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ACUTE ALCOHOL ATAXIA IN RELATION TO VESTIBULAR FUNCTION

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SUMMARY PAGE

THE PROBLEM

What is the role of the vestibular organ in causing ataxia during alcohol intoxication?

FINDINGS

Severe loss of vestibular function appears to lessen rather than to enhance the intoxicating effects of alcohol on postural equilibrium functioning. The severity and duration of ataxia induced were generally less than that observed previously in vestibular-intact individuals. The superimposition of an "acute alcohol ataxia" on vestibular-impaired individuals appears to depend upon the degree to which nonvestibular functions can be made to compensate for the initial characteristic vestibular ataxia. The likelihood that vestibular defect is compensable via nonvestibular learning processes in one subject with outstanding performance capabilities is discussed.

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INTRODUCTION

In a previous study of individuals with unimpaired vestibular function (3), moderate intoxication was sufficient to induce a significant decrement in motor performance as revealed by a quantitative postural equilibrium test (PET) battery and a clinical ataxia test (CAT) battery (10). Because the combination of graded tasks constituting the PET and CAT batteries was known from previous studies to be differentially sensitive to clinical or experimentally induced vestibular dysfunction (4,5,10,12,13), it had been expected that the sensitivity of the test batteries would likewise be present under conditions of alcohol intoxication. It was anticipated that alcohol would yield greater performance decrements on the more vestibular-dependent tasks (nonvisual walking and standing) than on the less vestibular-dependent tasks (visual walking and standing). It was found instead that the decrements were fairly uniform in terms of their magnitudes and rates of onset and recovery. Moreover, there were no systematic relationships between the extent of the decrements and the levels of baseline performances. The differential sensitivity of the tests may have been insufficient to detect nonuniform decrements, or these decrements may have been masked by certain nonvestibular effects of the alcohol.

Previous research has indicated that between-test differentials on the PET and CAT batteries are substantially greater for labyrinthine-defective individuals (LDs) than for normals (Appendix A). Because of this greater sensitivity, it was anticipated that by similarly testing LD individuals we could better assess the quantitative vestibular effects of alcohol on postural equilibrium functions.

This report presents findings of two experiments (I and II) with LD subjects; both are replications of the previously described study involving normal subjects (3). In Experiment II modifications were made to certain tests in the PET battery to adjust baseline performance levels.

EXPERIMENT I

PROCEDURE

Subjects

The participants were four males, 27,33,46, and 50 years of age who with others constitute a comprehensively studied group of LDs with virtual, if not complete, absence of vestibular function (7-9, 11, 14). Some previous clinical and experimental findings on the group are summarized in Appendix B.

METHODS

PET and CAT Batteries. The distinct tests comprising the batteries were administered in the following sequence: 1) Sharpened Romberg (SR) consisting of standing on the floor with eyes closed for 60 seconds; 2) PET battery (Short Version)*; 3) standing on one (each) leg on the floor with eyes closed for 30 seconds (SOLEC-R and SOLEC-L Tests); and 4) walking a 12-foot line on the floor with eyes closed (WALEC), scored as inches of deviation from the line. The PET consisted of 1) walking with eyes open (Walk E/O Test)+ on a $\frac{3}{4}$-inch by 8-foot rail, scored as number of steps (maximum of five steps per trial); 2) standing with eyes open (Stand E/O Test) on the $\frac{3}{4}$-inch rail, scored to the nearest second (maximum of 60 seconds per trial); and 3) standing with eyes closed (Stand E/C Test) on a $2\frac{1}{4}$ by 30-inch rail, also scored to the nearest second with a maximum of 60 seconds per trial.

The body position required of all subjects was: a) body erect or nearly erect, b) arms folded against chest, and c) feet, shoes on, tandemly aligned heel-to-toe (SOLEC excepted).

The best three trials out of five constituted the scoring of the PET battery, with maximum scores obtainable being 15 (steps) on the Walk E/O and 180 (seconds) on the Stand E/O and Stand E/C tests. Four SR and five SOLEC trials were administered. A perfect first trial on the SR was weighted 4, and a score of 240 assigned; a perfect first trial on the SOLEC was weighted 5, and a score of 150 assigned. Perfect scores on later trials were assigned progressively less weight; as soon as subjects received a perfect score, they were no longer tested but were given a perfect score for the remaining trials (10). The WALEC was scored in terms of the best two of three scorable trials.

