TECHNICAL REPORT STANDARD TITLE PAGE

1. Report No.	2. Government Accession No.	3. Recipient's Catalog No.
FAA-AM-71-16		

4. Title and Subtitle	5. Report Date
ALCOHOL AND DISORIENTATION-RELATED RESPONSES. II. NYSTAGMUS AND "VERTIGO" DURING ANGULAR ACCELERATION	April 1971
	6. Performing Organization Code
7. Author(s)	8. Performing Organization Report No.
David J. Schroeder, Ph.D.	
9. Performing Organization Name and Address	10. Work Unit No.
FAA Civil Aeromedical Institute P. O. Box 25082 Oklahoma City, Oklahoma 73125	11. Contract or Grant No.
	13. Type of Report and Period Covered
12. Sponsoring Agency Name and Address	OAM Report
Office of Aviation Medicine Federal Aviation Administration 800 Independence Ave., S. W. Washington, D. C. 20590	14. Sponsoring Agency Code

15. Supplementary Notes

This study was conducted under tasks AM-B-69-PSY-10, AM-A-70-PSY-10, and AM-A-71-PSY-27.

16. Abstract

The integrity of the visual and vestibular systems is important in the maintenance of orientation during flight. Although alcohol is known to affect the vestibular system through the development of a positional alcohol nystagmus, information concerning the effects of alcohol on "vertigo" and eye-movement (nystagmus) responses to angular acceleration is contradictory. Several investigators have reported that alcohol enhances these responses, while others have reported suppressive effects. This study was designed to investigate the effect of alcohol ingestion on both "vertigo" and nystagmic responses to angular stimulation. Responses were obtained (a) with and without visual fixation, and (b) with the alertness of the subjects controlled.

When recorded in total darkness, the nystagmic response to rotatory stimulation was suppressed by the alcohol. When visual fixation was allowed, a high-frequency low-amplitude nystagmus to rotation was obtained following alcohol ingestion; there was little or no response prior to drinking. This apparent enhancement of the response was not due to an increase in vestibular sensitivity but, rather, to the suppressive effect of alcohol on the ability of the subject to maintain adequate visual fixation. "Vertigo" sensations resulting from the rotatory stimuli evidenced only slight declines following alcohol ingestion.

17. Key Words	18. Distribution Statement
Alcohol, Nystagmus, Vestibular, Visual Fixation	Availability is unlimited. Document may be released to the National Technical Information Service, Springfield, Virginia 22151, for sale to the public.

19. Security Classif. (of this report)	20. Security Classif. (of this page)	21. No. of Pages	22. Price
Unclassified	Unclassified	27	$3.00

Form DOT F 1700.7 (8-69)

ALCOHOL AND DISORIENTATION-RELATED RESPONSES. II. NYSTAGMUS AND "VERTIGO" DURING ANGULAR ACCELERATION

The integrity of the visual and vestibular systems is important in the maintenance of orientation during flight. Although alcohol is known to affect the vestibular system through the development of a positional alcohol nystagmus, information concerning the effects of alcohol on "vertigo" and eye-movement (nystagmus) responses to angular acceleration is contradictory. Although Barany[1] noted no change in the duration of the nystagmic response following alcohol ingestion, he reported that the subjective sensations were weakened. Manz[15] found a prolonged duration of post-rotatory nystagmus, and later studies by Taschen[24 25], Schweitzer[23], and Schulte and Roth[22] all indicated that the nystagmic response was enhanced following alcohol ingestion.

Contrary to the preceding results, Forster[12] reported that alcohol depressed the nystagmic response to rotatory stimulation. Subsequent studies by Bochenek and Ormerod[3], Mizoi, Ishido, and Ohga[16], Ey[11], and Di Guinta and Rosa[10] supported the view that alcohol exerts a suppressive effect on nystagmus.

These differences in reported results might be attributed in part to the presence or absence of visual stimuli. The studies showing suppression were notably lacking in visual stimuli, while those studies reporting enhancement or no change included visual stimuli. An additional factor which may have influenced the findings concerns the effects of alertness on the nystagmic response. Studies by Collins[5 6 9] have shown that variations in alertness will alter the nystagmic responses to rotatory stimulation and that these variations can be manipulated by appropriate instructions. Under "alert" conditions the nystagmic response is of higher amplitude and of longer duration than that obtained when the subject is relaxed and in a "reverie" state.

The present study was designed to investigate the influence of alcohol on the subjective and nystagmic responses to rotatory stimulation: (a) with and without visual fixation, and (b) with the alertness of the subjects controlled by instructions. Additional information was obtained concerning the effect of alcohol on a proposed relationship between the duration of the rotation-induced turning experience and the duration of the spiral aftereffect.[17-20]

I. Method.

A. *Subjects.* Subjects were 30 male college students with no previous laboratory experience involving vestibular stimulation. They ranged from 21 to 30 years of age and were randomly placed in three groups of ten each. Of the three groups, one was designated as "high alcohol," one as "moderate alcohol," and the other served as a control group with no alcohol.

B. *Apparatus.* 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 head movements.

An eight-inch spiral, attached to a variable speed motor, provided the stimuli for the spiral aftereffect (SAE). The device was located seven feet in front of the subject in a well-lighted laboratory room. The speed of rotation was 80 rpm and the stimulus duration was 15 seconds.

Data presented here were submitted to the Psychology Department, University of Oklahoma, in partial fulfillment of the requirements for the degree of Doctor of Philosophy under the guidance of Dr. William E. Collins. The assistance of Gail Kranz, Ruth Ann Mertens, Cynthia Cochran, Carlyn Manley, and Nancy Rice in the conduct of the study, of Dr. Earl Folk and Mrs. Rosalie Melton for some of the statistical analyses, and of Dr. Delbert Lacefield and Mrs. Pat Roberts in the collection and analysis of the blood samples, is gratefully acknowledged.

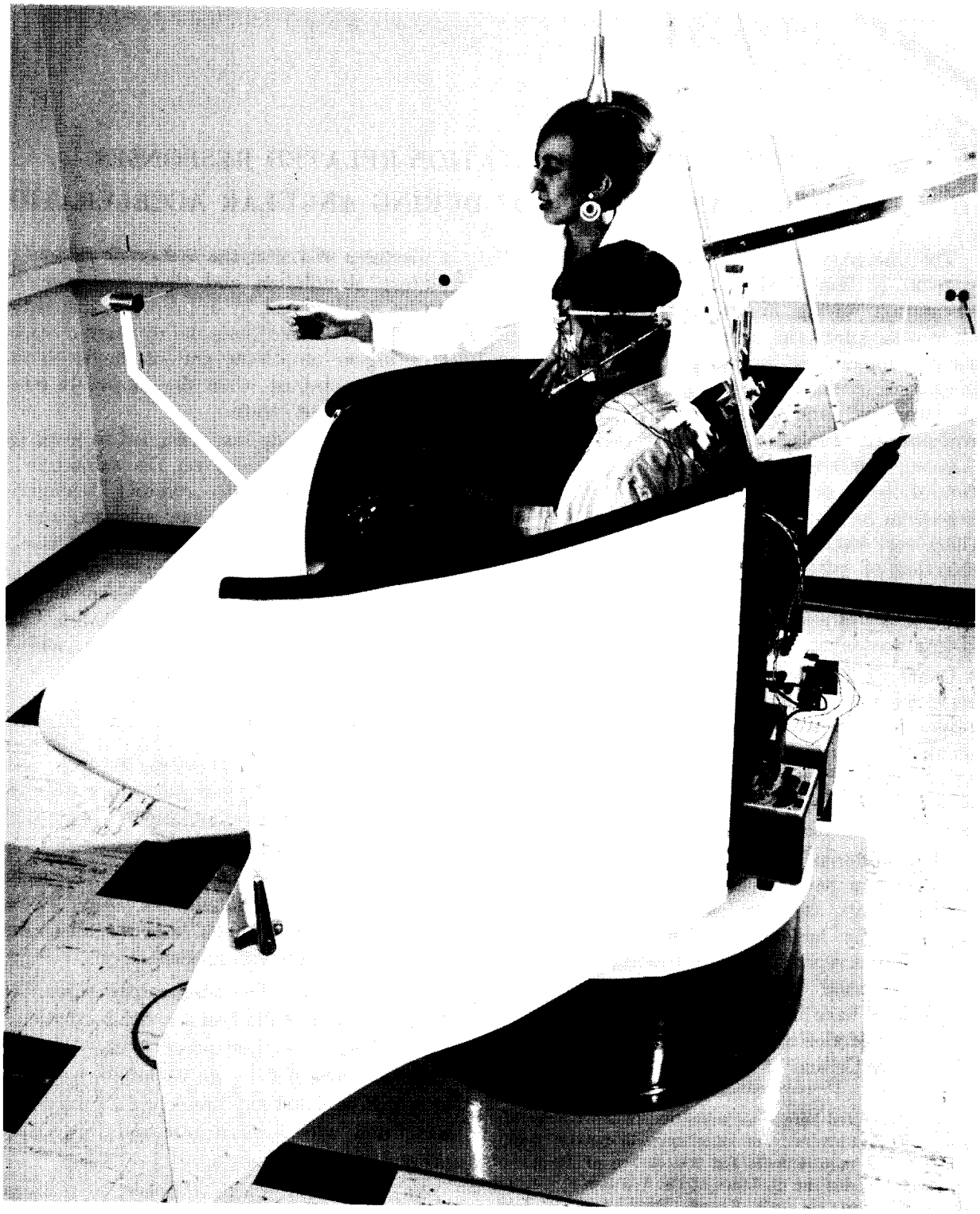

FIGURE 1. Modified Stille-Werner RS–3 chair used to administer the rotatory stimuli. The head rest and bite board were used to restrict the subjects' head movements and to keep the horizontal canals in the plane of rotation. The cross-shaped device at the front of the rotator contains the calibration 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.

C. *Recording.* Silver disc electrodes were taped by the outer canthi of the eyes to record horizontal components of eye movements. An electrode on the forehead served as a ground. Eye movements were amplified and recorded on an Offner Type T electroencephalograph using a 3-second time constant. The subjects' signals concerning their sensations of rotation were made by depression of a microswitch, and these signals were also recorded.

Eye movement calibrations were obtained by asking each subject to sweep his eyes between two small, dim, flashing lights (subtending a visual angle of 15°) attached to the front of the rotatory device. A switch, located inside the cabin, allowed the subject to turn the lights off and on in an otherwise totally dark room, thereby permitting calibrations both prior to the accelerations and during constant velocity prior to the deceleration.

II. Procedure.

The 30 subjects received identical trials during what are here termed Pre, Post I, and Post II sessions. These respective sessions were held the day before drinking, and 45 minutes and four hours after drinking.

The subjects received three practice trials of counter-clockwise (CCW) rotations in total darkness. These practice trials were used to train the subjects in the technique of estimating rotational velocity from turning sensations.[2 9 13]

A. *Pre Trials.* Following practice, the subjects received three experimental trials of clockwise (CW) rotations separated by 5-minute rest periods. The accelerations on these three Pre trials were 5°/sec² for 12 seconds followed by rotation at constant velocity (60°/sec) for three minutes. Deceleration began after an eye-movement calibration was obtained. On trials 1 and 2, a brake deceleration of 120°/ sec² for 0.5 sec was used; on trial 3 continuous deceleration at the rate of 5°/sec² was employed.

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 (MA) task in the form of a continuous division problem.[5 9] Different proplems were given for the acceleration and deceleration periods and the subject's answers were recorded (approximately 1½ minutes 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 experiences as he had during the training period. Thus, the subject signaled with a key press (KP) 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 subsequent turning experience (secondary sensation). The order in which these tasks were presented was counterbalanced among subjects.

