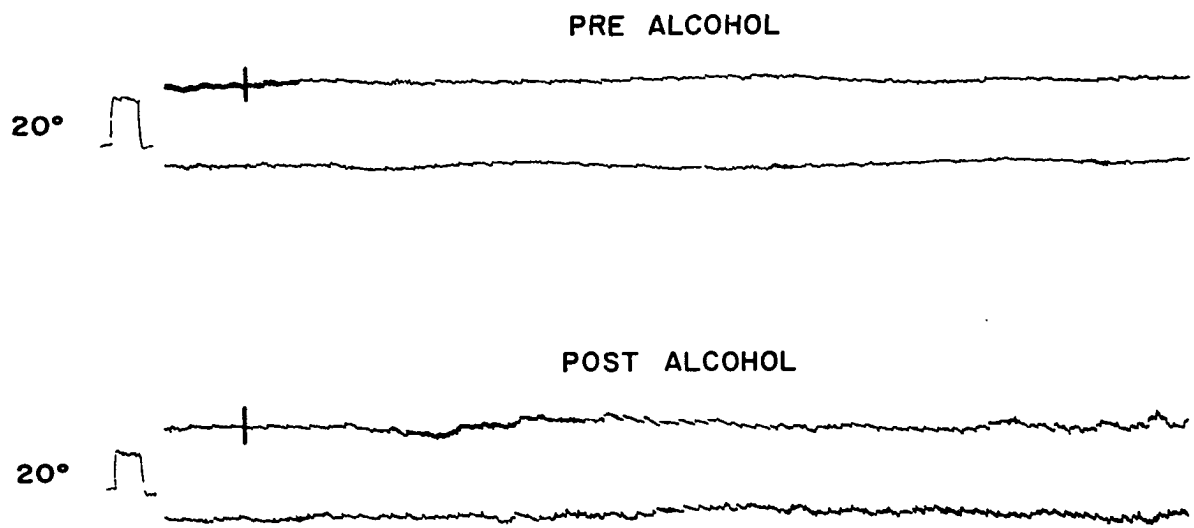

Figure 40. A portion of the tracings of the nystagmic response of a subject (DS) to a 30-sec Lc calorization. The caloric irrigation was administered with the room lights on while the subject fixated on a target. The vertical bar marks the end of the stimulus period. The calibration appearing before each of the trials represents 20° of eye movement. Changes in the nystagmic response following alcohol ingestion are the same as those noted in Figure 38.

and pedestrians when head and body movements occur as the result of vehicular movements.

Suppression of the visual fixation center is also evident in the work of Rashbass and Russell (1961) in which several caloric trials were administered before and after the administration of a barbiturate (sodium amytal). Using the ENG technique, the nystagmic response was recorded in total darkness and under conditions allowing visual fixation in the light. In comparing a drug and a non-drug trial, the authors report that there was no change in the nystagmic response under dark conditions. When visual fixation was allowed, there was no evidence of a response under the non-drug condition; however, after administration of the drug, the nystagmic response was vigorous and nearly identical to that obtained in darkness. These data imply that a drug which suppresses central nervous system functioning also inhibits the visual fixation center. Further information is needed to determine if all CNS depressants have a similar effect on the ability of the subject to maintain visual fixation following vestibular stimulation.

Influence of Brief Periods of Visual Fixation

Although the number of subjects in each of the separate light groups was small (N=5), the data seem to agree with previous findings by Guedry, Collins, and Sheffey (1961) and by Collins (1968a, b). The following conclusions are based on visual analyses of the tracings rather than on any statistical treatment of the data.

Brief periods of visual fixation, during or after angular stimulation, shortened the duration and overall intensity of the primary

nystagmus. As the primary response was shortened, the secondary response (a nystagmus in the opposite direction) began sooner and became more intense when compared to the secondary response in total darkness.

Visual fixation, according to Collins (1968a, b), will also change the subject's rotatory sensations. Under normal conditions, when the subject was accelerated in total darkness, the primary rotatory sensation was relatively long. Following the end of the primary sensation, some subjects reported a weak secondary sensation (i.e., an experience of turning in the opposite direction). Under the "light" conditions in the present study, the primary sensations were shortened. Several subjects indicated that they failed to experience any sensations following the cessation of the light, while others reported rather strong secondary sensations. In general, the "light" interval reduced the intensity and duration of the primary sensation, and increased the intensity and duration of the secondary sensation. The trends observed in these data are in agreement with the conclusions reached by Collins (1968a, b).