Pre-experimental practice sessions were held to stabilize baseline performance levels. Experimental testing took place at 30, 60, 120, 180, 270, 360, 420, and 480 minutes following the administration of alcohol.

History of Drinking Questionnaire. Administered prior to alcohol stimulation, this questionnaire consisted of open-ended autobiographical questions relating to duration, frequency, and amount of social drinking; the types of alcohol consumed; the circumstances surrounding alcohol consumption; intoxication effects experienced in relation to before- versus after-meal drinking; and several related questions in rating scale form.

Alcohol Stimulus. On an empty stomach, each subject consumed during a fifteen-minute period 80-proof vodka on the first experimental day and 100-proof vodka on the second experimental day (48 hours later). The mixture was four parts orange juice to one part vodka in the amount of 2.2 cc per kilogram body weight.

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*A Long Version, which employs six rails of varying widths, from which the Short Version evolved, was described fully together with the Short Version in a previous publication (10).

+Formerly called the Walk H/T Test.

Blood Samples. Blood was taken from the ante-cubital vein prior to alcohol consumption and again at approximately 30, 60, 180, and 270 minutes following alcohol intake. Blood alcohol levels were determined utilizing Natelson's microtechniques (18).

Electronystagmography. Positional alcohol nystagmus (PAN) was recorded on a direct writing two-channel AC-recorder (Sanborn), with a time constant of two seconds. Electrodes were placed near the lateral canthus of each eye and above and below the left eye, and a ground electrode was placed on the forehead. All recordings were made with subjects' eyes closed (1). Records were obtained at 30, 60, 120, 180, 270, 360, 420, and 480 minutes after alcohol intake, while subjects were supine on a couch with the head in alternate left and right positions.

Experimental Diary. The time-course of subjectively determined appearance and disappearance of behavioral changes along the time axis on each experimental day was obtained by means of a printed diary, or log, which subjects were carefully instructed to use (3).

Nourishment. Lunch was served four hours after alcohol intake. Light snacks consisting of cheese, crackers, or potato chips were permitted during a two-hour period preceding lunch.

RESULTS AND DISCUSSION

To facilitate quantitative comparisons of results in LDs with results in the previously-studied normals, the PET and CAT findings (PAN excepted) appear as a composite illustration (Figure 1).

No PAN was evidenced by any LD subject in either left or right lateral head positions. This negative finding corroborates earlier results of a different experiment in this laboratory with some of these LD subjects (15), as well as with results of experiments elsewhere (1) which attempted to elicit PAN in an analagous group of bilateral labyrinthine-defective individuals. Inasmuch as unilateral vestibular function was found to be sufficient to elicit both phases of PAN (1), it would be worthwhile to try to determine the extent to which 1) residual unilateral and, 2) bilateral vestibular function might be sufficient for the elicitation of PAN.

As evidenced by Figure 1, visual rail-walking was the only test on which LDs demonstrated performance decrements following alcohol administration. Maximum visual-rail-walking decrements approaching statistical significance ($P < .10$, by Mann-Whitney U test, ref. 17) occurred at about 70 minutes following the intake of 80-proof vodka and at 45 minutes after 100-proof ingestion. Two hours after ingestion of 80-proof and three hours after ingestion of 100-proof vodka the baseline performance levels were surpassed. Maximum improvements (over baseline levels) were seen between the third and fourth hour during the 80-proof experiment and between the sixth and seventh hour during the 100-proof experiment. These maximum improvements were not maintained, however, possibly due to the boredom of the experimental situation.

