Executive Functioning Early in Abstinence From Alcohol

Sandra Zinn, Roy Stein, and H. Scott Swartzwelder

Background: Executive dysfunction is among the cognitive impairments that may persist after abstinence in alcohol-dependent persons. The type(s) and extent of executive dysfunction early in abstinence have not been well characterized, but they may have important implications for the evolution of behavioral treatment strategies.

Methods: To determine which aspects of executive functioning were impaired in early abstinence, we administered memory and executive function tests to veterans who successively presented for treatment at an outpatient substance abuse clinic. We then compared the neuropsychological performance of these recovering alcoholics (*n* = 27) with that of age-matched primary care outpatients (*n* = 18). We also examined group differences in self-evaluation of cognitive decline and evaluated associations between drinking history and cognitive impairment in the index group.

Results: We found that the normal and alcohol-dependent groups differed on abstract reasoning, memory discrimination, and effectiveness on timed tasks. Patients in the alcohol-dependent sample were also more likely to perceive themselves as cognitively impaired. It is interesting to note that the duration of alcohol use did not relate to neuropsychological test performance, but recent quantity consumed and days of sobriety were associated with nonverbal abstract reasoning ability.

Conclusions: Executive functions are impaired early in abstinence and should, therefore, be taken into account when early behavioral treatments are being developed.

Key Words: Alcoholism, Cognition Disorders, Memory Disorders, Abstinence, Alcohol-Induced Disorders.

There is considerable evidence that prolonged, excessive alcohol use is associated with an increased likelihood of cognitive impairment that can persist after drinking cessation (Eckardt et al., 1995; Grant, 1987; Yohman et al., 1985). It has been estimated that 45% of alcohol-dependent individuals have residual deficits on neuropsychological testing after 3 weeks of abstinence, and as many as 15% retain deficits after 1 year of abstinence (Rourke and Loberg, 1996). In particular, abstract thinking, cognitive flexibility and persistence, and inhibition of competing responses seem to be frequently impaired after years of heavy alcohol consumption (Noel et al., 2001; Ratti et al., 2002). These functions are among those known as the executive functions—cognitive operations linked to the frontal cortex that guide complex behavior over time through planning, decision-making, and response control. Structural and physiological changes in relevant brain areas add to the evidence that executive dysfunction is a characteristic sequela of chronic heavy drinking. For example, chronic alcohol use causes atrophy of the frontal lobes (Krill et al., 1997; Kubota et al., 2001) and hypometabolism in the frontal cortex (Adams et al., 1993; Dao-Castellana et al., 1998; Demir et al., 2002), which is associated with specific neuropsychological deficits (Dao-Castellana et al., 1998). For a detailed review of frontal lobe and executive function changes associated with chronic alcoholism, see Moselhy et al. (2001).

In general, alcohol-related cognitive impairment seems to attenuate over time after cessation of drinking (De Soto et al., 1985; Grant et al., 1987; Munro et al., 2000; Reed et al., 1992), although factors such as age (Munro et al., 2000; Rourke and Grant, 1999), poor nutrition (Lotfi and Meyer, 1989; Skinner et al., 1989), and medical comorbidity (Adams and Grant, 1986; Edwin et al., 1999; Solomon and Malloy, 1992) seem to diminish the extent and prolong the time course of recovery. There is gathering evidence that executive functioning may also recover with the cessation of drinking, though systematic studies have been lacking. For...
example, abstracting abilities (Mann et al., 1999; Rourke and Grant, 1999), perceptual motor speed (Mann et al., 1999), and spatial abilities (Kish et al., 1980) show some recovery within several months of abstinence, but short-term, or working, memory has proved more resistant to recovery (Mann et al., 1999). This raises the question of whether some executive functions are related to the quantity or chronicity of intake, whereas others may be an invariant sequela of abuse or may exist premorbidly (Giancola and Moss, 1998).

The time course of cognitive recovery after cessation of drinking is also unclear, especially during the first few weeks. Some studies have shown partial recovery with 14 to 20 days of abstinence (Carlen et al., 1984), whereas others have concluded that cognition is relatively stable through early abstinence (Eckardt et al., 1979; Mann et al., 1999; Unkenstein and Bowden, 1991; Volkow et al., 1994). This could be important for treatment. If residual cognitive impairment after detoxification typically includes executive functions, learning, and memory (as well as visuospatial processing and perceptual/motor integration; Noel et al., 2001; Parsons, 1986; Rourke and Lobberg, 1996; Sullivan et al., 2000), a patient’s ability to use rehabilitative information is likely to be compromised during this period (Ihara et al., 2000; McCrady and Smith, 1986). It is clear that cognitive impairment affects the prognosis for treatment success (Parsons, 1983) and that moderate cognitive impairment compromises the learning of treatment content (Becker and Jaffe, 1984; Godding et al., 1992; Smith and McCrady, 1991).

