

Acute Alcohol Intoxication and Cognitive Functioning*

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ABSTRACT. Acute alcohol intoxication produces changes in the cognitive functioning of normal individuals. These changes appear similar *prima facie* to those exhibited by individuals who sustain prefrontal lobe damage during adulthood. In order to test the validity of this observation, and to control for the confounding effects of expectancy, 72 male subjects were administered a battery of neuropsychological tests, within the context of a balanced-placebo design. Each subject received one of three

widely different doses of alcohol. Analysis of the results of the cognitive test battery demonstrated that a high dose of alcohol detrimentally affects a number of functions associated with the prefrontal and temporal lobes, including planning, verbal fluency, memory and complex motor control. Expectancy does not appear to play a significant role in determining this effect. The implications of this pattern of impairment are analyzed and discussed. (*J. Stud. Alcohol* 51: 114-122, 1990)

THE EFFECTS of acute alcohol intoxication upon human cognition have been investigated for several decades. Jellinek and MacFarland (1940), who first attempted to systematically integrate knowledge about such effects, hypothesized that alcohol had less impact upon simple than upon complex cognitive functions. This hypothesis failed to evoke unequivocal support (Frankenhaeuser et al., 1962; Tarter et al., 1971), at least in part because no accurate standard for quantifying cognitive complexity exists. Attempts to clarify the confusion generated by this lack of standardization have been complicated because measures of cognitive ability vary idiosyncratically. This variance in approach makes comparison between different studies and integration of their findings difficult. Nonetheless, it has at least been demonstrated that alcohol intoxication differentially affects different aspects of cognition (Birnbaum et al., 1980; Ekman et al., 1964).

The use of neuropsychological tests to determine the effects of alcohol intoxication could conceivably help reduce such confusion. These tests offer the possibility of clarifying the nature of the relationship between intoxication and neuropsychological dysfunction. Neuropsychological testing offers the possibility that alteration in functioning at the behavioral level

may be ascribed to disruption of a specified cortical system, rather than causally linked to impairment in some reified cognitive structure.

The research reported in this article was based upon the hypothesis that the pattern of acute alcohol-induced neuropsychological impairment is analogous to that characteristic of patients suffering from prefrontal damage. Two considerations guided the development of this hypothesis. In the first place, many so-called complex cognitive abilities, apparently susceptible to interruption by alcohol, are dependent upon the activity of the prefrontal cortex (Luria, 1980). Individuals who have suffered prefrontal injury are impaired in the temporal organization of behavior and reduced in the ability to think abstractly. They plan and/or implement courses of action poorly and cannot modify such courses in accordance with their consequences (Eslinger and Damasio, 1985). Similar deficits—most often described in terms of “abstraction” and/or “planning”—are characteristic of acutely intoxicated individuals (Jones and Vega, 1972; Washburne, 1956; Zeichner and Pihl, 1979).

In the second place, individuals with verified lesions of the prefrontal area exhibit a pattern of overt behavior reminiscent of that characteristic of intoxicated individuals. Damage to the prefrontal cortex, which synthesizes information about the state of the external objective and internal subjective worlds, impairs the regulation of behavior (Luria, 1980). Those with such damage cannot perceive errors in their performance, fail to appreciate the impact they make on others and cannot criticize themselves accurately

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(Lezak, 1976). Washburne (1956) postulated that alcohol intoxication similarly reduces people's awareness of their own actions, making them unable to modify these actions in response to environmental demands. Hull (1981) has also proposed that alcohol intoxication interferes with encoding processes fundamental to the state of self-awareness. These processes include the evaluation of self-relevant information, including cues about appropriate forms of conduct and evaluation of behavior in the recent past.

Deficits in verbal ability have also been associated with alcohol intoxication and with prefrontal impairment. Although patients with left frontal lobe lesions often appear to possess intact speech upon superficial examination (Walsh, 1978), closer analysis reveals impoverishment (Lezak, 1976), adynamia and perseveration (Walsh, 1978). Prefrontal patients often offer extraneous associations during verbal tests (Luria et al., 1967). Clinical studies show that patients with left frontal damage perform significantly worse than controls on word-fluency tests (Benton, 1968; Miller, 1988; Milner, 1964; Ramier and Hécaen, 1970). Decreased verbal fluency is also characteristic of subjects acutely intoxicated by alcohol (Hartocollis and Johnson, 1956) and these individuals also alter their responses to free-association tests (Weingartner, 1977).

