

EFFECTS OF CONGENER AND NONCONGENER ALCOHOLIC BEVERAGES
ON A CLINICAL ATAXIA TEST BATTERY

David J. Schroeder, Ph.D.*
William E. Collins, Ph.D.
Civil Aeromedical Institute
Federal Aviation Administration
Oklahoma City, Oklahoma

*Veterans Administration Hospital
Topeka, Kansas



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16. Abstract As a screening procedure in aviation medicine, clinical examiners often use one or more tests of standing steadiness or gait to help assess neurological and vestibular soundness. Normal functioning of these mechanisms associated with proper body orientation has been traditionally regarded as critical to safety in piloting aircraft. It is clear that the ingestion of alcohol can disturb postural measures, and it is possible that the effects of alcohol may be manifested at significant stages subsequent to acute intoxication; i.e., during so-called "hangover" periods. This study was designed to investigate the performance of normally "heavy" and normally "light" young male drinkers on an ataxia test battery before and after they drank either a high-congener (bourbon) or low-congener (vodka) alcoholic beverage. To assess possible long-term effects of alcohol, testing was conducted 1, 3, 5, 9, 24, and 32 hours after drinking. With the exception of one walking test that showed inferior performance 1 hour after drinking and recovery thereafter, the measures of the ataxia test battery were about equally affected, showing decrements from 1 to 3 hours after drinking and a return to a normal plateau by the fifth postdrinking hour. Normally heavy drinkers tended to display less ataxia following drinking than did normally light drinkers. Comparisons of the low- and high-congener beverages failed to reveal any significant differential effects. There were also no indications of any significant impairment on ataxia tests during the hangover period.			
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EFFECTS OF CONGENER AND NONCONGENER ALCOHOLIC BEVERAGES ON A CLINICAL ATAXIA TEST BATTERY

I. Introduction.

As a screening procedure in aviation medicine, clinical examiners often use one or more tests of standing steadiness or gait to help assess neurological and vestibular soundness (4). Normal functioning of these mechanisms associated with proper body orientation has been traditionally regarded as critical to safety in piloting aircraft. It is clear that the ingestion of alcohol can disturb these postural measures, and it is possible that the effects of alcohol may be manifested at significant stages subsequent to acute intoxication; i.e., during so-called "hangover" periods.

The 1865 work of Immerman is cited by Goldberg (9) as one of the first scientific investigations of the disturbance in balance that occurs following drinking. Since that time, Miles (17), Carlson et al. (3), Goldberg (9), and Alha (1) have all made various measures of sway during the Romberg test (standing with feet together, eyes closed, arms at sides) as a measure of ataxia due to alcohol ingestion. While Goldberg (9) used both the Sharpened Romberg (the feet are placed in tandem instead of together) and the Romberg tests as his measures of ataxia, all of the earlier studies noted above and subsequent alcohol studies by Pihkanen (21) and Kelly et al. (16) used only a single measure, viz, the amount of sway during either the Romberg or the modified Romberg test. Begbie (2) obtained more precise measures of subject sway and oscillation with strain gauges while subjects attempted standing on an unstable platform; he noted that moderate amounts of alcohol yielding an average peak blood alcohol level (BAL) of 16 mg percent were sufficient to produce a significant deterioration in performance.

In 1966, Graybiel and Fregly (11) developed an ataxia battery that involved the use of a rail and provided several quantitative measures of postural equilibrium. Their battery tested ability to walk on the rail heel-to-toe (eyes open) and stand heel-to-toe (eyes open and eyes closed); in addition, on the floor, subjects performed the Sharpened Romberg, walked a line with eyes closed (WALEC), and stood on one leg with eyes closed (SOLEC). Fregly, Bergstedt, and Graybiel (7) used the battery with 13 naval flight students before and for several hours following the ingestion of either 80-proof or 100-proof vodka (yielding peak BALs of about 75 and 95 mg percent, respectively). The peak decrement in ataxia test performance occurred approximately 60-75 minutes following drinking. Recovery time was not identical for all of the measures and varied somewhat depending on the dose

(proof) of alcohol; only one of the seven measures was significantly depressed 4½ hours or longer after drinking either 80-proof (SOLEC-R) or the 100-proof vodka (standing on the rail, eyes open).

There is conflicting evidence available regarding the possible differential influence on human functioning of various types of alcoholic beverages. The differences are usually attributed to the detrimental action of congeners--the various substances other than ethyl alcohol (such as methanol, esters, aldehydes, etc.) found in many alcoholic beverages. Vodka is so low in congener content that it is often referred to as "noncongener." With regard to evidence for differential effects of some relevance to the present study (i.e., to mechanisms associated with posture and balance), Ryback and Dowd (22) and Dowd (5) reported that a high-congener alcohol produced larger increases than did vodka in ocular nystagmus and subjective responses to coriolis vestibular stimulation the morning after drinking. But data from studies by Hill, Collins, and Schroeder (12) and Hill, Schroeder, and Collins (13), dealing with the short- and long-term vestibular response, including coriolis stimulation and positional alcohol nystagmus (PAN), failed to reveal any significant differences in these responses.

Some studies have reported significant response differences between vodka and congener beverages when the latter have been "congener fortified." Thus, differences using "super-bourbon" have been reported for risk taking (15,23), using 4 times the normal congener levels, and for EEG and nystagmus (19,20), using 32 times the normal congener content.

