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The Association of Alcohol-Induced Blackouts and Grayouts to Blood Alcohol Concentrations

ABSTRACT: The primary aim of this study was to investigate the association between measured blood alcohol concentration (BAC) and the presence and degree of amnesia (no amnesia, grayout, or blackout) in actively drinking subjects. A secondary aim was to determine potential factors other than BAC that contribute to the alcohol-induced memory loss. An interview questionnaire was administered to subjects regarding a recent alcohol associated arrest with a documented BAC greater than 0.08 g/dL for either public intoxication, driving under the influence, or under age drinking was administered. Demographic variables collected included drinking history, family history of alcoholism, presence of previous alcohol-related memory loss during a drinking episode, and drinking behavior during the episode. Memory of the drinking episode was evaluated to determine if either an alcohol-induced grayout (partial anterograde amnesia) or blackout (complete anterograde amnesia) occurred. Differences in (1) mean total number of drinks ingested before arrest, (2) gulping of drinks, and (3) BAC at arrest were found for those having blackouts compared with no amnesia; while differences in drinking more than planned were found between the no amnesia and grayout groups. A strong linear relationship between BAC and predicted probability of memory loss, particularly for blackouts was obvious. This finding clinically concludes that subjects with BAC of 310 g/dL or greater have a 0.50 or greater probability of having an alcoholic blackout.

KEYWORDS: forensic science, anterograde amnesia, blood alcohol concentration, blackout and grayout

Trying to evaluate the credibility of defendants, plaintiffs, and witnesses whose testimony has been biased by the presence of alcohol in their system continually frustrates expert witnesses. Relatively few observational and survey studies have investigated the association of the blood alcohol concentrations (BAC) to memory. Thus, it is often difficult for an expert to opine to a jury whether or not a particular BAC purportedly claimed by a witness to have resulted in decreased memory for events associated with a crime ought to be believed.

Two types of amnesia have been associated with acute alcoholic intoxication. Goodwin (1) characterized these two types of memory loss as "en bloc" memory loss (blackout), referring to complete loss of memory occurring at some time during the drinking episode, and fragmentary memory loss (grayout) a partial impediment of memory for events occurring while a person is acutely intoxicated. Of these two types of memory loss, grayouts reportedly occur more frequently (2,3). Various ethical and practical considerations have hampered prospective research from determining the association between BAC and the occurrence of memory loss. One such study involving 10 patients diagnosed with alcoholism who were given alcohol in a monitored setting, found that a higher mean BAC was associated with blackouts in five of the 10 (4). Specifically the patients who experienced blackouts had significantly higher mean BACs (mean BAC = 0.279 g/dL) than those patients with no loss of memory (mean BAC = 0.181 g/dL). From these early results, a value of 0.25 g/dL has been hypothesized as a threshold BAC necessary for producing an alcoholic blackout (5).

However, alcoholic blackouts have been reported to occur at BACs below this threshold (6,7), Therefore, other factors probably contribute to the occurrence of alcohol-induced memory loss. Several variables have been reported to be associated with alcoholic blackouts. A structured interview given to 100 hospitalized alcoholics suggested that "gulping" drinks and drinking without eating contributed to the occurrence of blackouts (2). Similarly, in a small observational study of seven actively drinking alcoholic patients, the rate at which subjects drank was a key predictor of blackouts (6). Supporting this finding, Goodwin et al. (8) observed that those individuals whose BAC rose rapidly appeared more likely to experience a blackout. In addition, the National Longitudinal Survey of Youth indicated that blackout drinkers were more likely than nonblackout drinkers to have a significantly greater number of days that they drank alcohol, greater frequency of consuming six or more drinks, and a greater number of drinks on an average day (9). The survey found that males were roughly twice as likely as females to experience blackouts, and that the percentage of individuals reporting blackouts declined as the cohort aged.