When comparing the Pre, Post I, and Post II responses, there appears to be little evidence of any change as the result of alcohol ingestion. However, although changes in the total primary nystagmic output were slight, the nystagmus during the first 3-sec interval of darkness after the light was off appeared different following the consumption of alcohol. Comparison of the first 3-sec dark interval after the lights were extinguished (Figures 16 through 19) indicated that the primary nystagmic response was stronger for the Post I and Post II trials of both alcohol groups. Since there was no change in the response of the control group for the same intervals, the change may be attributed to the effect of alcohol.

Spiral Aftereffect

Alcohol ingestion failed to produce any significant changes in the duration of the spiral aftereffect (SAE). The duration values for all groups remained relatively stable throughout the three testing sessions.

Correlations between the duration of the SAE and the duration of the sensations resulting from the rotatory stimuli were calculated to provide further information concerning earlier work by Nilsson and Henriksson (1967), Reason (1968), Reason and Benson (1968), and Reason (1969). The significant correlations previously reported between the two measures, according to Reason (1968; 1969), reflect an individualized system of processing sensory information.

Most of the correlations between the duration of the SAE and the duration of the sensations resulting from either the 12-sec acceleration or the brake deceleration failed to reach significance in the present study. Only the high alcohol group evidenced a statistically significant (and positive) relationship between the two duration measures.

The only obvious difference between the responses of the high alcohol group and those of the control or the moderate alcohol group was the variability of their duration values for the SAE. The standard deviations for the high alcohol group were nearly double those of the other two groups (see Table 17). This lack of variability in the SAE measure was possibly the most significant factor in producing the low correlations.

Differences in results between this investigation and previous studies may also be due to differences in the procedures used to obtain

the response measures for the rotatory sensations. The duration of the rotatory sensations, in previous studies, was based on either the average duration of several successive trials at the same stimulus rate (Nilsson and Henriksson, 1967), or on the average obtained following several different stimulus rates (Reason, 1968; Reason and Benson, 1968). These additional data collected for each subject would tend to provide a more stable measure.

Differences were also apparent in the technique used to determine the duration of the rotatory sensations. Nilsson and Henriksson (1967) asked their subjects to call out when they no longer perceived any movement, while Reason (1968) and Reason and Benson (1968) asked their subjects to press a switch when they no longer had any sensation of rotation. In the present study, the subject was instructed to pay careful attention to his sensations and to signal the start and end of his rotatory experiences for the brake decelerations while signaling the start, each 90^{o} turn, and the end of his sensation for the 12-sec acceleration. Differences in instructions may have altered how the subjects approached the task and consequently how they signaled their sensations. Other differences in procedure between this investigation and the study by Nilsson and Henriksson (1967) are that the latter used the duration of the oculogyral illusion (apparent movement of a dim light following rotatory stimulation) as their response measure, and more intense rotatory stimuli (brake decelerations from a constant velocity of 116^{o}/sec, compared to our brake decelerations from a constant velocity of 60^{o}/sec).

CHAPTER V

SUMMARY

The present study was designed to investigate the effects of two levels of alcohol (1.25 and 2.5 cc of 100 proof vodka per kg of body weight) on the nystagmic and subjective responses to rotatory and to caloric stimuli. Two sets of three groups each (a total of six groups, each comprising ten subjects) were used; one set of three groups received rotatory stimulation and viewed the spiral aftereffect, the other set received caloric irrigations and optokinetic stimulation. In each set, two alcohol groups ("moderate" and "high") were tested the day before (Pre), 45 min after (Post I), and four hours after (Post II), the ingestion of a mixture of orange juice and vodka. The third group ("control") in each set was similarly tested, but consumed only orange juice.

Alcohol ingestion suppressed the nystagmic responses to both modes of vestibular stimulation. The high alcohol groups evidenced larger response declines for the Post I trials than did the moderate alcohol groups. At the time of the Post II session the responses had started to return to their Pre levels.

Changes in the subjective reports resulting from the caloric and rotatory stimuli were less conclusive. Although the Post I rotatory sensations were lower than those of the Pre level, the differences, in most

cases, were not statistically significant. There was little evidence of any suppressive effect of alcohol on the "vertigo" sensations resulting from the caloric irrigations.

