The effect of alcoholic intoxication upon calorically induced oculogyral illusion

ROBERT B. WELCH, DAVID J. SCHROEDER, JANET K. THURGATE,
CARLTON K. ERICKSON, RAYMOND L. HIGGINS, and JOHN S. WAIT
University of Kansas, Lawrence, Kansas 66045

Ten male and 10 female subjects, half of whom were classified as "heavy" and half as "light" drinkers, ingested a 4-1 combination of tonic water and 80-proof vodka in an amount calculated to produce a state of moderate intoxication (average estimated peak blood alcohol level = .075). This resulted in an increased sensitivity (latency, magnitude, and duration) to the calorically induced "oculogyral illusion" (OGI) relative to a placebo (tonic water) condition. "Heavy" and "light" drinkers did not differ significantly on any of the measures. It was speculated that the alcoholic enhancement of the OGI was due either to a decreased ability to suppress vestibular nystagmus when attempting to fixate a visual stimulus or to the expenditure of extra-normal effort in order to maintain fixation.

Acute alcoholic intoxication has been found to detrimentally affect a number of human visual visuomotor capacities, including critical flicker fusion (Goldberg, 1943), peripheral signal detection during a central attention task (Moskowitz & Sharma, 1974), and compensatory visual tracking during vestibular stimulation (Collins, Gilson, Schroeder, & Guedry, 1971). It is the sense of balance, however, which probably suffers most from the ingestion of alcohol. Control over both body orientation and vestibular-controlled eye movements (nystagmus) deteriorates significantly in the intoxicated subject (Schroeder, 1971, 1972; Wallgren & Barry, 1970). In fact, involuntary eye movements known as "positional alcohol nystagmus" (PAN) may be detected as long as 24-32 h after the cessation of drinking (Hill, Collins, & Schroeder, 1973).

Stimulation of the semicircular canals, whether by bodily rotation, galvanic (electrical) stimulation, or caloric (water) irrigation of the ear, induces both vestibular nystagmus and a sensation of turning. The former is an involuntary ocular oscillation, involving a relatively slow turn of the eyes in one direction, followed by a rapid shift in the opposite direction. Furthermore, intense vestibular stimulation in the presence of visual stimuli often leads to illusory motion of the field in the same direction as the rapid phase of the nystagmus. This experience was first systematically investigated by Graybiel and Hupp (1946), who termed it the "oculogyral illusion" (OGI). The basis of the OGI remains un-

clear. However, Whiteside, Graybiel, and Niven (1965) have argued that it is, at least in part, an outcome of the motor outflow emitted by the subject as he attempts to counteract the slow phase of the vestibular nystagmus which is drawing his eye away from the point of visual fixation. This is essentially the same explanation accepted by some authors (e.g., Gregory, 1973) for the illusory motion or displacement of the field experienced during attempts to actively rotate an immobilized eye. Whiteside et al. showed that vestibular nystagmus will continue for a relatively long period if the subject is in total darkness. However, with the addition of a visual fixation point, the nystagmus disappears, although a strong OGI is experienced. Finally, when the light is extinguished, nystagmus reappears. It is clear, then, that the provision of a fixation point soon leads to the suppression of vestibular nystagmus, and perhaps it is this active suppression which is largely responsible for the OGI. Nevertheless, at the outset, the nystagmic eye movements are generally too strong to be completely suppressed. Therefore, this involuntary motion of the eyes could contribute to the initial portion of the OGI by leading to retinal image motion which is incorrectly attributed to the external visual field.

The effects of alcohol upon vestibular nystagmus have been examined by Schroeder (1971, 1972), who found that acute alcoholic intoxication reduces the felt sensations of turning resulting from vestibular stimulation, as well as the ability to suppress vestibular nystagmus when a visual fixation point is available. It is not immediately obvious what the implications of these results are for the OGI, and therefore the primary aim of the present experiment was to fill this gap in our knowledge of the effects of alcohol upon vestibular functioning.