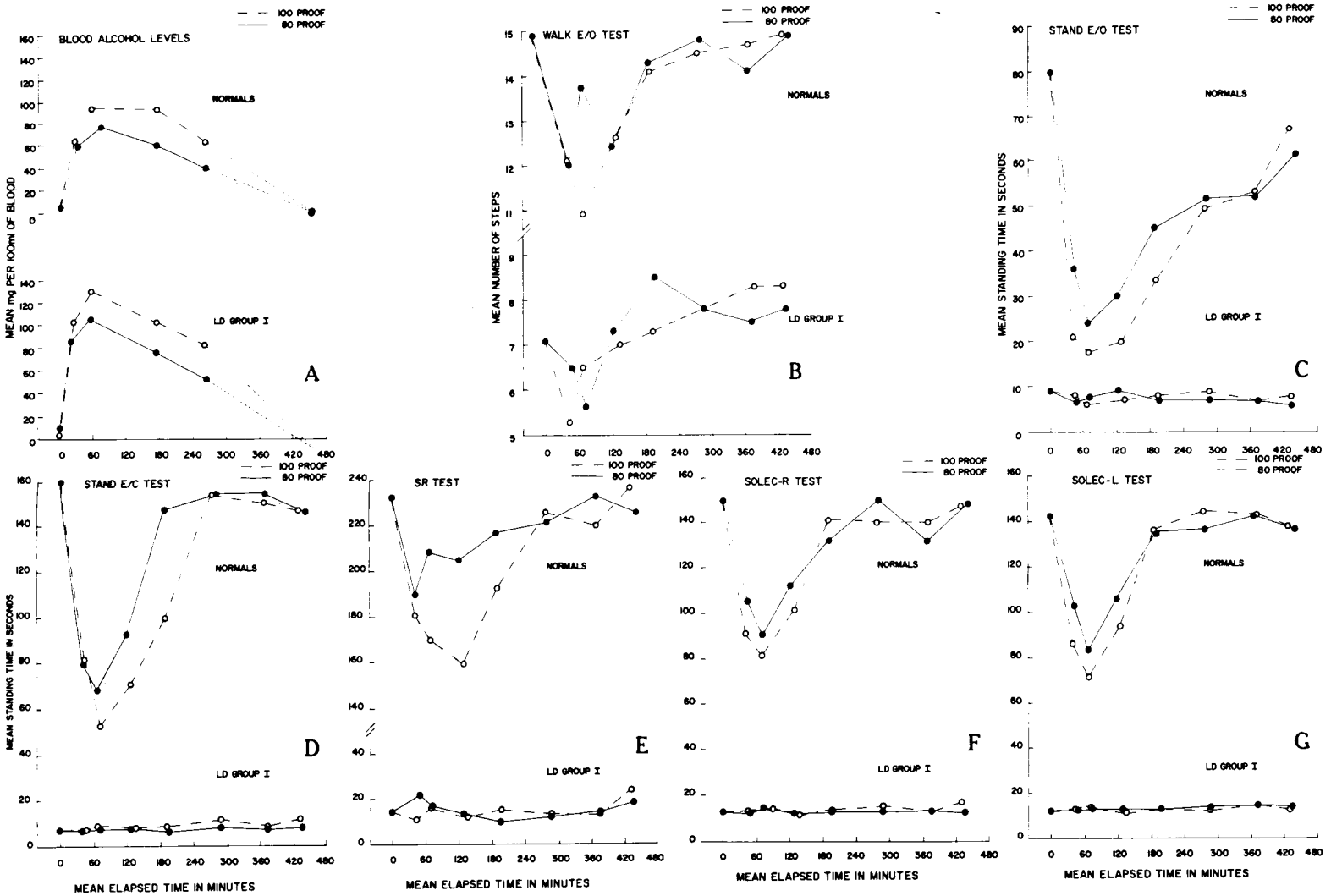

Figure 1

Comparisons of 80-proof and 100-proof vodka effects in a group of four bilateral labyrinthine-defective subjects (DO, GR, HA, MY) with a group of thirteen vestibular normal subjects: A) blood alcohol levels, B) walking with eyes open on a 3/4-inch-wide rail, C) standing with eyes open on the 3/4-inch rail, D) standing with eyes closed on a 2-1/4-inch-wide rail, E) standing on floor with eyes closed, F-G) standing on one leg (on floor with eyes closed).

That improvements over baseline levels were noted during the experiment may reflect either additional acquired compensation as a result of the oft-repeated experimental testing, or a rebound of activity of those structures suppressed by alcohol. Such a rebound has been posited as the explanation of PAN reversal during the hangover period (16). This pattern was not seen in the normal subjects, even on the task having insufficient "top" in terms of difficulty (visual rail-walking). Further investigation is required to determine whether the pattern is specific to LD subjects. Extending the duration of the experiment coupled with the administration of a more difficult visual rail-walking task might show similar results in normals.