In the present study, a variety of cognitive-behavioral tasks—some sensitive to particular types of neuropsychological impairment—were administered to the subject population. Those more sensitive to frontal function were included to provide a direct test of the hypothesis presented in this article. Nonfrontal and miscellaneous tests were administered to determine the specificity or generality of alcohol-induced impairment. Tests with well-established norms or tests that are apparently affected by highly specific forms of damage were included as first choices, but some in use for other reasons (sensitivity to expectancy, for example) were also included.

Dose and expectancy confound the effects of a drug, and play crucial roles in determining its behavioral sequelae (Marlatt and Rohsenow, 1981). Variation in response due to dose and expectancy was therefore directly measured. Subjects randomly received one of three widely differing doses of alcohol, under two conditions of expectancy, within the confines of a balanced-placebo design. A variety of techniques have been designed to ensure that subjects remain naive to condition and to therefore maintain the validity of the balanced-placebo design. Knight et al. (1986) have recently criticized these techniques. The present experiment was designed with these criticisms in mind, and was based on a modification of the balanced-placebo design devised by

Ross and Pihl (1989). The present study was additionally predicated upon the hypothesis that expectancy detrimentally affects performance. This secondary hypothesis was based on the assumption that alcohol intoxication provides an excuse for inappropriate or substandard behavior (Graham, 1980; Hull and Bond, 1986).

Method

Subjects

Seventy-two male university students between the ages of 18 and 34 were recruited as paid subjects by advertisement at McGill University. Moderate social drinkers (< 15 drinks/week) in good physical and mental health were selected for participation. The use of experienced drinkers in the sample ensured that all subjects were familiar with the effects of alcohol consumption. Exclusion criteria also included familiarity with psychological experimentation, and medical treatment that contraindicated alcohol consumption. Subjects were asked to refrain from consumption of alcohol for 24 hours and from food 4 hours before the experimental session.

Design and Procedure

Upon arrival at the laboratory, all subjects met the first of four experimental personnel. They were then required to sign an informed consent form. Each subject was then weighed and randomly assigned to one of the following six groups: told high dose, received high dose (THRH); told low dose, received high dose (TLRH); told high dose, received moderate dose (THRM); told low dose, received moderate dose (TLRM); told high dose, received low dose (THRL); and told low dose, received low dose (TLRL). High dose consisted of 1.32 ml of 95% pure pharmaceutical alcohol per kilogram of body weight; moderate dose of 0.66 ml/kg; and low dose, which was active placebo, of 0.132 ml/kg. Drinks for the high doses were mixed 1 part alcohol to 5 parts juice (3 parts grapefruit and 2 parts orange). The grapefruit juice helped mask the presence or absence of alcohol. Subjects in the moderate- and low-dose conditions received as much juice per kilogram of body weight as the subjects who received a high dose of alcohol. Subjects in each condition received instructions designed to maximize the effectiveness of the balanced-placebo design (Ross and Pihl, 1989). Each subject was given three drinks, and was asked to finish them within 20 minutes. After a 15-minute waiting period, each subject was given the first of three subjective intoxication scales and the first of three breath analyzer tests. Subjects completed their subjective

intoxication scales privately, to control for the effects of experimenter demand. The subjects were then taken to a separate testing area, where they were introduced to the remaining three experimenters, who were blind to the subjects' condition.

The following tests were divided equally among these three investigators, who administered them in random order to the subjects to control for differences in performance due to practice, fatigue, the effects of alcohol metabolism and central nervous system adaptation to alcohol intoxication.

Tests associated with the frontal cortex

Porteus Maze Test, Extension Series. This test measures planning and foresight (Porteus, 1959). Subjects are required to complete a series of mazes ranked according to difficulty. Individuals with lesions of the frontal lobes demonstrate impairments in their ability to correctly complete this test (Crown, 1952; Malmo, 1948; Mettler, 1952; Porteus and Kepner, 1944; Porteus and Peters, 1947).

