

Commentary: Alcoholic Blackout and Allegation of Amnesia During Criminal Acts

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The article by van Oorsouw and colleagues¹ presents interesting survey and experimental data, exploring the question of whether alcohol blackouts occur frequently or are used primarily as an excuse to minimize legal responsibility. The authors concluded that the results of their analysis indicated that blackouts during serious misbehavior are often reported outside the court, but the denial or claim of alcoholic blackout may serve a strategic function for the person who raises the claim.¹

Reports of alcoholic blackouts are well documented in the psychiatric literature. Lishman² notes that an alcoholic blackout consists of a dense amnesia for significant events which have occurred during a drinking episode, when at the time outward behavior perhaps seemed not disordered. Usually the gap extends for a period of several hours, but very occasionally, it may cover several days.² Goodwin *et al.*^{3,4} have presented detailed descriptions of the nature of blackouts in 64 subjects. This is one of the earliest research studies of alcoholic blackouts in American psychiatry. One fourth of their patients found themselves in strange places with no recollection of how they got there. The wives of two of these patients reported that they could tell when a blackout was in progress, by their husbands' glassy stare, belligerent behavior, or repetition of questions that showed that experiences were failing to register. These blackouts were described as *en bloc* and were distinguished from fragmentary memory losses, in which the subject was

unaware that events had been forgotten, until he or she was told about them later. An early investigation by Tarter and Schneider⁵ explored the possibility that alcoholics subject to blackouts might have some enduring impairment of memory when sober. Their results were uniformly negative for that question, and, on a wide battery of neuropsychological memory measurements, those with the highest incidence of blackouts performed as well while sober as those in whom blackouts were rare.

Goodwin and colleagues⁶ demonstrated further that volunteers trained and tested while under the effects of alcohol demonstrated some reproducible findings. During periods of moderate intoxication, registration was substantially normal, but short-term memory was considerably impaired. More severe intoxication caused a significant decrease in registration and a more profound diminishment in short-term memory. Subjects demonstrated poor ability to recall the events of the preceding day on days following moderate intoxication, and decreased recall was even more pronounced on days following severe intoxication. The diminishment of memory was also related to the duration of intoxication, in that it tended to become worse as the days of the experiment went by. Defects in 24-hour recall were also more frequent and severe. The worse the short-term memory had been, the greater the level of intoxication had been. Conversely, 24-hour recall was always normal in subjects who had shown intact short-term memory the day before. This study demonstrated further marked individual susceptibility to the same blood-alcohol level affecting each subject to a variable extent. Only 6 of the 13 subjects showed blackouts, which were defined as the ability to answer less

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than 30 percent of the questions concerning the previous day's activities. Three of the subjects were particularly vulnerable in this regard. No relationship was discovered to factors commonly thought to be neuropsychiatrically important, such as age, intelligence, history of head injury, presence of EEG abnormalities, duration of prior drinking history, or a pre-experiment history of delirium tremens.⁶ Lishman² points out that this investigation clearly restates the importance of an organic basis for the blackout. State-dependent effects are excluded in this particular setting, because 24-hour recall was tested while the subjects were still intoxicated. The correlates of individual vulnerability to blood-alcohol level, following the study of Goodwin *et al.*,⁶ remained an important question for further investigation.

As van Oorsouw and colleagues¹ reported, the prevalence of blackouts—particularly, among younger people—is higher than previously recognized. Fifty-one percent of American college students who had ever consumed alcohol reportedly had experienced a blackout at some point in their lives, and 40 percent had experienced one in the year before a recent survey.⁷ Even those preparing to be medical specialists have an apparent higher prevalence of blackouts than previously recognized. Thirty-five percent of pediatric residents in a recent survey admitted to having alcohol-associated blackouts.⁸ Van Oorsouw *et al.*¹ have reported to us the experiences among residents of The Netherlands, but recent evidence from Japan also indicates high levels of adolescent alcohol abuse and blackouts.⁹ Among American college students, fragmentary blackouts, in which memory for events is articulated, were far more common than blackouts of the *en bloc* type, in which a period of time is simply missing from memory.¹⁰ Furthermore, the prevalence of alcoholic blackouts is much more common among social drinkers than previously assumed. It is noteworthy that large amounts of alcohol—particularly if consumed rapidly—can produce either fragmentary or *en bloc* blackouts.¹¹

