

IN THE SUPREME COURT OF FLORIDA

THE FLORIDA BAR,
Complainant,

vs.

MYRON C. PREVATT, JR.
Respondent.

Case No. 77,271

TFB File No's. 88-00077-04C
and 91-00564-04C

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INITIAL BRIEF OF THE RESPONDENT,

MYRON C. PREVATT, JR.

ON APPEAL FROM A REPORT OF REFEREE

RICHARD E. WELTY, CHARTERED

By: *[Signature]*

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Appendix

1. Excerpt from "The Course of Alcoholism Four Years After Treatment," A Wiley-Interscience Publication, John Wiley & Sons, copyright 1980, **1981** by The Rand Corporation.

2. "Recovery: A Way of Life," by Miles Conway, from Alcoholism Treatment and Recovery, edited by Marshall J. Goby, PhD, copyright 1984, The Catholic Health Association of the United States.

3. "Brain Damage in Chronic Alcoholism," by Maria A. Ron, from Alcohol Related Problems Room for Manoeuvre, edited by Neville Krasner, J. S. Madden, and Robin Walker, copyright 1984, John Wiley & Sons.

4. "Cognitive Loss and Recovery in Long-Term Alcohol Abusers," by Jason Brandt, PhD; Nelson Butters, PhD; Christopher Ryan, PhD; and Roger Bayog, MD; from The Archives of General Psychiatry, Volume 40, April 1983.

STATEMENT OF THE CASE: This action was commenced by The Florida Bar against the Respondent, **MYRON C. PREVATT, JR.**, an attorney licensed to practice law in the State of Florida. Basically, there are two aspects of this action: (1) improper handling of his trust account; and (2) the improper handling of an estate. The Respondent stipulated to many of the violations, but specifically did not stipulate to any of the issues that dealt with criminal conduct. In his defense, he raised the issue of alcohol abuse. The Referee found against the Respondent on all issues, rejected his defense of alcohol abuse out of hand, and recommended that the Respondent be disbarred for a period of five years. Respondent takes this appeal.

AS TO THE IMPROPER HANDLING OF HIS TRUST ACCOUNT:

Respondent has very little to respond to in this matter. He stipulated to the facts stated in the Complaint, and no exception was taken to the report of The Bar's auditor. Respondent would only reiterate to this Court, as he did to the Referee, that he cooperated with The Bar in this investigation, he hired an accountant to help get the records in order, and there was never any indication of any funds missing from his accounts. The **accounts** were handled poorly, and there was testimony from his secretary, to explain that this was the system used by his father, but that his father was far more meticulous and was a hands on manager. Respondent, on the other hand, was always more eager to delegate the day-to-day operations to his staff, and did not do a good job of training them. Instead, he expected them to learn on their own. Combining this aspect of his management philosophy with his abuse of alcohol was the main cause of his poorly kept trust accounts.

Thus, he learned the trust account system from his father, but did not maintain the system in the personal way his father had. When The Bar rules changed to require more

detailed procedures, he was not mentally alert enough to make the changes. In his defense, however, the evidence clearly showed that he had begun working on this problem several years before The Bar stepped in. Actually, his trust accounts were in good order for the years that he had his computer program in place. **He was** in the process of going backwards in time to put the older accounts on the computer when the complaint **was** filed.

SUMMARY OF ARGUMENT AS TO REMAINING ASPECT OF THE ACTION

1. **ALCOHOL ABUSE IS A DEFENSE TO THIS ACTION.** To have a criminal act, one must show a criminal intent. The abuse of alcohol is a well-known defense to criminal conduct.

2. **THE EFFECTS OF LONG TERM ALCOHOL ABUSE ARE LONG LASTING.** The Referee's cavalier dismissal of the effect of alcohol abuse on the Respondent shows a total ignorance of the long term effects of alcohol abuse on the alcoholic, or an insensitivity to the problem of alcohol abuse.

ARGUMENT

I. ALCOHOL ABUSE AS A DEFENSE TO THIS ACTION

The Referee's remarks in his Report, at page 10, to-wit:

"While it is clear, and unchallenged that respondent was an abuser of alcohol and in fact was hospitalized in 1983 for medical complications associated with long term abuse, such condition does not account for the pattern, method, scope, nature nor duration of the misconduct present over this twelve (12) year process.