All 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 to pursue particular lines of thought. The subject was allowed to relax and daydream for 30 seconds prior to the acceleration, during the acceleration, and throughout the period of constant velocity until a few seconds before the eye-movement calibration was required; he was then told to make the calibration eye movements and to prepare himself for deceleration. The room lights were turned on during the first eight seconds of the deceleration for five subjects in each of the three groups, and for three seconds immediately after the chair was stopped for the remaining five subjects in each group. After the lights went out, the subject was asked to signal any further turning sensations 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 signaled 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 markers attached to the walls of the room while at a standstill in illumination. The use of the room lights was designed to provide data regarding the interaction of visual and vestibular stimulation.

B. *Post Trials.* Post I and Post II rotation trials were identical to the Pre trials but were

conducted on the following day after consumption of alcohol (or of the non-alcoholic control beverage).

C. *Visual Fixation Trials.* Seven additional subjects received angular stimulation while attempting to fixate visually. The rotatory stimuli for these subjects was again a 5°/sec² acceleration for 12 seconds, three minutes at constant velocity, and a brake deceleration. The only difference involved the use of visual fixation throughout the trials. During the acceleration with the room lights off, the subject fixated on a dimly illuminated target which rotated with him. The subject fixated on the same target following the brake deceleration; however, in this instance the room lights were turned on. These trials were administered a few minutes before and 45 minutes after drinking.

D. *Alcohol Ingestion and Blood Tests.* Subjects in the moderate and high alcohol groups drank a mixture of 100-proof Smirnoff vodka and orange juice. The mixture contained either 2.5 ml or 1.25 ml of vodka per kilogram of body weight for the high and moderate alcohol groups respectively. The control group drank orange juice with a few drops of rum extract added to alter the taste somewhat. Subjects who received rotatory stimulation while fixating on visual targets consumed a mixture which contained 2.00 ml of vodka per kilogram of body weight. All subjects consumed their drinks in a 15-minute period.

Venous blood samples of from three to five ml 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 gas chromatography.

E. *Scoring.* The nystagmus was scored with respect to (a) duration or the time from the start of the stimulus to the last nystagmic beat, (b) frequency or the number of nystagmus beats, (c) slow phase displacement or the total number of degrees of eye movement in a three-second period, and (d) slow phase velocity (degrees/sec) at peak slow phase displacement. Slow phase eye displacement was determined by measuring (in millimeters) the amplitude of each nystagmic beat from slow phase peak to baseline. These measures were summed for all the beats in each three-second interval and the sum converted, using the calibrations, into degrees of slow phase eye movement. From Bodin's[4] formula, the velocity of the slow phase eye movement was calculated from the average angle of the slow phase of the nystagmus during the three-second interval where the nystagmic output was maximum.

III. Results and Discussion.

A. *Blood Alcohol Levels (BALs).* Means and standard deviations for the blood alcohol levels are presented in Table 1. The mean level for the high alcohol group after one hour was 90 mg.%, nearly twice the average value (52 mg.%) reached by the moderate alcohol group. Four hours after ingestion, the mean BAL for the moderate alcohol group was reduced to 19 mg.%, while the average for the high alcohol group (65 mg.%) was above the values reached by the moderate alcohol group at either the 30-minute or the one-hour testing session.

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		Time Since the Ingestion of Alcohol		
		Thirty Minutes	One Hour	Four Hours
Moderate Alcohol	M	44.6	52.1	18.6
	SD	14.1	10.9	3.8
High Alcohol	M	64.2	90.0	64.7
	SD	19.7	18.0	10.1

B. *Alertness.* The mean response levels under the alert conditions in the KP and MA trials were, for most trials, higher than those under the relaxed (Rev) condition (Tables 2 through 5). Analyses of variance for all measures of the nystagmic responses to the angular accelerations revealed significant effects for "instructions" (Table 6). While supporting the conclusions reached in earlier studies[5 6 9] that alerting instructions produce a higher-amplitude, longer-duration response, the present data further indicate that alerting instructions result in an increase in the frequency and the slow phase velocity of vestibular eye movements. The nystagmic tracings presented in Figure 2 depict the effects of

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			

two different alertness levels (MA and Rev) on the nystagmic response to angular accelerations. The effects of alertness are also evident in the response curves for the slow phase displacement and the frequency of the nystagmic responses (Figures 3 through 8).

C. *Nystagmus Recorded in Darkness.* Means and standard deviations for the slow phase displacement, frequency, peak velocity, and duration of the nystagmic eye movements resulting from rotatory stimuli administered in total darkness are presented in Tables 2 through 5. The

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 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	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	32.1	32.3	35.1	29.4	32.4	36.8	25.3	26.0	31.7
		SD	10.1	9.7	9.5	11.8	8.0	13.7	8.4	12.4	14.4
	Dec	M	41.4	45.2	41.8	39.1	46.1	48.5	Interrupted by visual stimulation		
		SD	17.2	16.8	18.3	16.8	14.6	16.1			
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			

results of analyses of variance for the acceleration data are presented in Table 6. Since the "trial" factor was significant for all measures, t tests were conducted to determine where the significant changes occurred.

Although most of the measures evidenced some degree of Pre-to-Post I decline for the control group, only the decline in slow phase displacement for the MA acceleration (.01 level) and the decline in velocity for the KP acceleration (.05 level) were significant. Only one of the Pre-to-Post II differences was significant (Table 7).

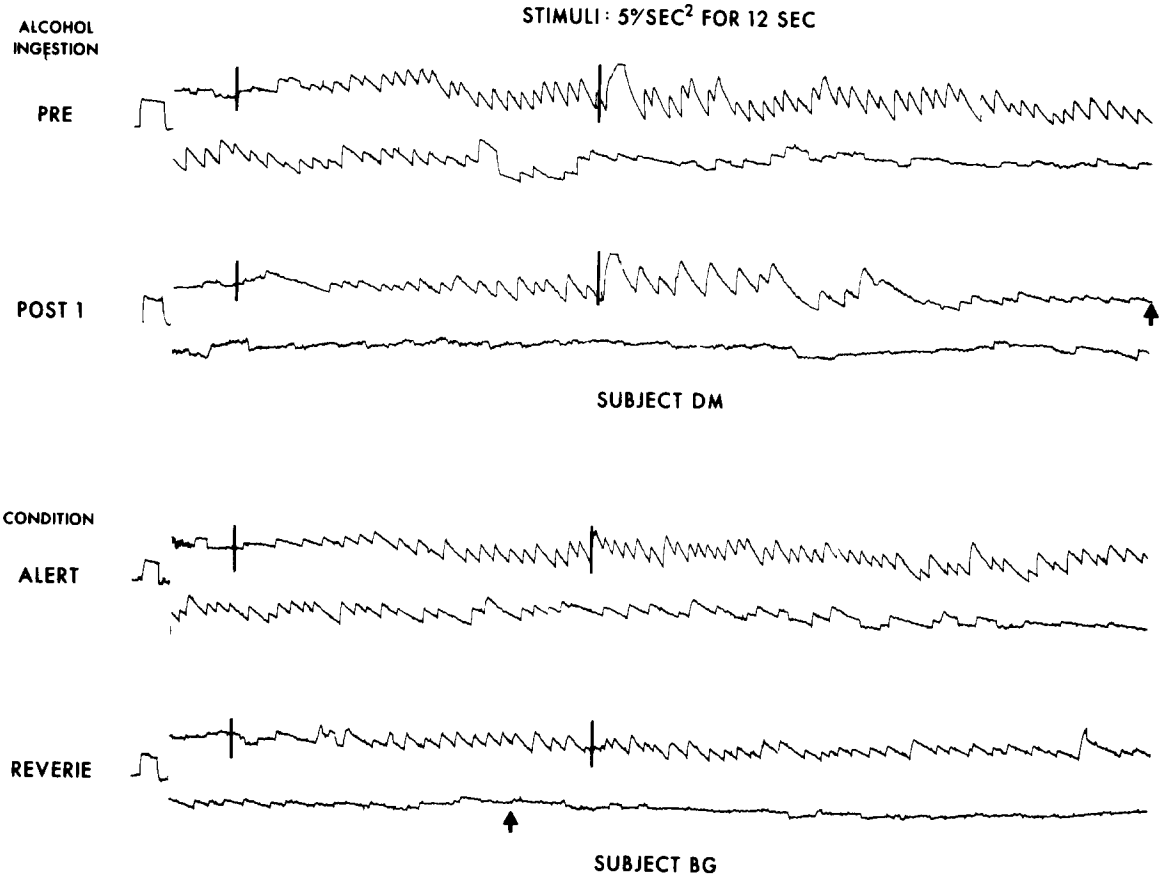

FIGURE 2. A portion of the nystagmic tracings, for two subjects, resulting from a 12-sec acceleration of 5°/sec². Calibrations (15° of eye movement) appear before each trial. The vertical bars demarcate the stimulus period and the arrows indicate the end of the primary nystagmic response. The tracings 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.

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

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

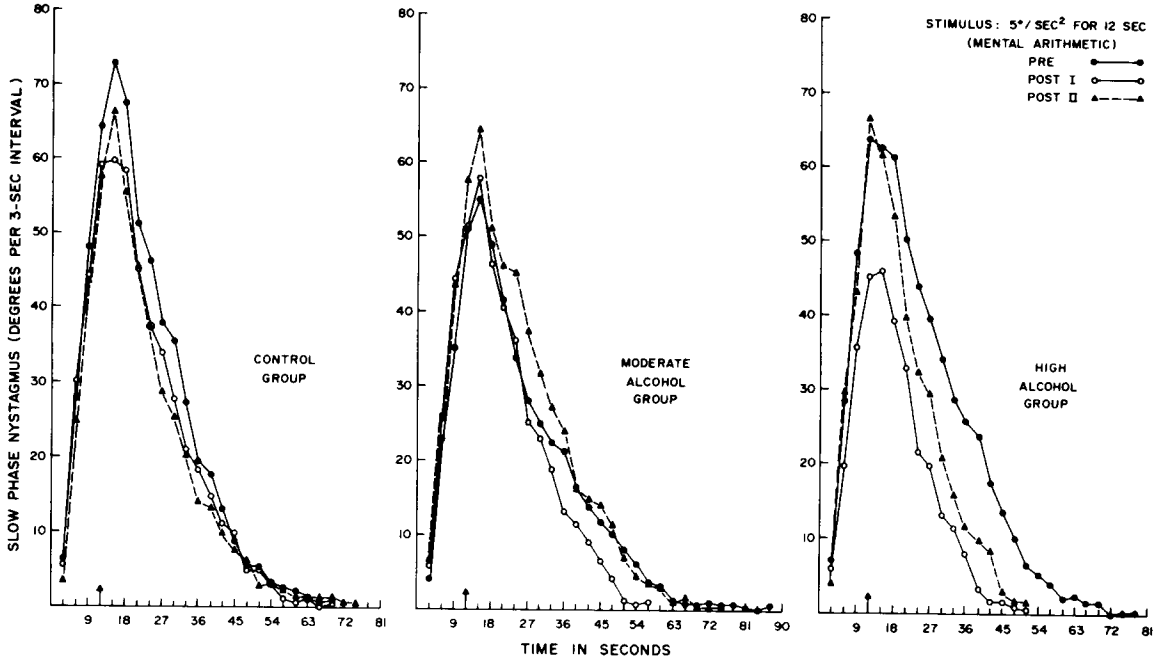

FIGURE 3. Response data for the average number of degrees of slow phase eye movement resulting from the angular accelerations (5°/sec² for 12 sec), under the Mental Arithmetic condition. Pre refers to the response recorded prior to the ingestion of alcohol, while the Post I and Post II data were obtained 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.