A resolution of the conflict among earlier studies of the effects of alcohol on the vestibular system was presented. Several previous investigators had reported that alcohol enhanced the nystagmic response while others claimed that it suppressed that response. The present study clearly indicates that, under conditions of total darkness, alcohol suppresses the nystagmic response to vestibular stimulation. However, under conditions where visual fixation was allowed, and where visual fixation was normally able to inhibit the nystagmic responses, the ingestion of alcohol allowed a strong nystagmic response to occur. The vestibular response was not enhanced, it was simply more evident because alcohol (directly or indirectly) inhibited the visual fixation system.

Data were also obtained concerning the effects of alcohol on optokinetic nystagmus. The changes in the nystagmic response, following alcohol ingestion, were similar to those noted for caloric and rotatory stimuli; the response was suppressed.

The effects of variations in subject alertness on the nystagmic responses to caloric and to rotatory stimuli were examined by means of different types of instructions. The results support previous studies demonstrating the enhancing effect of alerting instructions. Data from both modes of stimulation indicate that all measures (slow phase displacement, frequency, peak velocity, and duration) of the nystagmic response were of greater magnitude under "alert" conditions as compared with "reverie" states.

Additional information was obtained concerning the effects of alcohol on the duration of the spiral aftereffect (SAE). There were no significant changes in the SAE scores as a result of alcohol ingestion.

Information concerning the effects of brief periods of visual fixation on the nystagmic and subjective responses to rotatory stimuli was also obtained. Brief periods of visual fixation at the start (8-sec) of a deceleration, or after (3-sec) a deceleration was terminated, shortened the duration of the primary nystagmic response while enhancing the secondary nystagmic response. Similar, though weaker, effects were noted for the sensations resulting from the rotatory stimuli. There was no conclusive evidence of an interaction between alcohol and the effects of the brief intervals of light.

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APPENDIX

TABLE 38

Blood Alcohol Levels (in mg. per 100 ml.) for Subjects in the Moderate Alcohol and High Alcohol Groups. These Subjects Received Rotatory Stimulation and Viewed the Spiral Aftereffect.

Group	Subject	Time		
		Thirty Minutes	One Hour	Four Hours
Moderate Alcohol	KB	64	64	16
	DL	59	57	15
	TD	40	66	21
	OS	42	49	18
	HS	44	51	19
	EN	52	53	23
	ET	61	63	22
	BM	25	42	22
	SO	28	44	11
	RG	31	32	19
	Mean	44.6	52.1	18.6
High Alcohol	MM	54	80	71
	TB	30	79	71
	DM	43	104	68
	MMc	80	130	86
	LA	74	102	68
	JS	90	81	53
	DK	72	78	56
	JR	46	68	60
	PM	82	84	58
	TL	71	94	56
	Mean	64.2	90	64.7

TABLE 39

Blood Alcohol Levels (in mg. per 100 ml.) for Subjects in the Moderate Alcohol and High Alcohol Groups. These Subjects Received Caloric Irrigations and Optokinetic Stimulation.

Group	Subject	Time		
		Thirty Minutes	One Hour	Four Hours
Moderate Alcohol	MD	16	28	8
	SS	35	32	6
	PM	43	32	5
	KS	40	34	8
	JW	57	49	17
	TR	63	54	9
	RH	14	24	8
	EM	49	39	10
	BP	59	57	16
	RK	40	35	0
	Mean	41.6	38.4	8.7
High Alcohol	JJ	58	99	72
	BS	62	71	64
	MB	102	102	63
	TW	108	108	82
	AK	56	72	58
	JG	90	94	54
	DB	86	102	48
	CC	59	75	64
	BR	43	71	68
	WC	45	75	75
	Mean	70.9	86.9	64.8

TABLE 40

Results of the Analysis of Variance for the Slow Phase Nystagmus (in Degrees) Resulting From the 12-sec Accelerations of $5^{\circ}/sec^{2}$.

Source	df	MS	F
Groups (G)	2	89,062.40	0.62
Subject/Group G(S)	27	143,362.74	
Instruction (I)	2	850,919.48	32.12***
I x G	4	1,638.12	0.06
I x G(S)	54	26,494.86	
Trials (T)	2	251,793.73	15.98***
T x G	4	112,697.79	7.15**
T x G(S)	54	15,759.56	
I x T	4	5,424.32	0.72
I x T x G	8	4,178.30	0.56
I x T x G(S)	108	7,486.04	

* $p < .05$
** $p < .01$
*** $p < .001$

TABLE 41

Results of the Analysis of Variance for the Number of Nystagmic Eye Movements Resulting From the 12-sec Accelerations of $5°/sec^2$.