Executive function deficits are among the alcohol-related cognitive impairments that are most likely to affect rehabilitation success (Ihara et al., 2000). Impairment of executive functions in alcoholics has been associated with attribution from rehabilitation and higher rates of relapse (Miller, 1991), as well as with social difficulties such as increased marital disruption (Tuck and Jackson, 1991) and employment failure (Moriyama et al., 2002), all of which conspire toward poor treatment outcomes. Thus, this study was designed to assess the pattern of executive function deficits early in abstinence. To accomplish this, we examined cognitive operations (memory and executive functions) in veterans who successively presented for treatment at an outpatient substance abuse clinic. To determine which aspects of executive functioning were impaired in this cohort, we compared cognitive performance in these recovering alcoholics with cognitively normal age-matched outpatient controls.

**METHODS**

Participants

All participants were male veterans receiving treatment at the Durham Veterans Affairs Medical Center. (Due to the low frequency of women entering substance abuse treatment at our facility, only men were enrolled.) Alcohol-dependent participants were recruited by clinicians in the Substance Abuse Clinic during their initial treatment sessions. Twenty-seven veterans with alcohol dependence were recruited, and they participated in at least the initial testing session. Eligibility criteria for index participants included a clinical diagnosis of alcohol dependence with DSM-IV criteria, abstinence of 6 weeks or less, and no history of frequent inhalant or opiate use, schizophrenia, or active psychosis.

Given the high rates of head injury among abusers of alcohol (Solomon and Malloy, 1992), we included participants with this history (if not severe) and recorded its presence and severity to evaluate any effect on our outcome analyses. Head injury was reported in 33% of the controls and in 52% of the substance abuse patients. The pooled samples were grouped by the presence or absence of mild to moderate head injury, and then comparison of group means for neuropsychological test scores was performed with a t test. To ensure that there was no synergistic effect between alcohol consumption and the residual effects of head injury, score means for the substance abuse patients were examined separately. There were no significant differences in scores attributable to head injury in either case.

Control participants were recruited from the Primary Care Clinic at the Durham Veterans Affairs Medical Center. Primary care patients of participating physicians were contacted before their appointments, screened by a brief interview for substance abuse and exclusionary medical conditions, and invited to participate if they qualified. Patients with major depression, untreated sleep apnea, polypharmacy, severe head injury, or any chronic neurological condition affecting cognition (such as Alzheimer’s or Parkinson’s disease), chronic psychosis, or schizophrenia were excluded from both the alcohol-dependent and control groups. Medical charts were examined to corroborate participants’ self-reports.

Nineteen control participants were recruited. (One control participant was excluded from the analyses due to a borderline estimated IQ.) Participants received $10 for each test session attended. The study was approved by the local Division of Veterans Affairs Institutional Review Board.

Demographic characteristics for the two groups, including an estimate of participants’ premorbid IQ based on the Barona index (Barona and Chastain, 1986), are shown in Table 1. The groups were not significantly different in age or education. The substance abuse group had a higher proportion of African Americans. The mean estimated Full Scale IQ differed significantly between groups, as shown in Table 1. Two WAIS-III subtests (Similarities and Matrix Reasoning) were administered as part of the neuropsychological battery, and we examined the mean observed scores on these tests to validate the estimated group IQ differences by using a Wilcoxon summed ranks statistic. The controls had higher mean scores on these tests, but the group disparities for actual performance on these subtests from the IQ measure were far less significant (Similarities, \( p = 0.04 \); Matrix Reasoning, \( p = 0.02 \)) than those of the Barona formula estimate. The estimate may exaggerate IQ differences in our samples due to its dependence on race and occupational achievement.