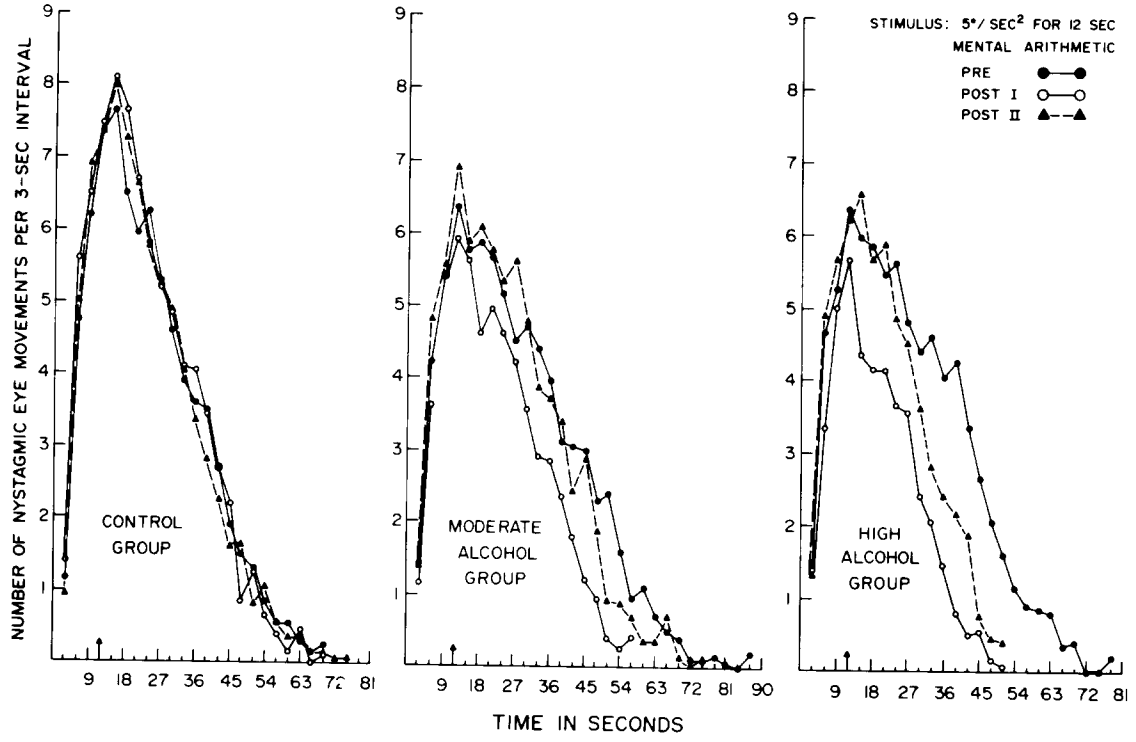

FIGURE 4. Response data for the average number of nystagmic eye movements resulting from the angular accelerations (5°/sec² for 12 sec), under the Mental Arithmetic condition. Symbols and markings are identical to those used in Figure 3.

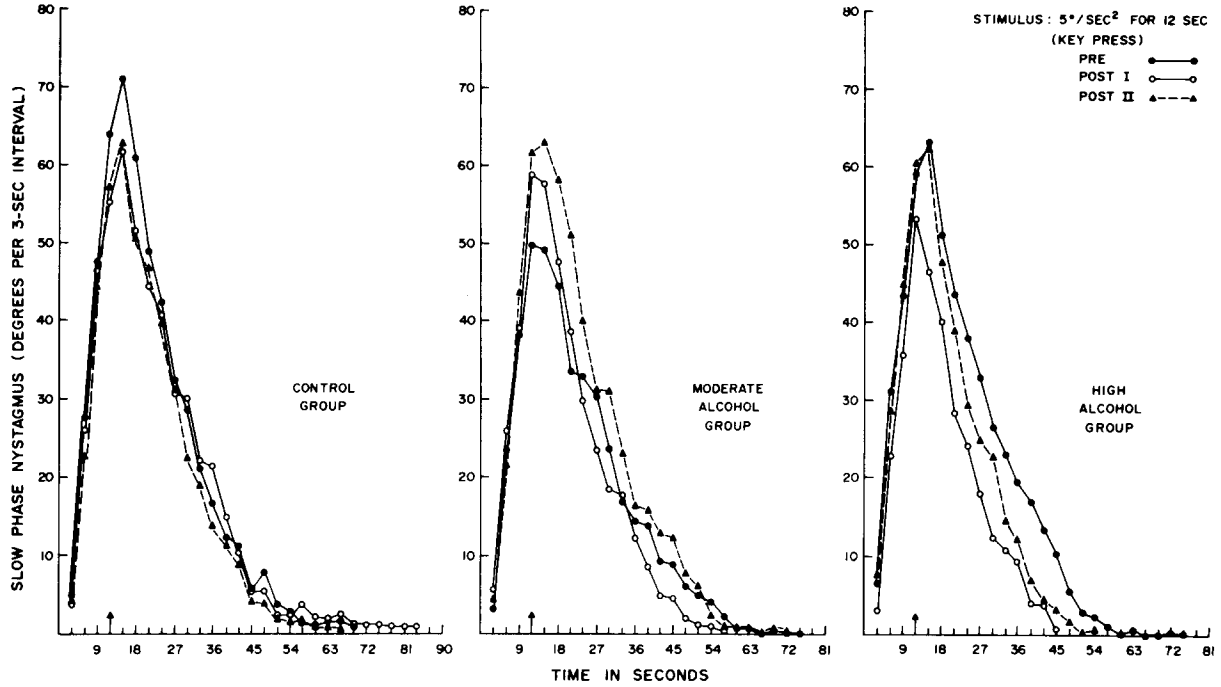

FIGURE 5. Response data for the average number of degrees of slow phase eye movement resulting from the angular accelerations (5°/sec² for 12 sec), under the Key Press condition. Symbols and markings are identical to those used in Figure 3.

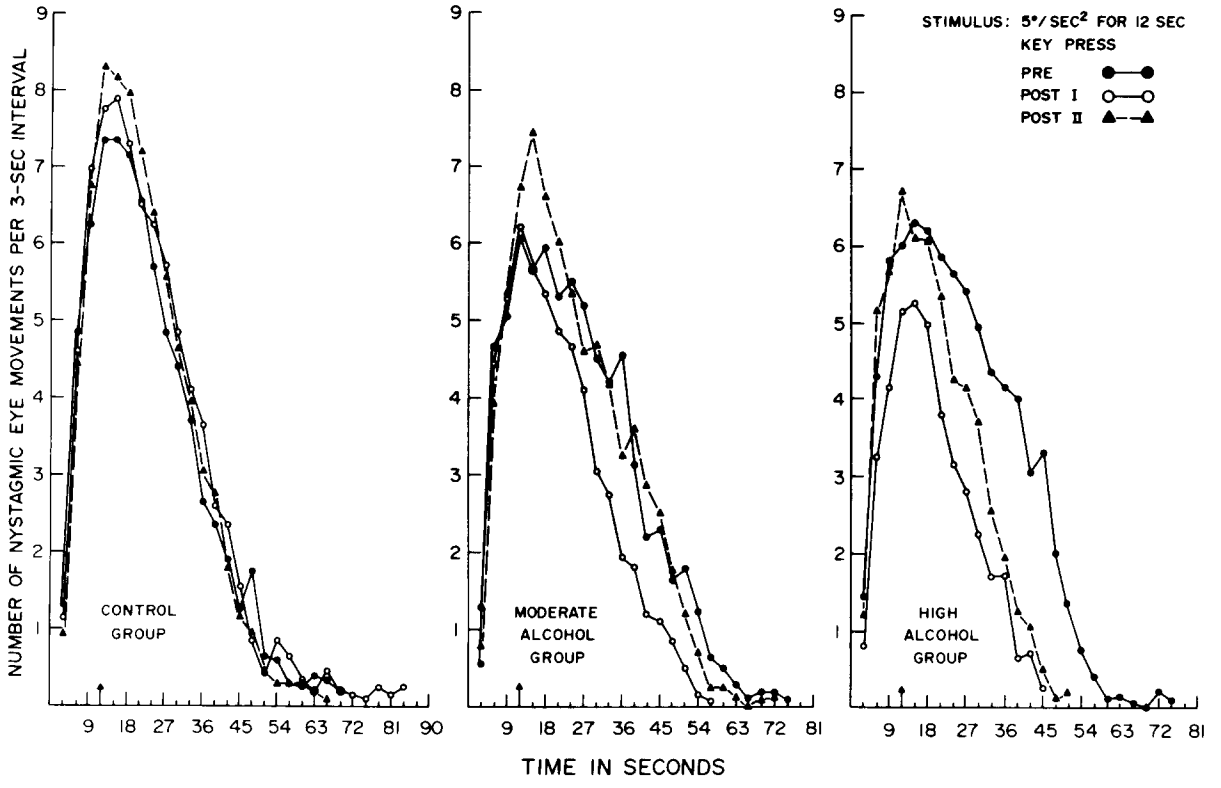

FIGURE 6. Response data for the average number of nystagmic eye movements resulting from the angular accelerations (5°/sec² for 12 sec), under the Key Press condition. Symbols and markings are identical to those used in Figure 3.

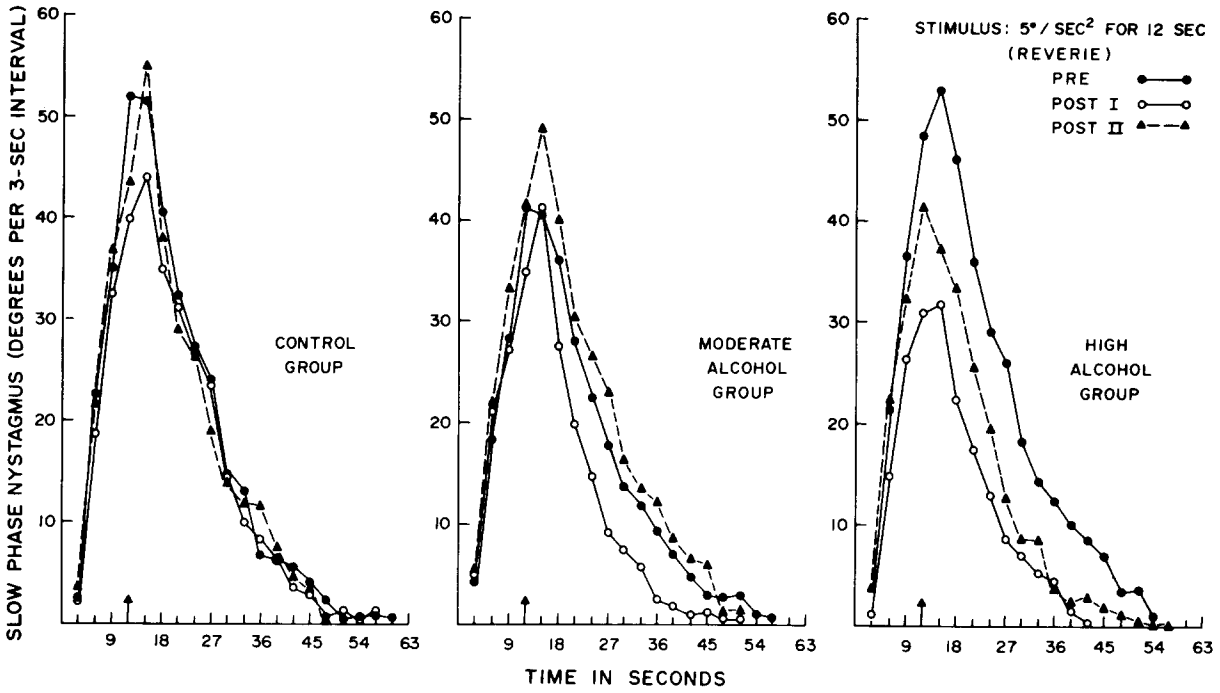

FIGURE 7. Response data for the average number of degrees of slow phase eye movement resulting from the angular accelerations (5°/sec² for 12 sec), under the Reverie (relaxed) condition. Symbols and markings are identical to those used in Figure 3.