Source	df	MS	F
Groups (G)	2	4956.49	1.85
Subject/Group G(S)	27	2682.92	
Instruction (I)	2	2290.69	6.67**
I x G	4	157.20	0.46
I x G(S)	54	343.57	
Trials (T)	2	6878.22	28.14***
T x G	4	2496.87	10.21***
T x G(S)	54	244.47	
I x T	4	31.57	0.35
I x T x G	8	52.22	0.58
I x T x G(S)	108	89.72	

* $p < .05$
** $p < .01$
*** $p < .001$

TABLE 42

Results of the Analysis of Variance for the Peak Velocity of the Slow Phase Nystagmus Resulting from the 12-sec Accelerations of $5°/sec^2$.

Source	df	MS	F
Groups (G)	2	751.42	1.40
Subject/Group G(S)	27	537.72	
Instruction (I)	2	1,674.10	15.94***
I x G	4	58.64	0.56
I x G(S)	54	105.00	
Trials (T)	2	838.48	8.44***
T x G	4	259.40	2.61*
T x G(S)	54	99.29	
I x T	4	16.19	0.45
I x T x G	8	26.67	0.74
I x T x G(S)	108	35.80	

* $p < .05$
** $p < .01$
*** $p < .001$

TABLE 43

Results of the Analysis of Variance for the Duration of the Slow Phase Nystagmus Resulting From the 12-sec Accelerations of $5°/sec^2$.

Source	df	MS	F
Groups (G)	2	1028.69	2.98
Subject/Group G(S)	27	345.76	
Instruction (I)	2	2506.36	36.02***
I x G	4	103.14	1.48
I x G(S)	54	69.57	
Trials (T)	2	2082.93	33.30***
T x G	4	623.77	9.97***
T x G(S)	54	62.56	
I x T	4	21.05	0.50
I x T x G	8	9.10	0.22
I x T x G(S)	108	42.15	

* $p < .05$
** $p < .01$
*** $p < .001$

TABLE 44

Total Number of Degrees of Subjective Displacement Resulting From the 12-sec Deceleration of $5^{o}/sec^{2}$. The Room Lights Were on for 8-sec at the Start of the Deceleration. Negative Values Refer to Secondary Sensations (CW).

Group	Subject	Pre	Post I	Post II
Control	BG	0	270	0
	CT	-990	- 90	360
	RH	+ 90	90	90
	DB	-450	0	-1440
	SH	+270	-900	+ 900
	Mean	-216	-126	- 18
Moderate Alcohol	BM	900	540	- 450
	RG	-360	-270	0
	KB	-360	- 90	- 630
	EN	-900	-450	- 720
	OS	+180	180	90
	Mean	-108	- 18	- 342
High Alcohol	TL	-180	180	- 450
	MM	270	180	360
	DM	270	360	360
	LA	-720	-540	0
	JS	-990	-990	- 180
	Mean	-270	-162	18

TABLE 45

Total Number of Degrees of Subjective Displacement Resulting From the 12-sec Deceleration of $5^{o}/sec^{2}$. Negative Values Refer to Sensations in the Secondary (CW) Direction.*

		Pre		Post I		Post II	
Group	Subject	Before	After	Before	After	Before	After
Control	MD	90	0	90	0	450	0
	BG	270	0	360	0	450	0
	MC	450	- 90	540	- 90	360	-540
	FS	360	-450	450	0	450	270
	TS	90	0	90	-270	90	-450
	Mean	252	-108	360	- 72	360	-144
Moderate Alcohol	SO	360	0	540	0	360	0
	DL	270	0	270	0	180	0
	TD	450	0	360	0	450	0
	ET	270	0	180	0	270	0
	HS	270	180	630	0	630	0
	Mean	340	36	396	0	378	0
High Alcohol	PM	270	0	360	0	270	0
	TB	360	0	270	0	450	0
	MM	180	0	0	0	270	0
	JR	360	0	360	0	90	0
	DK	450	0	270	0	360	0
	Mean	324	0	252	0	288	0

* The room lights were on for 3-sec after the end of the deceleration. "Before" refers to the sensation experienced prior to the onset of the lights while "after" refers to the sensations experienced after the end of the 3-sec interval of light.