Rey-Osterreith Complex Figure, Copy (1). Rey (1941) designed this complex figure to investigate perceptual organization and visual memory in brain-damaged patients (Lezak, 1976). The subject is first required to copy the figure from an original. Patients with prefrontal damage copy this figure (and complex figures in general) poorly (Lezak, 1976; Luria, 1980).

Thurstone Word Fluency Test. This test assesses the ability to generate words according to an abstract conceptual category. The subject must write as many words as he can beginning with the letter S in 5 minutes. Individuals with left frontal lobe damage consistently demonstrate significant impairments in word fluency (Benton, 1968; Miller, 1988; Milner, 1967; Ramier and Hécaen, 1970; Tow, 1955).

Self-Ordered Pointing Test, 12 Representational Drawings. The Self-Ordered Pointing Test measures organizational ability and sequencing of responses rather than the reproduction of sequences pre-organized by the experimenter (Petrides and Milner, 1982). Subjects must remember the location and sequence of each of 12 familiar pictures of objects presented 12 different ways. Individuals with frontal damage consistently manifest deficits in performance on this task. Individuals with temporal damage manifest deficits that vary with the severity of the lesion (Petrides and Milner, 1982).

Wisconsin Card Sorting Test. This test was devised to study "abstract behavior" and "shift of set" (Berg, 1948; Grant and Berg, 1948). The subject is given a deck of 64 cards, and is required to categorize these according to a plan established by the investigator. Individuals suffering from dorso-lateral frontal lesions perform poorly on this test (Milner, 1964).

Tests associated with the temporal cortex

Logical Memory, Immediate (I); Logical Memory, 25-Minute Delay (D). These Wechsler memory subtests assess the immediate and delayed recall of verbal ideas from an auditory presentation of two paragraphs. Individuals with temporal lobe damage perform poorly on these tests (Milner, 1975).

Rey-Osterreith Figure, Reproduction from Memory (2) & (3). After the subject has copied the Rey figure, he is subjected to a delay of 3 minutes and is asked to duplicate the figure from memory. The subject is then given a series of other tasks, and is finally asked to produce a final copy from memory 25 minutes later. Patients with right temporal lobe damage (particularly those with hippocampal lesions) have difficulty in completing this task (Milner, 1975).

Paired Associates, Difficult (D) and Easy (E). The Paired Associate subtest of the Wechsler Memory Scale assesses verbal retention (Lezak, 1976). The test consists of 10 word pairs; six forming easy associations and four forming word pairs not easily associated. Individuals with left temporal lesions have difficulty in completing this test (Milner, 1975). Additionally, there is evidence that organic brain damage particularly impairs recall of the difficult pairs (Walsh, 1978).

Tests associated with the parietal-occipital cortex

Albert's Simple Test of Visual Neglect. Subjects are given a pencil and a sheet of paper with a number of short lines printed on it, and are asked to bisect each line with another short line. Subjects who suffer from parietal damage perform particularly poorly on this test (Albert, 1973).

Apraxia Questionnaire. The apraxia questionnaire provides a general measure of ideomotor, ideational and buccofacial apraxia. Apraxia has been operationally defined by exclusion as a disorder of skilled movement not caused by weakness, akinesia, deaf-ferentiation, abnormal tone or posture, movement disorders, intellectual deterioration, poor comprehension or lack of cooperation (Heilman and Valenstein, 1985).

Miscellaneous tests

These tests were included in the battery for a variety of reasons. The information and vocabulary subtests of the WAIS provide a good measure of general intelligence but are not generally affected by frontal trauma in adulthood (Lezak, 1976). Analysis of the results of these tests would help determine whether alcohol affected the factor of "general intelligence" or if its action was more specific in nature.

The digit symbol subtest was included because it is sensitive to variations in motivation (Lezak, 1976). Alcohol in high doses impairs certain aspects of motor performance (Connors and Maisto, 1980; Hull and Bond, 1986; Lewis, 1969). A Reaction Time Test was included along with the Pursuit Rotor Test to aid in clarifying the nature of this impairment. Scores are assigned in tenths of a second for the reaction time test and in seconds for the Pursuit Rotor Test. The Young-Pihl Memory Test consists of a list of 40 words taken randomly from the Thorndike-Lorge AA lists (1944) read by the examiner at a rate of one per second. Subjects are given 90 seconds to recall and write down the words in any order, immediately after hearing the list. This test has proved sensitive to the effects of expectancy in the past (Young and Pihl, 1980).