A secondary goal was to examine the role of drinking experience in the effects of intoxication. Subjects were classified either as "heavy" or "light" drinkers on

This research was supported by a University of Kansas General Research Grant (3332-5038). The authors wish to thank Brad Barrett for assistance in testing and Kathe Koch for analysis of the breath samples. Reprint requests should be addressed to Robert B. Welch, Department of Psychology, University of Kansas, Lawrence, Kansas 66045. D. J. Schroeder is now at Veterans Administration Hospital, Topeka, Kansas 66622.

the basis of their responses to a "drinking inventory." It was assumed that the two types of drinkers would differ in their "tolerance" to alcohol and that this difference would be seen in a lesser effect of alcohol on the OGI for the "heavy" than for the "light" drinkers.

METHOD

Subjects

Ten male and 10 female students from the University of Kansas (ages 18-24) were selected for the study, based on their responses to the alcohol consumption inventory designed by Cahalan, Cisin, & Crossley (Note 1). This inventory asks about the subject's current frequency and rate of consumption of wines, distilled liquors, and beers, and allows him to be grouped into one of five drinking classes (heavy, moderate, light, infrequent, and abstinent). Equal halves of the male and female subjects were classified as either "heavy" or "light" social drinkers. Subjects categorized as heavy social drinkers reported currently drinking an average of 5-6 glasses of their favorite type of beverage (wines, distilled liquors, or beers) at least 2 or 3 times a month. Subjects classified as light social drinkers reported drinking an average of 1-2 glasses of their favorite beverage about once a month.

Participation in the experiment was voluntary, although each subject was paid \$10 after the second testing session. There was no evidence that any of the "heavy" social drinkers could be considered "problem" drinkers. People who indicated a susceptibility to motion sickness or who had ever suffered from a serious ear infection were excluded from the experiment.¹

Procedures

Prior to their arrival at the laboratory, all subjects were contacted and requested to abstain from the use of alcohol or other drugs for 24 h and from eating for 3 h preceding testing. (All subjects later indicated that they had complied with this request.)

Most subjects were run in pairs, in an alternating manner. In these cases, one subject was in the alcohol condition and the other in the placebo condition. Except during the induction and measurement of the OGI, the two were together and free to interact with one another. This procedure was used not only for efficiency but on the assumption that the subject in the placebo condition would be more likely to believe that he had ingested an alcoholic beverage if he was in the immediate presence of an actually intoxicated person. The experimenter who had administered the beverages was present at all times.

At the outset of the experiment, a given subject was run through the procedure of vestibular stimulation and measurement of the OGI on a practice basis. The subject lay on a cushioned table with his head in a 30-deg-inclined headrest. This head tilt was used in order to place the horizontal semi-circular canals in an upright position. The subject's head was turned to the left and 1 cc of 1°-2°C ice water was injected into the right ear, by means of a plastic syringe. He then covered his ear with a paper towel in order to keep the water from reemerging when his head was turned forward. This technique of vestibular stimulation has been referred to as the "minimal ice water caloric test" and is described in detail by Nelson (1969). Seven seconds after water injection, the head was turned forward to face a 7.6-cm equilateral triangular array of red light-emitting diodes (LEDs), located on a plane at a right angle to the subject's line of vision and subtending a visual angle of 6.04 deg. Aside from the LEDs, the room was in darkness. The subject's task was to fixate the center of the triangle and to indicate (1) when it first appeared to move (to the left for the right-ear irrigation), (2) how rapid the motion appeared to be when it had reached its peak velocity, and (3) how long the apparent motion lasted. The first measure, which is one of latency, was

taken by having the subject depress a button when he first saw the triangle move, thereby stopping a timer which had been set in motion by the experimenter simultaneous with the injection of the water. Peak intensity of the OGI was measured only qualitatively during this practice session, by means of the subject's verbal report. However, during the next OGI session (baseline), in which data were actually recorded, the subject was informed that his experience of peak apparent motion at that time should be considered to represent a magnitude of "100" and that experiences in subsequent sessions were to be described in terms of this referent. If, for example, he later experienced the triangular array as moving twice as fast as during the baseline period, he should describe it as "200." Duration of OGI was measured by a timer which ceased when the subject depressed the button which he had activated at the onset of apparent motion. Finally, the subject was asked to report whether the apparent motion was smooth or jerky and if the triangle appeared to tilt.