While various investigators have used different alcoholic beverages in their respective studies of ataxia, Pihkanen (21) was one of the first to attempt to compare the effects of different alcoholic beverages. He noted that static ataxia, as measured by modified Romberg performance (swaying was recorded) over a 4-hour postdrinking period, was nearly twice as great following the ingestion of brandy as it was after subjects drank a malt beverage (beer). However, the brandy trials were always first, the greatest difference occurred when the peak BALs were considerably different between brandy (124 mg percent) and beer (87 mg percent), and there was no control group. In comparing the effects of equivalent amounts of Canadian rye whiskey and Canadian beer ingested in a 25-minute period, Dussault and Chappel (6) found that Canadian whiskey produced a higher peak blood alcohol level and a greater amount of body sway (Romberg). Kalant, LeBlanc, Wilson, and Homatidis (14) were concerned that the differences in peak BALs noted by Dussault and Chappel (6) could have been due to the rapid rate of drinking that was required on an empty stomach. To test this assumption, Kalant et al. (14) compared the effects of equivalent amounts of Canadian rye whiskey, Canadian beer, and a sparkling table wine, consumed over a 4-hour drinking period, on physiological and sensorimotor responses. They found no significant differences in the peak blood alcohol levels or in the degree of impairment in body sway during the Romberg test.

Another variable of potential importance to postural equilibrium after drinking alcohol is the drinking history of the subject. Goldberg (9) is one of few authors who have attempted to relate performance changes following drinking to drinking history. Even though subjects classified as heavy and moderate drinkers ingested more alcohol during his study than did subjects classified as abstainers, Goldberg concluded that heavy drinkers evidenced only moderate ataxia.

To explicate the relationship between several of the above-mentioned variables, the present study of alcohol effects was designed to investigate differences in performance of subjects on a more recent quantitative ataxia test battery developed by Fregly and Graybiel (8). The latter have presented normative data for a quantitative ataxia battery that does not require the use of rails as their earlier one did (11) and hence is more readily adapted to a clinical setting. Variables assessed included: (i) The drinking habits of the subjects (heavy vs. light); (ii) the ingestion of a high-congener beverage (bourbon) vs. a relatively congener-free alcoholic beverage (vodka); and (iii) assessments made up to 32 hours after drinking since congeners have been implicated in some long-term effects of alcohol (19).

II. Method.

Subjects. On the basis of their responses (i) during interviews and (ii) to a questionnaire developed by Mulford and Miller (18), 25 men were selected as "heavy" drinkers and 25 as "light" drinkers from among several hundred university students between the ages of 21 and 29. The questionnaire (18) consists of 20 behaviorally defined statements scaled to distinguish five levels of drinking behavior. All of our "light" drinkers had to score on the two lowest levels while "heavy" drinkers had to score on the two highest levels. In addition, for a more objective measure, we used a scale based on the average monthly consumption of alcohol. Scores on this latter scale are based on the total number of ounces of various alcoholic beverages reportedly consumed by a given subject in a typical month multiplied by the percentage of alcohol in each beverage; e.g., 1 quart of 100-proof liquor (50 percent alcohol by volume) would yield a score of 16.0 (i.e., 32 oz x .50). The range of scores for our 25 "light" drinkers ranged from 0 to 4; those for our 25 heavy drinkers ranged from 50 to 240. These subjects were randomly placed into five groups comprising 10 subjects each; heavy drinkers given vodka, heavy drinkers given bourbon, light drinkers given vodka, light drinkers given bourbon, and a control group (5 heavy and 5 light drinkers) given a placebo drink.

Procedure. Measures of postural equilibrium were obtained by using the quantitative ataxia test battery developed by Fregly and Graybiel (8). The tests were conducted on a hard floor while the subject assumed an erect or nearly erect position, with his arms folded across his chest. The following tests were included:

1. Sharpened Romberg (SR). Each subject stood with his eyes closed and feet tandemly aligned, heel-to-toe for a period of 60 seconds. If the subject was successful on the first attempt, he was given the maximum score of 240 and no further trials were required. If he was unable to complete a 60-second standing time, additional trials (up to a maximum of four) were run until the subject was able to reach the 60-second criterion. A subject's score was then determined by subtracting the number of seconds he fell short of the standard from the maximum score of 240.
2. Walk a Line Eyes Closed (WALEC). After positioning himself at one end of a 12-foot line, the subject closed his eyes and walked in a heel-to-toe fashion, at a normal rate, to the end of the line. The deviation in inches from the end of the line represented his score for a trial. Trials during which the subject violated the heel-to-toe touching rule or tandem alignment were not scored. The best two trials out of three were used as the score for each subject.
3. Stand on One Leg Eyes Closed (SOLEC-R, SOLEC-L). While standing on his preferred leg, each subject folded his arms, flexed the other leg, and attempted to stand on the one leg for 30 seconds. Subjects were allowed to move their upper body or the flexed leg but were not allowed to move the standing foot in any way. The trial was started when the subject indicated that he was ready and closed his eyes. If the subject was unable to complete 30 seconds, additional trials were administered until criterion was reached or until five trials were conducted. Additional trials involved alteration with the nonpreferred leg. The scoring procedure was similar to that used for the SR test, but in this case, the maximum score was 150 seconds for each leg.
4. Walk on Floor Eyes Closed (WOFEC). Assuming the usual posture, the subject proceeded to close his eyes and walk 10 heel-to-toe steps beyond his first 2 steps in as straight a path as possible. Each subject's score was represented by the number of heel-to-toe steps successfully taken, up to a maximum of 10, on the best three out of five trials.