Indeed over the post 1983 years, respondent describes himself as a "recovering" alcoholic, yet significant and onerous abuses of the client/attorney process continued unabated. Alcoholism is not a mitigating factor in this case."

show either a total insensitivity to a person with a very common and very sad disease, a total ignorance of the effects of alcohol on the abuser, a complete lack of knowledge of the law **as** it pertains to alcohol abuse, or all of the above.

Abuse of alcohol is one of the few diseases that the law recognizes **as** a defense. There is even a jury instruction dealing with the subject. In criminal law, this is a fairly common defense, and there **is** no question that it was raised and proved at the hearing. In fact the Referee called it "unchallenged."

Beginning on **page 64** of the trial transcript, the Respondent's secretary, DIANE NETTLES, explains how she knew within one week of being hired that Respondent was an alcoholic. This was in 1974, and by the Respondent's own admission it became a problem in the late sixties or the early seventies." [Trial transcript at page 129].

As Ms Nettles's testimony indicates, the Respondent turned over the day-to-day operations of the office to his secretaries. There is no question that it was their good works that kept him from having any prior problems with The Bar or any other client.

As her testimony continues, she explains how Respondent's drinking became more and more of a problem with the functioning of the office. This testimony alone should have been sufficient to **show** that the Respondent **was** under the influence of alcohol to the extent that it **would** have been impossible for him to have the necessary intent to commit a criminal offense. This was not a man that got intoxicated on the weekend or went on a binge once in a while, This was a man who drank all the time. and the drinking affected his functioning as an attorney. It affected his ability to do his job.

One fact that needs **to be** restated here is that Respondent had been on the checking account of Mr. McKay for **several** years and was put on his savings account just before he went into the nursing facilities. Had Respondent wanted to actually steal the

money from Mr. McKay, he could have written a check and gone his way. **As it was**, he kept what he considered meticulous records of his transactions. **If** he wanted to be dishonest, he could have written all the relatives upon Mr. McKay's death and told them that all the funds had been used to care for Mr. McKay and his funeral. They would have probably believed him and no one would have been the wiser. That is not what he did. I am not trying to say that the way he handled the situation was correct, but he did have a plan formulated while he was drinking, and he continued to follow it.

I know that the relationship of Mr. McKay with the Respondent was one of attorney/client, and Respondent is painfully aware that this is the correct way he should have handled the situation. However, we must remember that this man was also a long-time friend of Respondent's and his family. Obviously, that is one reason that Respondent's name **was** on the checking account long before Mr. McKay became ill. Combined with this long personal relationship is the effect on the Respondent's thought **processes** caused by his abuse of alcohol.

The testimony **was** clear that Respondent gave over every other file to his secretaries to handle, but he did not turn **over** the McKay file to them. That is, **I assert**, because he felt **as if** he were taking care of an old family friend. This relationship was mure and we all know it, but then we are not functioning with the mental processes that are impaired by alcohol. The Bar would have you believe he handled the file himself because he wanted to steal the man's money. If that were his intent he didn't do a very good **job** of it. **As** pointed out earlier he could have just written himself a check and let the old man rot.

Instead he took care of his **old** friend, and made sure that he was cared for to the best of his ability. He kept records of all that he did, and although they were not perfect

records they were very complete, and not all that bad when you consider his abuse of alcohol. Again, I do not want to have this Court think that I **am** condoning the Respondent's actions, **because** we all know that this was not the proper way to handle this matter, but I do think we need to understand Respondent's thought process and realize that there was no criminal intent on the part of Respondent, His mind was adversely affected **by** the abuse of alcohol, and there could be no criminal intent.

Without the criminal intent, the worst aspects of The Bar's complaint and the Referee's findings **are** removed. We are left with the parts to which Respondent stipulated. Considering the Respondent's long history of alcohol abuse that is the only result one can reach, Without the intent there can be no criminal action, and there cannot **be** a criminal intent with the "...clear, and unchallenged..." fact that Respondent was an abuser of alcohol from about 1970 until 1983.

