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Cerebral damage associated with alcoholism

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Abstract

The neuroanatomical consequences of chronic ethanol ingestion were investigated using neuropsychological tests selectively sensitive to damage in circumscribed brain areas. The performance of a group of male alcoholics (N=15) with a self-reported drinking history less than ten years, and a group of male alcoholics (N=15) with a self-reported drinking history of ten or more years was compared to a group of male nonalcoholic controls (N=15). A multiple covariate analysis was done prior to analyzing group differences to determine what influence three covariates (age, education and socioeconomic status) had on the group performances. Results from this analysis indicated that some of the tests were significantly influenced by age or education but no significant Group x Covariate interactions were observed. In agreement with numerous neuropathological findings, the late stage alcoholics were found to be principally deteriorated on the tests of frontal lobe functioning, followed by lesser impairment on the tests of temporal lobe functioning, with no evidence of impairment on the tests of parietal lobe functioning. No significant group differences were found between the early stage alcoholics and the controls. Small but negative correlations were found between both the

duration of alcoholism and the average amount of alcohol consumed with each of the neuropsychological measures given in this study.)

Cerebral Damage Associated with Alcoholism

The chronic consumption of alcohol is a major public health problem affecting over nine million Americans (Chafetz, 1975). Alcoholism is estimated to cost the economy approximately 15 billion dollars yearly in lost work time, health and welfare services, property damage, medical expenses, and other incalculable expenses such as broken homes, wasted lives, loss to society, and human misery. Alcoholism also represents a serious medical problem and is involved in such medical complications as pellagra, nutritional amblyopia, peripheral polyneuropathies, fatty liver, cirrhosis of the liver, and damage to cardiac and skeletal muscles (Goodman & Gillman, 1975). Finally, there is a growing body of evidence suggesting that alcohol may directly disrupt the integrity of the central nervous system. The nature and extent of alcoholic brain damage has been investigated using two different types of methodology: a) by direct examinations of the pathophysiological results of alcoholism and b) through an analysis of the psychological deficits that appear to be common among alcoholics. Each of these methods leads to certain conclusions about the brain damage associated with alcoholism, and the outcomes of these two differing approaches

appear, at times, to be at variance with one another. However, in order to fully understand the nature of the evidenced brain damage, it is necessary to examine and attempt to integrate both methodological approaches.

Physical Evidence

One of the earliest investigations into the physical effects of alcohol on the central nervous system was conducted by Courville in 1955. Based on pathological evidence, he concluded that chronic alcoholism "is the most common cause of cerebral atrophy in the fifth and sixth decades of life" (p. 46). He reported cortical atrophy that was widespread, but more marked in the convolutions of the upper part of the dorsalateral surface of the frontal lobes, and accompanied by ventricular enlargement (suggesting subcortical atrophy) and thickening and opacity of the overlying leptomeninges. Courville also reported finding microscopic cell loss, proliferation of neuroglia elements, vascular changes, and swelling, fragmentation and disintegration of myelin. Courville conceptualized the cortical lesions as a direct consequence of ethanol toxicity rather than dietary deficiencies associated with chronic alcohol consumption. Similar neuropathological pictures of cortical atrophy in alcoholis, particularly involving the frontal lobes, have been reported by several other investigators (Alex-

ander, 1941; Lynch, 1960; Mancall, 1961; Stevenson, 1940; Umiker, 1949). Although these findings implicate brain damage as a result of chronic ethanol abuse, they cannot be construed as conclusive because they failed to parcel out such contributory factors as aging, dietary irregularities, length of drinking history, and type and amount of alcohol consumed.

In attempting to establish the frequency and extent of cerebral atrophy in earlier stages of alcoholism, several investigators have employed a neuroradiological technique known as the pneumoencephalogram. One of the earliest such studies was done by Tumarkin, Wilson, and Snyder (1955). Seven enlisted soldiers with an average drinking history of 11 years and a mean age of 32 were examined. Complete neurological physicals were taken to rule out all possible sources of brain damage other than alcoholism. Pneumoencephalographic findings revealed a diffuse bilateral cortical atrophy in the parietal region in six of the seven subjects, and frontal atrophy in four of these six (a mildly generalized atrophy was found in the seventh subject). Ventricular enlargement was found in three of the cases. In six of the subjects, abnormal slow-wave electroencephalographic patterns were observed in the frontal regions and in three of these subjects the same abnormal patterns were apparent in

the parieto-occipital areas. These results suggest that brain damage in alcoholics can occur in individuals whose age and neurological disposition would not seem to contribute to the abnormalities present. Interestingly, a positive correlation was found between cortical atrophy and impairment on the Digit Symbol and Digit Span Subtest of the Wechsler-Bellevue Intelligence Scale, suggesting that the brain damage was associated with psychological deficits. The authors concluded from their findings that brain damage in alcoholics may account for some of the difficulties encountered in the psychotherapeutic treatment of alcoholics.

Brewer and Perrett (1971) studied 33 male alcoholics and other heavy drinkers whose mean age was 50 and who drank approximately 3 liters of beer per day or its equivalent. Evidence of brain damage was sought using pneumoencephalograms, electroencephalograms, and psychometric testing that included the Benton Visual Retention Test and the Wechsler Adult Intelligence Scale. Results of the pneumoencephalographic examinations indicated that cortical atrophy was present in 30 of the 33 subjects and that ventricular enlargement was present in 24. In 70% of all the cases with atrophy, the atrophy was rated as significant and in 58%, the psychometric tests supported the diagnosis of brain damage. In the

majority of the cases with atrophy (19 out of 30) both the frontal and parietal lobes were involved. Out of the remaining 11, nine showed only frontal atrophy and two only parietal. From these findings, the authors concluded that diffuse cortical atrophy rather than localized atrophy is more characteristic of the alcoholic, however, in cases when the atrophy is localized, the frontal lobes are typically more involved than the parietal lobes. Electroencephalographic data were relatively unhelpful in identifying cortical disruption. The authors concluded that brain damage in alcoholics was probably more common than previously realized and that it probably contributes to the frequently observed psychological disabilities in these patients. Other pneumoencephalographic studies with alcoholics have reported similar findings of cortical and subcortical atrophy (Aguirre, 1970; Haug, 1968; Iivanainen, 1975; Skillicorn, 1955). Interestingly, Haug's (1968) pneumoencephalographic study of 60 alcoholics disclosed a positive correlation between cerebral atrophy and length of drinking history.

More recently, a few investigators have employed computerized tomography (CT) scans to visualize the cerebral structures (ventricles and cortical sulci) in alcoholics. Fox, Ramsey, Huckman, and Proske (1976)

compared the CT scans of 60 controls to a group of 12 alcoholics with various neurological symptoms such as withdrawal seizures, peripheral neuropathy, hepatic encephalopathy, headaches, tremulousness and depressed state of consciousness. Results of the investigation indicated that the alcoholics (eight out of 12) had significantly enlarged ventricles when compared to the controls, while only two of the 12 alcoholics were found to have enlarged sulci indicative of cerebral atrophy. Unfortunately, the results of this study are complicated by several factors that may reduce the generalizability of the findings to chronic alcoholics in general, including the use of alcoholics with neurological conditions that may have contributed to the observed outcomes, the failure to adequately control for the duration of alcoholism in the subjects and the effects of liver disease. Carlen, Wortzman, Holgate, Wilkinson, and Rankin (1978) examined the CT scans of eight alcoholics with a mean drinking history of 19 years. None of the subjects used in the study had a history of liver disease (including ascites, jaundice, and hepatic encephalopathy), severe head trauma requiring hospitalization, obvious malnutrition, or any other generalized disease. Contrary to the results of the Fox et al. study, alcoholics in this study were found to have a greater degree of gener-

alized cerebral atrophy than ventricular atrophy. Interestingly, repeated CT scans (a minimum of 26.5 weeks between the first and second scan) disclosed a decrease in cerebral atrophy in four of the eight alcoholics that was associated with abstinence and clinical improvement. From these findings, the authors concluded that chronic alcohol abuse (all subjects had a heavy drinking history greater than 10 years) can lead to neuronal damage via some unknown mechanism. However, upon the removal of this neurotoxin, regrowth of the damaged neurons, glia elements, and vascular tissues can occur over the subsequent weeks and months that would account for the decreased atrophy and concomitant psychological improvement. Although the results of this study are highly suggestive, it is difficult to determine what changes in the cortical structures may be accounting for the apparent regeneration or decreased atrophy, and whether it is of any functional significance. Increase in tissue mass does not in any reliable way provide evidence of neuronal plasticity in the central nervous system and may merely represent glial and collagenous scaring, dysfunctional axonal sprouting and chromatolysis.

