

European Addiction Research, 2004, 10 (3), 95-98.

A Comparison of Alcohol Abusers who have and have not Experienced
Blackouts.

John F. O'Mahony

University of New England and St. Patrick's Hospital, Dublin

Keywords: Alcohol Abuse, Blackout, Cognitive Impairment

Running head: Correlates of Alcoholic Blackout Experience

Correspondence to: John F. O'Mahony, School of Psychology,

University of New England, Armidale, NSW, 2351, Australia.

Email: jomahony@metz.une.edu.au

Telephone: 61 2 6773 3764

Fax: + 61 2 6773 3820

Abstract

This study examined the proposition that the experience of alcoholic blackouts is related to other indices of cerebral involvement in the alcoholic process. In particular it was suggested that those who experience alcoholic blackouts would also show signs of the effects of alcoholic damage in the form of lower scores on cognitive functions vulnerable to the effects of long-term alcohol abuse. While some evidence was found linking the experience of blackout to severity of the alcohol problem no relationship was found between cognitive functioning and the experience of blackout. The possibility that different mechanisms may underlie the various cognitive sequelae of alcohol abuse is raised.

A comparison between alcohol abusers who have and have not experienced
blackouts

Introduction

Blackouts, periods of time when intoxicated for which the person has little or no memory [1], are common among drinkers of all sorts, from those with relatively modest consumption to the severely alcoholic [2, 3, 4]. They are characterised by Lishman [1] as involving “ a dense amnesia for significant events which have occurred during a drinking episode, when at the time outward behaviour perhaps seemed little disordered” (p. 595). While they can occur early on in drinking careers [5, 6, 7], there are some indications that they become more frequent with severity of alcohol problems and duration of alcohol consumption [2, 4, 8, 9,10].

The mechanism underlying blackouts is unknown. There is some evidence that they may be related to the rapidity of the rise in blood alcohol level [11].

As mentioned above, blackouts may be more common among late stage drinkers and cognitive impairment (and presumably underlying cerebral derangement of some sort) is also more common among the same group [see e.g., 12]. However, not all heavy drinkers or the alcoholically dependent suffer from blackouts and not all suffer from intellectual impairment even

after long drinking careers. It is possible that those already intellectually compromised may be more susceptible to blackouts. Or, it is possible that both reflect a greater cerebral sensitivity to the effects of alcohol. In either case there should be an association between blackouts and intellectual impairment. To the author's knowledge, only one study has examined this proposition. Tarter and Schneider [8] compared alcoholics with high ($N = 23$) and low ($N = 27$) incidence of blackouts on four measures of learning and memory including the well known Wechsler Memory Scale [13] and found no difference between the groups. However, since this is the only study of its kind, it may be timely to revisit the question.

Method

Subjects

The study was approved by the Institutional Ethics Committee of St. Patrick's Hospital, Dublin and was in compliance with the Helsinki Declaration of 1975, as revised in 1983.

The sample consisted of 67 alcohol abusing inpatients of an Irish private psychiatric hospital who had been referred for examination of suspected cognitive impairment secondary to alcohol abuse. All had received a firm diagnosis of the alcohol dependence syndrome (ADS) according to ICD-9 criteria [14]. The concept of alcohol dependence syndrome is very

similar to alcohol dependence of DSM, for which it was a model. All patients were attending the hospital alcohol treatment program.

Exclusion criteria for the study were: (i) a primary hospital diagnosis other than that of alcohol dependence syndrome (ii) history of head injury, cerebrovascular accident or illness (including frank liver disease) which could potentially affect neuropsychological functioning (iii) history of psychotropic drug abuse, apart from alcohol (iv) less than three weeks abstinence from alcohol prior to neuropsychological examination. Of the sample, 76% were male; the majority (61%) were from socio-economic groups I and II - by the classification scheme of the Central Statistics Office [15] - , 24% were single, 61% married, 14% separated or widowed. The mean duration of problem drinking was 9.1 years (SD = 6.5) and subjects had spent an average of 136 days (SD = 202) in a psychiatric hospital for alcohol problems over 2.5 hospitalisations (SD = 3.1). A majority of the sample, 60%, were considered probably cognitively impaired by their referring physicians and the referral was for confirmation/disconfirmation of this clinical impression; for the remainder, there was merely a query about intellectual status. It was not usual hospital practice to refer intellectually disabled persons clinically considered to suffer from alcoholic dementia, Korsakoff's syndrome or hepatic encephalopathy for neuropsychological evaluation. Psychometric examination led to some 70% of the patients being judged impaired. Written, informed consent was obtained from participants at the time of neuropsychological

testing.