Because various medications may affect cognition, we examined the medical records of both index and control participants to determine whether any of these medications was being taken at or near the time of testing. Selective serotonin reuptake inhibitor–type antidepressants were the most frequent psychotropic medications taken. None of the substance abuse patients was taking benzodiazepines, although one control patient was. The number of cognition-affecting medications was tabulated for each subject, and the frequency of such medication use for substance abuse patients and controls was evaluated with a \( \chi^2 \) test. There was no significant difference in medication use between the substance abuse and control groups that would be expected to account for any neuropsychological test score differences found. Eleven of the substance abusers were

| Table 1. Participant Characteristics by Group |

<table>
<thead>
<tr>
<th>Demographic variable</th>
<th>Substance abuse patients (n = 27)</th>
<th>Controls (n = 18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age, years (SD)</td>
<td>50.8 (5.6)</td>
<td>52.4 (8.5)</td>
</tr>
<tr>
<td>Race (% Caucasian)*</td>
<td>22.2%</td>
<td>77.8%</td>
</tr>
<tr>
<td>Mean years of education (SD)</td>
<td>12.6 (2.1)</td>
<td>13.4 (2.5)</td>
</tr>
<tr>
<td>Estimated Full Scale IQ (SD)*</td>
<td>99.0 (8.1)</td>
<td>106.5 (7.8)</td>
</tr>
</tbody>
</table>

* Significantly different, \( p < 0.005 \).
taking no cognition-impairing medications, whereas 16 were taking 1 or more of these. For the control participants, 7 were not taking any of these medications, and 11 were taking one or more.

Test Administration

Testing was conducted within 1 week of treatment intake for the substance abuse patients. All participants were first interviewed about their background and daily functioning. As part of this interview, participants were asked self-evaluative questions about their cognition, such as “Do you believe your thinking is as good as it’s always been?” and “When you are trying to solve a problem, are you more easily frustrated than you used to be?” A history of head injury was taken. Participants with an alcohol abuse history were also interviewed to obtain a detailed drinking history. The participants were then administered a fixed-order neuropsychological battery of seven tests. All instruments were administered by the principal investigator or a trained research assistant. Standardized administration of the instruments was ensured through training and rote use of printed administration instructions.

Instruments

To pursue our examination of which aspects of executive functioning might reflect the exigencies of early abstinence, we selected measures of fluency, abstract reasoning, cognitive flexibility, and psychomotor speed. Both reasoning and fluency have been associated with impaired functioning of the frontal cortex in alcoholic patients, as noted previously. Because it has been hypothesized that right-hemisphere (or nonverbal) functions are more frequently affected than left-hemisphere functions (Clifford, 1986), we assessed both verbal and nonverbal aspects of abstract reasoning, fluency, and memory. Fluency tests require rapid production of variations on a theme (words beginning with the same letter or line patterns connecting dots) while avoiding repetition. We used the Controlled Oral Word Association Test (Borkowski et al., 1967) to assess verbal fluency and the Ruff Figural Fluency Test (RFFT) (Ruff et al. 1987, 1994) to assess nonverbal fluency.

Several measures of abstract reasoning have shown performance deficits in patients with alcoholism, as noted previously. Lezak (1995) notes that concrete thinking and reduced cognitive flexibility are common with generalized brain damage. Two subtests from the WAIS-III (Wechsler, 1997) were used to assess abstract reasoning. Similarities is a well known measure of verbal concept formation that requires the respondent to draw increasingly abstract relationships between concepts. Matrix Reasoning is similar to Raven’s Progressive Matrices and assesses increasingly complex visual analogies by having the respondent fill in the missing part of a pattern from five choices. To assess psychomotor speed and cognitive flexibility, we administered the Trail Making Test (Armitage, 1945). Trails Part A is a more direct measure of psychomotor speed, whereas Trails Part B, with its set-switching demands, also reflects cognitive flexibility. The RFFT perseveration score provided another index of cognitive flexibility.

We also examined whether verbal and nonverbal memory were differentially affected by chronic alcohol abuse. Verbal memory was assessed with the Hopkins Verbal Learning Test (HVLT) (Benedict et al., 1998; Brandt, 1991). The HVLT is a verbal list learning task that uses three semantically related categories, three learning trials, and both a free recall and a recognition memory trial after a 30-min delay. The number of words recalled on HVLT trial 4 were used to index delayed memory function. Indices of memory discrimination, typically considered to be dependent on executive functions, were provided by recognition accuracy and the number of intrusion words on recall for the HVLT.

Nonverbal memory was assessed with the Rey Osterrieth Complex Figure (ROCF) test (Rey and Osterrieth, 1993). The ROCF is a complex figure construction task with an unwarned delayed recall. (We did not administer an immediate recall trial.) Delayed spatial memory was measured by the ROCF delayed recall accuracy score. An organization score for the ROCF (Zinn and Swartzwelder, 2002) was used to assess organizational strategy, an executive function–based component of visual memory (Gaffan et al., 2002; Lange et al., 2000).