TABLE 46

Results of the Analysis of Variance for the Slow Phase Nystagmus (in Degrees) resulting From the Caloric Irrigations.

Source	df	MS	F
Groups (G)	2	288,282.76	0.11
Subject/Group G (S)	27	2,794,422.73	
Temperature (Te)	1	86,115.27	0.30
Te x G	2	27,914.68	0.10
Te x G (S)	27	286,573.58	
Instruction (I)	1	3,687,501.59	41.52***
I x G	2	371,901.16	4.19*
I x G (S)	27	88,812.57	
Trials (Tr)	2	4,077,802.16	16.33***
Tr x G	4	562,830.97	2.25
Tr x G (S)	54	249,730.15	
Te x I	1	37,181.44	0.68
Te x I x G	2	3,545.72	0.07
Te x I x G (S)	27	54,317.62	
Te x Tr	2	38,481.69	0.44
Te x Tr x G	4	48,972.69	0.56
Te x Tr x G (S)	54	87,815.88	
I x Tr	2	5,169.19	0.08
I x Tr x G	4	53,967.56	0.87
I x Tr x G (S)	54	61,687.92	
I x Tr x Te	2	164,657.44	2.82
I x Tr x Te x G	4	7,443.47	0.13
I x Tr x Te x G (S)	54	58,385.37	

* $p < .05$
** $p < .01$
*** $p < .001$

TABLE 47

Results of the Analysis of Variance for the Number of Nystagmic Eye Movements Resulting From the Caloric Irrigations.

Source	df	MS	F
Groups (G)	2	118,169.21	2.49
Subject/Group G (S)	27	47,486.40	
Temperature (Te)	1	3,198.13	0.97
Te x G	2	1,048.66	0.32
Te x G (S)	27	3,307.82	
Instructions (I)	1	9,312.66	6.98*
I x G	2	1,835.96	1.38
I x G (S)	27	1,334.74	
Trials (Tr)	2	49,153.98	17.10***
Tr x G	4	12,417.60	4.32*
Tr x G (S)	54	2,874.71	
Te x I	1	.63	0.00
Te x I x G	2	545.28	1.04
Te x I x G (S)	27	526.42	
Te x Tr	2	3,113.88	1.52
Te x Tr x G	4	764.85	0.37
Te x Tr x G (S)	54	2,047.75	
I x Tr	2	1,494.75	1.99
I x Tr x G	4	713.76	0.95
I x Tr x G (S)	54	752.74	
I x Tr x Te	2	644.00	0.86
I x Tr x Te x G	4	759.73	1.02
I x Tr x Te x G (S)	54	747.87	

* $p < .05$
** $p < .01$
*** $p < .001$

TABLE 48

Results of the Analysis of Variance for the Peak Velocity (deg/sec) of the Slow Phase Nystagmus Resulting From the Caloric Irrigations.

Source	df	MS	F
Group (G)	2	397.96	0.60
Subject/Group G (S)	27	665.20	
Temperature (Te)	1	336.98	2.96
Te x G	2	1.95	0.02
Te x G (S)	27	113.79	
Instruction (I)	1	148.87	6.67*
I x G	2	32.31	1.45
I x G (S)	27	22.32	
Trials (Tr)	2	169.89	4.02*
Tr x G	4	103.72	2.45
Tr x G (S)	54	42.27	
Te x I	1	31.74	1.12
Te x I x G	2	7.81	0.28
Te x I x G (S)	27	28.40	
Te x Tr	2	4.05	0.11
Te x Tr x G	4	27.27	0.74
Te x Tr x G (S)	54	36.84	
I x Tr	2	4.69	0.24
I x Tr x G	4	10.68	0.55
I x Tr x G (S)	54	19.50	
I x Tr x Te	2	18.68	0.63
I x Tr x Te x G	4	13.26	0.45
I x Tr x Te x G (S)	54	29.51	

* $p < .05$
** $p < .01$
*** $p < .001$

TABLE 49

Results of the Analysis of Variance for the Duration of the Slow Phase Nystagmus Resulting From the Caloric Irrigations.