Measures

The examination schedule consisted of sub-tests from the WAIS and the WAIS-R. The WAIS was used with thirty-eight subjects, the WAIS-R, over a somewhat later period, with twenty-nine. The sub-tests used were Digit Symbol, Block Design and Digit Span, selected for their established sensitivity as measures of alcoholic cognitive impairment [16]. Vocabulary and Picture Completion were also included as indicators of premorbid intellectual functioning [17]. Memory was measured by Russell's [18] adaptation of the Logical Memory and Visual Reproduction sub-tests of the Wechsler Memory Scale [13]. Russell's adaptation involves the addition of delayed memory trials to the original procedure. A gist scoring system similar to that of Schwartz and Ivnik [19] was used to score both trials of Logical Memory. Trail Making Tests A and B, which are also sensitive to the effects of alcohol abuse [12], were also administered. Most subjects were examined by the author, an experienced clinical psychologist; a few were examined by pre-doctoral clinical psychology trainees under his direct supervision.

The mean number of definite alcohol-free days prior to neuropsychological examination was 47 (SD = 37). The exclusion minimum was 21 alcohol-free days. Hospital practice was to order laboratory tests for

most of those referred for neuropsychological assessment. The measure of liver function in the standard hospital package of laboratory tests was GGT. The examiner was blind to the results of the GGT test, which were later taken from the general hospital file. Blood samples for GGT testing were given during the first few days of hospitalisation. It was thought that this test might serve as an index of physical susceptibility to the deleterious effects of alcohol.

Other relevant information was also gathered, either from interview or the hospital file. These included aspects of drinking history: approximate duration of alcohol abuse, presence of withdrawal symptoms, blackouts, morning drinking, loss of control over drinking and the presence of physical complications, such as gastroenteritis or frank liver disease, considered to be associated with alcohol abuse. Blackouts, of particular interest in this paper, were coded as present if the criteria of Lishman [1] (a dense amnesia for a time when outward behaviour was not disordered) were met.

Data treatment

All cognitive test scores were age-scaled and transformed to yield a mean of 10 and standard deviation of 3. The norms used for this were manual norms [20, 21] for the WAIS and WAIS-R variables, distributions provided by Abikoff et al. [22] for Logical Memory, by Russell [23] for Visual

Reproduction, and by Davies [24] for Trail Making, A and B. The drinking quality and medical variables were all coded as present (i.e. abnormal) or absent. Abnormal GGT was defined by hospital policy as > 56 IU/L. The author made an overall clinical judgement of the presence or absence of intellectual impairment on the basis of the neuropsychological data.

Results

Blackouts and severity of alcohol problem

Hospital file data indicated that 58% (39 of 67) of the sample reported blackouts. The presence or absence of blackouts was cross-tabulated against various measures of alcohol problem severity. The presence of blackouts was associated to some degree with some indices of severity (withdrawal symptoms and loss of control), but not with duration of problem drinking, physical complications or abnormal liver function. Table 1 presents these data.

Insert Table 1 about here

Blackouts and cognitive functioning

The participants in this research were, taken collectively, clearly intellectually impaired. Their scores on alcohol-vulnerable cognitive measures

were substantially below normative values for the general population. For example, age-scaled scores for the memory and the trails variables were all 7 or below (on a scale of Mean, 10; SD, 3). This is in sharp contrast to the mean age-scaled score of 11.7 for the Wais/Wais-R Vocabulary sub-scale, an index resistant to alcoholic deterioration.