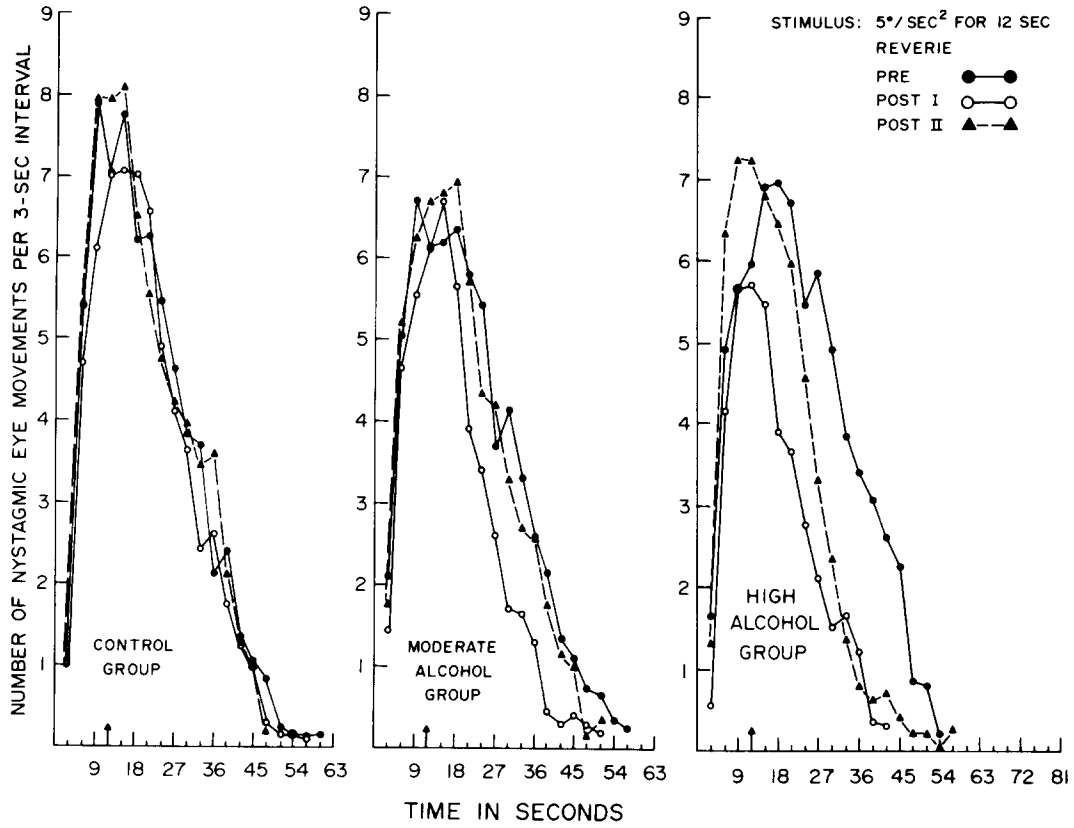

FIGURE 8. Response data for the average number of nystagmic eye movements resulting from the angular accelerations (5°/sec² for 12 sec), under the Reverie (relaxed) condition. Symbols and markings are identical to those used in Figure 3.

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.

Measure	Comparison	Condition				
		Mental Arithmetic		Key Press		Reverie
		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

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

The Pre-to-Post I declines in nystagmus for the moderate alcohol group were larger than those noted for the control group. All of the significant declines resulting from the accelerations (.05–.001 levels) occurred for the frequency and duration measures in Table 8. The Post II values were very near the Pre levels.

The suppressive effect of alcohol was most evident in the responses of the high alcohol group. With the exception of two of the "peak velocity" measures for the brake decelerations, all of the changes in slow phase displacement, frequency, peak velocity, and duration were statistically significant (.05–.001 levels). Even though there was considerable recovery at the Post II testing session, most of the frequency and duration scores were still significantly below their Pre levels (Table 9).

Thus, alcohol ingestion suppresses the nystagmic response to angular stimuli when the re-

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.

Measure	Comparison	Condition				
		Mental Arithmetic		Key Press		Reverie
		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.

Measure	Comparison	Condition				
		Mental Arithmetic		Key Press		Reverie
		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

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

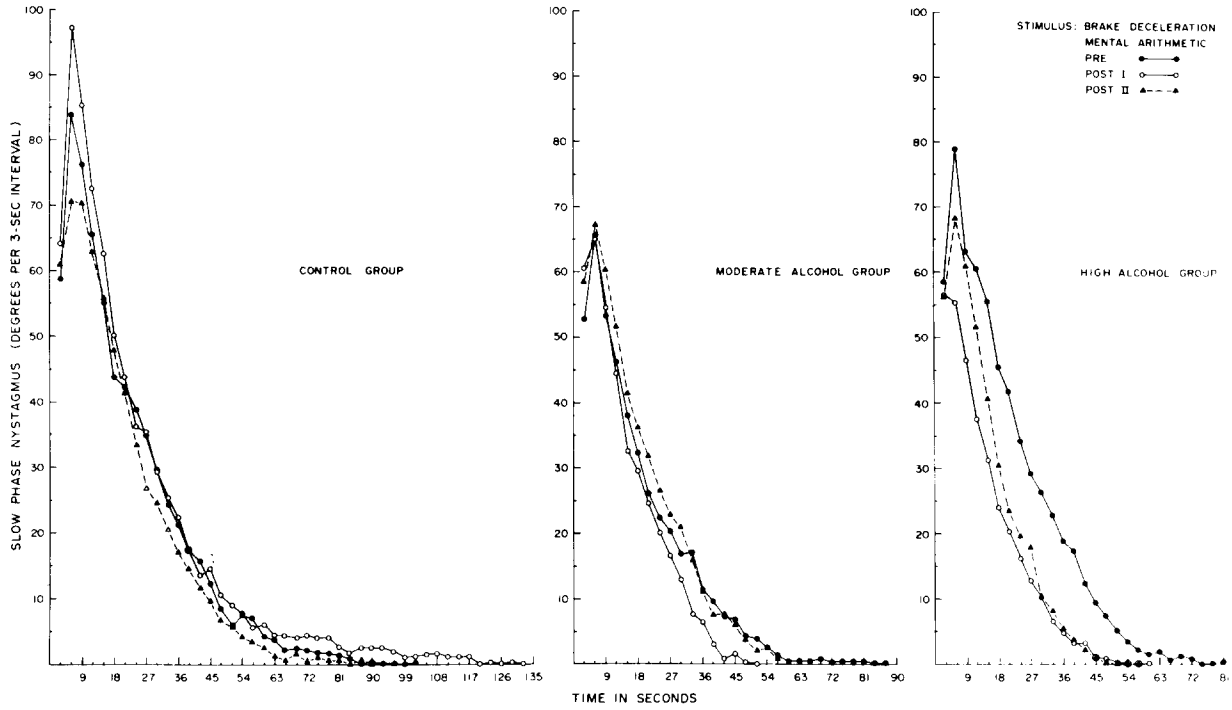

FIGURE 9. Response data for the average number of degrees of slow phase nystagmus resulting from the brake decelerations, under the Mental Arithmetic condition. Since the deceleration (from 60°/sec) was very rapid (approximately 0.5 sec), the values are plotted from the start of the deceleration. Symbols and markings are identical to those used in Figure 3.

sponse is recorded in total darkness. The extent of the suppression appears related to the amount of alcohol ingested; i.e., the high alcohol group evidenced larger declines than the moderate alcohol group. These effects of alcohol are apparent in the nystagmus response curves for the

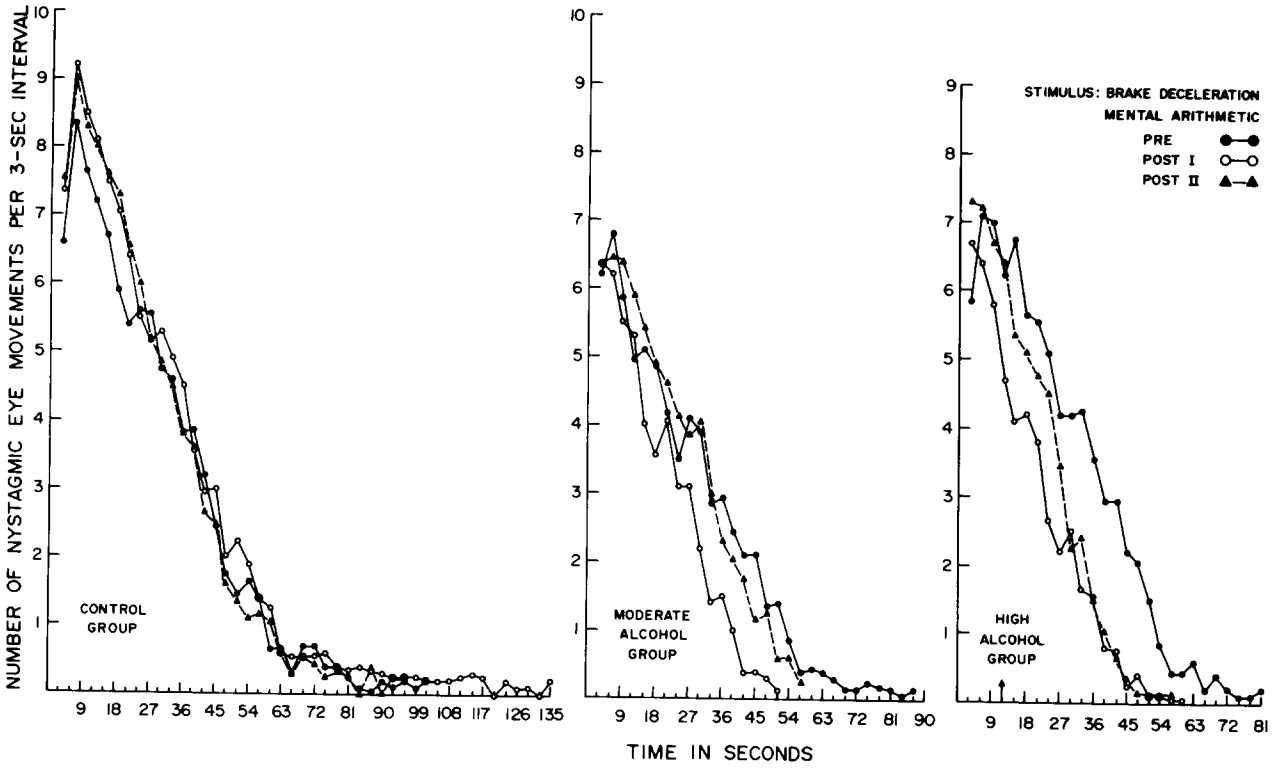

FIGURE 10. Response data for the average number of nystagmic eye movements resulting from the brake decelerations under the Mental Arithmetic condition. Symbols and markings are identical to those used in Figure 3.

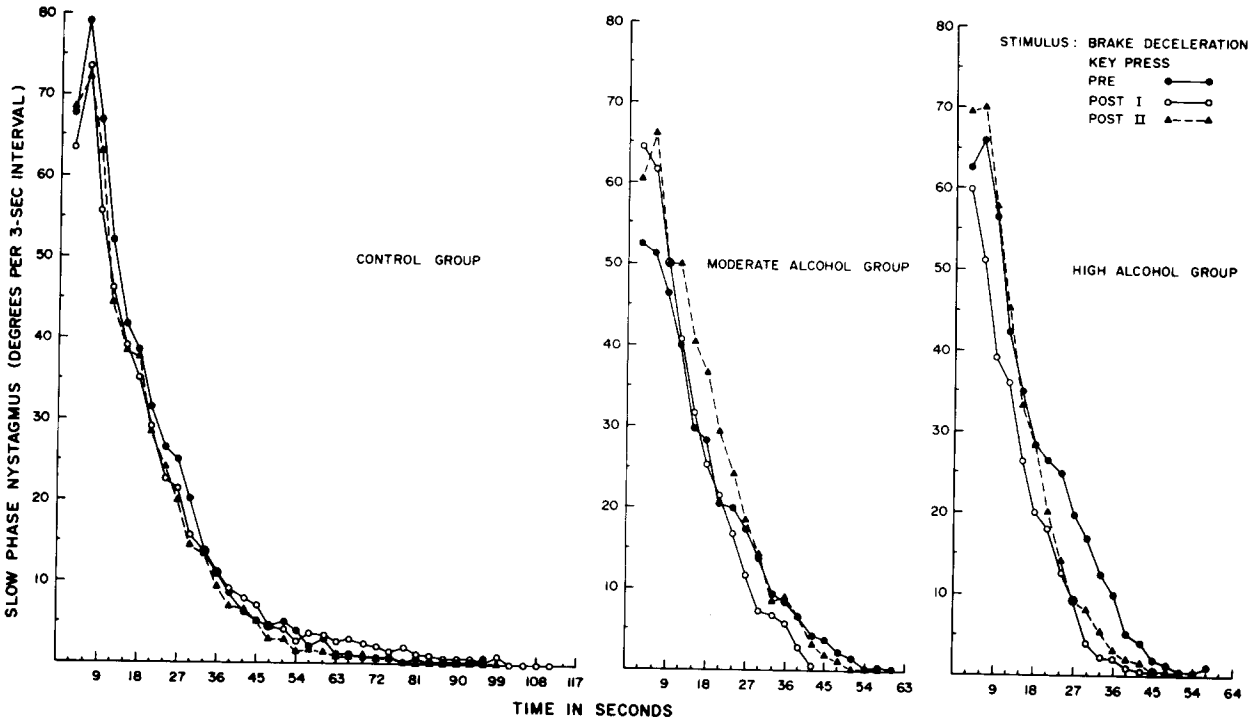

FIGURE 11. Response data for the average number of degrees of slow phase nystagmus resulting from the brake decelerations under the Key Press condition. Symbols and markings are identical to those used in Figure 3.