A final approach to studying the physical effects of chronic ethanol consumption on the integrity of the brain comes from the work of a team of investigators who

measured regional cerebral blood flow (RCBF) in a group of 60 alcoholics with a mean age of 45.2 years (Berglund & Ingvar, 1976). The purpose of their study was to determine if alcoholics displayed a reduction in mean hemispheric blood flow and whether such a reduction would be associated with duration of alcoholism. The results of the study indicated a significant reduction in cerebral blood flow in the anterior temporal and lower frontal regions in the older alcoholic group (above 45 years), while the parietal region appeared to be the least affected area. Also, the results disclosed a successive fall in the mean hemispheric RCBF (based on the average of eight regional measurements) with advancing age. The authors attributed this reduction to the duration of alcoholism rather than to increasing age because prior studies on cerebral blood flow have not reported a reduction with advancing age; however, the RCBF technique may not be comparable to other types of measures of cerebral blood flow. Nevertheless, Berglund and Ingvar's reported results are highly suggestive of pathological changes in cerebral functioning associated with chronic ethanol abuse.

The physical findings of the neuropathological, neuroradiological, computerized tomography scans, and regional blood flow studies clearly provide evidence of cortical and subcortical atrophy (as indicated by ventricular en-

largement) associated with alcoholism. While these studies indicate that the frontal lobes are damaged more frequently and severely than the rest of the brain, there is clear evidence of widespread cortical disruption as well (Brewer & Perett,1971; Carlen et al., 1978; Courville, 1955; Tumarkin et al., 1955).

Psychological Evidence

Researchers have employed the results of neuropsychological tests with alcoholics to speculate on the neuroanatomical substrates involved in alcoholic brain damage. Unfortunately, the interpretation of much of the neuropsychological evidence collected is debatable because many of the neuropsychological tests that have been used to assess the extent and location of brain damage in alcoholics do not have generally agreed upon implications for brain damage. Tarter (1975, 1976) has identified three major neuropsychological hypotheses of alcoholic brain damage: (a) diffuse or generalized damage (Brosin, 1967; Chafetz, 1967); (b) impairment that is lateralized to the right cerebral hemisphere (Jones & Parsons, 1971; Parsons, Tarter & Edelberg, 1972); and (c) impairment of the frontal-limbic-diencephalic system (Tarter, 1972, 1973, 1975, 1976).

Diffuse hypothesis. Sandok (1975) describes the psychological effects of diffuse brain damage, regardless

of the cause and severity, as disturbances in orientation, memory, intellect, judgment, and affect. Lezak (1976) characterizes the effects as "...non-specific slowing; diminished ability for complex mental activities, such as reasoning, planning and abstract conceptualizations; general impairment of memory, concentration, and attention; and generalized mental deterioration" (p. 150).

Relevant neuropsychological outcomes for the diffuse hypothesis has been sought from the results of intelligence testing and memory impairment in alcoholics. However, generalized intellectual deterioration associated with diffuse brain damage in alcoholics has not been experimentally documented. Using the Wechsler-Bellevue Intelligence Scale, Fitzhugh, Fitzhugh and Reitan (1965) studied a group of 35 chronic alcoholics with a mean age of 40.5 years. The alcoholics' mean IQ of 109 was very similar to that of a matched group of normal controls (IQ=113) and somewhat higher than a group of brain damaged subjects (IQ=99). Tarter, Buonpane and Wynant (1975) examined a group of 40 alcoholics on a battery of cognitive tests thought to measure factorially pure aspects of intelligence and found no evidence of impaired cognition when compared to a group of 20 psychiatric patients. Numerous other studies that have tested alcoholics with either the Wechsler-Bellevue Intelligence Scale or the Wechsler Adult

Intelligence Scale have also reported intact intellectual functioning (Bauer & Johnson, 1957; Fitzhugh, Fitzhugh & Reitan, 1960; Goldstein, Neuringer & Klappersack, 1970; Murphy, 1953; Peters, 1956; Smith, Burt & Chapman, 1973; Wechsler, 1941, 1958). In fact, Kaldegg (1956) reported that 50% of the alcoholics examined in his study were found to have an IQ greater than 120. Studies employing the Shipley-Hartford Test, a vocabulary and verbal abstracting test that provides an estimate of the WAIS Full Scale IQ, have also found the IQ of alcoholics to be well within the average range (Jones, 1971; Parsons, 1974; Smith, Johnson & Burdick, 1971; Smith & Layden, 1972; Tarter, 1973; Tarter & Jones, 1971). The performance of alcoholics, however, on the Raven's Progressive Matrices, a nonverbal measure of intelligence, has been found to be below what would be expected in the absence of intellectual impairment (Jones, 1971; Jones & Parsons, 1972; Page & Schaub, 1977; Parsons, 1974). Unfortunately, research suggests that this test does not adequately serve as a measure of general intellectual ability (Lezak, 1976).

The failure to find a generalized intellectual deterioration in alcoholics, as measured by intelligence testing, has been interpreted as evidence that alcoholics do not suffer from diffuse brain atrophy. Neuropsychologists,

however, do not all agree that the use of intelligence testing to establish the presence or absence of generalized brain deterioration is warranted. In many cases of brain damage, including those of considerable extent, lowered general intelligence scores have not been observed (Maher, 1963). Furthermore, the effect of a lesion on general intelligence is more severe the greater its rate of growth (Golden, 1978). In cases of slowly advancing atrophy, as would be speculated in alcoholism, the cognitive disruptions are likely to be smaller in magnitude than in more rapidly advancing damage.

The results of testing alcoholics on memory capacities have also been far from conclusive. Several investigators (Berglund & Sonesson, 1976; Brewer & Perrett, 1971; Claeson & Carlsson, 1970; Page & Linden, 1974) have found alcoholics to be impaired on the Benton Visual Retention Test (a measure of design memory). However, other investigators (Berglund & Sonesson, 1976; Donovan, Queisser & O'Leary, 1976; May, Urquhart & Watts, 1970) have found no evidence of impaired retention on the Graham-Kendall Memory for Designs Test, a similar measure of design memory. Verbal memorizing abilities in alcoholics, as measured by paired associates tasks, have not been found to be impaired (Berglund & Sonesson, 1976; Claeson & Carlsson, 1970; Jonsson, Cronholm & Izikowitz, 1962). The

results of testing alcoholics for recall on verbal serial learning tasks, on the other hand, has revealed a deficit in the number of words recalled correctly (Allen, Faillace & Reynolds, 1971; Weingartner & Faillace, 1971; Weigartner, Faillace & Markley, 1971). Observed memory deficits in alcoholics may be consistent with expected memory impairment in response to diffuse brain damage. Such memory deficits, however, may also be consistent with temporal lobe dysfunctioning, for which physical evidence was found in Berglund and Ingvar's (1976) study on regional cerebral blood flow in alcoholics.

Collectively, the neuropsychological evidence does not support or refute a theory of diffuse brain damage in alcoholics. Intelligence testing does not appear to be an appropriate way to assess the theory at all, and equivocal memory impairment may be a result of localized temporal as well as diffuse cerebral damage. However, there is much physical evidence of widespread cortical disruption consistent with a diffuse hypothesis of alcoholic brain damage (Carlen et al., 1978; Courville, 1955; Brewer & Perrett, 1971; Tumarkin et al., 1955). The cause of this apparent discrepancy between physical and psychological evidence may be the slowly advancing and chronic nature of the cortical disruption associated with alcoholism, as was pointed out with regard to the lack

of impairment in alcoholics on intelligence testing. Interestingly, on tests that are known to be very sensitive to any type of brain damage (e.g., Digit Symbol on the WAIS, the Category Test of the Halstead-Reitan Battery), alcoholics have been consistently found to be impaired, providing neuropsychological as well as neurophysical support for diffuse brain damage in alcoholics.