II. THE **EFFECTS** OF LONG-TERM ALCOHOL **ABUSE** ARE LONG LASTING

The Respondent admits that this aspect of the case was not pursued before the Referee **as** strongly **as** it should have been. The treatises that were presented to the **Referee** did not elaborate on this subject, and Respondent assumed that the Referee had more knowledge in this **area** than is shown by his report. However, a reading of his report displays a total lack of understanding concerning the problems of the alcoholic, or a total insensitivity to the problems manifested by this disease. I am going to vote for the former, because the Referee handled the attorneys and the parties with utmost respect at both hearings. **Also**, as **seen** in the next to the **last** paragraph on page 10 of his report he goes to great pains to put quotation marks around the word recovering to describe the Respondent **as** a "recovering" alcoholic. This one action appears to me to show a lack of knowledge of the disease.

Of course, the Respondent is a recovering alcoholic! Once an alcoholic always an alcoholic. Respondent's claim to fame is that he is a recovering alcoholic, which simply means that he has taken the cure and has **been** sober since February 1983. In that the Referee **did** not appear to know that this was the way in which alcoholics classify themselves, he made what I consider some poor assessments of the situation and of the evidence presented in this **case**.

The Referee implies that the Respondent was admitted to the hospital in 1983 for the treatment of the disease, and upon his release he was **as** capable **as** anyone **else** to return to work and to carry on his life. Nothing could be further from the truth, and it **was** not what **was** set forth in the evidence presented to the Referee at the hearing.

Ms Nettles testified **as** to Respondent's abuse of alcohol and how it had affected him and his work. Beginning on page **68** of the hearing transcript **she** explains that the drinking, which **she** spotted almost immediately after coming to work for the Respondent, continued to increase and affected the operation of the office. **It** got to the point that the Respondent would not even come into the office, but would call and see if there were any appointments that required his presence. When he failed to even call in one day, **she** went to his house and found him in his old school bus converted to a camper, and he was completely at the bottom of the barrel. He was swollen and could not even get back into the bus **after** he fell out. What she saw was the culmination of close to 15 years of alcohol abuse.

The Respondent was admitted to the hospital and was very close to death. His body was toxic and any more alcohol or a delay of another day may have been fatal. He was treated for cirrhosis and hepatitis. Sixteen **days** after being admitted to the hospital,

he was released. **As** soon thereafter **as** he could be admitted, he entered the Veterans Administration Hospital for the 28 day alcohol treatment.

To read the Referee's report, one would think that Respondent would then return to work a new man, with all of his faculties, and ready to right any mistake that he had made in the past. Such is not the **case** with alcoholics. Not only do they often have the medical problems of cirrhosis and hepatitis to contend with, neither of which is easy to recover from, but they **also** have the extra baggage of attempting to put their lives back together after years of drinking.

As any of **us** who have ever been drunk even one time can attest to, over indulgence in the **use** of alcohol affects the brain and the thought processes. Medical studies have confirmed that this is **a** major problem with the recovering alcoholic. They have, to use the vernacular, pickled their brains in alcohol, and not only **do** they have to dry-out, i.e., remove **all** the alcohol from the system, but they also have to get the brain to function properly again. This is what the Referee did not appear to understand, and an understanding **of** this problem **is necessary** to understand the problems that Respondent faced after he **was** released from the Veterans Administration Hospital. This knowledge, the Respondent assumed, was in the Referee's area of knowledge, When it became obvious that it was not, Respondent sought more detailed medical studies to buttress this brief. That **is** the reason an extension of time was sought.

Several studies, or at least parts of them, are included in the appendix to this brief. Each discusses to some degree cognitive impairment marked by problems in concentrating and remembering. In other words, not only does the alcoholic not return to work **ready** to correct the wrongs perpetrated in the past, he doesn't even remember his mistakes,

About a year after Respondent returned to full-time work, Mr. McKay died. The Respondent had only received one contact from any relative at the time Mr. McKay went into the nursing home, and one letter from a nephew after Mr. McKay passed away. That is to say that there were no squeaking wheels here looking for **grease**. The recovering alcoholic (Respondent) was busy attempting to move forward in his life, and to program his mind in an attempt to fill in the void caused by his years of heavy drinking. **As** the studies indicate, such improvement takes place over a long period.

An excellent article dealing with this problem is from The Archives of General Psychiatry, Volume 40, 1983. The article is titled, "Cognitive **Loss** and Recovery in Long-Term Alcohol **Abusers**," authored by Jason Bennet, PhD; Nelson Butters, PhD; Christopher Ryan, PhD; and Roger Bayog, MD. This deals with the effects on the mental processes of the long-term (10 years or more) abuser of alcohol. Respondent certainly fits this description.