Additional information concerning the administration and scoring of the test battery appears in the article by Fregly and Graybiel (8).

The entire ataxia test battery was administered to subjects on two practice occasions prior to the experimental day. For the experiment the predrinking administration of the test battery occurred at about 0900 soon after the subjects arrived in the laboratory. Postdrinking sessions were conducted 1, 3, 5, 9, 24, and 32 hours following the end of drinking. Meals were eaten at the laboratory and all subjects slept at the Institute.

Subjects started drinking their respective beverages shortly after completing the test battery, at approximately 1000. Each of the alcohol subjects received 2.2 ml of liquor (100-proof Smirnoff vodka or 101-proof Wild Turkey bourbon) per kg of body weight. The alcohol was added to orange juice to a total volume of 1,100 ml. Subjects in the control group received 1,100 ml of orange juice to which a drop or two of rum extract was added to give a rum odor and flavor. Subjects were told that they would receive "some" alcohol and were instructed to spread their drinking over the 30-minute drinking period. In order to ascertain blood alcohol levels, venous blood samples were drawn prior to drinking and 1 and 4 hours following the end of drinking. Subjects in the control group had only one sample drawn, prior to drinking.

III. Results.

Mean blood alcohol levels are presented in Table 1. There was no evidence of alcohol in the blood of any of the subjects prior to the start of the study and, as is evident from the tabulated blood alcohol values, there were no statistically significant differences between any of the groups for either the 1- or the 4-hour samples.

TABLE 1. Mean Blood Alcohol Levels (%)

<u>Beverage</u>	<u>Drinking Habits</u>	<u>Postdrinking Sessions</u>	
		<u>1-H</u>	<u>3-H</u>
Bourbon	Light	.077	.050
	Heavy	.086	.045
Vodka	Light	.085	.055
	Heavy	.083	.053

Means and standard deviations for the various ataxia measures for each group are presented in Table 2. For ease of interpretation, "change" scores of each postdrinking session from the predrinking (baseline) session were computed in percentages and plotted in two ways: One set of graphs compared the control group with all light drinkers and all heavy drinkers; the other set compared controls with all subjects given bourbon and all subjects given vodka.

Two types of statistical analyses were conducted. Simple analyses of variance were performed on the scores for each group on each test to assess within-group changes. Additionally, overall analyses (all groups) were conducted on difference scores for each test (subtracting each postdrinking score from the predrinking score) to assess between-group differences. Data yielding significant F ratios were further analyzed by Tukey's HSD test.

TABLE 2. Means and Standard Deviations for Scores Obtained on the Ataxia Test Battery

Test	Group		Hours After Drinking							Test	Group		Hours After Drinking						
			Pre	1	3	5	9	24	32				Pre	1	3	5	9	24	32
<u>SHARPENED ROMBERG</u>	Control	M	188	179	191	213	184	172	216	<u>SOLEC-R</u>	Control	M	94	88	103	107	110	131	108
		SD	54	31	48	23	73	65	51			SD	41	48	45	45	51	22	47
	Bourbon Lights	M	232	138	139	226	226	224	233		Bourbon Lights	M	133	60	64	137	113	138	142
		SD	17	102	92	34	46	47	16			SD	23	54	51	20	52	32	14
	Bourbon Heavies	M	224	151	198	217	234	240	233		Bourbon Heavies	M	124	86	100	123	130	143	128
SD		39	89	79	50	18	0	23	SD			47	55	50	36	29	16	37	
<u>WALEC</u>	Control	M	232	106	136	226	223	240	223	<u>SOLEC-L</u>	Control	M	134	54	85	126	145	146	140
		SD	17	99	95	28	29	0	27			SD	26	43	49	46	12	10	18
	Vodka Lights	M	203	117	152	189	217	218	216		Vodka Lights	M	98	55	86	98	90	114	119
		SD	56	74	75	79	59	51	64			SD	52	36	46	47	41	45	47
	Bourbon Heavies	M	38	42	30	29	24	18	20		<u>SOLEC-L</u>	Control	M	76	105	110	109	121	103
SD		32	34	20	24	12	17	26	SD				44	42	46	42	44	43	44
Bourbon Lights	M	22	56	40	15	8	12	5	Bourbon Lights	M		134	53	69	135	121	136	126	
	SD	16	37	33	13	6	6	3		SD		35	56	47	21	41	22	40	
Bourbon Heavies	M	17	44	16	15	18	13	11	Bourbon Heavies	M		118	60	116	127	137	129	142	
	SD	14	33	19	8	17	12	8		SD		39	35	49	29	19	36	19	
<u>WOFEC</u>	Control	M	27	52	23	22	10	12	10	<u>SOLEC-L</u>	Vodka Lights	M	128	51	71	121	123	144	145
		SD	25	36	14	25	8	10	5			SD	37	37	50	38	40	15	11
	Vodka Heavies	M	14	28	41	14	12	13	13		Vodka Heavies	M	118	57	101	103	111	111	104
		SD	7	33	36	7	6	10	13			SD	39	38	57	48	51	47	47
	Control	M	27	29	27	28	29	29	30		<u>SOLEC-L</u>	Control	M	27	29	27	28	29	29
SD		4	2	5	4	2	2	1	SD				4	2	5	4	2	2	1
Bourbon Lights	M	30	25	29	30	30	30	30	Bourbon Lights	M		30	25	29	30	30	30	30	
	SD	0	9	2	0	0	0	0		SD		0	9	2	0	0	0	0	
Bourbon Heavies	M	30	27	29	30	30	30	30	Bourbon Heavies	M		30	27	29	30	30	30	30	
	SD	1	4	2	2	0	0	0		SD		1	4	2	2	0	0	0	
Vodka Lights	M	30	23	29	30	30	30	29	Vodka Lights	M	30	23	29	30	30	30	29		
	SD	0	8	3	0	0	0	2		SD	0	8	3	0	0	0	2		
Vodka Heavies	M	29	24	28	30	30	30	30	Vodka Heavies	M	29	24	28	30	30	30	30		
	SD	4	4	4	0	0	2	0		SD	4	4	4	0	0	2	0		