Right lateralized hypothesis. The second neuropsychological hypothesis of brain damage in alcoholics postulates the damage as lateralized with greater impairment in the right hemisphere. While there does not appear to be any pathophysiological evidence for this theory, neuropsychological data has been interpreted to provide some support. For example, many neuropsychologists believe that large differences (greater than 15 points) between verbal IQ and performance IQ can be used to infer the presence and hemispheric location of brain damage. Alcoholics exhibit a small but consistent decrement in performance IQ relative to verbal IQ (less than 10 points) which is seen by some investigators as indicative of right hemisphere damage (Kaldegg, 1956; Long & McLachlan, 1974; Teicher & Singer, 1946; Wechsler, 1941). Verbal-performance IQ differences, however, have only been established as diagnostic aids in cases with acute lateralized damage, not in cases with chronic impairment, as characteristically

seen in alcoholic deterioration (Fitzhugh, Fitzhugh & Reitan, 1962; Reitan & Davison, 1974). Furthermore, nonverbal information may have a lower threshold of disruption than the well codified and greatly rehearsed linguistic symbols (Chandler & Parsons, 1977), which could account for a reduced performance IQ although damage might be diffuse.

A second source of data which has been used to support the right hemisphere theory of alcoholic deterioration comes from the results of alcoholics' performance on the WAIS subtests. Fitzhugh et al. (1965) reported that alcoholics performed significantly below a control group on the Block Design Subtest of the WAIS (a visuospatial test believed to be sensitive to right hemisphere functioning). Similar findings were reported by Goldstein, Neuringer and Klappersack (1970). In a review of 12 studies occurring between 1946 and 1971, however, Kleinknecht and Goldstein (1972) found Block Design performance in alcoholics to be impaired in only 25% of the studies employing this measure. Further, although extremely poor performance on the Block Design is associated with right hemisphere damage, it can also be depressed by any kind of brain damage (Lezak, 1976). Several studies (Hirschenfang, Silber & Benton, 1967, 1968; Silber, Hirschenfang & Benton, 1968) demonstrate a lack of impairment by alcoholics on

the Bender-Gestalt (a visuographic task) which is also believed to be sensitive to right hemisphere functioning. The absence of impaired visuospatial and visuographic functioning argues against a theory of lateralized right cerebral damage in alcoholics.

In the Kleinknecht and Goldstein review, the only WAIS subtest scores that were consistently depressed in alcoholics were the Digit Symbol (depressed in 66% of the studies employing this measure) and the Object Assembly Subtest (depressed in 50% of the studies employing this measure). Performance on the Digit Symbol Subtest, however, is consistently sensitive to the effects of any type of brain damage and may even be depressed when the damage is minimal (Lezak, 1976). Although the Object Assembly Subtest appears to be sensitive to right hemisphere damage, performance of this subtest is, to a large extent, determined by the subject's speed in responding. Poor performance by alcoholics on the Object Assembly may in part be explained by alcoholic peripheral neuropathy (characterized by motor weakness, sensory loss and pain in the extremities) which may adversely affect the alcoholic's response speed. In addition, this problem may account for the alcoholics' performance decrements observed on the Block Design and Digit Symbol Subtests, which also require intact perceptual-motor skills. Klein-

knecht and Goldstein noted that none of the reviewed studies attempted to control for this possibility. Therefore, the results of the Wechsler Subtests with alcoholics are far from conclusive in establishing the presence of right hemisphere damage.

The hypothesis of right lateralized brain damage in alcoholics has also been investigated by employing a dichotic listening technique for verbal material. Goodglass and Peck (1972) observed that alcoholics recalled items presented to the left ear more poorly than a group of normal controls. Tarter (1975, 1976) concluded that the results of this study indicated the possibility of increased vulnerability in the right hemisphere of alcoholics. Employing a similar technique, however, Chandler, Vega and Parsons (1973) observed right-ear superiority in recall for all the groups they tested (alcoholics without a possible history of brain injury, alcoholics with a possible history of brain injury and matched groups of normal controls). More errors of confabulation and repetition occurred in the group of alcoholics suspected of having brain damage. Interestingly, the authors interpreted this finding as a possible indication of left frontal damage.

In summary, a theory of right lateralized brain damage in alcoholics is not supported by physical or neuropsychological investigations. Poor test interpreta-

tions, failures in replications, and failures to control for such contributory factors as peripheral neuropathy make the theory unlikely. Finally, this theory does not offer a neurological basis for the possible right hemisphere selectivity of alcohol.

Frontal-limbic-diencephalic hypothesis. The third neuropsychological hypothesis of alcoholic brain damage asserts that alcoholics suffer from damage to the frontal-limbic-diencephalic system. Although this hypothesis implies the involvement of a variety of subcortical structures (the hippocampus, amygdala, septum, mammillary body, thalamus, and hypothalamus), it has principally been investigated by observing frontal lobe functioning in alcoholics. As previously mentioned, neuropathological and neuroradiological research frequently reports damage to the frontal lobes that is consistent with this hypothesis. In particular, the neuropathological work of Courville (1955) indicated that atrophy of the dorsalateral aspects of the frontal lobes is a primary characteristic of chronic alcoholism. The broadest collection of neuropsychological evidence for this theory has been compiled by Tarter (1975, 1976). Impairments of alcoholics on tests of abstraction are particularly important to the conclusions of Tarter and others regarding frontal damage due to alcoholism. Neuropsychologically, lesions in the dorsolateral portion

of the frontal cortex are thought to result in greater impairment of abstracting ability than damage to the posterior portion of the brain (Milner, 1963). The tests of abstraction used in studying alcoholics have been the Category Test from the Halstead Reitan Neuropsychological Battery, the Wisconsin Card Sorting Test (verbal and non-verbal forms), the Shipley-Hartford Test, and a modified version of the Levine Hypothesis Test.

Performance on the Category Test is thought to reflect competence in non-verbal abstraction, or the ability to process new information to solve unique problems when compared to prior information. Fitzhugh, Fitzhugh and Reitan (1960, 1965) reported that a group of 35 hospitalized alcoholics demonstrated significant impairment on this test when compared to a group of controls, but did not differ significantly form a group of hospitalized brain damaged subjects, which included a vast array of brain injuries ranging from convulsive disorder due to tertiary syphilis to cerebroarteriosclerosis. Similarly, Jones and Parsons (1971) found that a group of 40 alcoholics were significantly impaired on the Category Test when compared to a group of controls but did not differ significantly from a group of brain damaged patients (including equal numbers of left, right and diffusely damaged). They also found greater chronicity

of alcoholism to be associated with poorer test performance. Other investigators have also found alcoholics to be impaired on the Category Test (Jones & Parsons, 1972; Long & McLachlan, 1974; Shaw & O'Leary, 1977).

With respect to anatomical locality, the interpretation of these findings is unclear. Although the Category Test has been suggested as an accurate measure of frontal lobe functioning, Halstead (1947) has pointed out that in cases of chronic lesions, as in alcoholics, the test results are far less clear. Chapman and Woff (1959), upon re-evaluation of Halstead's findings, reported that the destruction of an equivalent mass of cerebral tissue in nonfrontal areas resulted in as great an impairment in abstracting ability (as measured by the Category Test) as in subjects with frontal lobe lesions. Since larger amounts of cerebral tissue had been removed in the frontal than in the nonfrontal subjects in Halstead's studies, Chapman and Woff suggested that the greater loss of abstract functions in the frontal subjects initially observed by Halstead was possibly an artifact. Finally, current information (Lezak, 1976; Reitan, Note 1) suggests that the Category Test is highly sensitive to the effects of brain damage per se and thereby serves as a good indicator of cerebral intactness rather than a a measure of frontal lobe functioning. The established poor performance on

on this test for alcoholics may therefore be a clear indicator of cerebral deficits in general, but not necessarily frontal lobe dysfunctioning in particular.

The Wisconsin Card Sorting Test (WCST), another test of abstraction frequently given to alcoholics, requires a subject to sort a group of stimulus cards that have symbols differing in shape, color, and number according to a principle that must be deduced from the examiner's "right" or "wrong" response for a given trial. After ten cards have been sorted correctly, the examiner shifts the principle, and again the patient must deduce this change from the examiner's response. Subjects may demonstrate difficulties in sorting according to category (concept acquisition), in shifting when the category changes (perseveration), and in maintaining a cognitive set (losing track of the present category after completing several correct shifts). Tarter and Parsons (1971) administered the WCST to a group of alcoholics and found them to be inferior on this task when compared to a group of normal controls. The alcoholics were impaired at maintaining a cognitive set, and this propensity was associated with chronicity of alcoholism. Tarter (1973) administered the WCST to a group of short-term alcoholics (admitted alcoholism of less than 10 years) and a group of long-term alcoholics (admitted alcoholism of greater

than 10 years). When compared to a group of normal controls, the long-term alcoholics were impaired at shifting and maintaining a cognitive set. The short-term alcoholics, however, were only deficient at maintaining a cognitive set. Neither group of alcoholics demonstrated impaired concept aquisition. Tarter concluded from his study that the WCST testing pattern in alcoholics is consistent with neuropathological impairment of the frontal regions of the brain. Finally, a verbal form of the WCST, in which printed words were substituted for geometric symbols, was administered to a group of alcoholics to determine their level of verbal abstraction (Jones, Note 2). The performance of alcoholics was not impaired on this task.