The above findings suggest that a high level of blood alcohol is required, but not sufficient, to produce an alcoholic grayout or blackout. Further, while the previous mentioned correlates have been examined individually, no study to date has incorporated all these factors as part of a single study design. Therefore, the primary aim of this study was to investigate the association between measured BACs and the presence and degree of amnesia (no amnesia, grayout, or blackout) in actively drinking subjects. A secondary aim was to determine potential factors other than BAC that contribute to the alcohol-induced memory loss.

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Methods

This was an observational study of data collected by interview from subjects 18 years or older who were recruited from the Iowa City community, the Mid-Eastern Council on Chemical Abuse (MECCA), and the chemical dependency services at the University of Iowa Hospitals and Clinics. To be included in the study, patients had to have a documented BAC of 0.08% or greater resulting from a drinking episode that resulted in them being arrested for either under age drinking, driving under the influence or public intoxication. The interviews occurred within 6 months of the arrest date. Included patients were required to present documentation, i.e., official court record that indicated the BAC at the time of the arrest. All subjects provided written informed consent for study participation. The University of Iowa Human Subjects Institutional Review Board approved the study.

The interview questionnaire required about 15 min to administer, and included demographic variables, subjects' alcohol-use history, family history of alcoholism, age at first alcohol consumption, history of head injuries with and without loss of consciousness, and presence of previous alcohol-related memory loss during a drinking episode. Subjects were also questioned specifically about the incident in which the BAC was obtained and documented, and asked to quantify and qualify the drinks they had consumed. A drink was defined as one 12 ounces of beer, 1.25 ounces of liquor, or 4 ounces of wine. Subjects were also queried regarding: the length of time over which alcohol intake occurred, whether they had consumed the drinks by sipping or gulping, whether they consumed the alcohol on a full or empty stomach, whether any other legal or illegal substances were used during the time period of the drinking episode, and whether they had vomited at any time during the drinking episode. Lastly, the amnesia subject were asked to detail the drinking episode, from the time of the first drink to the last event remembered from that episode. Subjects were asked to self-describe a timeline of events that occurred during the drinking episode, starting at the time the first drink was consumed. From this provided timeline, the "last event remembered" was defined as the last event that could clearly be recalled by the subject with details of the time, place and activities of the event that had occurred during the drinking episode.

To assess the relationship between BAC and memory loss, stepwise logistic regression models for two dependent variables, (1) grayout or blackout and (2) blackout only, were constructed utilizing the previously described independent variables. Predicted values for the presence of a grayout or blackout and a blackout from the multivariate models were then plotted against BAC. All statistical analyses were performed utilizing SAS for Windows version 9.1 (SAS Institute, Cary, NC). For each analysis, a p-value less than 0.05 was considered statistically significant. One-way analysis of variance was used to determine differences between the amnesia groups on continuous variables, and χ^2 tests were used to compare the groups on dichotomous variables.

Results

History

A total of 65 subjects with a documented arrest and a corresponding blood alcohol level were recruited for the study. There were 51 males aged 31.0 ± 12.0 and 14 females aged 30.5 ± 10.2 years at the time of the interview. Ethnically, there were 58 Caucasians, four American Indians, and three African Americans. Thirty-one (48%) of the subjects reported a positive family history having at least one first degree relative with alcoholism (mean = 1.1 ± 1.5). The average age when alcohol was consumed (outside of the context of a religious ceremony) was 14.7 ± 2.6 years old. Twenty (31%) subjects described a history of closed head injury with loss of consciousness occurring outside of a drinking episode. Three of these 20 subjects lost consciousness for 1–24 h while the remaining patients experienced a 5–10 min loss of consciousness. Subjects reported that they ingested an average 36 ± 49 (range = 0–245) drinks per week. Fifty (77%) of the subjects reported awakening the next day after a drinking episode and not being able to remember the events of the previous day within the previous year. Subject estimates of the number of lifetime episodes of alcohol-induced amnesia ranged from 0 to 100 (22 ± 31). For the 2 weeks before their arrest, subjects reported drinking ranged from 1 to 14 days (6.8 ± 4.9, SD).