Source	df	MS	F
Groups (G)	2	27,691.27	3.43*
Subject/Group G (S)	27	8,072.70	
Temperature (Te)	1	4,185.43	2.45
Te x G	2	545.22	0.32
Te x G (S)	27	1,710.74	
Instruction (I)	1	9,338.11	6.23*
I x G	2	590.49	0.39
I x G (S)	27	1,499.19	
Trials (Tr)	2	48,650.35	30.96***
Tr x G	4	1,240.43	0.79
Tr x G (S)	54	1,571.61	
Te x I	1	1,066.06	0.77
Te x I x G	2	205.32	0.15
Te x I x G (S)	27	1,399.11	
Te x Tr	2	625.22	0.46
Te x Tr x G	4	821.33	0.60
Te x Tr x G (S)	54	1,359.09	
I x Tr	2	2,343.99	2.34
I x Tr x G	4	956.86	0.96
I x Tr x G (S)	54	1,000.31	
I x Tr x Te	2	272.68	0.26
I x Tr x Te x G	4	1,722.94	1.65
I x Tr x Te x G (S)	54	1,041.64	

* $p < .05$
** $p < .01$
*** $p < .001$

TABLE 50

Results of the Analysis of Variance for the Intensity Ratings of the Subjects' "Vertigo" Sensations Resulting From the Caloric Irrigations.

Source	df	MS	F
Groups (G)	2	2,475.16	0.59
Subject/Group G (S)	27	4,173.80	
Temperature (Te)	1	30,673.10	8.83**
Te x G	2	3,643.88	1.05
Te x G (S)	27	3,473.56	
Instruction (I)	1	35.47	0.05
I x G	2	829.75	1.20
I x G (S)	27	691.84	
Trials (Tr)	2	46,341.70	21.67***
Tr x G	4	2,551.66	1.19
Tr x G (S)	54	2,138.57	
Te x I	1	11.75	0.03
Te x I x G	2	415.11	1.11
Te x I x G (S)	27	372.60	
Te x Tr	2	9,481.37	6.81**
Te x Tr x G	4	1,121.43	0.81
Te x Tr x G (S)	54	1,392.76	
I x Tr	2	411.69	0.82
I x Tr x G	4	454.07	0.90
I x Tr x G (S)	54	502.17	
I x Tr x Te	2	900.13	2.69
I x Tr x Te x G	4	222.32	0.66
I x Tr x Te x G (S)	54	335.21	

* $p < .05$
** $p < .01$
*** $p < .001$

TABLE 51

Results of the Analyses of Variance for the Slow Phase Nystagmus Resulting From Optokinetic Stimulation. Stimuli Were of 15 or of 60 Seconds Duration. The Sources of Variance Are Designated as T (Trial) and E (Error).

				Slow Phase Displacement (Degrees)			
				Lights On		Lights Off	
Group	Stim. (Sec)		df	Mean Sq.	F	Mean Sq.	F
Control	15	T	2	234.74	0.38	18.18	0.03
		E	27	612.41		520.68	
	60	T	2	561.51	0.05	3256.00	0.36
		E	27	11048.68		8927.99	
Moderate Alcohol	15	T	2	854.34	0.88	677.18	0.84
		E	27	970.57		810.13	
	60	T	2	37559.15	3.46*	47341.68	3.57*
		E	27	10845.42		13269.40	
High Alcohol	15	T	2	6921.09	5.45**	8362.71	5.26*
		E	27	1270.64		1589.55	
	60	T	2	109287.52	4.00*	148986.06	4.12*
		E	27	27353.98		36188.42	

* $p < .05$
** $p < .01$

TABLE 52

Results of the Analyses of Variance for Number of Nystagmic Eye Movements Resulting From Optokinetic Stimulation. Stimuli Were of 15 or of 60 Seconds Duration. The Sources of Variance Are Designated as T (Trial) and E (Error).

				Frequency			
				Lights On		Lights Off	
Group	Stim. (Sec)		df	Mean Sq.	F	Mean Sq.	F
Control	15	T	2	7.60	0.17	3.70	0.12
		E	27	43.44		30.50	
	60	T	2	58.38	0.12	88.66	0.30
		E	27	495.46		295.53	
Moderate Alcohol	15	T	2	77.70	0.95	30.57	0.44
		E	27	81.72		68.83	
	60	T	2	1032.70	1.02	1099.13	1.17
		E	27	1015.12		935.60	
High Alcohol	15	T	2	403.45	7.29**	565.90	8.27**
		E	27	55.34		68.40	
	60	T	2	10107.35	8.27**	12137.34	8.46**
		E	27	1222.70		1434.38	

* $p < .05$
** $p < .01$