Several MANOVA's were run to detect any association between cognitive functioning and the presence of blackouts. Since different versions of the Wechsler Intelligence Scale were used, analyses were conducted separately for the WAIS and WAIS-R groups. Five subscales were included in the analysis (Digit Span, Vocabulary, Picture Completion, Block Design, and Digit Symbol). Neither analysis was significant (For the WAIS group, $(F(5,28) = .70, p = .63)$; for the WAIS-R group, $(F(5,23) = .17, p = .97)$). A MANOVA for the Trails and the memory variables conducted for the whole group showed little difference as a function of the presence of blackouts ($F(7,47) = .56, p = .79$).

Discussion

This study replicates some previous findings. The presence of blackouts was related to some measures of severity of problem – withdrawal symptoms and loss of control. However, contrary to the findings of some

others [8, 9], blackouts were not related to duration of problem. The hypotheses that blackouts either reflect a general vulnerability to the cerebral consequences of alcohol abuse or are associated with other forms of more enduring cognitive impairment did not receive any support. In this finding the present study agrees with Tarter and Schneider [8], which to the author's knowledge is the only other study to have examined this relationship.

The lack of a relationship between duration of problem drinking and the presence of blackouts is interesting in the light of the finding that blackouts may occur early on in drinking careers [5, 7]. This, and the findings of the current study are consistent with Lishman's [25] suggestion that there may be different and independent routes to the several forms of alcohol-related cerebral impairment. One possibility is that the locus of the short-term blackout effect is in the hippocampus [26, 27] whereas the cognitive inefficiencies frequently found among alcoholics [28] may be more related to enduring damage to the frontal cortex as a consequence of long term alcohol abuse [29].

The results are somewhat limited by methodological concerns. It could perhaps be argued that presence/absence of blackouts is a rather crude measure and information regarding age of onset; number, duration and intensity might have yielded more sensitive and powerful indices of blackout. Certainly, it would be useful to have such indices, but they were not included

in the original coding. However, blackout is a phenomenon that does not occur in all heavy drinkers/alcoholics and a presence/absence measure is a defensible index of susceptibility. It is also possible that relationships between blackout and cognition might have been better demonstrated through more extensive and sophisticated examinations of cognitive functioning rather than the relatively brief and clinical measures used in this study. A further limitation is the relatively small number for the analysis of cognitive variables which would not have maximised power.

Nonetheless the data presented here suggest that alcoholic blackouts and more enduring alcohol-related cognitive impairment are not closely related phenomena. Blackouts are a remarkably under researched phenomenon and the author believes with Lishman [1] that “correlates of individual vulnerability (to blackout) remain an important question for further investigation.” (p. 595).

Acknowledgement

This research was supported by a grant from the Friends of St. Patrick's
Hospital, Dublin.

References

- 1 Lishman WA: Organic Psychiatry ed 3. Oxford, Blackwell, 1997.
- 2 Goodwin DW, Crane JB, Guze SB: Alcoholic "blackouts": A review and clinical study of 100 alcoholics. *Am J Psychiatry* 1969; 126:191-198.
- 3 Goodwin DW: Two species of alcoholic "blackout". *Am J Psychiatry* 1971; 127:1165-1670.
- 4 Zucker DK, Austin FM, Branchey L: Variables associated with alcoholic blackouts in men. *Am J Drug Alcohol Abuse* 1985;11: 295-302.
- 5 Anthenelli RM, Klein JL, Tsuang JW, Smith TL, Schuckit MA: The prognostic importance of blackouts among young men. *J Stud Alcohol* 1994;55:290-295.
- 6 Jennison KM, Johnson KA: Drinking-induced blackouts among young adults: Results from a national longitudinal study. *Int J Addict* 1994;29: 23-51.
- 7 Buelow G, Koeppe J: Psychological consequences of alcohol induced blackout among college students. *J Alcohol Drug Educ* 1995;40:10-20
- 8 Tarter, RE, Schneider DU: Blackouts: Relationship with memory capacity and alcoholism history. *Arch Gen Psychiatry* 1976;33:1492-1496.
- 9 Poilolainen K: Blackouts increase with age, social class and the frequency of intoxication. *Acta Neurol Scand* 1982;66:555-560.