While the study by van Oorsouw *et al.*¹ provided no information about cognitive functioning in those who reported blackouts, there is recent information to suggest that many elements of cognition are not affected among persons who report blackouts, while other cognitive areas seem preferentially affected. Recent studies suggest that most *en bloc* blackouts in-

volve concurrent use of illicit substances, and poly-substance abuse is also reported at fairly significant levels in those reporting fragmentary blackouts.¹² Another study found some evidence linking the experience of blackouts to the severity of alcohol abuse. However, no relationship was found between cognitive function and the experience of blackouts in this recent investigation.¹³ Because most tests of cognition are made after the fact, it is difficult to determine what cognition is like during intoxication. Recent findings suggest that fragmentary blackouts result from poor retrieval and that individual differences in retrieval emerge after alcohol is consumed. Those who reported past fragmentary blackouts and consumed alcohol displayed marked difficulty with recall of a narrative when this was attempted both during intoxication and after detoxification, as well as with a source memory task presented during intoxication.¹⁴

Potential Biological Mechanisms

Most researchers in the area of alcohol abuse and alcohol-induced cognitive disorders point out the individual differences noted among those who do and do not experience blackouts. Recent genetic studies have added substantially to our understanding of individual differences among those who abuse alcohol and report blackouts. Nelson and colleagues¹⁵ recently reported a lifetime history of blackouts in 39.3 percent of women and 52.4 percent of men who were members of the Young Adult Australian Twin Register. This sample contained 2,324 monozygotic and dizygotic twin pairs with a mean age of 29.9 years. Both twins' responses were coded for blackout questions and for frequency of intoxication. The heritability of lifetime blackouts was 52.5 percent and that of having had three or more blackouts in a year was 57.8 percent. Models that controlled for frequency of intoxication found evidence of a substantial genetic contribution, unique to risk for the blackouts, and a significant component of genetic risks shared with frequency of intoxication. These findings may offer important additional avenues to investigate individual susceptibility to alcohol-related memory and learning dysfunction. Other biological factors may play a role in memory disruption following ingestion of alcohol. Moderate doses of ethanol can modify rapid-eye-movement (REM) sleep architecture by reducing the number of REMs and/or REM

densities, as well as minutes of REM sleep, particularly in the first half of the night. These modifications result in memory impairment for recently learned cognitive procedural material. Alcohol also appears to have a subtle effect on Stage II sleep, as well, since memory for a stage II-sensitive motor-procedural task was impaired in a recent Canadian research study.¹⁶ A recent within-subjects research design, involving an alcohol challenge and a no-alcohol condition, revealed that an alcohol challenge separated automatic and effortful memory processes in volunteers. Free recall was significantly lower in the alcohol than in the no-alcohol condition.¹⁷

Findings in recent studies in which alcohol's effects were reduced to the brain tissue level and the high prevalence of alcohol use among young people worldwide was considered are consistent with the view that the hippocampus is more sensitive to the acute effects of ethanol during adolescence and may be more susceptible to the neurotoxic effects of ethanol during this developmental period.¹⁸ Working memory, which is an attentional modality in the brain rather than a memory modality, seems significantly unaffected by chronic alcohol consumption, whereas cognitive measures, such as response shifting and inhibition, are affected.¹⁹ Moreover, a statistically significant loss of 37 percent of glial cells has been found globally in the hippocampus of alcoholics, when compared with control subjects. This gives rise to further considerations of the hippocampus and associated frontal brain areas as anatomic locations primarily involving learning and memory, which are selectively affected by high-dose or chronic alcohol exposure.²⁰ These findings in humans are consistent with recent findings in animals. Long-term potentiation in the rat hippocampus is reversibly depressed by chronic intermittent ethanol exposure.²¹ The number of cholinergic neurons in the basal forebrain is reduced both in humans and rats following chronic alcohol consumption.²² The progenitor cells, which proliferate throughout life and form neurons, astrocytes, and oligodendrocytes, are inhibited in adult male rats by binge-alcohol exposure.²³ Moreover, the effect of acute alcohol on learning an auditory tone has been shown to be hippocampus-dependent in rats. These human and nonhuman findings collectively support recent theories that forensic implications are probably hippocampally mediated.