Sharpened Romberg (SR). While subjects in the control group evidenced little postdrinking change in standing time for the SR (Figure 1), scores for subjects in each of the alcohol groups yielded significant F ratios ($p < .01$ in all cases). A significant pretest to posttest decline in performance (32-54 percent) 1 hour after drinking was obtained for each alcohol group ($p < .05 - .001$). For both groups of light drinkers but for neither group of heavy drinkers, the scores 3 hours after drinking were still significantly ($p < .01$) below predrinking levels. Moreover, for all the alcohol groups, scores 1 hour after drinking were significantly poorer ($p < .05 - .001$) than scores for each of the last four sessions.

Overall statistical comparisons of the difference scores for the various groups yielded three significant effects. The Vodka-Lights ($p < .01$), Bourbon-Lights ($p < .05$), and Vodka-Heavies ($p < .05$) had poorer scores 1 hour after drinking than the controls. Performance by light drinkers in both the bourbon and vodka groups remained significantly poorer ($p < .01$) than that of control subjects through the 3-hour postdrinking session. Clear recovery for all alcohol groups was evident by the fifth postdrinking hour.

Walk a Line, Eyes Closed (WALEC). Simple analyses of variance of scores for each group yielded significant declines ($p < .01 - .001$) for all alcohol groups and a significant improvement ($p < .05$) for the control group. With respect to the latter, the 24-hour session was significantly better than the predrinking session as the control group showed a general improvement across sessions. For three of the four alcohol groups (Vodka-Heavies excepted), scores 1 hour after drinking were significantly below scores for all other sessions. The major decline for the Vodka-Heavies occurred 3 hours after drinking at which time performance was significantly poorer than baseline and the 9-, 24-, and 32-hour sessions.

Change scores in WALEC performance are presented in Figure 2 where higher scores represent greater deviation from straightline walking, hence poorer performance. While subjects in the control group evidenced a slight increase (10 percent) in walking deviation from baseline to the 1-hour postdrinking session, their performance improved on subsequent trials so that by the last two testing sessions, 24 and 32 hours after drinking, they displayed approximately 50 percent less deviation than baseline in their locomotion. In contrast, deviation from straightline walking by subjects in the alcohol groups 1 hour after drinking was 94-136 percent greater than before drinking. In spite of these large decrements in performance 1 hour after drinking, the variability in overall performance was sufficiently great that only the difference between the control group and Bourbon-Lights was statistically significant ($p < .05$). Performance by the Bourbon-Heavies and Vodka-Lights improved notably from 1 to 3 hours after drinking; they exhibited slightly less deviation (8 percent and 15 percent) in walking the line than prior to drinking (Table 2). At the same time the Bourbon-Lights and Vodka-Heavies still evidenced 82 percent and 198 percent greater deviation than