That decrements in performances by the LD group were observed only on the visual rail-walking task is consistent with the finding that visual rail-walking is the only task for which the LD pre-experimental performance level was above the alcohol-depressed level shown in the normal group. For the visual rail-standing task and all of the nonvisual (floor and rail) tasks, the characteristic noncompensable decrements shown pre-experimentally were far greater than the severity of decrements due to alcohol in the normals; i.e., performances were initially so poor as to prevent further decrement even with potent alcohol stimulation.*

Although the magnitude of blood alcohol concentration was somewhat higher in LDs than in normals (Figure 1), the time-course was much the same for both groups. Likewise parallel relationships between the rate of rising blood alcohol levels and the rate of decrease in visual rail-walking performance scores were present in both LDs and normals. Despite the higher blood alcohol concentration level in the LD group, there was no corresponding increase in performance decrements. This finding suggests a differential effect of alcohol on the rail-walking performances of normals and LDs with similar blood alcohol concentrations. This may in part be attributable to the initially lower baseline performances of LDs, or to the superiority of LDs over normals in using nonvestibular cues in difficult walking and standing tasks.

The history questionnaire responses indicated minimal alcohol consumption in three subjects and irregular drinking in the fourth subject. Two subjects reported a two- to four-year history of drinking, and the remaining two a 28- to 30-year history of drinking. All other questionnaire responses were similar to those given by the normal subjects.

The behavioral effects of the alcohol upon three of the LDs were similar to those observed in normals; i.e., they were typical of alcohol intoxication. The fourth subject reported that he merely felt relaxed and fatigued. Interestingly, one subject reported the onset of tinnitus, and another noted a characteristic increased pitch of his chronic tinnitus--a phenomenon, the subject reported, which allows him to gauge the extent of his social drinking.

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* The characteristic vestibular ataxia of the LDs is so intense that none was able to meet the minimum criterion for a scorable trial on the WALEC Test.

Full intoxicating effects were felt within sixty minutes of alcohol intake, and included in one or more subjects the following: blurred vision, instability, relaxation, feeling of well-being, lessened physical control, mellowness, and elevated mood. These symptoms were markedly less some one and one-half hours after intake and were replaced by sleepiness, fatigue, and boredom. Within four hours of alcohol intake all experimentally induced symptoms had disappeared.

Notably absent were such symptoms as headache (except in one subject), tremor, and nausea which were indicated in the history questionnaire as being effects induced by alcohol in doses sufficient to produce hangover. Other alcohol effects on the LDs were disclosed by interviews with the subjects and included heightened pain sensitivity (one subject), tendency toward feelings of detachment from the surroundings (one subject), increased talkativeness (one subject), and improved balance test performances (two subjects). Effects of the 100-proof vodka were reported as being about equal to the 80-proof effects. Neither dosage produced hangover in any LD subject. In general, the behavioral effects were somewhat less to considerably less in the LDs than in the normals (3), even in the two LDs with as brief a drinking history as that reported by the normals.

The present findings permit the following tentative conclusions:

1. The superimposition of an "acute alcohol ataxia" in individuals with markedly impaired vestibular function appears to depend upon the degree to which nonvestibular functions can be made to compensate for the baseline characteristic vestibular ataxia.

2. The ataxia induced by medium doses of alcohol was generally of lesser severity and duration in individuals with pronounced bilateral labyrinthine defects than in vestibular intact individuals.

3. Severe loss of vestibular function, rather than increasing the intoxicating effects of alcohol on postural equilibrium functioning, appears to lessen the effects.

4. The qualitative behavioral changes reported by the LD subjects in response to alcohol appear to be of lesser severity and duration than in vestibular normal subjects.

5. PAN appears nonelicitable in individuals with total or nearly total absence of vestibular function.

EXPERIMENT II

Because baseline levels of performance are essential to define the extent of decrements as well as for between-group comparability of alcohol effects, three other members (ages 25,37, and 38) of the same group of LDs (Appendix B) were studied with the following changes in experimental conditions: 1) the partially compensable visual rail-walking and minimally compensable visual rail-standing tasks (Appendix A) were made easier by the use of wider rails, allowing the LD subjects to approach "normal" performance in terms of a relative baseline, and 2) one of the standardized nonvisual tasks performed on the floor (Sharpened Romberg) was repeated.*

METHOD

To equate baseline levels of performance difficulty with normals as much as possible, "optimum" widths of rails for the walking and standing-with-eyes-open tasks were selected from the standardized Long Version of the PET battery. These were of sufficient width merely to permit several repeatable perfect scores during the morning and afternoon practice sessions held for four days immediately preceding the experimental day. To each individually determined optimum rail were added several successively narrow rails to provide a "top" to each "tailor-made" PET battery. Unlike previous studies which employed the Long or Short Versions of the PET battery, within-group differences in baseline performance scores were thus highly controlled, each subject's baseline representing a performance near his maximum, but of sufficient difficulty to yield a decrement under less than standard conditions.