Because the samples were age matched, we used raw test scores for between-group comparisons of neuropsychological variables. Comparison of neuropsychological test scores for those older than 50 years with those aged 50 years or less (by using a t test) revealed no differences. To minimize any potential confounding of age in examining drinking history and addiction severity relationships, however, we used age-corrected test scores, standardized with commonly accepted normative samples for each test, in these analyses.

The Addiction Severity Index (ASI; McLellan et al., 1985) is a well validated instrument assessing substance abuse patient functioning in multiple life domains (Alterman et al., 1994; Hodgins and El-Guebaly, 1992; Stoeffelmayr et al., 1994). For this assessment, substance-abusing patients are administered a structured interview to determine the severity of their addiction problem by assessing its effect on seven areas of potential difficulty: medical, psychiatric, legal, family, employment, alcohol, and other drug use. The ASI was administered at treatment intake by the trained clinicians of the Substance Abuse Clinic. We examined both the addiction severity score and the composite score, a weighted sum reflecting current status and functioning in the seven dimensions assessed by the ASI.

Drink History

Participants recruited from the Substance Abuse Clinic were interviewed regarding their drinking history. We asked them at what age they began using alcohol, how many years they had been dependent drinkers, and their weekly average intake over the past 6 months. We also asked these participants the date of their last drink, which we validated by using the medical chart reports of their clinic intake. We used self-report of years of dependency as the measure of chronicity. The average number of years of dependency for our sample was 29.9 (range, 17–50). Average age of drinking onset was 14 years (SD, 3.7 years). Our measure of recent quantity consumed, the weekly average intake for each participant, was converted into standard drinks. Mean weekly intake was 12.9 (SD, 11.0) drinks. The minimum reported recent intake was 1.52, and the maximum was 46.3. We defined abstinence duration as the number of days from the last drink to the date of testing. Participants were sober for 21.7 days on average at the time of testing; however, one had been abstinent for only 1 day, and one had been abstinent for 58 days. (We examined the test scores of the individual who had been abstinent only 1 day, but these were typically within 1 SD for the mean of the group, so the data were included in the analyses.) The addiction severity composite score from the ASI was also used as a measure of drink history. This index reflects the consequences of substance abuse, as well as quantity and use history.

Statistical Tests

Comparisons of demographic characteristics between controls and alcohol abusers were conducted with t tests or χ² tests, as appropriate. Between-group differences for cognitive functioning were examined with t tests for normally distributed raw test scores, whereas cognitive function indices from which the data were not normally distributed were assessed with the nonparametric Wilcoxon summed ranks test by using the probability value for a one-sided t test (predicting worse scores for the alcohol abuse sample). To examine the relationship between cognitive functioning and both drinking history and addiction severity, Spearman correlations were established between age-corrected test scores and the variables described previously. To reduce type II error, we conducted correlations only on test score variables that were different between controls and substance abusers.

RESULTS

Significant differences in cognitive performance were found between recovering patients and controls for several
indices of executive functioning (Table 2). These are presented by the cognitive function assessed. Given the possibility that IQ differences might account for the disparity between groups, we also performed analyses of covariance (ANCOVAs) with estimated verbal or performance IQ (as appropriate) as a covariate predicting each test score that showed a significant difference between controls and patients with alcoholism. These ANCOVA results are reported as each test result is described. For direct contrast of the scores, graphs showing both samples’ scores standardized as z scores, using the manual norms for each test (Heaton norms for Trail Making), presented in Figs. 1 and 2.

Fluency

Only nonverbal fluency showed group differences; verbal fluency was similar in the two groups. Figural fluency (assessed by number of unique designs produced) was impaired in the substance abuse group relative to the control group. When estimated performance IQ was used as a covariate in an ANCOVA model, it was a significant predictor of the figural fluency score ($p = 0.003$), but the effect of group status remained ($p < 0.001$).

Abstract Reasoning

Both verbal and nonverbal reasoning differed between groups. The sample of alcohol-dependent patients scored significantly worse on verbal abstraction, as measured by the Similarities subtest, than did the controls. Abstract reasoning for visual material, assessed by Matrix Reasoning, was worse in the substance abuse patients. Our check for the potential confound of premorbid IQ suggested that premorbid IQ was a more potent predictor of reasoning performance in both modalities than group status, although group status remained a significant predictor. For prediction of both verbal and nonverbal reasoning, estimated IQ (verbal for Similarities; performance for Matrix Reasoning) was significant at $p < 0.001$, whereas group status in each case had a $p$ value of 0.02.