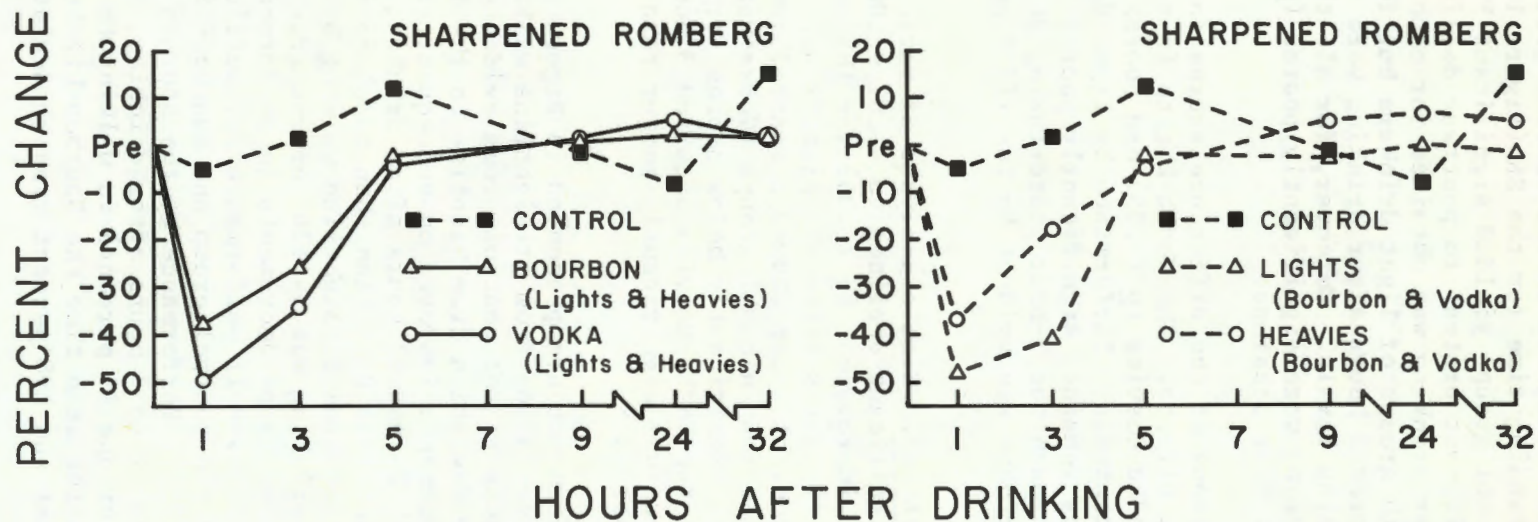


Figure 1. Percent change of postdrinking from predrinking scores for the Sharpened Romberg test.

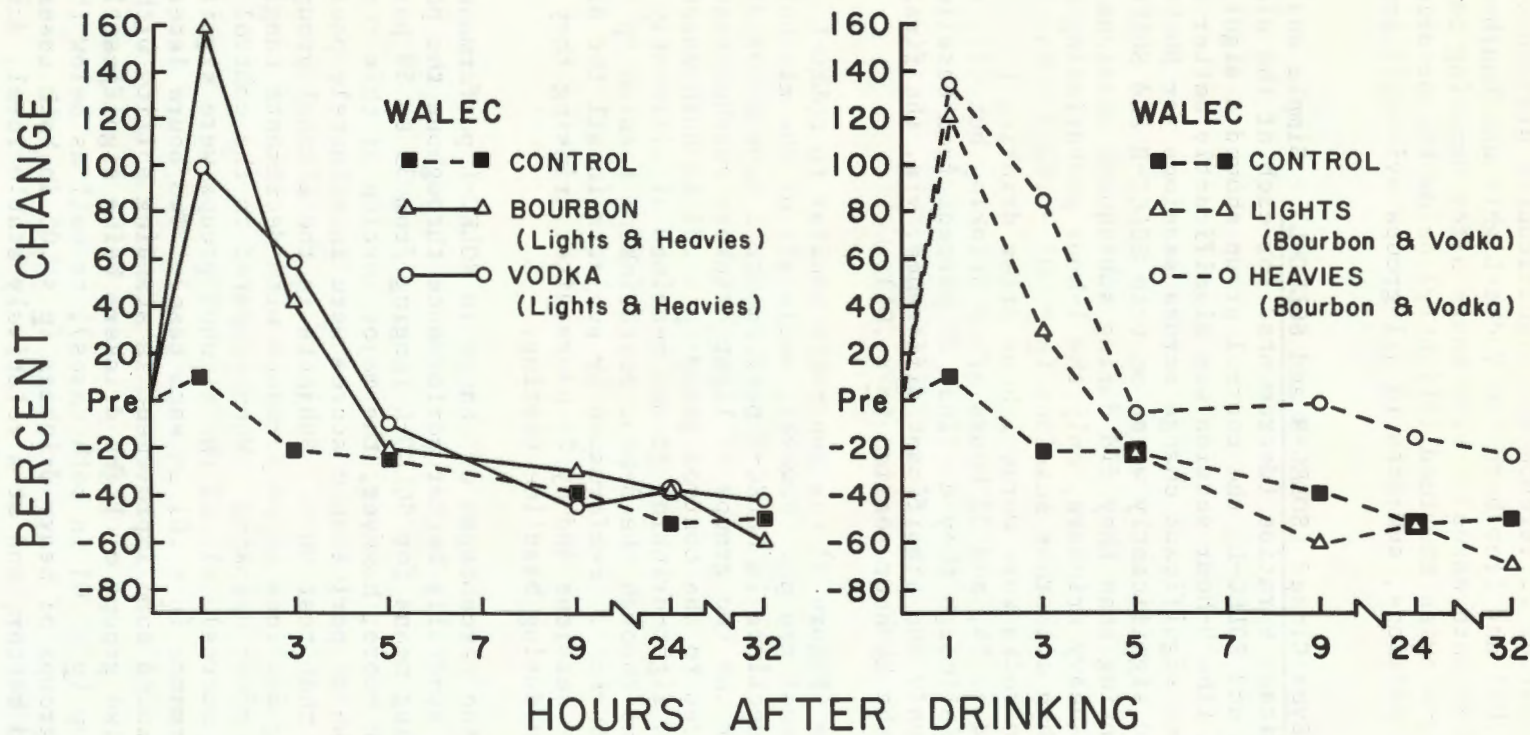


Figure 2. Percent change of postdrinking from predrinking scores for the Walk a Line, Eyes Closed (WALEC) test.

during predrinking; the latter difference was significantly different from the control group ($p < .01$) and from both the Vodka-Lights and Bourbon-Heavies groups ($p < .05$ in both cases). Five hours after drinking only the performance of the Vodka-Heavies remained (slightly) below the predrinking level. Over subsequent sessions, subjects in all groups evidenced improvement in performance.