All subjects also practiced the SR test as often as they did the PET battery. During both the experiment and the practice sessions the tests were administered in the following sequence: 1) SR, 2) walking with eyes open on rails, and 3) standing with eyes open on rails.

As before, blood alcohol levels were determined,+ the drinking history questionnaire was administered, and a log of the subjects' behavior was kept. Testing for PAN was not repeated because of negative findings in Experiment I. Moreover, as only one experimental day was deemed sufficient to realize the purposes of the additional study, 100-proof vodka was chosen so as to maximize comparability of the findings with previous results. In contrast with the eight-hour duration previously employed, the present experiment was limited to six hours duration; blood sampling and performance testing were done at the same intervals as in Experiment I.

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*Fuller realization of this experimental design was hampered by nonresolution of the methodological dilemma that even the simplest nonvisual tasks failed to improve with practice and could not be made easier, since these were performed on the widest platform available--the floor.

+Blood was drawn from the ear lobe at the intervals indicated in Experiment I, and blood alcohol concentrations were determined by a microtechnique using gas chromatography (2).

RESULTS AND DISCUSSION

The over-all mean baseline performance scores shown in composite Figure 2 represent the pre-experimental performances of the three LD subjects on their individually-tailored rails. Visual rail-walking performance decrements were again observed in the LDs. The magnitude of the decrements, the rate at which maximum decrements occurred, and the onset and recovery periods were nearly identical to previous results despite the reduced level of difficulty of the task. In the normals (Figures 1 and 2) maximum decrement occurred later (70 minutes after alcohol intake versus 33-45 minutes in the LDs), the magnitude of maximum decrement was greater (mean loss of 4 steps versus a loss of only 2 steps in the LDs), and recovery was later (7 hours versus $2\frac{1}{4}$ - 3 hours in the LDs) under the influence of the 100-proof vodka.

Unlike the previous LD group, the second LD group showed visual rail-standing performance decrements, remarkably similar to those shown by normals, at least in terms of magnitude (maximum loss of 55 seconds in the LDs versus 62 seconds in the normals) (Figure 2). In the normals, maximum decrements in the visual rail-standing occurred later (at 70 minutes after alcohol intake versus 33 minutes in the LDs), as did recovery of losses (7 hours versus $2\frac{1}{2}$ hours in the LDs). As with the previous LD group, the performances on the visual tasks eventually surpassed the baseline levels.

A substantially higher SR baseline level (Table I) than that observed in the previous LD group (Figure 1), was a totally unexpected finding, due primarily to the overwhelming improvement with practice of one exceptional subject, ZA. This subject had several perfect scores on the first trial of the SR test during several of the practice sessions. His sporadic perfect performances were typical of vestibular intact individuals in that minimum limb, body, or head movements were necessary to maintain his erect standing position.

Table I

Influences of 100-Proof Vodka on the Sharpened Romberg Test Performances of The Three Labyrinthine-Defective Subjects of Experiment II

		Individual Performances				
		Scores During Alcohol Stimulation				
Subject	Baseline Score	34'*	72'	134'	184'	272'
JO	18.7	11	17	19	12	16
PE	18.9	14	10	22	33	31
ZA	152.0	32	60	138	93	28

*Elapsed time in minutes since alcohol intake.