Unfortunately, the difficulties on the non-verbal form of the WCST observed in Tarter's alcoholics have not been clearly established as reliable indicators of frontal brain damage. Although Milner (1964) reported that subjects with superior frontal lesions made more perseverative errors than the control subjects, Drewe (1974) reported that difficulties with shifting were observed as a common occurrence in brain damaged individuals regardless of the side or site of the lesion. Consistent with Milner's findings (1963, 1964), Drewe also reported that patients with frontal lobe damage most

often demonstrated impaired concept aquisition on the WCST. Concept aquisition has not, however been judged as impaired in alcoholics (Chapman & Wolff, 1959; Tarter, 1973) in contradiction to the hypothesized frontal lobe damage of alcoholism.

The Shipley-Hartford, a portion of which assesses verbal abstracting ability, has also been given to groups of alcoholics. Basically, this verbal test is designed to measure two classes of intellectual functions, one of which is insensitive to the effects of brain damage (vocabulary), while the other (verbal abstraction) is vulnerable to brain damage. Ornstein (1977) found the performance of a group of alcoholics on this test to be far below what might be expected in the absence of brain damage. He concluded from this finding that alcoholics suffer mild organic brain impairment. Similar results were reported by Page and Schaub (1977). Although this test has been used as a screening technique for organicity, other research suggests that it fails to discriminate between brain damaged and normal subjects (Parker, 1957; Savage, 1970). Jones (1971) administered this test to a group of 30 chronic alcoholics and hospitalized normal controls. The results of this study indicated that alcoholics did not differ significantly from the normal controls on this task. Similar results were observed in

a study by Tarter and Jones (1971). The inconsistent results and limited usefulness of this procedure in identifying brain damage in general minimize the interpretive value it has with respect to alcoholism.

In general, tests of abstraction require intact memory ability in that a subject must recall, from one trial to the next, the set of appropriate responses that is necessary for solving the problem at hand. To obtain a pure measure of abstraction, Oscar-Berman (1973) tested a group of alcoholic patients, including Korsakoff patients, on Eimas' (1970) modified version of the Levine-Hypothesis Test in which the memory component has been parcelled out. Basically, this procedure requires a subject to formulate a solution to a set of two-choice visual discrimination problems where a series of stimulus cards are presented (each having 2 backgrounds of different colors, letters, sizes of letters and different sides of the card) and the subject determines which stimulus is the correct solution on an outcome trial from four previously presented cards. To parcel out the effects of memory, arrow markers were placed over a correct solution and left in view of the subject (in the standardized version each card is turned over after each response). Oscar-Berman found the alcoholics to be more impaired on this task than a group of normal and

brain damaged controls. Korsakoff patients, however, demonstrated the greatest impairment, which led her to conclude that alcoholics were approaching a Korsakoff condition, a syndrome associated with damage to the medial aspects of the temporal lobes, interference with limbic system pathways, and damage to the mammillary bodies (Berkow & Talbott, 1977). Klisz and Parsons (1977) administered the same test to a group of alcoholics and also found them to be deficient on this abstraction task. In the same study, they also investigated abstraction as it related to duration of alcoholism, but found no difference between the performance of alcoholics with different durations of alcoholism. The results of the Oscar-Berman and Klisz and Parsons studies indicate that alcoholics demonstrate impaired abstract functioning on the Levine Hypothesis Test. Although the authors do not speculate on the locus of brain damage responsible for this deficit, it is consistent with the frontal lobe model of abstraction in alcoholism.

At the core of the difficulty in interpreting the results of tests of abstraction with alcoholics is the fact that the very global nature of abstraction tasks makes it unlikely that only frontal lobes are involved. Lezak (1976) points out that higher order cognitive functions, such as abstraction, have not been related

to specific neuroanatomical systems. In fact, patients with moderate to severe or diffuse damage tend to do poorly on all tests of abstraction regardless of the mode of presentation or channel of response. To illustrate this point, research has suggested that subcortical atrophy, as indicated by ventricular enlargement, may also be responsible for impaired abstract functioning. Matthews and Booker (1972) reported that performance on the Category Test was significantly poorer for patients with enlarged ventricles when compared to patients with smaller ventricles. This finding was observed in subjects with unilateral as well as bilateral ventricular enlargement. The authors concluded that impairment of higher order cognitive functions was related more to the total number of inadequately functioning neurons rather than to the localization of these neurons (be they focally isolated or distributed diffusely throughout the hemispheres). Similar results have been reported by Willanger, Thygesen and Nielsen (1968).

Overall, investigative work on abstract functioning in alcoholics has not proven to be a heuristic method in determining the validity of the frontal-limbic-diencephalic hypothesis of alcoholism. Although there is reason to believe that alcoholics demonstrate impaired abstract functioning (e.g., Jones & Parsons, 1971, 1972; Klisz &

Parsons, 1977; Oscar-Berman, 1973), there is little reason to believe that damage to the frontal area is solely responsible for this deficit. This point becomes even more dramatic when one attempts to appreciate the neuroanatomical substrates of abstraction. Testing alcoholics on cognitive functions that are not clearly related to specific neuroanatomical structures, as is the case with abstraction, tells the researcher nothing about the locus of brain damage; at most it presents a sensitive measure for the possibility of brain damage. Perhaps a more fruitful approach to investigating neuropsychological hypotheses of alcoholic brain damage would be to assess the performance of alcoholics on psychological tests that appear to be highly sensitive to damage in selective brain regions. but not to brain damage in general. In this way, the integrity of cerebral regions could be outlined and compared against each other to give a more complete neuropsychological picture of the alcoholic brain.

Purpose

The purpose of the present study was to further investigate the three neuropsychological hypotheses of alcoholic brain damage using measures selectively sensitive to damage in circumscribed brain areas. A comparative analysis of the performance of alcoholics on six

neuropsychological measures was done in an effort to outline the pattern of cerebral deficits with respect to each theory. Each of the six measures is believed sensitive to only one homologous brain region (right or left frontal, temporal, and parietal). According to each theory, one would expect to find different testing patterns on these six measures for alcoholics when compared to controls. For example, the diffuse hypothesis would predict the approximately equal impairment on all six measures. The right lateralized hypothesis would predict deficits in performance on each of the right hemisphere tests while the measures for left hemisphere functioning would appear unimpaired relative to the controls. Finally, the frontal-limbic-diencephalic hypothesis would predict only the appearance of frontal test deficits.

As previously mentioned, physical evidence clearly demonstrates atrophied frontal lobes as common in alcoholics (Brewer & Perrett, 1971; Courville, 1955; Lynch, 1960; Mancall, 1961; Warner, 1934). However, there is also considerable reason to believe that the atrophy is also widespread throughout the hemispheres (Brewer & Perrett, 1971; Courville, 1955). As such, these findings indicate that frontal lobe atrophy is the principle area of involvement in the alcoholic brain,

and that the atrophy spreads to adjacent cortical regions equally throughout each hemisphere, suggesting a fourth model of alcoholic brain damage that can be conceptualized as the diffuse frontal-limbic-diencephalic hypothesis. Therefore, it was hypothesized that the performance of alcoholics on the six measures relative to the controls will be deteriorated principally in the tests of frontal lobe functioning, followed by lesser impairment in the temporal lobes, and with little, if any, impairment of the parietal lobes.

Each measure was also examined with respect to chronicity of alcoholism and average daily consumption of alcohol. Prior research (Fitzhugh et al., 1960, 1965; Tarter, 1973; Tarter & Jones, 1971) suggests that these factors may play an important role in the evidenced cerebral damage, with greater chronicity and consumption producing greater damage. As such, it was expected that the performance of late stage alcoholics (drinking history greater than 10 years) would be worse than the performance of early stage alcoholics (drinking history less than 10 years) and significantly worse than the performance of nonalcoholics. Finally, the effects of 3 independent variables (age, education, and socioeconomic status) on each of the dependent measures was examined. Evidence suggest that the effects of the

three independent variables will vary across dependent measures and experimental conditions (Parsons & Prigatano; 1978). For example, age may correlate with the performance of the controls on a given measure but not with the performance of the two alcoholic groups.