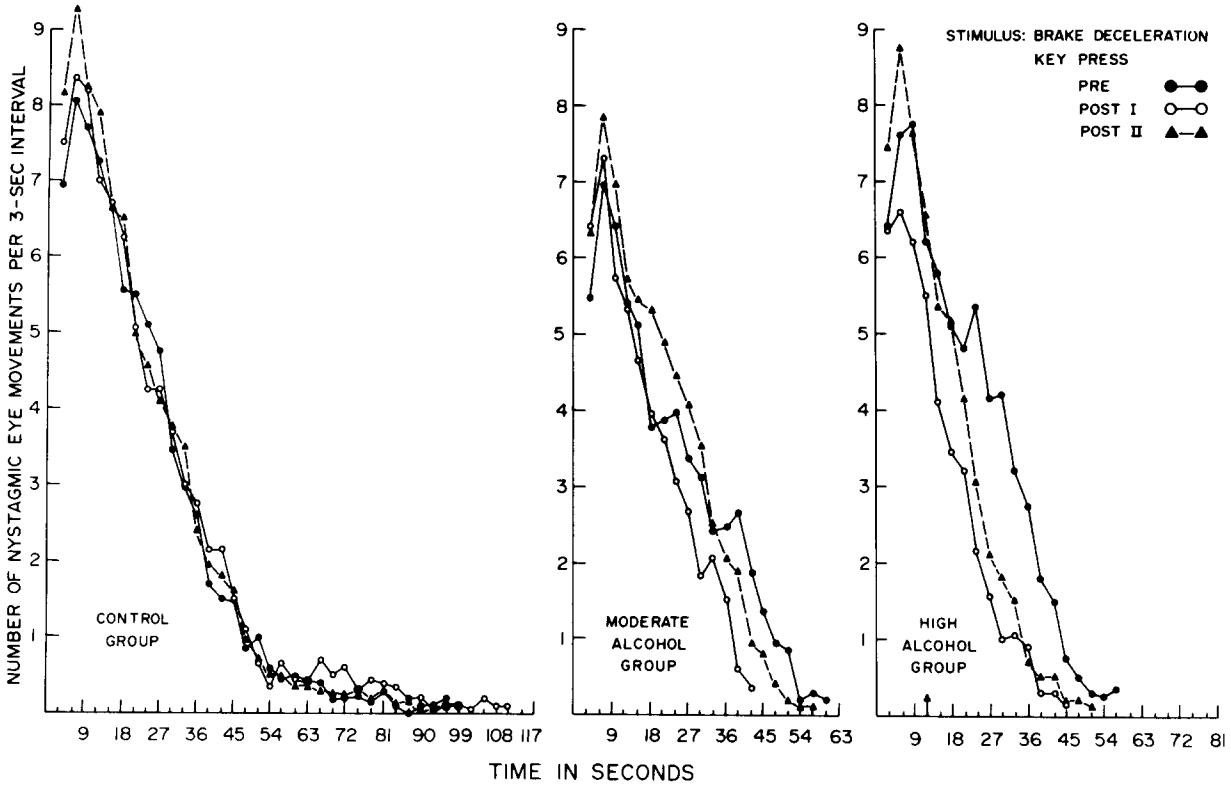

FIGURE 12. Response data for the average number of nystagmic eye movements resulting from the brake decelerations under the Key Press condition. Symbols and markings are identical to those used in Figure 3.

various stimuli (Figures 3 through 8) and in the nystagmus tracings presented in Figure 2.

Changes in the nystagmic responses from the brake decelerations, following alcohol ingestion, were similar to those noted for the accelerations (Tables 2 through 5 and Figures 9 through 12).

These data support the earlier studies[3 10 11 12 16] which showed that alcohol ingestion suppressed the nystagmic response to rotatory stimuli. However, these results fail to support other investigations[22 23 24 25] where alcohol enhanced vestibular nystagmus.

D. *Resolution of Conflicting Results from Previous Studies.* Data from seven additional subjects who received rotatory stimulation while fixating on visual targets provide a basis for resolving conflicting reports in the literature. Tracings from a subject's nystagmic response to the acceleration while fixating on the target lights in an otherwise dark room, and from the brake decelerations while fixating on the target with the room lights on, are presented in Figures 13 and 14. It is apparent from both figures that the subject was able to suppress the recorded nystagmic response by visual fixation when alcohol was not involved. However, following the ingestion of alcohol, a low-amplitude, high-frequency nystagmus was elicited by both stimulus rates.

What was reported in several previous studies as an enhanced vestibular response to acceleratory stimuli following the ingestion of alcohol can now be explained by the interference of alcohol with the subject's ability to maintain visual fixation. The inability to fixate permits a more vigorous nystagmic response to manifest itself. This interference by alcohol was also apparent in the subjects' reports concerning their attempts to fixate; greater effort and more time was required to bring the target into focus under the alcohol condition. (Similar effects have been obtained using caloric vestibular stimulation.)[21] Data from a study by Jatho[14] offer additional support to this view that alcohol interferes with the visual fixation mechanism. Jatho[14] reported that recovery movements of the eyes following head shaking took a considerably longer time after the ingestion of alcohol.

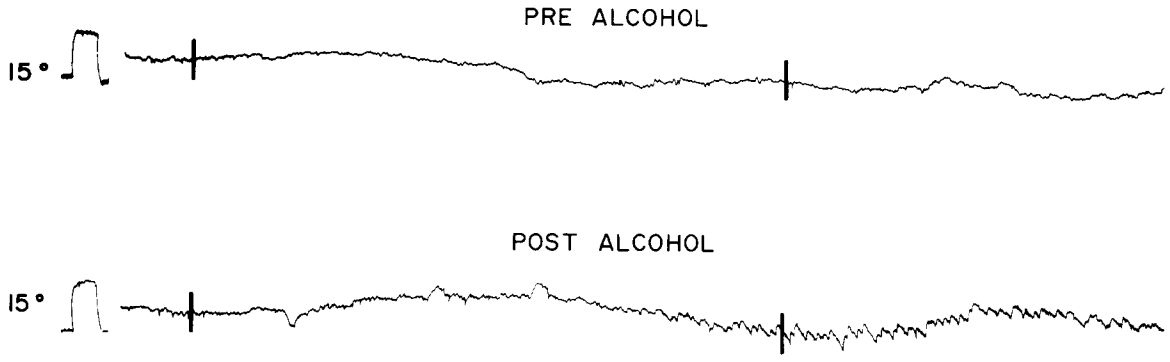

FIGURE 13. A portion of the tracings of the nystagmic response of a subject to a 12-sec acceleration of 5°/sec² during visual fixation of a dimly lit target-light. The vertical bars indicate the beginning and end of the stimulus. The calibration, representing 15° of eye movement, appears before each trial. The subject's response was recorded before and 45 min after the ingestion of 2 ml 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.

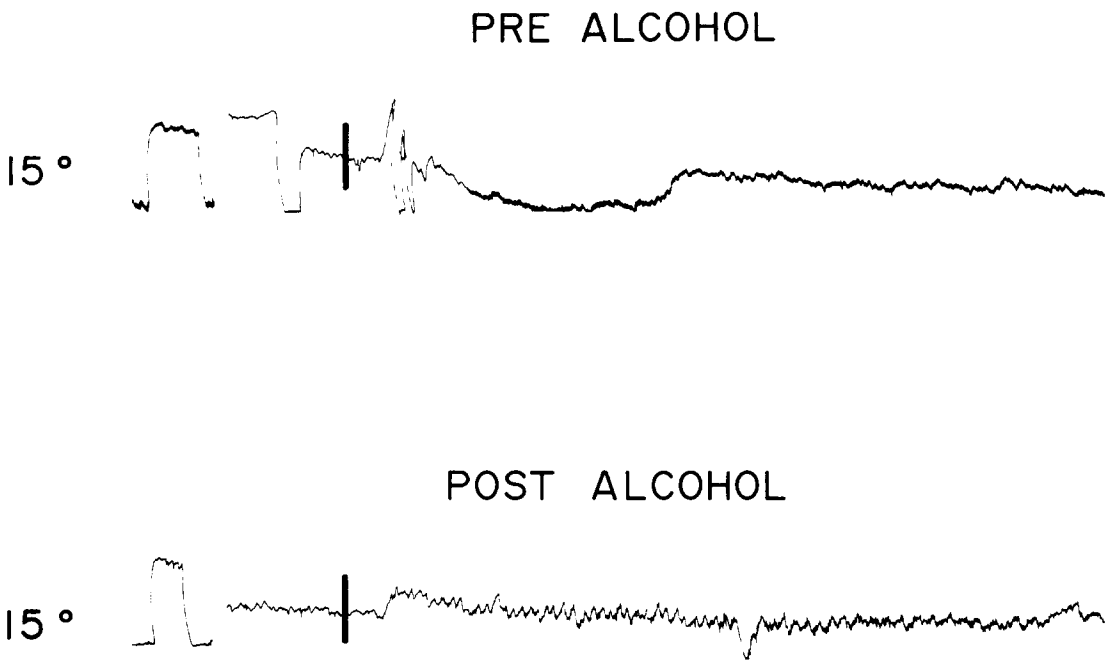

FIGURE 14. A portion of the tracings of the nystagmic response of a subject to a brake deceleration, following two min of constant velocity at 60°/sec. The vertical bar indicates the start of the deceleration and the point where the room lights were turned on. The subject was instructed to fixate on 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 13.

E. *Subjective Data from the Accelerations and Decelerations in Darkness.* Means and standard deviations for the total displacement (in degrees) and duration of the rotatory sensations resulting from the accelerations, and for the duration of the rotatory sensations resulting from the decelerations are presented in Tables 10 and 11. Response curves for the subjects' estimations of turning velocity during the accelerations are presented in Figure 15. Analyses of variance (Table 12) were applied to these data.

TABLE 10.—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 Alcohol	M	1323	1224	1287
	SD	511.0	728.9	578.7
High Alcohol	M	1107	945	918
	SD	395.8	360.6	257.4

1. Accelerations. Pre to Post I declines in mean experienced velocity and duration of the turning sensations for the moderate and the high alcohol groups were larger than those for the control group. Although the largest declines occurred for the high alcohol group, the F ratios for these data were not significant (Table 12). Even though the Post II means for the velocity and duration measures of the rotatory sensations evidenced some recovery from the Post I level, they were still below Pre levels.

2. Decelerations. Results of the analysis of variance (Table 12) indicated that changes across trials in the duration of the turning sensations resulting from the decelerations were significant. The Pre to Post I declines in the durations, according to t tests, were significant for both alcohol groups but not for the control group (Table 13). None of the Pre-Post II differences were significant. That the declines in the duration measures were significant for the decelerations, but not for the accelerations, may be due to the fact that, during the acceleration, the subjects were required to make signals concerning the velocity of their sensations between the start and end of their turning sensations, whereas they needed only to signal the start and end of their turning experience during the deceleration. The additional signals may have maintained alertness and thereby prevented the response from decaying as rapidly as it did under the acceleration condition.