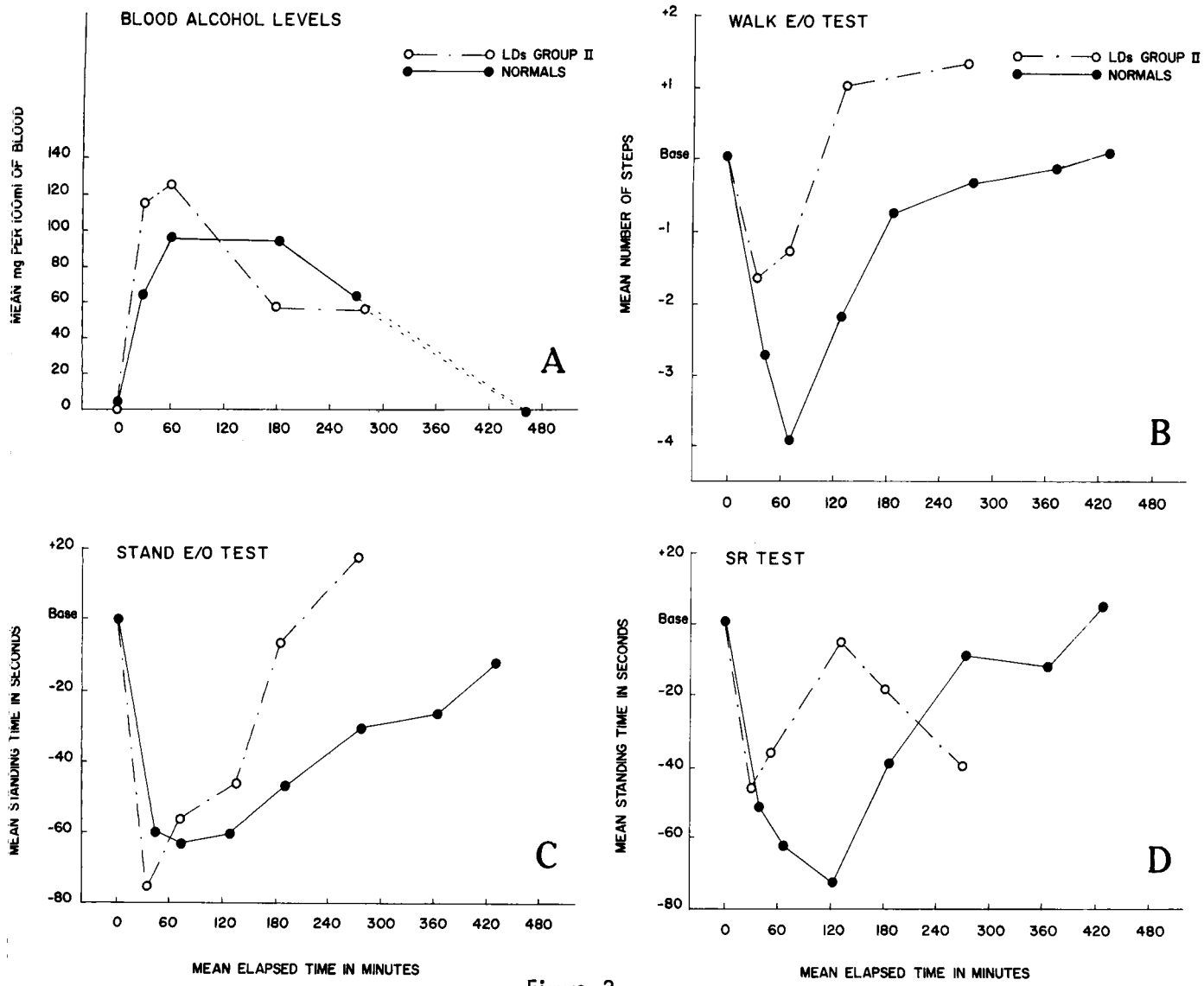

Figure 2

Comparison of 100-proof vodka effects in a group of three bilateral labyrinthine-defective subjects (JO, PE, and ZA) with a group of thirteen vestibular normal subjects: A) blood alcohol levels, B) rail walking with eyes open, C) rail standing with eyes closed, and D) standing on floor with eyes closed.

Had subject ZA consistently maintained perfect SR performances throughout the practice period, the mean baseline level of this LD group (Figure 2) would have been considerably higher although not so high as the mean baseline scores in normals (232.5) nor even so high as the maximally reduced mean scores of the normals (159.5) during 100-proof intoxication. Even subject ZA's baseline level (mean score of 152) was less than the alcohol-depressed SR level in the normals. Nevertheless, mean SR performance decrements during alcohol intoxication occurred in all three LDs, with the greatest decrement shown by subject ZA (Table I). As was found with the visual rail tasks, maximum SR decrements occurred much sooner and recovered much sooner than in the normals. Within the LD group subject ZA was least able to maintain his recovery (Table I). His final score reverted to the level of maximum decrement due to alcohol, but this probably reflected his extreme drowsiness (he slept considerably between testing), in conjunction with a severe headache for which he required aspirin. The SR performances of subjects JO and PE regained and surpassed their baseline levels, a finding consistent with the rebound possibility discussed above.