Method

Subjects

Two groups of 15 right handed male alcoholics served as experimental subjects. The first group (early stage alcoholics) was comprised of seven inpatients from the San Joaquin County Residential Recovery Center and eight inpatients from the San Joaquin County Recovery House. Subjects in this group had a self-reported drinking history of less than 10 years (M=5.6; SD=2.4; Range=1-9), a mean age of 30.3 (SD=11.7; Range=17-61) years, and a mean education of 12 (SD=2.5; Range=8-16) years. The second group (late stage alcoholics) was comprised of five inpatients from the San Joaquin County Residential Recovery Center and ten inpatients from the San Joaquin County Recovery House. Subjects in this group had a self-reported drinking history of 10 or more years (M=20.9; SD=7.9; Range =10-34), a mean age of 44.6 (SD=8.4; Range= 35-60) years, and a mean education of 11 (SD= 1.6; Range= 8-14) years. Fifteen nonalcoholic right handed males with a mean age of 32.6 (SD=13.4; Range=18-61) years,

and a mean education of 12.7 (SD=3.0; Range=7-18) years, all of whom were nonacademic personnel at the University of the Pacific, served as control subjects in the experiment.

All potential subjects were interviewed for a determination of right handedness and for information about prior disorders other than alcoholism that might cause impaired neuropsychological functioning. Subjects with a positive self-reported history of cerebral infections (meningitis, encephalitis, and cerebral syphilis), hospitalized head injuries (concussion, contusion, lacerations, and hematomas), circulatory disorders (cerebral arteriosclerosis, embolism, hemorrhages, aneurysms, hypertension, chronic cardiovascular disease, and cardiac failure), epilepsy or other convulsive disorders, metabolic disturbances (thyroid, pituitary and adrenal disorders, vitamin deficiency, diabetes, uremia, and pellegra), intracranial neoplasm, multiple sclerosis, Huntington's Chorea, Parkinsonism, Alzheimer's,Creutzfeldt's, Krapelin's, Pick's and Stern's disease were excluded from the study because of the possible etiological role of these disorders in cerebral dysfunctioning. Individuals with a history of psychoses or psychoneurosis were excluded from the study as well. Alcoholics and controls with evidence of polyneuropathy (characterized by reported

peripheral tingling, numbness, and burning pain in the feet and/or hands) were also excluded.

All potential control subjects were given the Michigan Alcoholism Screening Test to reduce the likelihood of selecting alcoholics for controls. Individuals scoring above the range for normal drinkers (a score of 5 or greater) were excluded from the study. Alcoholics with an admitted history of habitual drug usage other than alcohol were also excluded from the study.

Instruments

Frontal tests. To determine left frontal lobe functioning, each subject was given the Thurstone's Word Fluency Test. Milner (1964) reported this procedure to be selectively sensitive to lesions in the dominant (i.e., left for right handed subjects) frontal lobe. The subject is required to write down as many words as possible beginning with the letter 'S' for a period of 5 minutes. Following this, the subject is asked to write down as many four-letter words beginning with the letter 'C' as possible during a 4 minute trial. A subject's score is the total number of correct words produced.

Right frontal lobe functioning was assessed by performance on the Test of Three-Dimensional Constructional Praxis. Benton (1968) reported that this test

is differentially selective to the effects of right frontal lobe dysfunctioning. The procedure requires the subject to reproduce three complex structures, one made of 6 blocks, one made of 8 blocks and the other of 15 blocks, using a tray of assorted blocks. The subject's score is based on the number of errors (substitutions, omissions, and displacements) subtracted from a possible perfect score of 29, with the remainder as the obtained score.

Temporal tests. Left temporal lobe functioning was determined by using the Logical Memory portion of the Wechsler Memory Scale. Several investigators (Blakemore & Falconer, 1967; Meyer & Yates, 1955; Milner, 1967; Milner & Teuber, 1968) have found deficits in such verbal memory tests to be associated with left temporal lobe damage. In this test, the subject is required to recall as much information as possible from two single paragraph stories read by the examiner. The first story contains 24 bits of information, while the second contains 22. A subject's score is the average number of bits recalled for each paragraph (e.g., the first line of the paragraph is scored as follows: Anna Thompson/of South/ Boston). To assess both short- and long-term memory capacities, two versions of the same test were administered (Russell, 1975). The first required the subject

to recall the information immediately following the presentation of the story, while the second required a 30 minute delay between presentation and recall. Therefore, each subject provided two scores indicative of left temporal functioning, one for short-term verbal memory and the other for long-term. This procedure provided a more comprehensive assessment of left temporal lobe functioning.

The Visual Reproduction portion of the Wechsler Memory Scale was utilized to assess right temporal lobe functioning. Several investigators (Kimura, 1963; Milner, 1962, 1967; Warrington & James, 1967a) have reported impairment on such tests of memory for nonverbal stimuli to be indicative of selective damage to the right temporal lobe. In this test, the subject receives a 10 second exposure to each of three stimulus cards and is asked to reproduce the designs, using paper and pencil, in as much detail as possible following this timed exposure. A subject's score is based on the likeness of the drawing to the actual stimulus, with a possible high score of 14. As with the test for left temporal functioning, two versions of this test were also administered (Russell, 1975). The first required the immediate design recall (short-term) following its presentation, while the second required a 30 minute delay (long-term) be-

tween presentation and recall. Again, this more comprehensive procedure provided two scores of right temporal lobe functioning for each subject.

Parietal tests. Left parietal lobe functioning was determined by the performance of subjects on the Arithmetic subtest of the Wechsler Adult Intelligence Test. McFie (1975) reports that a left parietal lesion results in a significantly impaired performance on this subtest. This test consists of 14 arithmetic story problems that the subject is required to answer without benefit of paper and pencil. A subject's score is based on the number of correct answers given within a defined period of time for each problem; bonus points can also be accumulated for rapid responses on the last 4 items, allowing for a total possible score of 18.

The Visual Retention Test was employed to assess right parietal lobe functioning. Warrington (1967b) found patients with right parietal lesions to be significantly impaired on the two-second administration of this task. This multiple choice recognition task presents twenty 5 x 5 inch white cards with four blackened squares variously positioned on the face of each card such that no two cards are alike. After a two second exposure to each stimulus card, the subject is asked to identify the stimulus from a set of four similar figures.

The subject's score is the total number of correct responses out of a possible 20.

Procedure

All potential control subjects were identified by supervisors within various nonacademic departments at the University of the Pacific (operations and maintenance, custodial operations, auxilary services, and site development offices). All potential alcoholic subjects were identified by the director at each of the two alcoholic rehabilitation centers. To allow ample time for detoxification, the alcoholics were not solicited for the study until they were 14 days past medical detoxification. All subjects were contacted directly by the examiner and asked to voluntarily participate in the study. When possible, such requests were made in private rooms - otherwise, subjects were contacted by phone at their work spaces. No mention of brain damage was made when subjects were being asked to participate in the study, for fear of defensive postures that might interfere with the subject's test behaviors. The following is a verbatim set of statements that was used to solicit the subjects for the study.

> As a graduate student in psychology at the University of the Pacific, I've become very interested in the problems an alcoholic

faces on the road to recovery. The study that I am doing now is trying to find what kinds of things cause some alcoholics difficulty. If we knew that, maybe better rehabilitation programs could be developed that are sensitive to the alcoholic's special needs. What I want to do, in order to determine these things, is give some simple tests to several alcoholics and non-alcoholics. Then I will try to compare these two groups of people to see if there are any consistent differences. The tests I will give you are not for determining your intelligence or your personality, they are just ways of looking at how people solve problems of different kinds. Altogether, the tests will take about 1 hour of your time. I think you'll find them rather easy and maybe even fun to do. If you in fact do decide to be in the study, I will be glad to mail the results of the study to you when I have finished. If at any time you don't want to finish the testing, you are of course free to stop. I will do my best to make the testing a pleasant experience. Do you have

any questions?