3. Overview. The data from the decelerations support the conclusion reached by Barany[1]; alcohol ingestion tends to suppress the sensations resulting from rotatory stimulation. Moreover, some declines were evident for the velocity and duration of the turning sensations from the accelerations, although they were not statistically significant. This difference in the magnitude of decline of the turning experience between the accelerations and decelerations may be due to differences in instructions concerning attention to (and signals of) rotatory sensations.

F. *Spiral Aftereffect Data.* Means and standard deviations for the spiral aftereffect (SAE)

TABLE 11.—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.

Group		Pre		Post I		Post II	
		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

TABLE 12.—Results of the Analyses of Variance for the Displacement (in Degrees) and Duration (in Seconds) of the Subjective Reactions During the KP Rotation Stimuli.

Source	df	Subjective Displacement (Accelerations)		Duration (Accelerations)		Duration (Decelerations)	
		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 13.—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

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

TABLE 14.—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 Alcohol	M	15.3	13.0	14.1
	SD	7.2	5.4	6.1
High Alcohol	M	14.2	13.8	15.1
	SD	10.1	11.9	16.1

are presented in Table 14. Comparisons of the Pre, Post I, and Post II values reveal little change as the result of the ingestion of alcohol.

The relationship between the duration of the SAE and the duration resulting from the accelerations and decelerations of the rotatory stimuli was investigated. Only the correlations for the high alcohol group were significant (Table 15). Thus, these data offer only partial support to the studies by Reason,[18 19] Reason and Benson,[20] and Nilsson and Henrikson.[17] The lack of significant correlations among the measures may be due to the lack of appreciable variability in the SAE duration values for the control and moderate alcohol groups; or to several differences in experimental technique between earlier studies and this one.

G. *Subjective Data from the Deceleration which Involved Brief Intervals of Light.* The analysis of the data concerning the subjective sensations resulting from the decelerations using either a three-second or an eight-second period of room illumination was based on visual observation rather than on any statistical treatment of the data. There were no dramatic changes in the response which could be attributed to the ingestion of alcohol.

When compared to the experiences reported in total darkness (using a comparable angular stimulus) the primary sensations from the "light" decelerations were much shorter. The "light" intervals also tended to increase the occurrence and intensity of secondary sensations (i.e., a renewal of sensations which follows the end of the primary response and is in the opposite direction). These differences are evident when you compare Figures 15 and 16.

Under dark conditions, the secondary sensation, in most cases, was either not present or was too weak to be signaled. Even with the small number (five) of subjects in each of the subgroups, the data appeared to agree with results obtained in studies by Collins.[7 8]

H. *Nystagmus Data from the Deceleration which Involved Brief Intervals of Light.* Means and standard deviations for the slow phase displacement and frequency of the primary and

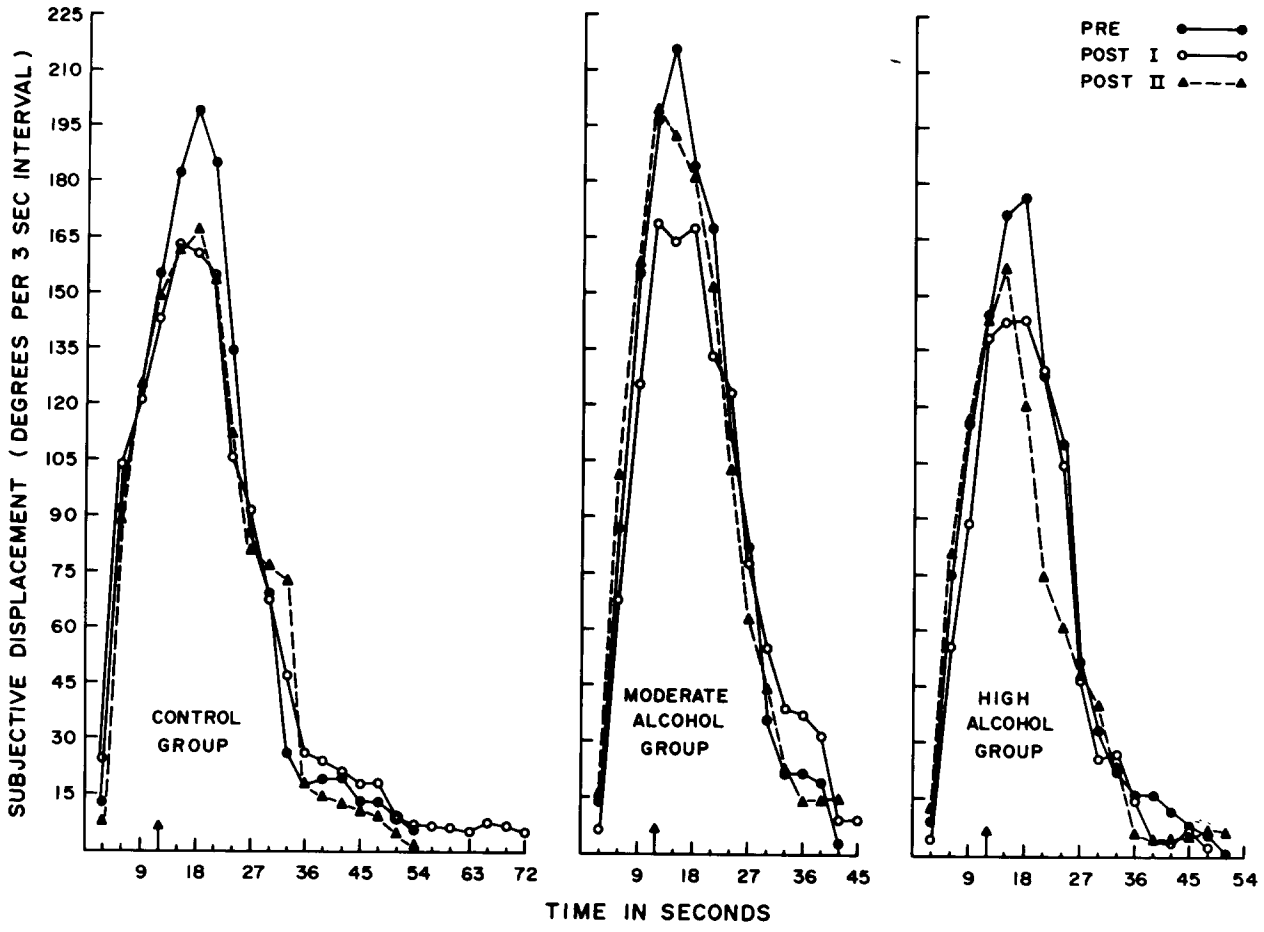

FIGURE 15. Response data for the average number of degrees of subjective displacement resulting from the accelerations (5°/sec² for 12 sec). Pre refers to the response recorded prior to the ingestion of alcohol, while the Post I and Post II data were obtained, respectively, 45 min and four hours after 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 15.—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.

Group	Acceleration Durations			Deceleration Durations		
	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**

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

secondary nystagmus resulting from the prolonged (light) decelerations are presented in Tables 16 and 17. Similar computations for the duration of the primary nystagmus are presented in Table 18; for comparative purposes, nystagmus duration values for a comparable stimulus (an acceleration) in total darkness are also presented. Each value represents a mean for five subjects since each group was divided into a three-second and an eight-second light sub-group.

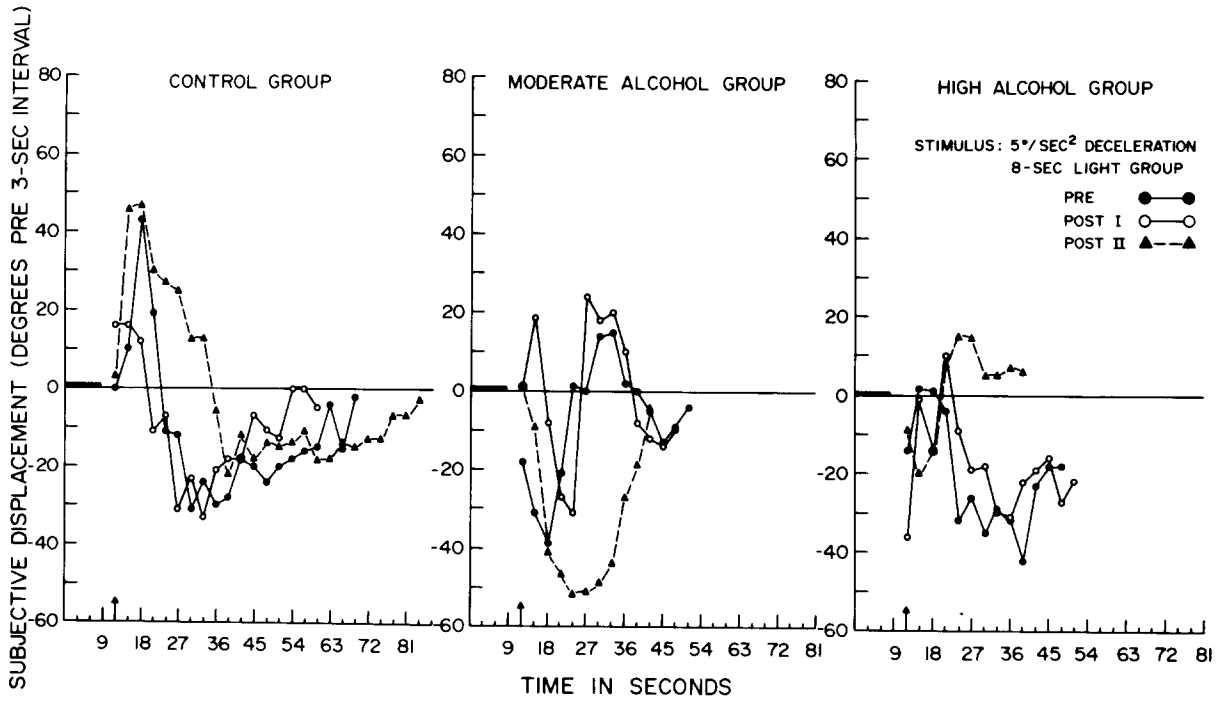

FIGURE 16. Response data for the average number of degrees of subjective displacement resulting from the 12-sec deceleration of 5°/sec². The room lights were turned on at the start of the deceleration and remained on for 8 sec. Points plotted above the zero line represent the primary response, while those below represent the secondary response. Symbols and markings are identical to those used in Figure 15.