After the practice session, the subject spent 15 min in what will hereafter be referred to as the "drinking room." This waiting period was instituted in order to reduce the likelihood of a carry-over effect from the practice water-injection experience to the baseline measures. During this waiting period, the subject filled out several paper-and-pencil tests, which, however, are not relevant to the present report. Upon returning to the "testing room," a baseline OGI measure was taken. Next, the subject went back to the drinking room, where a different experimenter provided him with either an alcoholic beverage or the placebo. The amount of liquid for a given subject was calculated according to body weight, as indicated by bathroom scales. Body weights of the subjects ranged from 115 to 182 lb for females and from 130 to 204 lb for males. The alcoholic beverage consisted of a 4-1 ratio of tonic water and 80-proof vodka (i.e., 40% w/v ethanol diluted to a final concentration of 8% w/v). This particular combination is commonly used in alcohol studies (e.g., Marlatt, Demming, & Reid, 1973) and was judged by several observers in our laboratory to be difficult to discriminate from the placebo beverage (tonic water) on the basis of taste. The amount of alcohol fed to a subject was calculated to produce a blood alcohol concentration (BAC) level of .1%, the minimum level of alcohol required to qualify a person as legally intoxicated in most states.² The total volume of liquid ingested by the subjects ranged from 15.5 to 31.0 oz for females and from 22.0 to 34.0 oz for males. The subject was given either the vodka drink or the tonic water, and was told in each case that he would be drinking an alcoholic beverage and could expect to become somewhat intoxicated. The liquid was divided equally into four plastic cups, each containing five ice cubes. The drinking period lasted precisely 32 min, during which the subject was to drink at an even rate, such that the contents of one cup would be consumed every 8 min. The experimenter provided occasional reminders if it was apparent that the subject was drinking either too slowly or too rapidly. Conversation was allowed and a television set was available for viewing, if desired.

At the completion of the 32-min drinking period, the subject was taken to the testing room, where he immediately underwent the caloric stimulation and measurement of the latency, intensity, and duration of the OGI. The experimenter was "blind" to the subject's drinking condition. After a 40-min wait in the drinking room, these tests were repeated and a sample of the subject's breath was obtained by means of a Mobat "Sober-Meter" (Model SM-7),³ which includes a reservoir bag, a balloon, and a plastic bag designed to hold precisely 2,100 ml of alveolar air. The latter is defined as the air collected at the end of a forced expiration which has passed through the bronchi and the oral-pharyngeal cavities. As the air was expelled, its alcohol content was trapped in a section of dry silica gel, which was later analyzed for alcohol concentration (as described in the next section). A breath sample was taken along with each of the remaining two OGI tests, the latter occurring at 80 and 120 min after the cessation of drinking. At the end of the experiment,

the subject was escorted or driven home, regardless of the drinking condition in which he had participated, and was requested not to drive an automobile for the remainder of the evening.

Each subject was used as his own control in the placebo and alcohol conditions, the order of which was counterbalanced. His second session usually occurred 1 week after the first and, besides the nature of the beverage, differed only in the omission of the practice OGI period. At the completion of this second session, the subject was questioned about his experiences, debriefed, and paid for his participation.

During the debriefing it was found, not surprisingly, that all of the subjects had become aware of the fact that more alcohol had been administered in one condition than in the other. Furthermore, about one-fourth of them suspected that one of the conditions had not included any alcohol at all. The latter insight occurred only during or after the second session. Furthermore, the basis for this observation was not a difference in the taste of the alcohol and placebo beverages, but a subjective evaluation of experienced intoxication at some point in time (roughly 20 min) subsequent to the drinking period. Thus, it appears that, for a few subjects, there was a partial breakdown in the attempt to maintain naiveté about the absence of alcohol in one of the conditions. However, it is unlikely that this failure seriously confounded the results. That is, because the present task of assessing the apparent motion of a point of light in the dark was presumably quite unfamiliar to the subjects, it does not seem reasonable that their belief that they had (had not) recently drunk an alcoholic beverage would lead to a systematic expectation about how they "should" respond.

Measurement of Breath Alcohol

Analysis of the silica gel tubes from the breath collection kit was based upon the method of Glendening, Rush, and Duffett (1971). Briefly, it involved emptying the sample silica gel crystals into 60-ml serum bottles, adding .35 g potassium carbonate crystals into the dry bottle, stoppering the bottle, adding exactly 1 ml of distilled water through the stopper with a needle and syringe, and incubating at 34°C for 30-45 min, with gentle shaking. Standards were made by substituting 1 ml of 100 mg% ethanol for the 1 ml of water in the serum bottle. New silica gel crystals and potassium carbonate (.35 g of each) were added to the standard serum bottles, which were then shaken and incubated in the same manner as the subjects' samples.