All subjects were given standardized instructions relevant to the tests, and completed all tests within 1.5 hours. Subjects were administered the second and third breath analyzer tests and subjective intoxication scales at the midpoint and conclusion of the testing session. Each subject was then debriefed and, once his blood alcohol concentration had fallen below 0.04, was paid \$25 and allowed to leave. Subjects were not allowed cigarettes during the testing session to control for any potential interaction between the chemicals in cigarette smoke and alcohol.

Results

As the hypothesis to be tested varied according to the test, univariate analysis of variance was employed separately for each measure. No significant interaction effects emerged from any analysis.

Blood alcohol concentration

All subjects in the high-dose condition maintained BACs above the legal limit. Means, standard deviations and significance levels are presented in Table 1. Significant differences between dosage groups were found in the first ($F = 256.8$, 2/63 df), second ($F = 217.5$, 2/63 df) and final BAC reading ($F = 491.2$, 2/63 df).

Subjective intoxication

Analysis of the subjective intoxication scale indicated that the expectancy manipulation was successful. Means and standard deviations are listed in Table 2. Within dosage groups, subjects told that they were receiving a high dose of alcohol rated themselves as more intoxicated than subjects told they were receiving a low dose. Initial ($F = 4.15$, 1/65 df, $p < .05$) and mid-session ratings ($F = 4.38$, 1/65 df, $p < .05$) were significant, while final ratings ($F = 3.28$, 1/65

TABLE 1. Blood alcohol concentrations

	DOSAGE GROUP					
	High dose		Medium dose		Low dose	
	Mean	SD	Mean	SD	Mean	SD
One	.093 ^{a,b}	.021	.044 ^c	.009	.003	.0005
Two	.103 ^{a,b}	.027	.031 ^c	.008	.002	.0006
Three	.083 ^{a,b}	.013	.022 ^c	.009	.002	.0005

Note: All reported differences are significant at $p < .01$.

^a Represents a significant difference between the high- and low-dose conditions.

^b Represents a significant difference between the high- and medium-dose conditions.

^c Represents a significant difference between the medium- and low-dose conditions.

df, $p < .07$) approached significance. Further analysis (Tukey Test) carried out on the subjective intoxication ratings indicated that the first ratings carried out by subjects in the low-dose condition differed significantly from those completed by subjects in the medium- ($p < .01$) and high-dose ($p < .01$) conditions. Significant differences between the high- and medium-dose conditions ($p < .05$), high- and low-dose conditions ($p < .01$) and medium- and low-dose conditions ($p < .01$) also emerged from analysis of the mid-session rating scale, although these differences disappeared by the end of the testing period.

Effects of expectancy

Only two of more than 20 tests proved sensitive to expectancy effects, albeit not as predicted. Subjects told they were receiving a high dose of alcohol scored significantly higher on the Digit Symbol ($F = 5.03$, 2/63 df, $p < .05$) and Young-Pihl Memory Tests ($F = 11.16$, 1/64 df, $p < .001$) than subjects who were told that they were receiving a low dose of alcohol. For the sake of clarity, and since expectancy

TABLE 2. Subjective intoxication measures

	Minutes	NUMBER OF BEER EQUIVALENTS SELF-REPORTED			
		Told high dose		Told low dose	
		Mean	SD	Mean	SD
High dose:	30	3.73	1.19	3.53	1.29
	60	3.10	1.19	2.74	1.64
	90	2.35	1.06	1.86	0.67
Medium dose:	30	3.28	0.82	2.93	1.22
	60	2.63	1.06	1.88	1.01
	90	1.48	0.48	1.34	0.51
Low dose:	30	2.71	0.75	1.77	0.71
	60	1.62	0.91	1.10	0.29
	90	1.13	0.32	1.00	0.00

Note: See Method section for levels of significance.

effects were otherwise minimal, further analyses were carried out on means collapsed within dosage groups.