Stand on One Leg, Eyes Closed (SOLEC-R and SOLEC-L). Simple analyses of variance yielded significant F ratios (decrements) for each of the alcohol groups for both SOLEC-R and SOLEC-L; the control group showed a significant improvement for SOLEC-L (the 9-hour session was significantly better than baseline, $p < .05$) and no significant change across sessions for SOLEC-R. Light drinkers performed significantly worse on both SOLEC-R and SOLEC-L 1 and 3 hours after drinking than they did during subsequent sessions and prior to drinking. For heavy drinkers, only the 1-hour postdrinking session for SOLEC-L was worse than all other sessions ($p < .05 - .001$). For SOLEC-R, (i) heavy drinkers given vodka were worse 1 hour after drinking than they were during baseline and 5, 24, and 32 hours after drinking but (ii) heavy drinkers given bourbon (although they declined 30 percent from baseline 1 hour after drinking) showed only one significant difference, viz, the first posttest differed from the 24-hour session ($p < .01$).

SOLEC-R performance (Figure 3) was generally similar to SOLEC-L performance for the alcohol groups. However, while all of the alcohol groups evidenced considerable declines in SOLEC-R performance 1 hour after drinking, only the differences for the two groups of light drinkers reached statistical significance when compared to the control group ($p < .01$ in both cases). Performance by these two light-drinking groups remained significantly below that of the control group through the 3-hour postdrinking session ($p < .05$ for vodka and $p < .01$ for bourbon). Performance by subjects in all the alcohol groups improved in later sessions and by 24 hours after drinking they were 4-16 percent better than during baseline testing.

Figure 3 reflects the percentages of change in SOLEC-L performance. Control subjects showed generally better performance throughout the postdrinking sessions with postdrinking means for SOLEC-L ranging from 35 to 59 percent better than the baseline score; however, the major portion of this increase is due to two subjects whose performance scores were inordinately poor in the predrinking session for that test only. Subjects in the alcohol groups displayed significant postdrinking declines in performance with decrements ranging from 49 to 60 percent 1 hour after drinking. When compared to the control group (analysis of difference scores), all of the alcohol groups were significantly poorer in overall performance ($p < .01$ in each case). Two hours later all alcohol groups had evidenced some improvement in standing ability with only the performance of the two groups of light drinkers being significantly below that of the control group ($p < .01$ in both cases), as well as below the performance of the two groups of heavy drinkers ($p < .05$ in both cases). All alcohol groups performed better, and at a relatively stable level, during later sessions.