One milliliter of head space was withdrawn from each sample or standard bottle and injected into a Varian Series 2440 gas chromatograph, fitted with an aluminum column, 2 m x 4.8 mm, packed with Poropak QS, 80-100 mesh. Helium flow rate was 25 ml/min, and column temperature was 170°C. Under these conditions, ethanol peak retention time was ~80 sec. Sample peak heights were compared directly with standard peak heights to quantify ethanol in alveolar air.

RESULTS

Blood Alcohol Measurements

The gas chromatographic estimates of blood alcohol were taken within a relatively short period of time after the collection of the samples. (It was observed that silica gel alcohol concentrations did not deteriorate during periods of up to 12 days, the longest interval examined.) The results of the analysis for the alcohol condition, broken down according to heavy and light drinkers, is seen in Figure 1. This and the subsequent analyses were carried out on the data from all but one subject, whose responses on several of the OGI measures were so extreme as to clearly qualify him as an "out-

Figure 1. Breath alcohol levels for heavy and light drinkers as a function of minutes after drinking. ($N = 9$, rather than 10, for each of the points representing the light drinkers).

lier." Typically, his scores were greater than 2 SDs above or below the mean of the distribution. According to a mixed analysis of variance (Sex by Drinking History by Tests) for groups of unequal N , only the change in breath alcohol over the tests (not including baseline) was statistically significant, $F(2,30) = 11.5$, $p < .001$. It can be seen in Figure 1 that the blood alcohol level increased rapidly at first and then began a gradual decline, which is typical of blood alcohol curves.⁴ It is also clear that the subjects, as a group, were never brought to the .1% level to which the present experiment had aspired. Perhaps the breath analysis procedure used here led to an underestimation of the actual BAC achieved. In any event, whether or not the subjects were brought to the legal level of "drunkenness" is irrelevant for the purposes of this study.

Oculogyral Illusion (OGI) Measures

All subjects experienced the OGI. For many, it included not only apparent motion of the triangular array to the left but also a jerky component and an apparent counterclockwise tilt. However, there appeared to be no relationship between these qualitative visual experiences and type of subject or drinking condition.

Contrary to expectation, in none of the analyses did drinking history prove to be a statistically significant factor, nor, more importantly, did it interact with the placebo/alcohol manipulation. As a consequence, the data from the heavy and light drinkers were combined for the pictorial presentation seen in Figures 2, 3, and 4. These figures present the change over tests in OGI as a function of drinking condition (alcohol/placebo) and sex for the latency, intensity, and duration measures, respectively. A mixed analysis of variance (Drinking Condition by Sex by Drinking History by Test) was carried out for each of the dependent variables. With respect to latency (Figure 2), statistically significant main effects were found for both Sex, $F(1,16) = 14.56$, $p \leq .022$, and Drinking Condition, $F(1,16) = 19.20$,

Figure 2. OGI latency (1/100 min) for males and females as a function of drinking condition and minutes after drinking. (In this and the subsequent figures, $N = 9$, rather than 10, for each of the points representing the males.)

$p \leq .001$. As seen in Figure 2, latencies were shorter for females than for males and for alcohol as compared to placebo. None of the interactions was statistically significant. Analysis of the OGI intensity measure (Figure 3) revealed a significant effect for Drinking Condition, $F(1,16) = 19.60$, $p < .001$ (with the greater intensities being found for the alcohol condition), for Test, $F(4,64) = 6.05$, $p < .001$, and for the Drinking Condition by Test interaction, $F(4,64) = 4.18$, $p \leq .005$. As seen in Figure 3, the ingestion of alcohol led to a sharp increase in the apparent intensity of the OGI and then a gradual decrease, whereas the placebo condition was characterized by a slow decline in intensity. Finally, the analysis of the duration measure (Figure 4) revealed a marginally significant main effect for Drinking Condition, $F(1,16) = 3.96$, $p < .065$, with the alcohol condition apparently inducing the greater duration. The only other significant effect was the Sex by Drinking Condition by Test interaction, $F(4,46) = 3.68$, $p < .009$. The nature of this interaction may be seen in Figure 4; females in the placebo condition revealed a decline in OGI duration over tests, while little or no change occurred for them in the drinking condition or for males in either of the conditions.