After deciding to participate, each subject was immediately given an interview to rule out the possibility of complicating medical or psychological conditions (see Appendix A) and handedness (see Appendix B, Reitan, Note 3) that might obfuscate the interpretations of the results. Each control subject was screened for undiagnosed alcoholesm at that time (see Appendix C). Also, each alcoholic was asked to estimate the duration of his alcoholism and the average daily amount of ethanol consumed during the last three years of their abusive drinking period. After determining a subject was appropriate for inclusion in the study, a mutually agreeable appointment for the testing session was set up between the subject and the experimenter within a period that did not exceed 7 days from the initial interview. To assure voluntary participation in the study, each subject was asked to sign a consent form (see Appendix D) before scheduling the testing session. The following is a verbatim set of statements that were used during the interview:

> Now that you have decided to be in the study, I would like to schedule an appointment for the testing within the next 7 days. But before we do that, I need to ask you

several questions about your health and past history. It is important for me to ask these questions to be sure that there is nothing in your medical background that will interfere with the testing procedures. This will only take a few minutes.

Alcoholic subjects were tested in a private quiet room provided by each treatment facility. Control subjects tested under similar conditions in a small sound shielded room located adjacent to the main office of the Psychology Department at the University of the Pacific. Each subject was given standardized instructions and feedback as provided by the test publishers. Otherwise, all subjects routinely received positive feedback about their test performance to discourage any feelings of inadequacy that might impair their test performance (e.g., "good", "you are doing just fine", etc.).

Individual testing administrations took approximately 1 hour for the entire procedure. To allow for the 30 minute delay required by the temporal tests, the tests were given in the following order: Logical Memory (Immediate Recall), Visual Reproduction (Immediate Recall), Word Fluency, Arithmetic, Logical Memory (Delayed Recall), Visual Reproduction (Delayed Recall), Three-Dimensional Construction Praxis, and the Visual

Retention Test. The interposed tests were quite different from either memory test in order to prevent contamination and interference with memory recall. To discourage fatigue and restlessness, each subject was allowed a 5-10 min. break period between every other test. During these breaks, the subject was allowed to visit the bathroom, to smoke, to drink water, and encouraged to engage in friendly conversation. Also, any questions about the tests were answered during these breaks. At the close of the testing session, each subject was thanked for their participation in the study. Any questions the subject may have had were answered at that time. For those subjects that indicated that they would like to be furnished with a copy of the results, a brief (not exceeding one typed page) non-technical description of the experimental outcome will be mailed to a designated address.

Results

Tests for the subjects were scored by a person blind to the conditions of the subjects and naive to the purpose of the study. Group means and standard deviations for the raw scores on each measure appear in Table 1. A multiple covariate analysis (Tatsuoka, 1971) was performed on the data to test the relationship between the three possible covariates (age, education, and socioeconomic

Table 1

Mean (±SD) Raw Scores of

Alcoholics and Nonalcoholic Controls

Test	Controls	Early Stage Alcoholic	Late Stage Alcoholic
Word Fluency	51.1 ± 16.2	43.1 ± 13.1	32.5 ± 16.4
3-D Cons. Prax.	27.9 ± 2.1	28.2 ± 1.2	25.5 ± 3.4
Log. Mem.-Im.	9.1 ± 3.6	8.0 ± 3.1	6.6 ± 2.3
Log. Mem.-Del.	7.9 ± 3.3	7.1 ± 2.5	5.3 ± 2.9
Vis. Repro.-Im.	12.5 ± 2.7	11.2 ± 2.2	9.0 ± 3.1
Vis. Repro.-Del.	10.5 ± 3.8	10.3 ± 2.8	7.3 ± 3.7
Arithmetic	11.8 ± 4.3	10.7 ± 2.9	10.3 ± 2.6
Vis. Reten.	15.7 ± 2.2	14.3 ± 2.1	14.2 ± 2.3

status) and each dependent measure. Scatter diagrams for each covariate and dependent measure were done to test the assumption of linearity inherent in this approach. Based on visual analysis of these diagrams it was concluded that a linear model best described any relationships between the covariates and the dependent measures. Results of the covariate analysis can be seen in Table 2. To estimate the strength of the relationship between each covariate and the dependent measures, simple correlation coefficients were calculated for all the subjects combined. The results of this analysis can be seen in Table 3. A comparison between Table 2 and Table 3 indicates that there is not a perfect correspondence between the significant F tests and the significant simple correlations for the covariates on the dependent measures. The differences between these tables is a function of testing partial correlations in Table 2 which takes into account associations between covariates, versus testing simple correlations in Table 3 which do not consider associations between covariates.

To test the assumption of homogeniety of regression across groups, each dependent measure was analyzed for the possibility of a Group x Covariate interaction. For all measures, none of the interactions approached a level of significance, indicating that the regression

Table 2

F Tests of Significance for Three Covariates on Eight Neuropsychological Tests

Test	Covariates		
	Age	Education	Socioeconomic Status
Word Fluency	NS	9.21**	NS
3-D Cons. Prax.	NS	NS	NS
Log. Mem.-Im.	NS	8.69**	NS
Log. Mem.-Del.	NS	5.81*	NS
Vis. Repro.-Im.	15.08**	NS	NS
Vis. Repro.-Del.	20.88**	NS	NS
Arithmetic	NS	5.42*	NS
Vis. Reten.	NS	4.92*	NS

df = 1/39
*p<.05
**p<.01

Table 3

Correlation Coefficients Between
Three Covariates and Eight Neuropsychological Tests
for All Subjects Combined

Test	Covariates		
	Age	Education	Socioeconomic Status
Word Fluency	-.031	.572**	.232
3-D Cons. Prax.	-.381*	.214	-.068
Log. Mem.-Im.	-.293	.409**	-.010
Log. Mem.-Del.	-.316*	.394**	.029
Vis. Mem.-Im.	-.529**	.238	.005
Vis. Mem.-Del.	-.564**	.287	-.007
Arithmetic	.095	.504**	.372*
Vis. Reten.	-.027	.380*	.119

df = 43
*p<.05
**p<.01

coefficients for the three groups on the covariates were equal. The results of the multiple covariate analysis and the test for Group x Covariate interactions reduced the overall data analysis for group differences to a one-way analysis of covariance (with 3 levels) for seven of the dependent measures and a one-way analysis of variance (with 3 levels) for the eighth measure. The results of these analyses can be seen in Table 4.

The general trends of the performances of the three groups may be seen in Figure 1. Both the raw and the adjusted mean T scores (a standardized score with a mean of 50 and a standard deviation of 10; defined as T=10z+50) on each dependent measure are presented. Mean T scores from the raw data were calculated using the mean and standard deviation of the control group on each measure. Adjusted mean T scores were calculated using the mean age (32.6 years) or education (12.7 years) from the control group for those measures warranting adjustment.

To determine the roles duration of alcoholism and amount of alcohol consumption might play in the alcoholic's test results, simple and multiple correlation coefficients were calculated for the alcoholics combined. The results of this analysis can be seen in Table 5.

Discussion

As expected, the late stage alcoholics performed

Table 4

F Tests of Significance for Early Stage Alcoholics (ES), Late Stage Alcoholics (LS) and Controls (C) on Eight Neuropsychological Tests

Tests	Covariate	Overall F	Multiple Comparisons			
			C vs ES	C vs LS	C vs (ES+LS)	ES vs LS
Word Fluency	Education	3.27*	NS	6.53*	4.72*	NS
3-D Cons. Prax.	None	5.89[a]**	NS	7.89[b]**	NS	9.68[b]**
Log. Mem.-Im.	Education	NS	NS	NS	NS	NS
Log. Mem.-Del.	Education	NS	NS	3.35≠	NS	NS
Vis. Repro.-Im.	Age	2.91≠	NS	5.23*	5.54*	NS
Vis. Repro.-Del.	Age	NS	NS	NS	NS	NS
Arithmetic	Education	NS	NS	NS	NS	NS
Vis. Reten.	Education	NS	NS	NS	NS	NS