Interpretation of the unexpected findings on subject ZA is dependent upon accounting for his startling improvement in SR test performance during the practice sessions. In view of the nonvisual nature of the task, such a high degree of compensation necessarily reflects either residual vestibular function, a greater ability to compensate through nonvestibular functions than is generally considered possible (6), or an interaction of residual vestibular function with utilization of nonvestibular cues. Albeit so great a compensation by means of nonvestibular functions alone seems unlikely, nevertheless a vestibular source of compensation, either residual semicircular canal and/or otolith functioning, seems more unlikely. ZA has failed repeatedly to show any objective or subjective vestibular responses to brief or sustained rotation (7,8,11,14) or to intense, sustained ice-water caloric stimulation.* Moreover, although he is one of the six LDs who have perceived the oculogravic illusion (9), he no longer perceived the illusion when tested under water in another experiment. Furthermore, his counterrolling index is poorer than that of his cohorts (8). Thus all other testing of vestibular function to date indicates certain loss of canal functioning and extreme, if not total, loss of otolith functioning in this subject. On the other hand, ZA was afflicted by meningitis considerably earlier in life than all except one of his cohorts (Appendix B), thus affording him a greater opportunity for proprioceptive sources of compensation for his labyrinthine defect. In addition, he is an individual with outstanding motor coordinative abilities. Present experimental findings therefore most likely reflect nonvestibular rather than vestibular origins of his improved SR performance and the superimposition of an acute alcohol ataxia upon his previous nonvisual ataxia.

The blood alcohol levels of the LDs of Experiment II during the earliest periods of sampling (Figure 2) were quite similar to those calculated for those of Experiment I (Figure 1); in the succeeding two periods of sampling the blood levels were more similar to those of the normals. Since rate and extent of behavioral changes are functions of the rising blood alcohol concentrations (16), the greater concentration in the LDs than in the

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*Unpublished data, 1966.

normals would reasonably be expected to reflect more rather than less decrements and slower rather than faster recovery than seen in the normals. The between-group differences observed, with LDs showing smaller performance decrements and more rapid recovery, warrant the suggestion that gross vestibular dysfunction may lessen the alcohol effects upon the skills studied.

The subjects' accounts of the 100-proof effects were limited to physical complaints. These included, in one or more subjects, numbness, unsteadiness, double vision, muscular incoordination, sluggishness, "very slight dizziness," fatigue, and sleepiness. The uniform outstanding complaint was that of numbness. None of the subjects experienced morning aftereffects of the experimental alcohol intoxication.

Generally, results of Experiment II support the previous results with LDs. Decreases in levels of performance on the visual tasks were again found to be less than those observed previously in normals. Ability to compensate performance skills on a nonvisual task did occur unexpectedly in one LD subject, seemingly due to outstanding utilization of nonvestibular (tactile-kinesthetic) cues in that individual. The tentative conclusion from the initial study with LDs that absence or near absence of vestibular function appears to lessen the effects of alcohol was reinforced in the repeat experiment.

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APPENDIX A

A DEFINITION OF VESTIBULAR ATAXIA

In the presence of known or ascertainable severe vestibular defect, vestibular ataxia is defined as initial 1st percentile performance levels in relation to normative standards on the PET and CAT batteries (main text references 4, 5, 10, 13).

Ability to compensate for the characteristic vestibular ataxia of bilateral labyrinthine-defective subjects was established with a practice regimen (10), and in terms of type and severity may be classified as follows:

Type I. Partially compensable ataxia (ataxic visual rail walking), as indicated by marked improvements with extended practice in the ability to walk with eyes open on rails of various widths. (All subjects' initial scores improved considerably, and the improvements of some subjects attained a level commensurate with average, or better, initial performance levels of vestibular normal individuals.)