DISCUSSION

The main finding in the present experiment was that the ingestion of an alcoholic beverage to the point of moderate intoxication increases or maintains sensitivity to the visual effects of vestibular stimulation. Specifically, the alcohol condition led to a decrease in latency, an increase in magnitude, and an increase in duration of the OGI. A number of previous investigators (e.g., Jones & Vega, 1972) have demonstrated a greater behavioral effect of alcohol when BAC is increasing than when it is decreasing. The present experiment, although not specifically designed to assess this variable, did include measures timed to coincide with the ascending

and descending limbs of the BAC curve. It is possible that the strong effect of intoxication on OGI intensity (Figure 3) at the first test, followed by a decreased effect on the second and third tests, represents further evidence of the especially great impact of an increasing concentration of alcohol in the bloodstream. Such an effect is not apparent, however, for the other two OGI measures (Figures 3 and 4).

No relationship was obtained between "typical" drinking rate and the effects of intoxication on any of the OGI measures. In retrospect, it is clear that the present measure of drinking practices was insufficient as an indicator of alcohol tolerance, in that it failed to assess the length of time for which the subject had been engaged in his current drinking pattern. Thus, it is likely that some, or perhaps most, of the relatively young individuals who served as subjects in the present experiment had been drinking at their current level for only a few years. Future attempts to ascertain the relationship between "drinking history" and the effects of alcoholic intoxication should use older subjects as well as a more comprehensive and rigorous measuring device, such as the structured interview devised by Ewing and Rouse (Note 2).

Figure 3. OGI intensity (magnitude estimates) for males and females as a function of drinking condition and minutes after drinking.

Figure 4. OGI duration (1/100 min) for males and females as a function of drinking condition and minutes after drinking.

Thus, the important question concerns the mechanism by which alcohol affects the OGI. On the one hand, it may be proposed that the enhancement of this visual illusion results from the intoxicated subject's presumed difficulty in suppressing his vestibular nystagmus, as demonstrated by Schroeder (1971). It is possible, as indicated previously, that the retinal image motion which results from the initially uncontrollable nystagmus is a factor in the OGI. Guedry, Gilson, Schroeder, and Collins (1975) have speculated that the locus of control over vestibular nystagmus is in the flocculonodular lobes of the cerebellum. It is possible, therefore, that the physiological basis of the alcohol-impaired ability to inhibit nystagmus may involve a depletion of guanosine-3', 5'-cyclic monophosphate (cyclic-GMP) in the cerebellum, as shown by Redos, Catravas, and Hunt (1976) with single doses of ethanol in rats. Since there is suggestive evidence that cyclic-GMP is localized in Purkinje cells (Mao, Guidotti, & Landis, 1975), and since these cells provide inhibitory input to other brain areas to control body musculature, it is tempting to speculate that ethanol's effect on cerebellar cyclic-GMP may be responsible for the loss of oculomotor coordination resulting in the impaired control over calorically induced nystagmus which may have occurred in the present experiment.

An alternate interpretation of the present findings is that, as a result of alcoholic intoxication, the eye muscles fail to respond to effector commands as efficiently as under normal circumstances and therefore an unusual amount of effort is required to restrain their nystagmic movement. Such increased efference would presumably lead to an increased OGI.

Clearly, it will be necessary to monitor the movements of subjects' eyes in order to determine the role of oculomotor activity in the alcoholic enhancement of the OGI.

REFERENCE NOTE

1. Cahalan, D., Cisin, I. H., & Crossley, H. M. *American drinking practices*. New Brunswick, N.J.: Rutgers Center of Alcohol Studies, Monograph No. 6, 1969.
2. Ewing, J. A., & Rouse, B. A. *Measuring alcohol consumption: The "alcohol quotient."* Paper presented at the 30th International Congress on Alcoholism and Drug Dependence, Amsterdam, 1972.