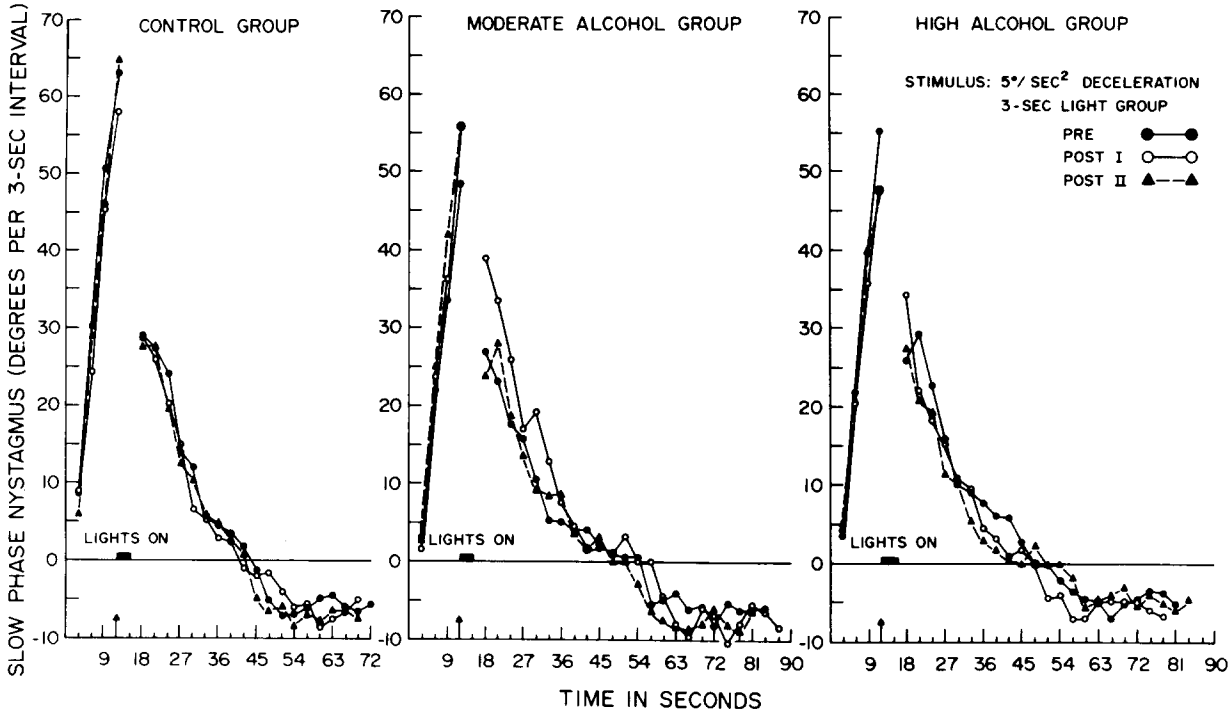

FIGURE 17. Response data for the average number of degrees of slow phase eye movement resulting from the 12-sec deceleration of 5°/sec². Immediately following the end of the deceleration, the room lights were turned on for 3 sec, during which time the subject fixated on a target. The short bar above 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.

TABLE 16.—Means and Standard Deviations for the Slow Phase Displacement of Primary and Secondary Nystagmus (in Degrees) Resulting from the 12-sec Deceleration of 5°/sec². 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 17.—Means and Standard Deviations for the Number of Primary and Secondary Nystagmic Eye Movements Resulting from the 12 sec Deceleration of 5°/sec². 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 18.—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.

Group	Interval		Deceleration			Acceleration		
			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

Because of the small number of subjects in each of the sub-groups, the data should be interpreted with caution. Due to the nature of the data, a visual analysis rather than statistical treatment was used.

Changes in the nystagmic response, which occurred as a result of the brief intervals of light, are evident in comparison of the nystagmus response curves from the light decelerations and dark accelerations (compare Figures 3 through 8 with Figures 17 through 20). The primary nystagmic responses under both light conditions were weaker than those obtained under dark conditions. The suppressive effect of the light was also evident in the duration of the primary nystagmus; mean duration values for the dark acceleration were much longer (Table 18). While the primary nystagmic response was weakened and shortened, the secondary nystagmic response (like the secondary sensation, the secondary nystagmic response follows the primary nystagmic response and is in the opposite direction) began sooner and was of greater amplitude, see Figure 21. Under dark conditions, the secondary nystagmic response was too weak and infrequent to score adequately in most cases. These suppressive effects of the brief intervals of light on the primary nystagmic response and their enhancing effects on secondary nystagmus support findings in earlier studies.[7,8]

The small number of subjects in each of the sub-groups made any analysis of the effects of alcohol difficult. Comparisons of the duration of the primary nystagmic response between the dark and light conditions indicate that the only trials where the values for the light condition were as long or longer than the dark condition occurred following alcohol ingestion (Table 18). These data provide some support for the idea that visual fixation, following alcohol ingestion, was less effective in suppressing the primary nystagmic response.

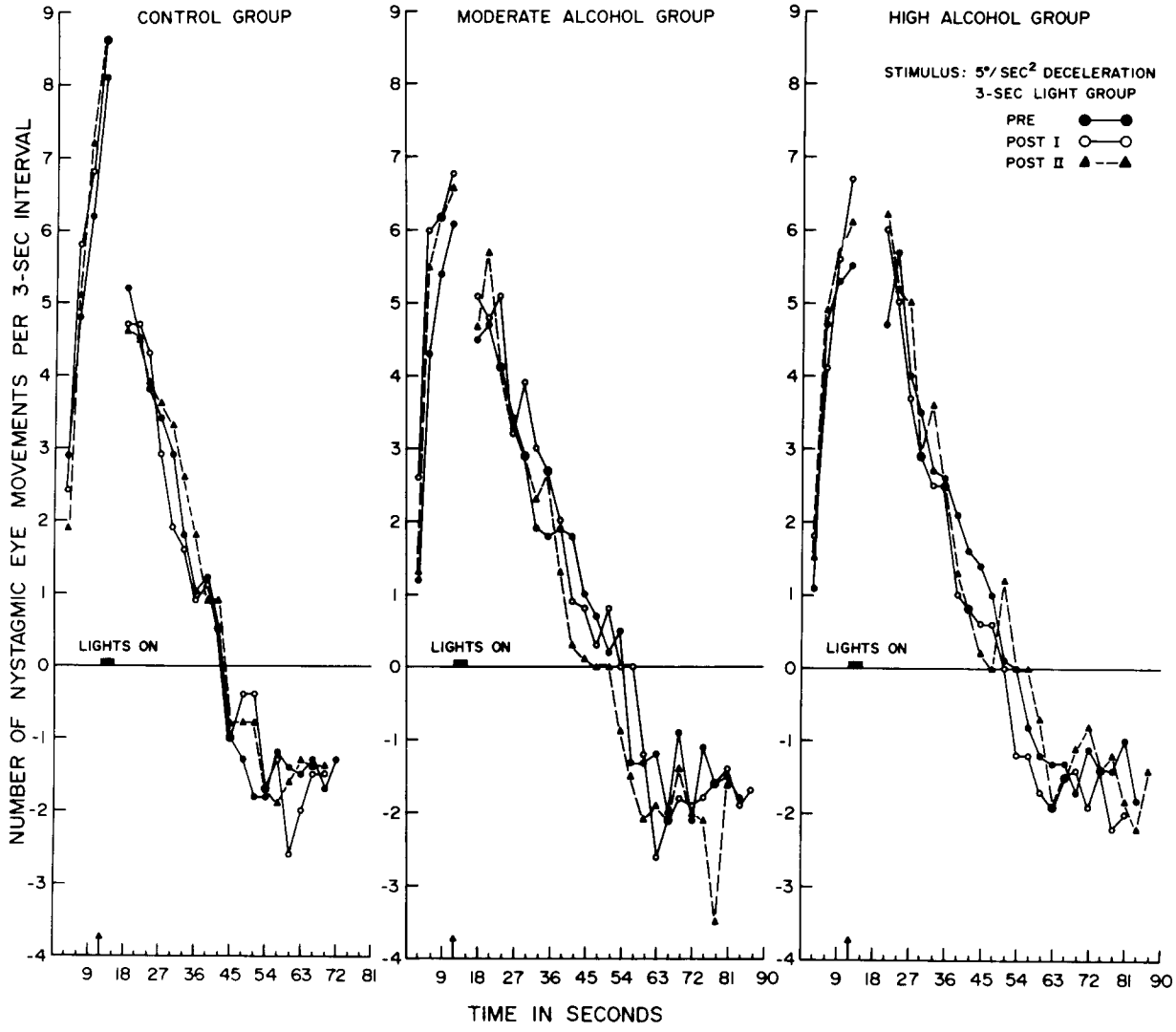

FIGURE 18. Response data for the average number of nystagmic eye movements resulting from the 12-sec deceleration of 5°/sec². Following deceleration, room lights were turned on for 3 sec, during which time the subject fixated on a target. Symbols and markings are identical to those used in Figure 17.

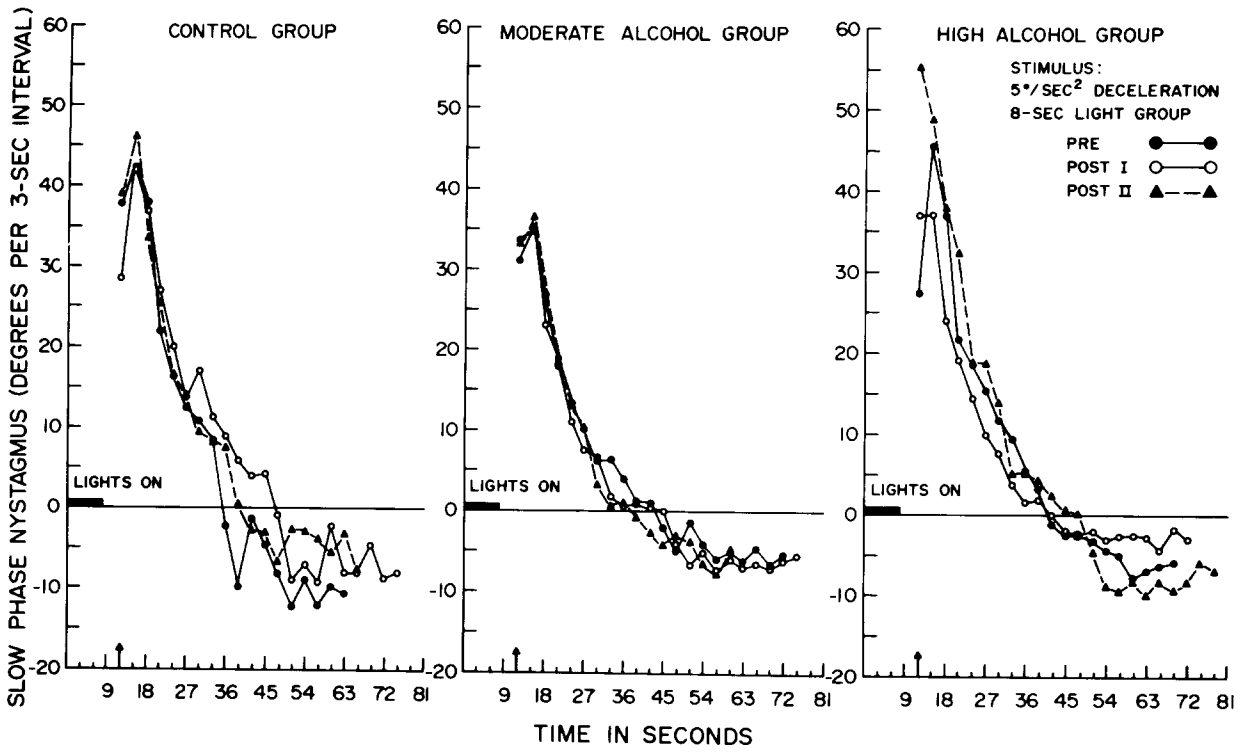

FIGURE 19. Response data for the average number of degrees of slow phase eye movement resulting from the 12-sec deceleration of 5°/sec². The room lights were turned on at the start of the deceleration and remained on for 8 sec. Symbols and markings are identical to those used in Figure 17.