Alcoholic Blackouts and Forensic Psychiatry

Diagnostically, the DSM-IV-TR recognizes organic amnesia and psychogenic amnesia. There are three major amnesia diagnostic possibilities, according to the DSM.³⁴ The first is amnesic disorder due to a general medical condition (294.0), and the second is substance-induced, persisting amnesic disorder (292.83). An amnesic disorder that does not meet the criteria for the first two conditions is listed as amnesic disorder, not otherwise specified (294.8). Memory dysfunction due to a general medical condition is further subdivided into transient or chronic, whereas the substance-induced disorder is not. However, the substance-induced persisting amnesic disorder is specific to the substance or substances in question.

There was a high degree of amnesia reported in a forensic psychiatric population.²⁵ Leaving alcoholic blackouts aside, amnesia has been reported to some degree in 40 to 70 percent of homicides.²⁶⁻²⁹ This, of course, presents a particular dilemma for the psychiatrist examining a person who reports blackouts after the crime. Alcohol abuse is involved at very high rates in homicides and other crimes in most industrialized countries. Van Oorsouw *et al.*¹ and others remind us that, in many instances, offenders claim excessive alcohol consumption as an explanation for their amnesia. Whether or not the examination takes place without the benefit of knowing the blood-alcohol concentration following the alleged offense, the examining psychiatrist is in a significantly compromised position to draw conclusions regarding alleged alcohol-induced blackouts. Van Oorsouw and colleagues¹ report interesting data when the blood-alcohol concentration (BAC) is considered. The average measured BAC was 190 mg/dL, with a range of 66 to 350 mg/dL. The mean BAC for offenders claiming blackout was slightly lower—180 mg/dL. This may be a BAC insufficient to induce a blackout in susceptible individuals. For instance, based on subjective reports, the data of van Oorsouw *et al.*¹ estimate BACs for blackout episodes in the first survey at extremely high levels (260 mg/dL). They point out that it is possible the alcohol dosage was overestimated. However, there is a much higher frequency of alcoholic blackouts reported in their first survey, in which BAC was extrapolated with a predictive formula (the Widmark equation), than in the traffic-

control survey, in which BAC was empirically measured.

The question for forensic psychiatry is: what difference does it make if a person reports amnesia during a criminal act due to alcohol consumption? Most legal statutes in the United States do not allow voluntary intoxication as a defense for committing a crime.³⁰ Moreover, most of the evidence of a blackout is provided by subjective recall from the accused and so may be of questionable veracity.³¹

*Wilson v. U.S.*³² is instructive regarding issues of alcoholic blackouts. Mr. Wilson had a permanent retrograde amnesia, with no recollection of the events charged in his indictment as a result of a head injury suffered in a car chase. His competence was an issue at trial, but the trial court found him to be competent, pointing out that he had the capacity to understand the details of the case by relying on other sources of information. The U. S. Court of Appeals accepted the trial court's analysis of what was required for competence, based on the *Dusky* standard.³³ The case was remanded, and Judge Leventhal pointed out that it is a common occurrence for a person to be convicted of negligent homicide, even though his memory is impaired by intoxication at the time of the crime. It is likely that a similar legal analysis would apply to alcoholic blackouts raised as a defense for, or explanation of, illegal or negligent acts.

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