Type II. Minimally compensable ataxia (ataxic visual rail standing), as indicated by only slight improvements with extended practice in the ability to stand with eyes open on rails of various widths. (Initial 1st percentile performance levels were maintained despite considerable raw score improvements.)

Type III. Totally noncompensable ataxia (ataxic nonvisual rail or floor walking and standing), as indicated by lack of improvements with extended practice in the ability to stand with eyes closed on rails of various widths or even in the ability to walk or stand with eyes closed on the floor. (Initial 1st percentile levels and the raw scores themselves were virtually unchanged.)

This classification was promulgated by virtue of the uniform stringent body position required of subjects in the testing of these representative abilities subserving postural equilibrium functioning, i.e., body erect, arms folded against chest, and feet (shoes on) tandemly aligned heel-to-toe.

The observation that visual railwalking ability proved in general to be highly but not totally compensable after extended practice underscores the observation that a true vestibular ataxia prevented better than 1st percentile level performance scores in the majority of the subjects upon initial testing.* Indeed, the compensation demonstrated was generally "hard won" and somewhat less indicative of the skills tested than the improved performance scores would indicate. For example, the LDs readily discovered and exhaustively exploited such "gimmicks" as rapid staccato-like walking patterns to increase their momentum, and they persistently used extraordinarily careful starting positions. With the exception of one outstanding subject (ZA), who scored initially at the 50th percentile level, little of the ease, poise, and grace of smooth, continuous, well-coordinated movements at average walking speeds, so characteristic of vestibular intact individuals during unpracticed rail walking, were present in the improved, practiced performances of the LD subjects.

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*Three exceptional LDs scored initially at the 7th, 10th, and 50th percentile levels.

APPENDIX B

CLINICAL FINDINGS IN SEVEN DEAF SUBJECTS WITH BILATERAL LABYRINTHINE DEFECTS

Clinical Findings in Seven Deaf Subjects with Bilateral Labyrinthine Defects

		Auricular Defects		Hearing*		Nystagmus Response Caloric Test		C-R Index** Max. Tilt‡	
Subject	Age	Etiology	Age Onset	R	L	R	L	50°	75°
DO	46	Meningitis	13	Nil	Nil	Nil	Nil	74	74
GR	50	Mastoiditis	12	Nil	160	Nil	Nil	60	—
HA	33	Meningitis	13	Nil	Nil	Nil	Nil	47	53
MY	27	Meningitis	8	Nil	Nil	Nil	Positive+	63	82
JO	38	Meningitis	7½	Nil	Nil	Nil	Nil	126	176
PE	37	Meningitis	12	Nil	Nil	Nil	Nil	21	30
ZA	25	Meningitis	3½	≥135	≥130	Nil	Nil	22	36

* Thresholds of response to high intensity noise (max. 160 db.)

+ Irrigation 40 sec. water temp 7.9° C.

**One-half the sum of maximum counterroll (min. of arc) with right and left body tilt at 25°, 50°, and 75°.

‡ Angular displacement of body from vertical in frontal plane.

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Security Classification

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ACUTE ALCOHOL ATAXIA IN RELATION TO VESTIBULAR FUNCTION

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5. AUTHOR(S) *(Last name, first name, initial)*

Fregly, Alfred R.; and Graybiel, Ashton, Capt MC USN

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13. ABSTRACT

Determination of alcohol effects on postural equilibrium of bilateral labyrinthine defective individuals was made to aid in the elucidation of the functional role of the vestibular organ in man. Generally, severity and duration of the intoxicating effects were found to be less than that observed in a previous study on vestibular-intact individuals. The superimposition of an "acute alcohol ataxia" on vestibular-impaired individuals appears to depend upon the degree to which nonvestibular functions can be made to compensate for the initial characteristic vestibular ataxia.

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Ataxia						
Deafness						
Labyrinthine defects						
Postural equilibrium						
Psychomotor skills						
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Determination of alcohol effects on postural equilibrium of bilateral labyrinthine defective individuals was made to aid in the elucidation of the functional role of the vestibular organ in man. Generally, severity and duration of the intoxicating effects were found to be less than those observed in a previous study on vestibular-intact individuals. The superimposition of an "acute alcohol ataxia" on vestibular-impaired individuals appears to depend upon the degree to which nonvestibular functions can be made to compensate for the initial characteristic vestibular ataxia.

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Vestibular apparatus

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