Drinking Episodes

Self reported total average number of drinks before the arrest was 15.3 ± 9.8, range = 2–51. The duration of the drinking episodes ranged from 0.5 to 12 h (4.7 ± 2.7). Forty-one (63%) of the patients described gulping their drinks rather than sipping before the arrest. Five patients recollected vomiting during the drinking episode while 32 drank on an empty stomach and 41 drank more than they originally planned.

Amnesia

Based on the subjects' histories of the first event they recalled at the beginning of the drinking episode leading to arrest and the last event they were able to recall during the drinking episode, 20 (31%) subjects described blackouts, 13 (20%) described grayouts, and 32 number (49%) reported no amnesic episode. These three groups did not differ with respect to age, gender, ethnicity, family history of alcoholism, age of first drink, closed head injury with loss of consciousness history, drinks per week, days of drinking for the 2 weeks before arrest, lifetime amnesia history, and hours of consuming alcohol, vomiting, or drinking on an empty stomach with the arrest event. The time onset of drinking and time to the 12 grayouts (5.0+/ − 2.4 h) and 20 blackouts (5.2 ± 4.0 h) was compared and found to be similar between both groups (unpaired t-test with Welch correction = 0.1943, $p = 0.8473$, df = 29). Differences were found among the groups with respect to (1) total number of drinks ($F = 6.88$, $p = 0.002$); (2) gulping of drinks ($F = 4.32$, $p = 0.017$); and (3) drinking more than planned ($F = 6.72$, $p = 0.002$). Additionally, BAC s at arrest differed among the three groups ($F = 4.60$, $p = 0.014$). Follow-up analyses were performed for the above variables using the Tukey–Kramer HSD method. Differences for three of the four previously mentioned significant factors were found to be limited between the no amnesia and the blackout group. Specifically, differences ($p < 0.05$) in (1) mean total number of drinks ingested before arrest, (2) gulping of drinks, and (3) BAC at arrest were found for those having blackouts compared with no amnesia; while differences in drinking more than planned were found between the no amnesia and grayout groups. These data are summarized in Table 1.

All variables in univariate analyses associated with a p-value of < 0.15 were included in the multivariate analyses (stepwise logistic regression); including: age, average number of drinks per week, along with total number of drinks, drinking on empty stomach, gulping, drinking more than planned, hours consuming, and BAC associated with the arrest event. Two separate models were tested, the first modeling for (1) amnesia (grayout or blackout), and the second for (2) blackouts only. For ease of interpretation of

TABLE 1—*Differences in subject characteristics between memory statuses.*

	No Amnesia ($n = 32$)	Grayout ($n = 13$)	Blackout ($n = 20$)	p-Value
Mean age ± SD (years)	33.5 ± 11.6	25.3 ± 9.8	30.4 ± 11.7	= 0.09
Male, number, (%)	24 (75%)	12 (92%)	15 (75%)	= 0.39
White, number, (%)	26 (81%)	12 (92%)	20 (100%)	= 0.20
Mean age ± SD (years) of first alcohol drink	15.1 ± 2.9	15.0 ± 2.3	13.9 ± 2.1	= 0.24
Mean alcoholic drinks ± SD per week	33.6 ± 55.5	17.2 ± 11.8	53.8 ± 50.3	= 0.10
Days drinking ± SD last 2 Weeks	6.8 ± 5.1	5.0 ± 4.5	7.9 ± 4.7	= 0.25
Lifetime episodes of amnesia ± SD	16.8 ± 33.6	17.4 ± 14.8	32.0 ± 32.2	= 0.20
History of head injury	10 (31%)	4 (31%)	6 (30%)	= 0.99
Vomited, number, (%)	1 (3%)	1 (8%)	3 (15%)	= 0.29
Empty stomach, number, (%)	14 (44%)	4 (31%)	14 (70%)	= 0.06
Gulped, number, (%)	15 (47%)*	9 (69%)	17 (85%)*	= 0.02
Drank more than planned, number, (%)	15 (47%)*	13 (100%)*	13 (65%)	< .01
Relatives with ETOH abuse ± SD	1.0 ± 1.2	0.6 ± 1.2	1.5 ± 2.0	= 0.20
Time of consumption ± SD (hours)	4.0 ± 2.1	5.5 ± 2.8	5.4 ± 3.2	= 0.10
Mean total drinks associated with arrest ± SD	11.4 ± 8.2*	17.2 ± 9.0	20.8 ± 9.9*	< 0.01
Mean blood alcohol level % ± SD (%)	0.18 ± 0.07*	0.22 ± 0.05	0.23 ± 0.05*	= 0.01