Tests associated with the frontal cortex

Four of six measures of frontal lobe functioning proved sensitive to the effects of intoxication induced by a high dose of alcohol. Means, standard deviations and levels of significance are presented in Table 3. The quantitative ($F = 8.55$, 2/64 df, $p < .001$) and qualitative ($F = 6.79$, 2/64 df, $p < .002$) score analyses of the Porteus Maze demonstrated that performance on this test was significantly impaired by the effects of alcohol intoxication. The quantitative score is awarded in terms of mental age. Maximum score is 17. Half a year is subtracted for each unsuccessful trial. Lowest possible qualitative score is 100. Scores on the Thurstone Word Fluency Test ($F = 4.20$, 2/62 df, $p < .05$) also were significantly negatively affected by alcohol intoxication. Scores reported are number of words generated. Intoxicated subjects also demonstrated significant impairments in their ability to reproduce the Rey-Osterreith complex figure (reproduction from copy: $F = 4.85$, 2/63 df, $p < .01$). Maximum possible score is 35. Tukey Tests were carried out subsequent to the ANOVA (see Table 3). The results of these tests indicated a strong detrimental high-dose effect of alcohol intoxication. The Self-Ordered Pointing and Wisconsin Card Sort Tests did not appear to be sensitive to the effects of alcohol consumption. The Self-Ordered Pointing scores reported are total errors in three trials. The Wisconsin Card Sort scores are total errors.

Tests not directly associated with the frontal cortex

Five of 14 measures of nonfrontal function also proved sensitive to the effects of high-dose alcohol intoxication. Analysis of the Logical Memory subtest demonstrated that alcohol intoxication significantly impaired delayed recall of verbal information ($F = 6.99$, 2/64 df, $p < .002$). Intoxicated subjects were also impaired in their ability to reproduce the Rey-Osterreith figure after a delay (3-minute delay: $F = 12.77$, 2/64 df, $p < .001$; 25-minute delay: $F = 15.21$, 2/64 df, $p < .001$). Alcohol consumption significantly impaired recall of the difficult pairs of the Paired Associates ($F = 3.32$, 2/64 df, $p < .05$), while the level of impairment approached significance for recall of the easy pairs ($F = 2.52$, 2/64 df, $p < .09$). The Pursuit-Rotor Test also proved sensitive to the detrimental effects of alcohol intoxication ($F = 6.41$, 2/62 df, $p < .003$). Means, standard deviations and levels of significance for all nonfrontal tests except for the test of visual neglect and the Apraxia Questionnaire are presented in Table 3. No

subjects made any errors while carrying out the tasks associated with these two tests.

Discussion

The present study tested two hypotheses: (1) that a similar neuropsychological deficit pattern underlies prefrontal lobe syndrome and alcohol intoxication, and (2) that alcohol expectancy detrimentally affects neuropsychological performance. The results provide mixed support for the first hypothesis at the high-dose level, although it is apparent that alcohol intoxication severely disrupts those functions associated with the temporal lobe as well. The experimental subjects demonstrated significant levels of impairment, after consuming a high dose of alcohol, on four of six measures of prefrontal lobe function and on four of six measures of temporal function. Functions associated with the parietal-occipital lobe remained intact, as did those assessed by the miscellaneous tests (with the exception of the Pursuit Rotor Test). The second hypothesis did not receive support. The expectancy manipulation employed during this study was successful, according to measures of subjective intoxication. Nonetheless, expectancy played a minimal role in determining the effect of alcohol upon neuropsychological functioning and, when it did play a role, it was in the opposite direction to that predicted. The fact that functional tolerance to alcohol intoxication can be learned (Hoffman and Tabakoff, 1985) may account for the direction of the expectancy effect seen in this study. If experienced drinkers receive accurate information about the dose of alcohol they are consuming they may be able to adapt and adjust to that dose if circumstances demand it.

The study is interesting because it demonstrates that alcohol intoxication per se impairs certain aspects of neuropsychological functioning. Cognitive skills dependent upon the prefrontal cortex suffer deterioration. Such skills are crucial to successful completion of the Porteus Mazes (Porteus, 1959), the Rey copy (Lhermitte et al., 1972) and to the copying of complex figures in general (Luria and Tsvetkova, 1964). Although individuals with parietal lobe damage also have difficulty copying complex figures (Walsh, 1978), the pattern of impairments induced by alcohol intoxication is not suggestive of constructional apraxia. Additionally, Albert (1973) found that constructional apraxia was highly correlated with visual neglect, and alcohol intoxication did not produce visual neglect in the present study. Impairments in word fluency are also associated with damage to the frontal lobe (left, anterior to Broca's area) (Benton, 1968; Milner, 1967; Ramier and Hécaen, 1970; Tow, 1955). Since the prefrontal cortex is responsible for the planning and