Note: df = 2/41 overall F test; df = 1/41 specific comparisons

[a]df = 2/42

[b]df = 1/42

*p < .05

**p < .01

≠p < .10

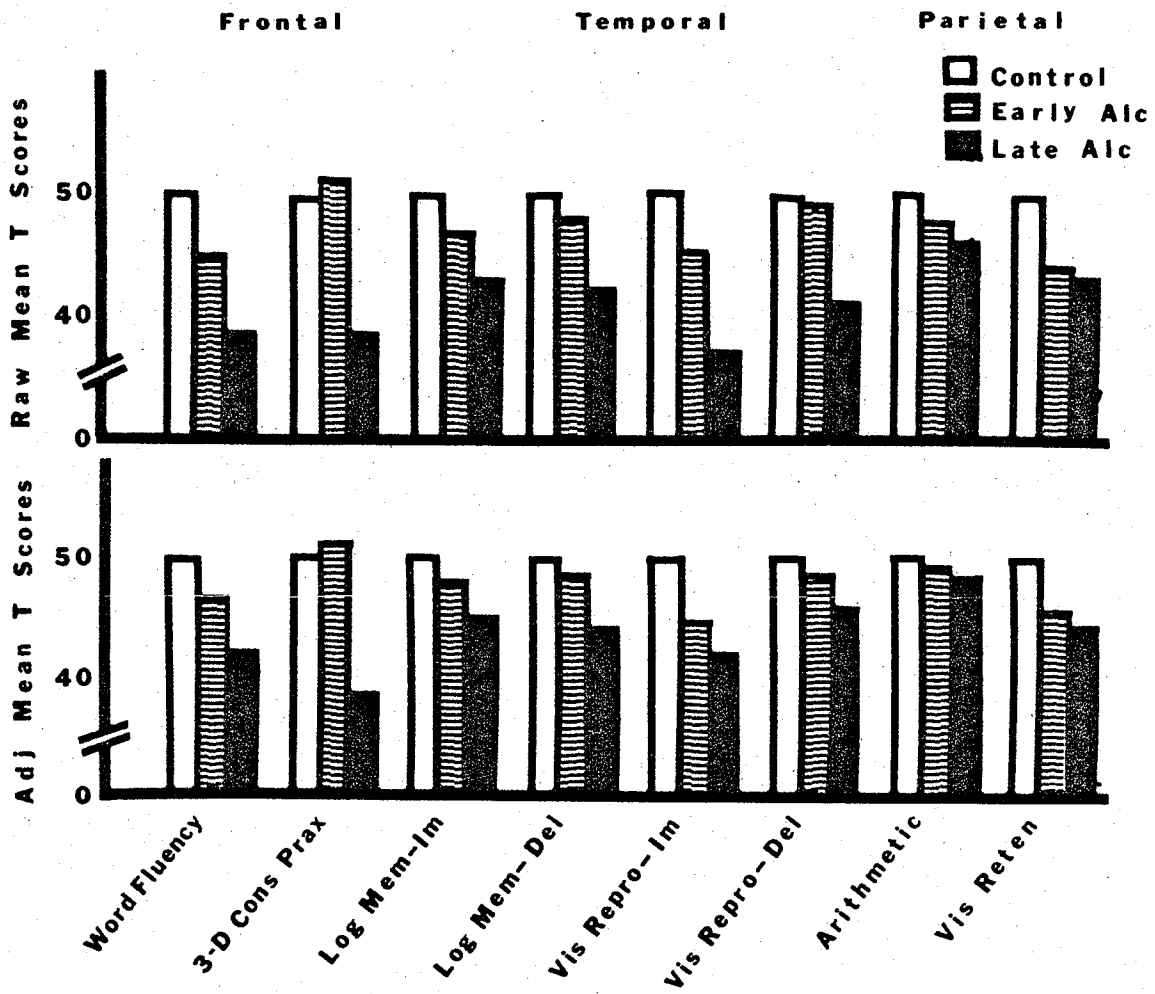

Figure 1. Raw and Adjusted Mean T Scores for Early Stage Alcoholics, Late Stage Alcoholics, and Controls on Eight Neuropsycholigical Tests.

Table 5

Simple and Multiple Correlation Coefficients for Alcoholics Combined on Eight Neuropsychological Tests

Measures	Neuropsychological Tests							
	Word Fluency	3-D Cons. Prax.	Log. Mem. Im.	Log. Mem. Del.	Vis. Repro. Im.	Vis. Repro. Del.	Arith.	Vis. Reten.
Years[a]	-.168	-.432*	-.266	-.317	-.395*	-.528**	-.046	-.176
Amount	-.192	-.630**	-.221	-.282	.104	.022	-.281	-.303
Years x Amount[b]	.224	.679**	.305	.372	.459*	.561**	.284	.316

[a]df for simple correlation = 28

[b]df for multiple correlation = 2/27

*p .05

**p .01

below the level of the controls on all eight measures given (See Figure 1). The difference between the late stage alcoholics and the controls reached a level of significance for the two measures of frontal functioning and for two of the four measures of temporal functioning (Logical Memory - Delayed and the Visual Reproduction - Immediate). There was no significant difference between the late stage alcoholics and the controls on the two measures of parietal functioning. Although none of the differences between the early stage alcoholics and the controls reached significance, the early stage alcoholics performed below the level of the controls on seven of the eight measures given. Interestingly, the late stage alcoholics performed below the level of early stage alcoholics on the measures of frontal and temporal functioning, however, the differences between these two groups was only significant for the Three Dimensional Constructional Praxis Test. As predicted, the general trend of the adjusted mean T scores indicates that the late stage alcoholics were found to be most strongly deteriorated on the tests of frontal lobe functioning, followed by lesser impairment on the tests of temporal functioning, with no evidenced impairment on the tests of parietal lobe functioning. This impairment gradient is consistent with the findings of neuropathological investigations

in which frontal lobe atrophy has commonly been found in alcoholics (Brewer & Perrett, 1971; Courville, 1955; Lynch, 1960; Mancall, 1961; Warner, 1934) and with those studies reporting disruption of adjacent cortical structures (Berglund & Ingvar, 1976; Brewer & Perrett, 1971; Carlen et al., 1978; Courville, 1955; Tumarkin et al., 1955). The findings of this study support a model of alcoholic brain damage that can be conceptualized as the diffuse frontal-limbic-diencephalic hypothesis.

In partial support of the right brain hypothesis of alcoholic brain damage, the late stage alcoholics in this study displayed greater impairment (based on adjusted mean T scores) on those measures associated with the right hemisphere. For the temporal lobe functioning tests on which alcoholic subjects were impaired, the greatest impairment was found to be on those measures associated with the right lobe (Visual Reproduction - Immediate vs. Logical Memory - Delayed). On the frontal tests, the late stage alcoholics showed greater difficulty with the right lobe measure (the Test of Three Dimensional Constructional Praxis) than with the left lobe measure (Word Fluency). However, this relationship is exactly opposite for the early stage alcoholics, who displayed greater difficulty with the left lobe measure. This apparent performance discrepancy between right and

left hemisphere tasks may in part be related to differences in disruption thresholds. Figural and/or nonverbal information that is associated with the right hemisphere may have a lower threshold of disruption than well codified and rehearsed verbal information that is associated with the left hemisphere (Chandler & Parsons, 1977). Also, the tests used in this study were not equated for item difficulty and reliability (Parsons & Prigatano, 1978). Therefore, it is not possible to determine what role these factors played in contributing to the performance differences on the left versus right hemisphere tasks.

Consistent with prior research on the effects of chronicity (Fitzhugh et al., 1960, 1965; Jones, 1971a, Jones & Parsons, 1971; Tarter, 1973; Tarter & Jones, 1971; Tarter & Parsons, 1971) the late stage alcoholics performed below the level of the controls on all eight tests given, with significant differences occurring on the measures of frontal functioning and two measures of temporal functioning. The early stage alcoholics also generally performed below the level of the controls, however, none of these differences reached a level of significance. Although there were few significant correlations found between duration and amount of consumption with each dependent measure, the general trend indicates that both

amount and years of consumption have a negative influence on the tests given. Other studies (Klisz & Parsons, 1977; Long & McLachlan, 1974; Tarter & Jones, 1971) attempting to relate alcoholic chronicity with psychological test performances have also generally found weak associations. However, the self reported measure of drinking history used in this study may have been too crude for the determination of precise correlations. A more extensive interview may be required to obtain more accurate estimations of duration of alcoholism before correct relationships with test performances can be determined.

The outcome of the multiple covariate analysis clearly provides evidence for the effects of age and education as influencing factors in the dependent measures used in this study. Also, none of the dependent measures were significantly influenced by more than one of these covariates. Socioeconomic status (average yearly income), however, was not found to be an important influencing factor across the dependent measures. Because this study failed to include broader aspects in its measure of socioeconomic status (e.g., occupation, specific acquired skills, and environmental factors) it is uncertain what impact this variable as broadly defined may have had.