*Differences between groups, Tukey method.

odds ratios (ORs), BAC levels were converted from g/dL to mg/L (BAC(mg/mL) = BAC (g/dL) × 100). In the first model, subjects with greater consumption time and higher BAC levels were found to have increased risks for amnesia (grayout or blackout) (OR = 1.41; 95% confidence interval (CI) = 1.06–1.87, $p = 0.018$; OR = 1.02; 95% CI = 1.01–1.03, $p = 0.009$, respectively) while subjects who were older had lower risks (OR = 0.90; 95% CI = 0.83–0.97, $p = 0.004$). In the second model, subjects with higher BAC levels were found to have higher risks for blackout (OR = 1.01; 95% CI = 1.00–1.02, $p = 0.018$). Model discrimination as measured by the c statistic was very good in both models ($c = 0.84$ and 0.76, respectively). The c statistic is numerically equivalent to the area under a receiver operator characteristic curve and represents the proportion of times that a given subject who had amnesia had a higher predicted risk of amnesia than a subject who did not have amnesia. The c statistic ranges from 0.5 to 1.0; a model with perfect discrimination has a c statistic equal to 1.0, while a model with no discrimination has a c statistic of 0.5, i.e., a coin flip.

The odds ratios associated with BAC are numerically close to 1.0. However, this is a mathematical artifact of converting BAC units from g/dL to mg/dL, or mg%. As stated above, this was done to facilitate ease of interpretation. Thus, an increase of 1 mg/dL in BAC in our stepwise logistic regression models increases the odds of a blackout or grayout by ~2% (OR = 1.02). We feel that readers should consider our findings important more for the c statistics, whose values of 0.84 and 0.76 show that models incorporating BAC information can be used to predict grayout/blackouts.

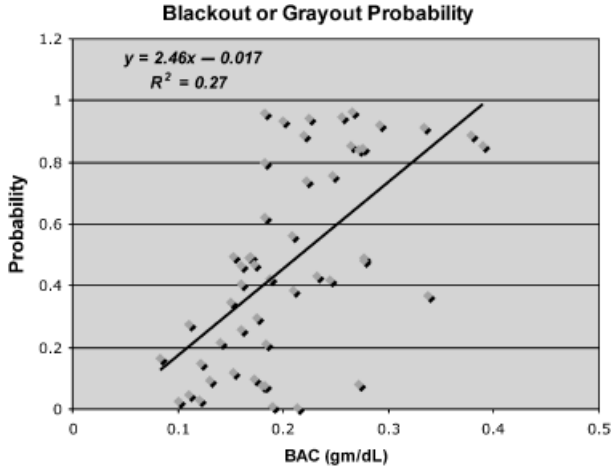

FIG. 1—*Probability of grayouts or blackouts as a function of the blood alcohol concentration (BAC) (g/dL) where probability = 2.46 (BAC)–0.02 ($R^2 = 0.27$).*

Discussion

The primary finding of the study was that there was a significant association in measured BAC with the level of amnesia reported by the subject during a recent drinking episode. Thus as the BAC increased the likelihood of a grayout or a blackout increased. Figure 1 shows the results of predicted probability for a grayouts or blackouts plotted against BAC; while Fig. 2 shows the results of predicted probability of a blackout plotted against BAC. As can be seen, there appears to be a strong linear relationship between BAC and predicted probability of memory loss, particularly for blackouts ($R^2 = 0.54$).