---

**Table 2.** Means and Standard Deviations of Neuropsychological Test Scores by Group

<table>
<thead>
<tr>
<th>Neuropsychological test score</th>
<th>Substance abuse patients ($n = 27$)</th>
<th>Controls ($n = 18$)</th>
<th>$p$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>COWA (total words)</td>
<td>39.2 (9.3)</td>
<td>43.8 (10.5)</td>
<td>0.13</td>
</tr>
<tr>
<td>Ruff Figural Fluency—uniques</td>
<td>58.3 (23.1)</td>
<td>81.3 (24.1)</td>
<td>0.002</td>
</tr>
<tr>
<td>Similarities</td>
<td>18.6 (4.5)</td>
<td>21.5 (5.0)</td>
<td>0.05</td>
</tr>
<tr>
<td>Matrix Reasoning</td>
<td>10.8 (5.5)</td>
<td>14.4 (5.5)</td>
<td>0.04</td>
</tr>
<tr>
<td>HVLT Learning (additional words)</td>
<td>2.9 (1.8)</td>
<td>2.8 (1.1)</td>
<td>0.44</td>
</tr>
<tr>
<td>HVLT Delayed Recall (No. words)</td>
<td>7.4 (2.5)</td>
<td>8.6 (1.9)</td>
<td>0.09</td>
</tr>
<tr>
<td>HVLT Intrusions</td>
<td>2.2 (2.5)</td>
<td>0.9 (1.2)</td>
<td>0.02</td>
</tr>
<tr>
<td>HVLT Recognition—true positives</td>
<td>10.9 (1.3)</td>
<td>11.4 (1.3)</td>
<td>0.24</td>
</tr>
<tr>
<td>HVLT Recognition—false positives</td>
<td>1.2 (1.4)</td>
<td>1.1 (0.8)</td>
<td>0.30</td>
</tr>
<tr>
<td>Rey Osterrieth—copy accuracy</td>
<td>27.6 (5.9)</td>
<td>29.9 (4.1)</td>
<td>0.15</td>
</tr>
<tr>
<td>Rey Osterrieth—delayed recall</td>
<td>13.9 (5.3)</td>
<td>17.3 (4.8)</td>
<td>0.03</td>
</tr>
<tr>
<td>Rey Osterrieth—organization</td>
<td>48.7 (24.6)</td>
<td>49.8 (29.0)</td>
<td>0.88</td>
</tr>
<tr>
<td>Ruff Figural Fluency—perseverations</td>
<td>18.5 (22.1)</td>
<td>16.7 (22.6)</td>
<td>0.48</td>
</tr>
<tr>
<td>Trails A (time in seconds)</td>
<td>43.6 (16.3)</td>
<td>33.8 (8.5)</td>
<td>0.01</td>
</tr>
<tr>
<td>Trails B (time in seconds)</td>
<td>151.3 (98.9)</td>
<td>84.1 (40.0)</td>
<td>0.003</td>
</tr>
</tbody>
</table>

COWA, Controlled Oral Word Association Test.

---

**Fig. 1.** Performance by group on memory tests.

**Fig. 2.** Performance by group on executive function tests.

---

**Memory**

Although learning and verbal memory were equivalent between groups, patients with alcohol dependence had more difficulty discriminating list words from other semantically related words during retrieval; this resulted in more intrusions during recall but adequate recognition of list words. Nonverbal memory, as reflected in ROCF delayed recall accuracy, was also poorer in the alcohol group. Esti-
significant correlation (\( r = 0.52; p = 0.005 \)). Thus, heavier drinkers were likely to have been sober longer at the time of testing.

Substance-abusing patients’ cognitive functioning did not, in general, show significant relationships to daily life difficulties rated in the ASI. Nonverbal reasoning was, however, related to employment difficulties: poorer reasoning was associated with greater difficulty in staying employed (\( r = -0.56; p = 0.008 \)). More intrusions (autodistraction) on word recall were related to family difficulties (\( r = 0.42; p = 0.05 \)). The Legal, Medical, and Psychiatric subscales of the ASI showed no significant relationships to neuropsychological performance.