The results of the covariate analysis also indicate

that each test given is differentially affected by age and education. Although neuropsychological investigators frequently rely on published norms (usually based on large samples) regarding the effects of these covariates, results from this study suggest that such effects may vary across samples and investigations. For example, both age and education are expected to have a significant influence on the Logical Memory - Delayed Recall Test. This expectation is consistent with the significant correlations found in this study between both these covariates and the Logical Memory - Delayed Recall Tests. However, results of the covariate analysis indicated that age did not significantly influence a subject's performance on the Logical Memory - Delayed Recall Test beyond the effects of education. Therefore, only education was used as a covariate in the analysis of group differences on this measure. Discrepancies between published norms and the present study for the influence of age as a covariate were also found on the Logical Memory - Immediate Recall Test, Word Fluency Test, and the Arithmetic Test. For the effects of education as a covariate, discrepancies between the published norms and the present study were found on the Test of Three Dimensional Constructional Praxis and the Visual Reproduction Tests. The results of the correlations

between each covariate and the dependent measures are generally consistent with the above findings. Interestingly, the effects of a covariate on a measure did not vary across the three groups employed in this study, indicating that the variance due to alcoholism was not large enough to reduce the effects of a covariate.

To reduce or possibly eliminate the effects of a covariate in neuropsychological testing, it has been recommended that researchers should equate groups being studied on the means and standard deviations of the covariate (Parsons & Prigatano, 1978). Although it is virtually impossible to exactly equate means and standard deviations among different groups, investigators are frequently content to manipulate such parameters by dropping or adding subjects to each group until there is no statistical difference between groups being studied. Not only does this procedure potentially introduce unwanted regression effects and an uneven distribution of extraneous variables across groups, it may also be an inadequate control. To illustrate this point, consider that the three groups in this experiment are reasonably equated on education. If education had not been used as a covariate in the data analysis for those measures it was significantly related to, a different experimental conclusion would have been warranted. As

such, this alternative conclusion would have indicated that the damage to the alcoholic brain was diffuse or generalized. For example, using a one-way analysis of variance to analyze the raw scores, significant group differences between the late stage alcoholics and the controls ($p<.05$) would have been found on four out of the five measures influenced by education (Word Fluency, Logical Memory - Immediate and Delayed, and the Visual Retention Test). However, the results of analyzing differences between these groups with education as a covariate, disclosed significant outcomes on only two of the five measures (Word Fluency and Logical Memory - Delayed). In fact, analyzing each dependent measure with an analysis of variance would have resulted in significant differences between the late stage alcoholics and the controls on seven of the eight measures given in this study (no significant outcomes were found on the Arithmetic Test). See Figure 1 for a comparison of raw versus adjusted mean T scores. These findings confirm the need in neuropsychological research for a data analysis that revolves around a careful statistical search for covariates that may significantly influence a subject's performance on a psychological measure (i.e., age, education, socioeconomic status, sex, etc.).

The results of the present investigation provide

strong evidence of cerebral damage associated with chronic alcoholism. Accordingly, this damage can best be described as a gradient that principally involves the frontal lobes of each hemisphere, with spreading occurring in the adjacent temporal areas. Although the tests selected for use in this study were believed to be reasonably sensitive to circumscribed brain regions, it may be argued that the destruction of any brain area may result in the disruption of a variety of intellectual skills. Therefore, the testing of alcoholics on measures with greater sensitivity may further resolve the localization issue of alcoholic brain damage. Furthermore, a more accurate localization profile may be determined by the testing of alcoholics on neuropsychological measures that are equated on item difficulty and reliability. The results of this study also indicate that the alcoholic's drinking history may relate to cognitive performance. Specifically, both the duration and amount consumed may negatively influence the alcoholic's cognitive abilities. It may be of future interest to determine how the age of onset of heavy drinking and both the amount and type of beverage consumed may interact with the cognitive performance of alcoholics. Further research is necessary to establish how the cognitive deficits observed in this study influence the alcoholic's adaptive capabilities, potential for recovery, and therapeutic success.

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Appendix A

Medical and Psychological Screening

Name_____Date_____

1. Have you ever been hospitalized or treated for an infection of the brain, spine or other nerves?
2. Have you ever been hospitalized for a head injury of any type?
3. Have you ever been knocked unconscious? If yes, how long did you remain so?
4. Have you ever been treated for or hospitalized for high blood pressure, heart problems, a stroke or other blood circulatory problems?
5. Do you now have or have you ever had sudden uncontrollable body tremors, muscular twitches or convulsions?
6. Have you ever noticed a brief loss of awareness?
7. Do you now have or have you ever been treated for vitamin deficiencies, diabetes, glandular problems or any other condition related to your body chemistry?
8. Have you ever been diagnosed to have a brain tumor or growth?
9. Has a medical doctor ever suggested that you might have premature ageing of the brain (senility)?
10. Can you think of any medical problem that you have had in the past or currently suffer from that I did

not ask you about?

11. Have you ever been admitted to a mental hospital for any period of time?

12. Are you currently seeing, or have you ever seen, a mental health professional for personal difficulties?

13. Do you ever suffer from a tingling, numbness, or burning pain in your feet or hands?

NOTE: Any and all affirmative answers to the above questions will be explored in greater detail to to determine if the subject has evidence of a condition warranting exclusion from the study.

Alcoholics Only

14. During the last three years you were known to have a drinking problem, how much did you drink on a typical day?

_____

15. Have you ever, or do you now, frequently use drugs other than alcohol? If so, what kind and how much?

_____

16. How long have you considered yourself to have a drinking problem?

_____

Appendix B

Lateral Dominance Examination

Name_____Date_____

1. Show me your right hand_____; left ear_____;
   right eye_____
2. Show me how you: throw a ball_____
   hammer a nail_____
   cut with a knife_____
   turn a door knob_____
   use scissors_____
   use an eraser_____
   write your name_____
3. Write your full name_____
   preferred hand (_____) _____seconds
   non-preferred hand (_____) _____seconds
4. Show me how you look through a telescope _____eye
   Aim this gun at the tip of my nose _____shoulder
   _____eye
5. Show me how you kick a football _____foot
   step on a bug _____foot

Appendix C

MAST

Name_____Date_____

1. Within the last two months have you felt that you are a normal drinker? YES NO
2. Within the last two months have you ever awakened the morning after some drinking the night before and found that you could not remember a part of the evening before? YES NO
3. Within the last two months has your wife or parents complained about your drinking? YES NO
4. Within the last two months have you been able to stop drinking without a struggle? YES NO
5. Within the last two months have you felt bad about your drinking? YES NO
6. Within the last two months have your friends felt you are a normal drinker? YES NO
7. Within the last two months have you tried to limit your drinking to certain times of the day or to certain places? YES NO
8. Within the last two months have you been able to stop drinking when you want to? YES NO
9. Within the last two months have you attended a meeting of Alcoholics Anonymous (AA)? YES NO

10. Within the last two months have you gotten into fights when drinking? YES NO

11. Within the last two months has your drinking created problems with you and your wife (other family members)? YES NO

12. Within the last two months has your wife (other family members) gone to anyone for help about your drinking? YES NO

13. Within the last two months have you lost any friends because of your drinking? YES NO

14. Within the last two months have you ever gotten into trouble at work because of your drinking? YES NO

15. Within the last two months have you lost a job because of your drinking? YES NO

16. Within the last two months have you neglected your obligations to your family or your work for two or more days in a row because you were drinking? YES NO

17. Within the last two months have you drank before noon? YES NO

18. Within the last two months have you been told that you have liver trouble? YES NO

19. Within the last two months have you had delerium tremens (DT's), severe shaking, heard voices, or

seen things that weren't there after heavy drinking? YES NO

20. Within the last two months have you gone to anyone for help about your drinking? YES NO

21. Within the last two months have you been hospitalized because of your drinking? YES NO

22. Within the last two months have you been a patient in a psychiatric ward where drinking was a part of the problem? YES NO

23. Within the last two months have you been seen at a psychiatric or mental health clinic, or gone to a doctor, social worker, or clergyman for help with an emotional problem in which drinking had played a part? YES NO

24. Within the last two months have you been arrested for drunken behavior? YES NO

25. Within the last two months have you been arrested for drunk driving or driving after drinking? YES NO

Subject's Total Score_____

All subjects with a score of 5 or greater are excluded from the study.

Appendix D

Consent Form

I, _____ (name), hereby agree to participate in John Bolter's study on alcoholism. The purpose and intent of the study has been explained to my satisfaction. I understand that the information I provide will be used only in connection with the study and that my identity will remain anonymous. I further understand that my participation will require approximately one hour and that if at any time I don't feel comfortable with being in the study, I am free to resign from the study. I have also been informed that the results of the study will be furnished to me by mail upon my request.

Signature_____

Date_____