The study populations studied to date that consider the association between BACs and memory dysfunction vary considerably. Despite the variability, many of the predictive factors for blackouts are replicated between studies. A structured interview with 100 hospitalized alcoholics of whom 36 experienced blackouts suggested that blackout-contributing factors were "gulping" drinks and

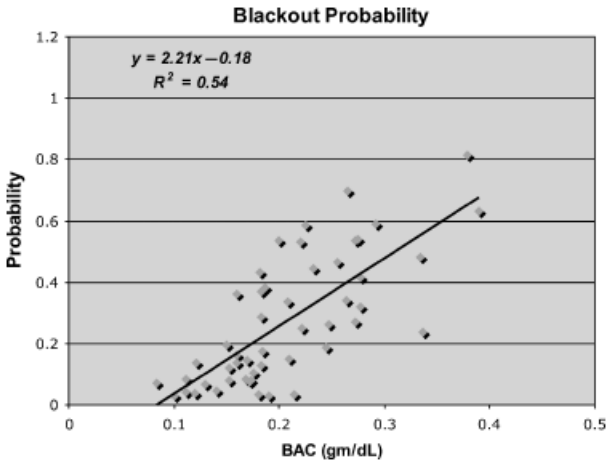

FIG. 2—*Probability of blackouts as a function of the blood alcohol concentration (BAC) (g/dL) where probability = 2.21 (BAC)–0.18 ($R^2 = 0.54$).*

drinking without eating (2). Our population differs from the Goodwin population because our subjects were not assessed for a diagnosis of alcoholism. The White et al. (3) population of 772 undergraduate college students of whom 68% were female does not reflect the alcohol abuse problem population. White et al. reported that grayouts were far more common than blackouts in their population. Our population differs from the White et al. cohort in that our subjects were primarily male and older who experienced more blackouts than grayouts. The White et al. finding of fewer blackouts suggests that these subjects were exposed to lower BAC. The longitudinal survey of Jennison and Johnson concluded that following age stratification of the study subjects, the blackout rate decreases as the age cohort increases its age. Our logistic regression analysis came to a similar conclusion. Jennison and Johnson also mention four additional significant variables related to blackouts, increased alcohol consumption, age of drinking onset, the number of alcoholic relatives, and the subject's ability to control drinking (9). Our data set found that these variables were not contributing factors. However, inspection of our data suggests that these differences may be a function of small sample size.

The role of concurrent substance abuse in blackouts while drinking has been difficult to ascertain whether it influences alcohol-induced memory (10). Our attempts to tease out these data failed. Because of our subjects had legal charges pending against them at the time of interview, they were reluctant to admit to any further substance abuse. This was borne out by the results of the Substance Abuse Subtle Screening Inventory (SASSI) interview (11). The SASSI is a widely used but infrequently studied screening test for substance misuse. Early on in the study the SASSI scores were extracted from the medical chart stemming from the consenting subjects mandated court-ordered substance abuse evaluation. These evaluations were far less likely to suggest abuse problems than the SASSI scores collected by the study interviewers for the later two thirds of the study. We concluded that the explanation for the more common negative SASSI scores associated with the court-ordered substance abuse evaluation was related to the consequences resulting from a positive score. A positive score indicated that the subject was required to complete a substance abuse treatment program before their case could be closed. On the other hand, there were no consequences for a positive SASSI score when administered by the study interviewers. Thus it was concluded that the SASSI data was biased and therefore not evaluable.

As shown in Fig. 2, the BAC is the independent variable that determines the probability of alcohol-induced blackouts ($R^2 = 0.54$). However, the BAC cannot discriminate nearly as well between no amnesia and the presence of a grayout or blackout as demonstrated by Fig. 1 ($R^2 = 0.27$). Thus from a forensic pharmacology frame of reference it is reasonable to conclude that a subject with BACs 0.305 g/dL has a 50% probability of being truthful if claiming that a blackout had occurred during the alleged incident. However, the lower coefficient of variation (R^2) of 0.27 for the grayout association suggests that attempts by expert witnesses to discriminate between the credibility of subjects' claims of grayouts versus no amnesia are problematic.

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