**DISCUSSION**

**Executive Function**

We found several deficits in executive functioning among treatment-seeking, recently abstinent alcohol-abusing patients relative to same-age control patients. Reasoning, nonverbal fluency, performance of timed complex tasks, and difficulty with discriminative memory were impaired in patients with alcohol dependency. However, executive functions were not consistently impaired across the board, nor were nonverbal functions uniformly depressed. For example, nonverbal fluency was impaired, whereas verbal fluency was not. Although this alcohol-abusing sample had greater difficulty in general with nonverbal, right hemisphere–related executive tasks (consistent with our hypotheses and other reports; Grant, 1987), they manifested mild verbally related performance deficits as well. Furthermore, although perseveration and strategy are thought to be dependent on the frontal cortex, the substance abuse group had few perseverations on the RFFT and did not differ in their organizational ability (on the ROCF) from the controls. It is not clear whether some of the variability in our results may be due to the inclusion of less-alcohol-dependent patients in our sample.

The neuropsychological variables showing the greatest discrepancy between alcohol-abusing patients and controls (RFFT unique designs and Trails Part B) are both timed tasks with a motor component, visual perception elements, and considerable working memory demands. This raises the possibility that some deficits found after chronic alcohol abuse may be due to a superimposition of mild performance decrements in different cognitive operations. Impairment on these tasks did not seem to be related to recent drinking patterns and may represent gradually acquired deficits that do not remit early in abstinence. The contribution of premorbid ability, represented by estimated performance IQ, to these cognitive efficiency tasks was minimal. Nonverbal or novel tasks may be more susceptible to the effects of chronic alcohol because of their reliance on global or generic processes that become less effective with chronic alcohol abuse and eventually erode to the point of deficit on a high-demand task.

Impairments in our sample were also observed on both

**Psychomotor Speed and Cognitive Flexibility**

Finally, patients with a history of alcohol abuse also took longer to complete each trial of the Trail Making Test than did controls. Trails Part A, an assessment of psychomotor speed, showed differences between controls and alcohol abuse patients. Part B, which indexes cognitive flexibility, demonstrated a highly significant difference between groups. It is interesting to note that estimated performance IQ accounted for a significant portion of the variance in Trail Making scores. For part A, both group status and Performance IQ (PIQ) were significant at \( p = 0.02 \); for part B, group status was significant at \( p = 0.007 \), whereas PIQ contributed to prediction at \( p = 0.04 \).

Participants’ perceptions of their cognitive status and problem-solving ability were consistent with these findings (Table 3). Patients who were chronic alcohol abusers more frequently reported that their thinking was worse than it used to be. These patients distinguished their general cognitive ability from their memory functioning, as predicted: fewer reported worse memory, and their rates of endorsement did not significantly differ from those of controls, although there was a trend nearing significance (\( p = 0.07 \)). The participants recovering from alcohol dependence also noted increased frustration at higher rates than the control participants.

Our prediction that recent drink quantity would be related to executive function tests with a psychomotor speed component was not supported. The only executive function measure to correlate with drink history was nonverbal abstract reasoning (Matrix Reasoning; Table 4). This score covaried with both recent quantity and days since the last drink. It is interesting to note that both correlations were positive—both higher quantities of recent drinking and a longer duration of sobriety were associated with a better Matrix Reasoning score (Figs. 3 and 4).

Examination of the association between the days of sobriety and recent drink quantity variables revealed a significant correlation (\( r = 0.52; p = 0.005 \)). Thus, heavier drinkers were likely to have been sober longer at the time of testing.

<table>
<thead>
<tr>
<th>Question</th>
<th>Substance abuse (% endorsing)</th>
<th>Controls (% endorsing)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Is your thinking worse than it used to be?</td>
<td>85.2*</td>
<td>44.4</td>
</tr>
<tr>
<td>Is your memory worse than it used to be?</td>
<td>48.2</td>
<td>22.2</td>
</tr>
<tr>
<td>Are you more easily frustrated than you used to be?</td>
<td>73.1**</td>
<td>44.4</td>
</tr>
<tr>
<td>Are you typically less patient than others?</td>
<td>55.6</td>
<td>44.4</td>
</tr>
</tbody>
</table>

* \( p < 0.01 \).
** \( p = 0.05 \).
verbal and nonverbal memory tests. Learning rates, delayed recall, and recognition were not impaired for verbal material. The verbal memory difficulty involved intrusions on free recall, with improvement on recognition. This pattern suggests that retrieval, but not learning or retention, is affected and thus implicates frontal rather than temporal cortical involvement, as predicted. Several studies have suggested that patients with impaired executive functioning have faulty retrieval strategies (Mangels et al., 1996; Moscovitch and Melo, 1997) and, hence, faulty discrimination between target words and distracters. The source of the impairment in figure recall for our sample was less clear. It seems likely that impaired encoding or retrieval strategies would account for some of the nonverbal memory deficit. Inadequate organization on construction, typically associated with executive dysfunction, can affect figure recall (Dawson and Grant, 2000), but there was no clear impairment of organizational quality in our sample. It may be that our measure of organization was less sensitive or reliable than those used in the Dawson and Grant study. Further research is needed to determine whether figural memory deficits in chronic abusers of alcohol may involve frontal, parietal, and temporal lobe–based processes.