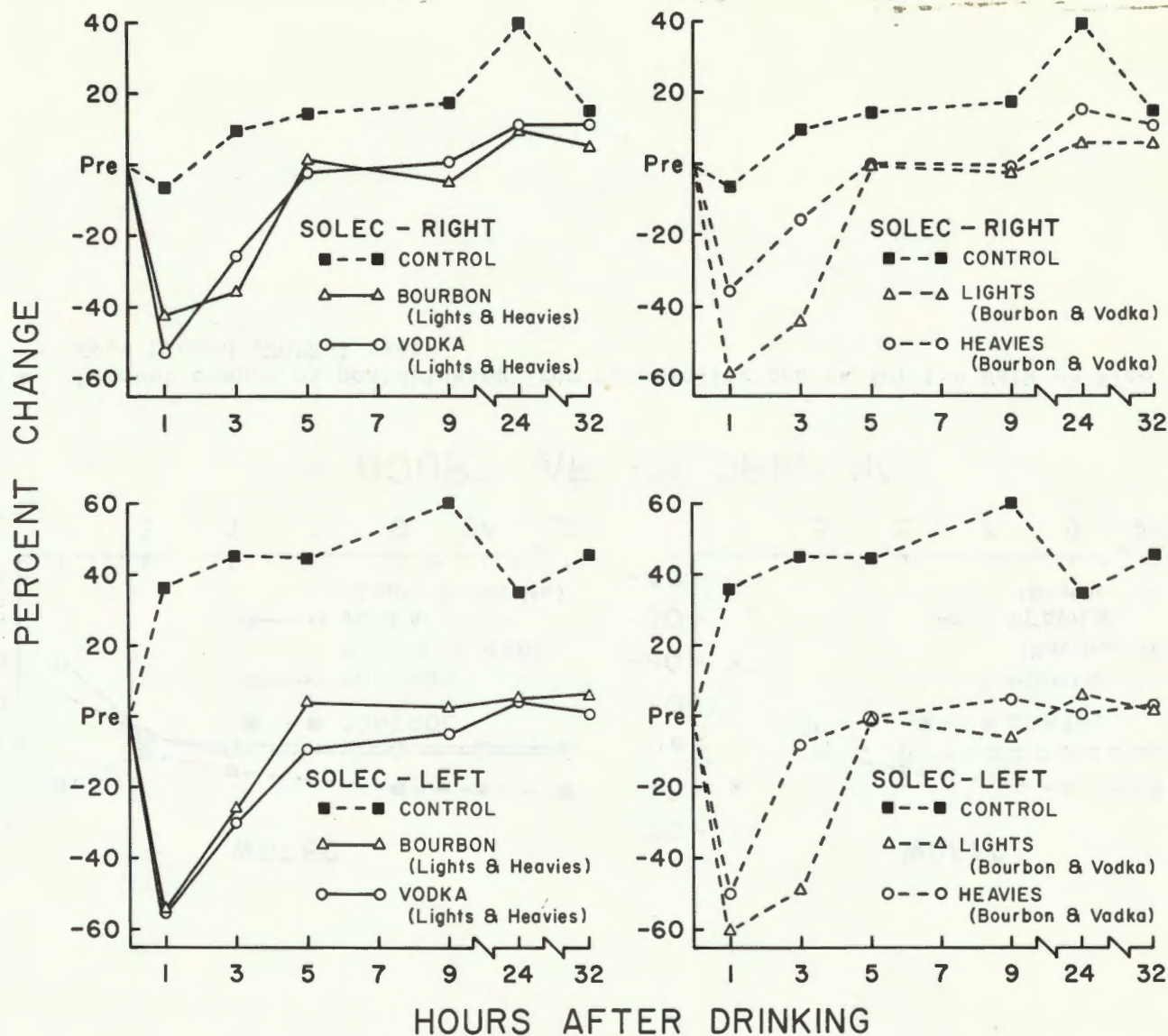


Figure 3. Percent change of postdrinking from predrinking scores for the Stand on One Leg, Eyes Closed tests for the right (SOLEC-R) and left (SOLEC-L) legs.

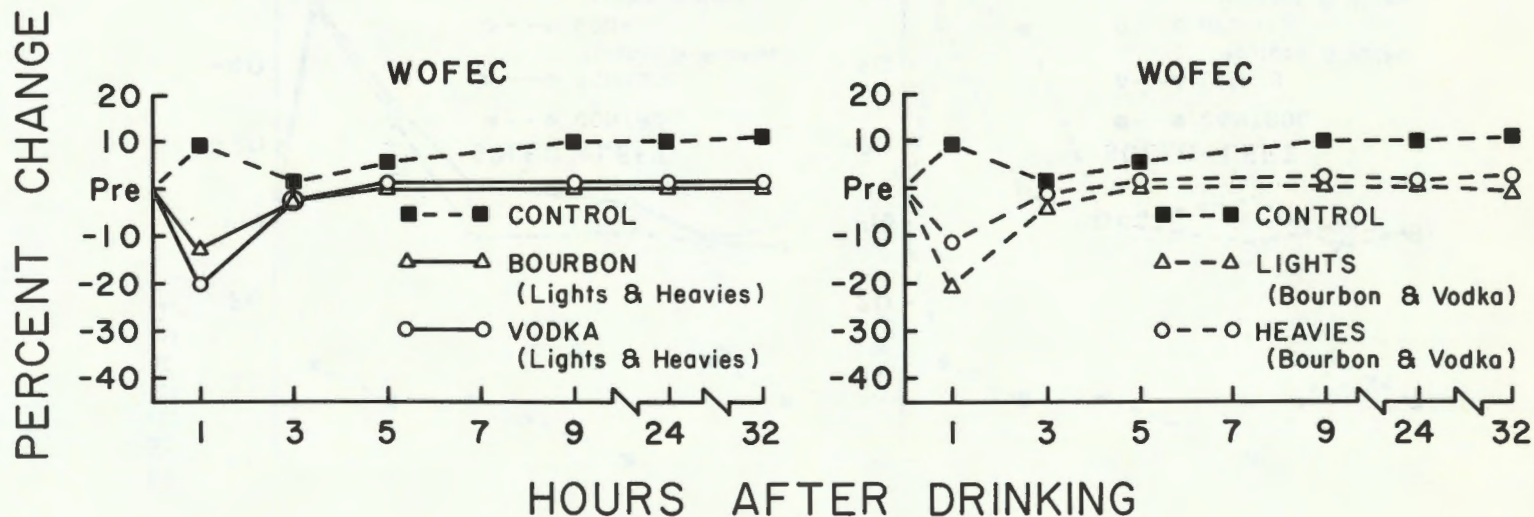


Figure 4. Percent change of postdrinking from predrinking scores for the Walk on Floor, Eyes Closed (WO FEC) test.

Walk on Floor, Eyes Closed (WOFEC). Control subjects displayed improved WOFEC performance on posttesting while an increase in ataxia during acute intoxication was evident for subjects given alcohol (Figure 4). Simple analyses of variance yielded no significant effects for the control group, i.e., essentially no change across sessions, but significant F ratios ($p < .01 - .001$) for all alcohol groups. Specifically, both groups of light drinkers and the heavy drinkers given bourbon had poorer performance 1 hour after drinking than they had prior to drinking. All alcohol groups did significantly better ($p < .05 - .001$) 9, 24, and 32 hours after drinking than they did 1 hour after drinking. In addition, (i) scores 5 hours after drinking were better ($p < .05 - .001$) than those of the first postdrinking session for all alcohol groups except the Bourbon-Heavies, and (ii) the light drinkers given vodka were significantly better ($p < .01$) 3 hours after drinking than they were 1 hour after drinking.

While the largest declines in performance 1 hour after drinking occurred for subjects in the two groups of light drinkers (18 percent and 25 percent for bourbon and vodka, respectively), overall statistical analyses of difference scores indicated that all of the alcohol groups were significantly poorer in performance than control subjects during the first postdrinking session ($p < .05$ level for the Bourbon-Heavies; $p < .01$ for the others). Recovery was rapid on this relatively gross measure of ataxia so that by the next testing session, mean scores for the alcohol subjects were very near their respective baseline levels and no other statistically significant findings were obtained.