REFERENCES

- AMERICAN MEDICAL ASSOCIATION, COMMITTEE ON MEDICOLEGAL PROBLEMS. *Chemical tests for intoxication, manual*. Chicago: American Medical Association, 1959.
- COLLINS, W. E., GILSON, R. D., SCHROEDER, D. J., & GUEDRY, F. E. Effects of alcohol ingestion on tracking performance during angular acceleration. *Journal of Applied Psychology*, 1971, 55, 559-563.
- GLENDENING, B. L., RUSH, A. C., & DUFFETT, N. D. The use of small gas chromatographs for breath testing of drunken drivers

- by the highway patrol utilizing the public health laboratory (Kansas). *Health Laboratory Science*, 1971, 8, 131-141.
- GOLDBERG, L. Quantitative studies on alcohol tolerance in man. *Acta Physiologica Scandinavica*, 1943, 5, Supplement 16.
- GRAYBIEL, A., & HUPP, D. The oculogyral illusion. *Journal of Aviation Medicine*, 1946, 17, 3-27.
- GREGORY, R. L. *Eye and brain*. New York: McGraw-Hill, 1973.
- GUEDRY, F. E., GILSON, R. D., SCHROEDER, D. J., & COLLINS, W. E. Some effects of alcohol on various aspects of oculomotor control. *Aviation Space and Environmental Medicine*, 1975, 46, 1008-1013.
- HILL, R. J., COLLINS, W. W., & SCHROEDER, D. J. Influence of alcohol on positional nystagmus over 32-hour periods. *Annals of Otolaryngology, Rhinology and Laryngology*, 1973, 82, 103-110.
- JONES, B. M., & VEGA, A. Cognitive performance measured on the ascending and descending limb of the blood alcohol curve. *Psychopharmacologia*, 1972, 23, 99-114.
- MAO, C. C., GUIDOTTI, A., & LANDIS, S. Cyclic GMP: Reduction of cerebellar concentrations in "nervous" mutant mice. *Brain Research*, 1975, 90, 335-339.
- MARLATT, G. A., DEMMING, B., & REID, J. B. Loss of control drinking in alcoholics: An experimental analogue. *Journal of Abnormal Psychology*, 1973, 81, 233-241.
- MOSKOWITZ, H., & SHARMA, S. Effects of alcohol on peripheral vision as a function of attention. *Human Factors*, 1974, 16, 174-180.
- NELSON, J. R. The minimal ice water caloric test. *Neurology*, 1969, 19, 577-585.
- REDOS, J. D., CATRAVAS, G. N., & HUNT, W. A. Ethanol-induced depletion of cerebellar guanosine-3', 5'-cyclic monophosphate levels. *Science*, 1976, 193, 58-59.
- SCHROEDER, D. J. Influence of alcohol on vestibular responses to angular accelerations. *Aerospace Medicine*, 1971, 42, 959-970.
- SCHROEDER, D. J. Some effects of alcohol on nystagmus and vertigo during caloric and optokinetic stimulation. *Annals of Otolaryngology, Rhinology and Laryngology*, 1972, 81, 218-229.
- SPECTOR, N. H. Alcohol breath tests: Gross errors in current methods of measuring alveolar gas concentrations. *Science*, 1970, 172, 57-59.
- WALLGREN, H., & BARRY, H., III. *Actions of alcohol* (Vol. I). *Biochemical, physiological, and psychological aspects*. New York: Elsevier Publishing, 1970.
- WHITESIDE, T. C. D., GRAYBIEL, A., & NIVEN, J. I. Visual illusions of movement. *Brain*, 1965, 88, 193-210.

NOTES

1. Actually, three additional subjects began the experiment. However, two were dropped due to a failure to experience the OGI during the baseline measures and one because of nausea early in the experiment.
2. The formula used was: $BAC = \{oz\ 100\% \text{ alcohol (weight)} / (oz\ body\ weight \times .68)\} \times 100$. It was derived from an American Medical Association report (1959).
3. The kit may be obtained from Luckey Laboratories, Inc., 7252 Osburn Road, San Bernardino, California 92404.
4. The initial portion of this curve would presumably have been smoother if blood alcohol concentration could have been accurately assessed immediately after the drinking period. However, it has been shown that breath samples taken within 20 min of drinking produce artificially inflated estimates of blood alcohol level due to the alcohol caught in the mucous membranes of the mouth and nasal passages (Spector, 1970).

(Received for publication July 20, 1976;
revision received January 31, 1977.)