**Self-Perception of Cognitive Deficits**

The picture of reduced cognitive power drawn by our findings is also supported by the personal experience of the alcohol abusers in our sample. These patients perceived their thinking as deteriorating over time and their frustration level as increasing, in comparison to the control sample. There was a trend toward perception of increased memory difficulties as well. These acknowledgments of impaired cognition suggest that patients have observed cognitive lapses on multiple occasions in their daily lives.

Although denial of consequences has been documented in persons with alcoholism, at least while intoxicated, our participants’ responses show that they are aware of some deleterious effects. It is not clear, however, that these perceptions of impairment are any more effective than other health concerns in motivating a reduction in drinking. Maintenance of sobriety does not seem directly related to neuropsychological variables, despite the compelling intuitive appeal of this idea. The treatment-adherence literature supports the ineffectiveness of knowledge as a sufficient prerequisite for healthy behavior in many persons with various medical problems; the multiple approaches to understanding lack of behavior change in that literature may be fruitful for the substance abuse population.

### Table 4. Correlations With Drink Indices (n = 27)

<table>
<thead>
<tr>
<th>Executive function measure</th>
<th>Years drinking</th>
<th>Recent quantity (in standard drink units)</th>
<th>Days sober</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verbal memory (HVLT Intrusions raw score)</td>
<td>−0.02 (0.92)</td>
<td>0.07 (0.74)</td>
<td>0.07 (0.73)</td>
</tr>
<tr>
<td>Verbal memory (HVLT true positives on recognition z score)</td>
<td>0.07 (0.74)</td>
<td>−0.19 (0.35)</td>
<td>0.01 (0.95)</td>
</tr>
<tr>
<td>Figural memory (RO figure delayed recall accuracy)</td>
<td>0.21 (0.30)</td>
<td>−0.17 (0.40)</td>
<td>−0.10 (0.62)</td>
</tr>
<tr>
<td>Figural fluency (RFFT Unique Designs z score)</td>
<td>0.04 (0.85)</td>
<td>0.06 (0.78)</td>
<td>0.26 (0.18)</td>
</tr>
<tr>
<td>Verbal abstract reasoning (Similarities scaled score)</td>
<td>0.11 (0.58)</td>
<td>−0.12 (0.58)</td>
<td>−0.12 (0.57)</td>
</tr>
<tr>
<td>Nonverbal abstract reasoning (Matrix Reasoning scaled score)</td>
<td>0.29 (0.16)</td>
<td>0.51 (0.008)</td>
<td>0.41 (0.04)</td>
</tr>
<tr>
<td>Trails A (t score)</td>
<td>0.00 (0.99)</td>
<td>0.01 (0.94)</td>
<td>0.01 (0.95)</td>
</tr>
<tr>
<td>Trails B (t score)</td>
<td>0.29 (0.15)</td>
<td>0.00 (0.99)</td>
<td>0.12 (0.54)</td>
</tr>
</tbody>
</table>

RO = Rey Osterrieth.
Data are r value (p value).

---

![Fig. 3. Correlations of Matrix Reasoning with duration of sobriety.](image1)

![Fig. 4. Correlations of Matrix Reasoning with average daily intake quantity.](image2)
It is reasonable to infer that these observed cognitive lapses would have affected real-world functioning in persons with chronic alcohol abuse. Cognitive impairment has been related to posttreatment employment success (Donovan et al., 1984; Moriyama et al., 2002); we found a correlation with an indicator of past employment success (on the ASI). Consistency in this finding may reflect the greater cognitive demands of work environments. However, we found little other evidence that measured cognitive impairment related to the effect of alcohol in domains of daily living. Studies attempting to link neuropsychological test performance with clinically relevant outcomes in this population have also been largely unsuccessful (Jin et al., 1998; Macciocchi et al., 1989). Executive dysfunction, in particular, is likely to be the result of complex processes, because significant associations with indices of drinking chronicity are rarely found [see Adams and Grant (1986) or Beatty et al. (2000) for discussions of these factors]. The development of models that propose more complex mediated relationships between consumption history and cognitive functioning is merited. The use of more ecologically valid neuropsychological instruments to measure executive dysfunction may also facilitate this line of investigation (Moriyama et al., 2002). It may be that other psychological constructs, such as depression or increased frustration, moderate the effect of cognitive impairment on real-world functioning.