- 10 Campbell WG, Hodgins DC: Alcohol-related blackouts in a medical practice. *Am J Drug Alcohol Abuse* 1993;19:369-376.
- 11 Rybach RS: Alcoholic amnesia: Observations on seven drinking inpatient alcoholics. *Q J Stud Alcohol* 1970;31:616-632.
- 12 Chelune GJ, Parker JB: Neuropsychological deficits associated with chronic alcohol abuse. *Clin Psychol Rev* 1981;1:181-195
- 13 Wechsler D: A standardised scale for clinical use. *J Psychol* 1945;19: 87-95.
- 14 Edwards G, Arif A, Hodgson R: Nomenclature and classification of drug and alcohol-related problems. *Br J Addict* 1982;77:3-20.
- 15 Central Statistics Office: *Classification of Occupations*. Dublin, Author, 1986.
- 16 Parsons OA, Farr SP: The neuropsychology of alcohol and drug use; In Filskov S, Boll TS (eds): *Handbook of Clinical Neuropsychology*. New York, Wiley, 1981, pp 320-365.
- 17 Lezak MD: *Neuropsychological Assessment*, ed 3. New York, Oxford University Press, 1995.
- 18 Russell EW: A multiple scoring method for the assessment of complex memory functions. *J Consult Clin Psychol* 1975;43:800-809.
- 19 Schwartz, MS, Ivnik RJ: Wechsler Memory Scale I: Toward a more objective and systematic scoring system for the logical memory and visual reproduction subtests. Paper presented at American Psychological Association

meeting. 1980 September; Montreal, Canada.

20 Wechsler D: Manual for the Wechsler Adult Intelligence Scale. New York, Psychological Corporation, 1955.

21 Wechsler D: Manual for the Wechsler Adult Intelligence Scale – Revised. New York, Psychological Corporation, 1981.

22 Abikoff H, Alvir J, Hong G, Sukoff R, Orazio J, Soloman S, Saravay J: Logical memory subtests of the Wechsler Memory Scale: Age and education norms and alternate-form reliability of two scoring systems. *J Clin Exp Neuropsychol* 1987;9:435-448.

23 Russell EW: Renorming Russell's version of the Wechsler Memory Scale. *J Clin Exp Neuropsychol* 1988;10:235-249.

24 Davies AD: The influence of age on Trail Making Test performance. *J Clin Psychol* 1968;24:96-98.

25 Lishman WA: Alcohol and the brain. *Br J Psychiat* 1990; 156: 635-644.

26 Givens B, Williams JM, Gill TM: Septohippocampal pathway as a site for the memory-impairing effects of ethanol. *Hippocampus* 2000;10:111-121.

27 White AM, Matthews DB, Best PJ: Ethanol, memory and hippocampal function: a review of recent findings. *Hippocampus* 2000;10:88-93.

28 Ellis RJ, Oscar-Berman M: Alcoholism, ageing and functional cerebral asymmetries. *Psychol Bull* 1989;106:128-147.

29 Kril JL, Halliday GM Brain shrinkage in alcoholics: A decade on and what we have learned. *Prog Neurobiol* 1999;58:381-387.

Correlates of Alcoholic Blackout Experience

1

Table 1. Problem severity and other characteristics of 67 alcohol dependent patients according to the presence (n =39) or absence (n = 28) of blackouts

Blackouts	Morning Drinking		Withdrawal Symptoms*		Loss of Control*		Physical Complications		GGT		Duration of Problem Drinking (Years)	Duration of Treatment (Days)
	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Normal	Abnormal		
Present n = 39	57	43	80	20	69	31	18	82	73	27	10.0 (7.4)	130 (191)
Absent n = 28	35	65	52	48	36	64	39	61	71	29	7.7 (4.9)	144 (219)

Note. The two duration variables are continuous. The remainder are dichotomous. Dichotomous variables are presented as percentages.

* $p < .05$. 2-tailed Chi Square test.

All other comparisons $p > .05$ by Chi Square or 2-tailed t test .