TABLE 3. Test of cognitive functioning

	High dose		DOSAGE GROUP Medium dose		Low dose	
	Mean	SD	Mean	SD	Mean	SD
<i>TESTS ASSOCIATED WITH THE FRONTAL CORTEX</i>						
<i>Porteus Maze</i>						
Quantitative	12.78 ^{a†}	2.11	14.83	1.62	14.42	1.56
Qualitative	75.40 ^{a†*}	23.4	53.6	26.3	49.6	26.9
Rey Osterreith (1)	30.58 ^{a†}	3.40	32.50	2.40	33.23	2.65
Word Fluency	39.09 ^{a*}	9.86	46.00	11.63	48.46	10.82
Self-Ordered Pointing	3.51	1.63	4.04	1.77	3.01	1.91
Wisconsin Card Sort	20.33	11.49	17.63	11.78	17.20	11.12
<i>TESTS ASSOCIATED WITH THE TEMPORAL CORTEX</i>						
Logical Memory (I)	9.43	2.84	11.04	3.07	10.87	2.98
Logical Memory (D)	6.86 ^{a†*}	2.74	9.27	3.11	9.91	2.69
Rey Osterreith (2)	13.40 ^{a†*}	4.63	20.28	6.40	21.78	6.62
Rey Osterreith (3)	13.06 ^{a†*}	3.87	19.93	6.60	21.77	6.40
Paired Associates (E)	16.51	1.92	17.45	1.32	16.96	1.35
Paired Associates (D)	6.53 ^{a*}	3.00	7.95	2.51	8.64	2.77
<i>MISCELLANEOUS TESTS</i>						
<i>WAIS</i>						
Information	23.70	2.70	23.59	2.82	23.23	3.35
Vocabulary	57.27	7.36	59.54	6.23	57.82	6.48
Digit Symbol	58.09	7.74	61.77	8.96	63.36	7.62
Reaction Time (1)	35.46	5.84	34.33	6.19	33.55	7.12
Reaction Time (2)	49.30	7.16	45.20	5.89	46.23	6.79
Reaction Time (3)	57.24	8.11	53.94	8.79	54.86	8.13
Young-Pihl Memory	6.42	1.79	7.31	2.67	7.64	2.10
Pursuit Rotor	9.52 ^{a†}	3.39	10.54	3.00	12.56	2.03

^a Represents a significant difference between the high- and low-dose conditions.

^b Represents a significant difference between the high- and medium-dose conditions.

* $p < .05$. † $p < .01$.

organization of behavior (Damasio, 1985), knowledge that alcohol impairs its functioning pharmacologically sheds an interesting light on consideration of intoxication.

Skills associated classically with the temporal cortex also deteriorate. Intoxicated subjects manifested deficits in all forms of delayed noncued memory (verbal and spatial) during this study, as well as performing poorly on the Wechsler Paired Associates Test. This pattern of impairment, overall, suggests that alcohol intoxication particularly affects the function of the hippocampus, which plays a key role in the transfer of information from short-term attention to long-term storage and/or in its retrieval (Gray, 1982; Luria, 1980; O'Keefe and Nadel, 1978). Extensive support for this idea is available within the relevant animal literature (Gray, 1982), which suggests that the benzodiazepines, barbiturates and alcohol act primarily on the septal-hippocampal system. Recent work by Begleiter and Porjesz (1988) also lends credence to this supposition. This is particularly

interesting given that the function of the hippocampus is not limited to memory, and that it and the septum play a role in eliciting a variety of cue-specific states of anxiety (Gray, 1982). These cues include novelty, threat of punishment and threat of frustrative nonreward (Gray, 1982). An alcohol-impaired septal-hippocampal system might well show reduced sensitivity to cues that would normally elicit anxiety and aid an individual in maintaining safety. Alcohol intoxication may allow people to engage in activities that would normally produce anxiety, and may produce cognitive impairments that limit their ability to act adaptively even if they do become anxious. These two factors may account in part for the pronounced tendency of intoxicated individuals to become both victims and victimizers.