Because alcoholism treatment approaches rely heavily on psychosocial components, their efficacy depends, in part, on the cognitive and psychological functioning of the patient. Deficits in executive functioning would thus seem to be a significant impediment to positive treatment outcomes. Indeed, executive function has been associated with employment outcomes in patients who were not in treatment (Moriyama et al., 2002). Thus, our findings of executive dysfunction early in abstinence suggest that clinicians might consider tailoring treatment approaches for recently detoxified alcoholics in ways that demand less of these functions—particularly in the domains of learning, reasoning, and cognitive flexibility—as has been suggested for cognitive dysfunction in general (McCrady and Smith, 1986). Clearly, these data cannot be interpreted directly to suggest particular alterations in treatment strategy, but they do imply that executive dysfunction early in abstinence may be a factor to consider. Our results suggest that alcohol-dependent individuals are aware of their impairment; it is likely that such individuals will accept a rationale for treatment approaches that help them compensate for their deficits.

Possible Influences of IQ

The relation of estimated premorbid ability to the cognitive impairments found is intriguing. Group differences in reasoning ability seemed largely due to estimated IQ for both verbal and nonverbal domains. It is unclear whether this stronger association for these functions in our data is associated with the use of subtests from the WAIS-III that have a high correlation with g (the general intelligence factor). Both of these subtests are among the four tests used in Wechsler’s brief battery to estimate IQ (Wechsler Abbreviated Scale of Intelligence; The Psychological Corporation © 1999). Thus it would seem that these scores represent intellectual potential, a purportedly stable aspect of cognition. A recent study by Donders et al. (2001) found that Matrix Reasoning did not seem to be sensitive to traumatic brain injury. However, our findings in toto suggest that the picture may be more complex.

Although, in general, drinking history was not associated with neuropsychological variables, nonverbal reasoning was associated with recent patterns of use. Our data showed that longer abstinence was associated with higher Matrix Reasoning scores. Other studies have found improvement in scores on different measures of nonverbal reasoning within weeks of abstinence (Cocchi and Chiavarini, 1995; Kish et al., 1980). This raises the question: how stable is IQ in chronic alcohol abusers? Because reasoning seems to be consistently affected by chronic drinking (Cocchi and Chiavarini, 1995; Kish et al., 1980), then measures of IQ during early treatment may not reflect true premorbid ability. Kish et al. (1980) found improvements in performance on three WAIS subtests with abstinence. WAIS-R performance subtests have also been affected early in detoxification (Blennerhassett et al., 1993). It is possible that although raw intellectual ability may remain fairly constant, demonstration of that ability on testing may be affected by generalized impairment or reduced cognitive efficiency from chronic alcohol abuse.

CONCLUSION

Our findings provide further documentation for an impairment of executive functions in persons who chronically abuse alcohol. Both psychomotor components and generalized inefficiencies may contribute to the deficits found. Premorbid ability and recent alcohol intake each seem to have a role in executive functioning early in abstinence, but their relations are not simple or universal. Although the size and demographic makeup of our sample may have affected the findings, it is clear that multiple factors need to be evaluated to map out the relationship between alcohol dependence and executive functioning. There may be different paths to cognitive impairment that involve family history, psychosocial environment, drink history, and individual differences in resilience. The external effect of executive functions on the daily lives of alcohol-dependent persons is likely to require similar complex modeling. The sometimes-subtle effects of executive dysfunction may be apparent only in large-scale longitudinal studies, as has been the case in mild traumatic brain injury (Kolitz et al., 2003). We believe that executive dysfunction early in abstinence is a challenge to treatment, and we encourage fur-
their research on the factors that may moderate its development, effect, and recovery.

ACKNOWLEDGMENTS

Special thanks to Nan Birchall, Pat Murphy, Bert Loro, Heather Klein, Ashlee Carter, and Linda Leimone for their invaluable assistance in conducting this study.

REFERENCES


Copyright © Research Society on Alcoholism. Unauthorized reproduction of this article is prohibited.