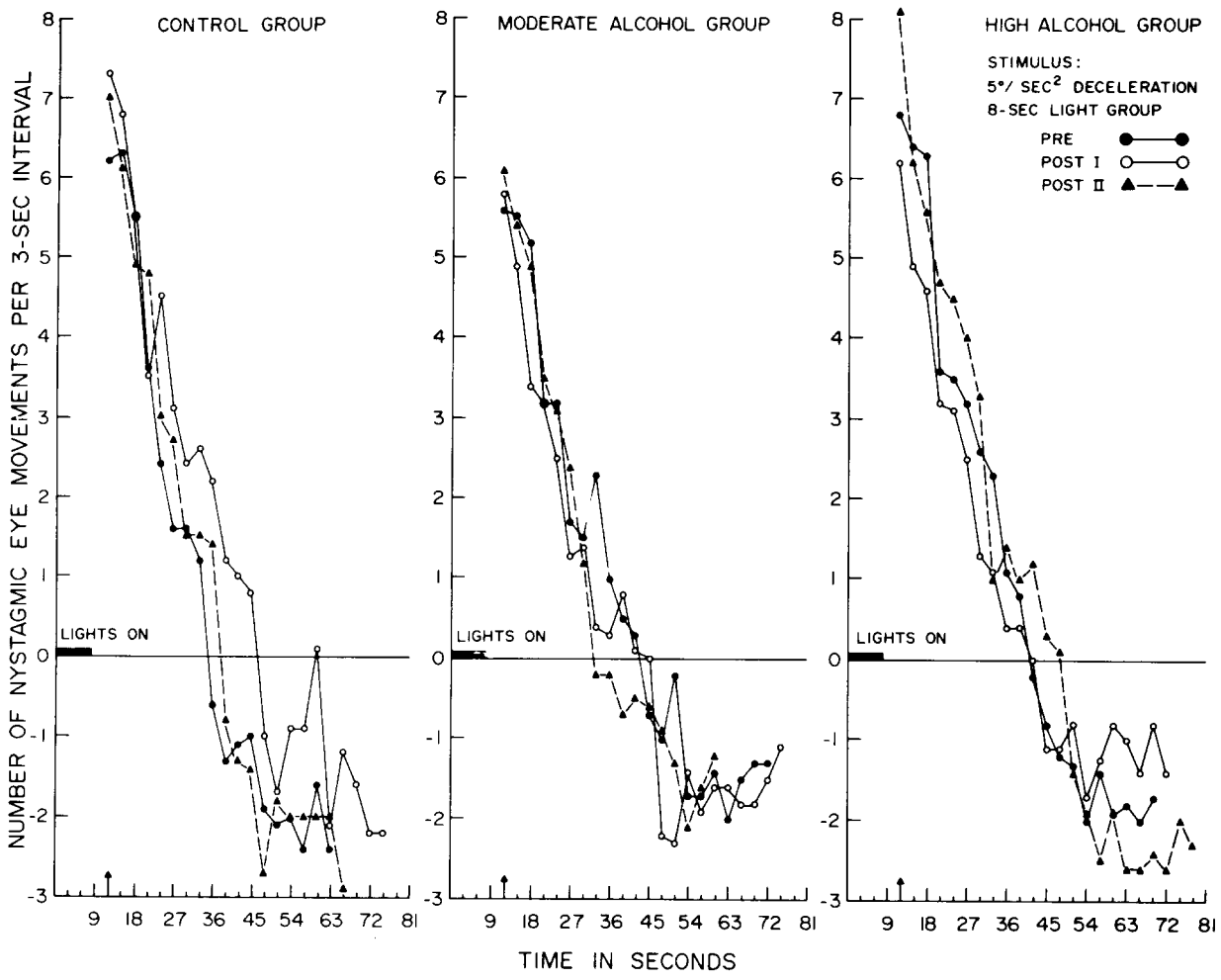

FIGURE 20. Response data for the average number of nystagmic eye movements resulting from the 12-sec deceleration of 5°/sec². Room lights were turned on at the start of the deceleration and remained on for 8 sec. Symbols and markings are identical to those used in Figure 17.

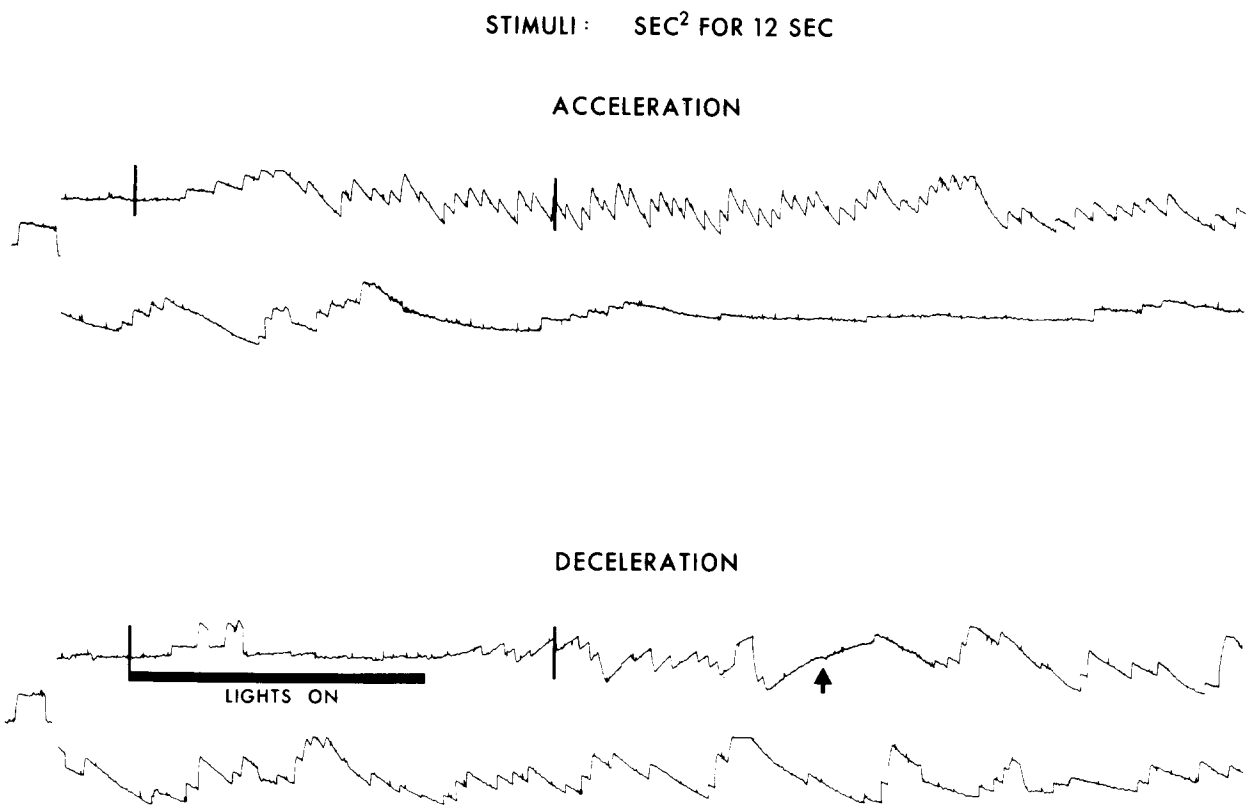

FIGURE 21. A portion of the nystagmic tracings for a subject's response to the dark acceleration and to the deceleration with the 8-sec period of illumination. The stimuli were comparable: a 12-sec acceleration and a 12-sec deceleration each at the rate of 5°/sec². Although the nystagmic responses are in opposite 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° 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.

REFERENCES

1. Barany, R.: Experimentelle Alkoholintoxication. MONATSCHRIFT FÜR OHRENKEILKUNDE UND LARYNGO-RHINOLOGIE, 45:959–961, 1911.
2. Békésy, G.: Subjective cupulometry. ARCHIVES OF OTOLARYNGOLOGY, 61:16–28, 1955.
3. Bochenek, Z. and F. C. Ormerod: The inhibitory action of certain substances on the response to vestibular stimulation. JOURNAL OF LARYNGOLOGY AND OTOLOGY, 76:39–44, 1962.
4. Bodin, M. A.: Some practical notes for the analysis of post-rotational nystagmus by determination of time constants of decays. Unpublished manuscript, 1966.
5. Collins, W. E.: Effects of mental set upon vestibular nystagmus. JOURNAL OF EXPERIMENTAL PSYCHOLOGY, 63:191–197. 1962.
6. Collins, W. E.: Manipulation of arousal and its effects upon human vestibular nystagmus induced by caloric irrigation and angular accelerations. AEROSPACE MEDICINE, 34:124–129, 1963.
7. Collins, W. E.: Modification of vestibular nystagmus and "vertigo" by means of visual stimulation. TRANSACTIONS OF THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, 72:962–979, 1968.
8. Collins, W. E.: Special effects of brief periods of visual fixation on nystagmus and sensations of turning. AEROSPACE MEDICINE, 39:257–266, 1968.
9. Collins, W. E.: Task-control of arousal and the effects of repeated unidirectional angular acceleration on human vestibular responses. ACTA OTOLARYNGOLOGICA SUPPLEMENTUM, 190, 1964.
10. DiGiunta, E. and D. Rosa: Rilievi nistagmografici post-rotatorii in corso di intossicazione alcoolica acuta. CLINICA OTO-RINO-LARINGOIATRICA (ROMA), 20:88–105, 1968.
11. Ey, W.: Storungen des oculo-vestibularen Systems bei alcoholisierten Personen. BERIGHT: DEUTSCH OPHTHALMOLOGISCHE GESSELSCHAFT, 65: 349–353, 1964.
12. Forster, B.: Elektronystagmographische Untersuchungen uber den Drehbeschleunigungs-nystagmus nach Alkoholgaben. DEUTSCHE ZEITSCHRIFT FUR DIE GESAMTE GERICHTLICHE MEDIZIN, 47:282–288, 1958.
13. Guedry, F. E. and L. S. Lauver: Vestibular reactions during prolonged constant angular acceleration. JOURNAL OF APPLIED PHYSIOLOGY, 16:215–220, 1961.
14. Jatho, K.: Die Wirkung der Alkoholintoxication auf den Vestibularapparant unter besonderer Berucksichtigung der Storungen der vestibular-okulomotorischen Regelfunktion. II. Mitteilung Ergebnisse experimenteller Untersuchungen. ZEITSCHRIFT FUR LARYNGOLOGIE, RHINOLOGIE, OTOLOGIE UND IHRE GRENZGEBLETE, 44:105–108, 1965.
15. Manz, Rudolf: Der Einfluss geringer Alkoholgaben auf Teilfunktionen von Auge and Ohr. DEUTSCHE ZEITSCHRIFT FUR DIE GESAMTE GERICHTLICHE MEDIZIN, 32:301–312, 1940.
16. Mizoi, Y., T. Ishido, and N. Ohga: Studies on post-rotatory nystagmus in alcohol intoxication. JAPANESE JOURNAL OF LEGAL MEDICINE, 16:247–260, 1962.
17. Nilsson, A. and N. G. Henriksson: The oculogyral illusion—a form of rotation after-effect—and its relation to the spiral after-effect in repeated trials. PSYCHOLOGICAL RESEARCH BULLETIN, 7:1–17, 1967.
18. Reason, J. T.: Individual differences in motion sickness susceptibility: A further test of the "receptivity" hypothesis. BRITISH JOURNAL OF PSYCHOLOGY, 60:321–328, 1969.
19. Reason, J. T.: Relations between motion sickness susceptibility, the spiral after-effect and loudness estimation. BRITISH JOURNAL OF PSYCHOLOGY, 59:385–393, 1968.
20. Reason, J. T. and A. J. Benson: Individual differences in the reported persistence of visual and labyrinthine after-sensations, and of exponentially decaying visual and auditory signals. BRITISH JOURNAL OF PSYCHOLOGY, 59:167–172, 1968.
21. Schroeder, D. J.: Alcohol and Disorientation-Related Responses. I. Nystagmus and Vertigo During Caloric and Optokinetic Stimulation. FAA OAM report 71–6, 1971.
22. Schulte, K. and H. Roth: Drehnystagmus un Blutalkohol-konzentration. ZENTRALBLATT FUER VERKEHRS-MEDIZIN, VERTEHRS—PSYCHOLOGIE UND AUGRENZENDE GEBIETE (Alfred/Leino, Germany), 3:141–144, 1957.
23. Schweitzer, H.: Graphologische, klinische und psychologische Untersuchungen unter Alkoholwirkung. ÖFFENTLICHE GESUNDHEITSDIENST (Stuttgart), 16:361–372, 1955.
24. Taschen, B.: Nystagmusprufungen zur Verstellung der Alkoholbeeinflussung. DEUTSCHE ZEITSCHRIFT FUR DIE GESAMTE GERICHTLICHE MEDIZIN, 44:436–437, 1955–1956.
25. Taschen, B.: Eine einfache Nystagmusprobe zur Festsellung der Alkoholbeeinflussung. MEDIZINISCHE MONATSSCHRIFT (Stuttgart), 9:25–27, 1955.