Certain other aspects of this study deserve consideration as well. The detrimental effect of alcohol upon what has been described as "motor" performance has been consistently demonstrated (Connors and Maisto, 1980; Hull and Bond, 1986; Lewis, 1969;

Vuchinich and Sobell, 1978). Researchers who study the effects of alcohol intoxication have described both the Pursuit Rotor and Reaction Time Tests as indicators of "motor performance" (Williams et al., 1981). However, in the present study, alcohol intoxication did not decrease reaction time (in three different paradigms) nor performance on the Digit Symbol WAIS-R subtest, which is sensitive to deficits in motor persistence and response speed (Lezak, 1976). However, it did impair complex motor performance, as measured with the Pursuit Rotor. Different parts of the central nervous system control different aspects of movement. Recent research at the Montreal Neurological Institute suggests that the frontal lobes are particularly important in governing complex acts of motor performance, but do not determine motor speed (Leonard et al., 1988). Luria (1980), in his discussion of the motor analyzer, located in the frontal cortex, has made similar suggestions. These ideas might help clarify the confusion about alcohol's effect on motor performance. Perhaps intoxication has a more profound effect upon the sequencing, organizing and control of motor action, rather than upon its speed of execution.

Alcohol intoxication did not produce deficits in "verbal" or "general" intelligence per se, at least insofar as these qualities are measured by the Wechsler Adult Intelligence Scale Vocabulary and Information subtests. These tests are robust in the face of even extensive brain damage, and are often used as indicators of premorbid ability (Lezak, 1976). Alcohol intoxication also had no effect on performance during the Self-Ordered Pointing Test. This test was validated on individuals with extensive and variable lesions of the frontal cortex (Petrides and Milner, 1982), and it may be that alcohol affects a system that does not participate in the functions necessary to complete this test successfully. There is no evidence that alcohol intoxication detrimentally affects those functions associated with the occipital-parietal lobes, insofar as the present study is concerned. Tests for the functioning of this region are somewhat subjective in nature, but in all cases the performance of the intoxicated and sober subjects was equally perfect. No subjects manifested any signs whatsoever of neglect, of apraxia associated with limb and buccofacial gestures and manipulations, or serial acts. In summary, it seems as though the similarities between those behaviors associated with alcohol intoxication and those associated with prefrontal damage are striking, but that the two conditions are certainly not identical.

The fundamental hypothesis underlying this study suffered from a number of faults. The concept of "frontal-lobe syndrome" itself has been criticized as

a general descriptor for the dysfunction associated with damage to the prefrontal areas of the frontal lobes. The concept is too simplistic. Locale of damage, even within an area as specific and apparently nonspecialized as the frontal lobes, still variably affects behavior (Damasio, 1985). Although dysfunction associated with circumscribed lesions may be usefully described in terms of lobe location, it may be more accurate to attribute drug effects to alterations in neuropsychological systems, which are not likely to be lobe-specific. The prefrontal divisions are supplied with rich connections to various limbic structures, including the hippocampus (Luria, 1980). The hippocampus plays a key role in memory consolidation, and the memory deficits demonstrated by intoxicated subjects in the present study suggest that its functions may be impaired by alcohol consumption. The orbital-frontal cortex and various structures in the limbic system act as an integrated unit (Luria, 1980) and subsume many of the functions that seemed to be impaired by alcohol during this study. This theory, admittedly post hoc, seems to fit the facts more accurately than the hypothesis offered at the beginning of the study. Nonetheless, the results of the present study are interesting for a variety of reasons. Research into the effects of alcohol upon behavior has lacked a testable unifying hypothesis since its inception—and the use of tests validated clinically and experimentally offers the possibility for deeper comprehension of intoxicated behavior. In addition, the fact that alcohol affects cognition at a purely pharmacological level is striking, although Hull and Bond (1986) suggested that the expectancy effects associated with alcohol use primarily affect social behavior and not information processing per se. In the present study, the effects of expectancy upon cognitive functioning were minimal. Situations that involve alcohol consumption may serve as an excuse for indulging in certain types of antisocial behavior; nonetheless, it is evident that alcohol intoxication pharmacologically induces neuropsychological impairment. It does not seem unreasonable to suppose that such impairment has serious behavioral consequences.

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