IV. Discussion.

Separate analyses of variance for each group on each of the five ataxia tests yielded only three nonsignificant F ratios across sessions; all three were for the control group. The two significant Fs ($p < .05$) for the control group were based on improved performance in later sessions. Significant F ratios were obtained for each of the four alcohol groups on every test ($p < .01 - .001$); further analyses of these 20 group-by-test differences revealed that 17 involved significantly poorer performance 1 hour after drinking than during baseline (and other) tests, another involved a significant decrement from baseline (and other sessions) 3 hours after drinking, and the remaining two involved significantly poorer scores 1 hour after drinking than during subsequent sessions. Thus, the decrements during sessions 1 hour and 3 hours after drinking were the only ones to yield significant effects with other sessions for the alcohol groups.

The acute effects of moderate alcohol ingestion were apparent in all of the ataxia measures. This increase in ataxia following drinking is consistent with the earlier findings of Miles (17), Carlson et al. (3), Goldberg (9), Alha (1), Pihkanen (21), and Kelly et al. (16). The detrimental effects of

alcohol had generally dissipated by the fifth hour after drinking. This recovery process was approximately identical for all measures of ataxia with the exception of the WOFE test which presented little difficulty for the subjects and showed recovery by the third hour after drinking. While Fregly, Bergstedt, and Graybiel (7) reported that there was still some indication of the influence of alcohol intoxication in the performance of subjects on the Sharpened Romberg as long as 6 hours after drinking, the performance levels for our intoxicated subjects were very near predrinking levels 5 hours after drinking.

In testing the differences between groups, significant overall effects for the five ataxia tests were obtained in every case 1 hour after drinking ($p < .05 - .01$), for four of the five tests 3 hours after drinking ($p < .01$ in all cases), and in one case 9 hours after drinking (SOFE-L; $p < .05$). These 10 significant overall Fs subsequently yielded 27 significant differences between groups (out of a possible 100); of these, 22 involved differences between the drinking groups and the control group (9, 7, 4, and 2 significant differences for the Bourbon-Lights, Vodka-Lights, Vodka-Heavies, and Bourbon-Heavies, respectively). Of the remaining five differences, three represented poorer scores for the Vodka-Heavies as compared with each of the other alcohol groups and one difference each involved poorer scores for the Vodka-Lights and the Bourbon-Lights (both vs. the Bourbon-Heavies). Thus, most of the obtained overall differences between groups involved poorer performance of the alcohol groups as compared with the control group, there were no differential effects attributable to congeners in the alcohol, and there were more decrements for light drinkers than for heavy drinkers.

In general, light drinkers displayed greater increases in ataxia following drinking than did heavy drinkers with the exception of performance on the WALEC test. There were also indications that the recovery process was slower for light drinkers than for heavy drinkers. It is unlikely that these differences between groups can be accounted for by the slight differences in mean BALs (4 mg percent) between the light and heavy drinkers. These results, moreover, are consistent with the findings of Goldberg (9), even though subjects in our sample of young men had only had a few years to develop their drinking (and coping) habits as compared with the 40- to 50-year-old adults in Goldberg's study.

While there were differences among the groups in the amount of ataxia produced, there was no convincing evidence that either bourbon or vodka produced greater ataxia. In this study, vodka more often resulted in poorer performance than did bourbon, however slight the differences within sessions and tests. What is clear, however, is that the high-congener bourbon failed to produce a greater effect than the "noncongener" vodka. These findings agree with conclusions reached in other vestibular-related studies by Hill, Collins, and Schroeder (12), and Hill, Schroeder, and Collins (13) where

bourbon, when compared to vodka, failed to elicit greater PAN, more hangover symptoms, or more nystagmus to angular or to coriolis stimulation. Perhaps congeners in larger amounts than the moderate levels used in our work are required to produce differential effects (cf. 19,20,22).

While there is little indication in our data of any ataxia disturbances during the hangover period, the chronic abuse of alcohol apparently does lead to a disturbance in gait (10). The group of alcoholics studied by Goldstein et al. (10) had been abstinent for at least a week, yet their ability to negotiate the Heath Rail Walking Test was significantly impaired from that of a control group. After being tested every second day for 20 days, their rail-walking ability reached the same level as that exhibited by control subjects during the initial (and final) session. If standardized values for the various tests were provided, subsequent impaired performance could be used to indicate individuals who may have a drinking problem. Fregly and Graybiel (8) have provided a set of norms for ataxia performance on the battery (military subjects) that could be used if an alcoholic comparison were available.

Since the effects of alcohol were fairly similar in all measures of the ataxia battery used in this study, it is not clear how much additional information is provided by the use of several tasks. In terms of the time required to complete the full battery, these findings would suggest that for routine clinical use, an examiner could continue to use performance on the Sharpened Romberg as an adequate measure of ataxia without losing an appreciable amount of information. Moreover, the Sharpened Romberg was the only test in this study that did not show some effects of learning (improvement) with repeated trials for control subjects. It is possible, of course, that the assorted measures of ataxia in the battery may be differentially affected by various neurological or otological problems, but no supportive data for such differentiation are currently available.

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