The article *is* detailed discussing short-term abstinence, long-term abstinence, prolonged abstinence, short-term memory, and long-term memory. Some of the findings and conclusions **are** discussed hereafter.

As mentioned earlier, the Referee did not seem to comprehend that though the Respondent was out of the hospital and **back** at work, he did not have full use of his mental processes for several years after returning to work.

The obvious conclusion to be drawn from these data is that recovery of cognitive function after cessation of drinking is not an all-or-nothing phenomenon. Improvement with prolonged abstinence was very notable on tasks requiring *short-term* retention of nonverbal and verbal information. In fact, alcoholics who remained sober for a minimum of five years performed, **as** a group, as well **as** nonalcoholics on our measures of STM. Dramatic improvements were **also** seen on tests of rapid symbol-to-number and number-to-symbol transcription, which may reflect recovery of motor regulation and/or increased psychomotor speed.

On the other hand,.... Even after almost seven years of continuous abstinence, long-term alcoholics displayed a profound deficit in the learning of novel associations....

... Ryan and associates examined recovery after extended periods of abstinence **and** did not find restitution of any memory **skills** with abstinence periods of one to five years. Berglund and co-workers in one of the few long-term follow-up studies of cognitive and social functioning in alcoholics, examined subjects an average of 3.7 **years** after inpatient alcoholism treatment. They found that subjects who had remained abstinent or **whose** alcohol consumption had significantly declined displayed slight improvements on several measures of intellectual functioning." **Id.**, at **page 440**.

Towards the **end** of that article the authors conclude:

"... The **results** of the present study clearly indicate that there are significant deficits to short-term retention, new learning **and** memory and visuoperceptual analysis in thoroughly detoxified long-term alcoholics." **Id.**, at **page 441**.

From this it is clear that the alcoholic has problems when he is drinking, and if he is lucky to stop drinking and remain sober, he does not recover instantly. He cannot function at what would be a normal level for those of us who have never been subject to the disease for years after he has stopped drinking. This must explain, at least partly, the law's view of the alcoholic. The Florida Bar and this Court have been instrumental in this field by forming Florida Lawyers Assistance, with which the Respondent has been associated since **he** returned to work.

The law recognizes the abuse of alcohol **as** a defense in criminal matters. Society has finally come around to realize that this **is** a disease we are dealing with, and not just a weakness of the individual. The individual alcoholic cannot take a drink and leave it alone **as** the vast majority of people can. Instead he continues *to* consume and always to his detriment,

Respondent **is** an alcoholic, thank goodness a recovering alcoholic. For him **as** long **as** he abstains from alcohol he will **be** recovering and he **will** continue to improve the functioning of his mind.

Respondent is concerned that **if** the sanction set by the Referee in this matter is not reduced, then other attorneys that are suffering from the same disease will not want to cooperate with The Bar **as** the Respondent has. During this investigation, the Respondent did everything he could to aid The Bar, its auditor, and its investigator.

CONCLUSION

Respondent **does** not expect to walk out of this proceeding unscathed. After all he stipulated to certain elements of The Bar's complaint, and has admitted that he handled his office trust accounts poorly and Mr. McKay's estate improperly. However, he **does** expect to **see** the defense of alcoholism given its proper measure.

Consideration must be given to the following factors: (1) the **Respondent's** alcohol abuse, the proximate cause for his problem with The Bar, (2) his cooperation during the entire process, (3) the fact that restitution has been made to the **McKay Estate**, and **(4)** that no monies **were** missing from his trust accounts. **After** consideration, one must realize the sanction of disbarment for five years is too harsh. This is especially true if the alcohol defense removes the criminal aspects from the Referee's report.

After reviewing **the** proceeding and taking into account the medical evidence concerning the long-term effect of recovery from the disease, the Respondent thinks that a one year suspension would **be** nearer to the sanction that **this** Court should impose.

**THE COURSE OF
ALCOHOLISM
FOUR YEARS AFTER TREATMENT**

**J. MICHAEL POLICH
DAVID J. ARMOR
HARRIET B. BRAIKER**

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with life conditions among abusive drinkers. A more formal statement of the relationship between heavy alcohol consumption and general dissatisfaction is contained in the model of stressful life events. According to this view, alcoholism (or a marked increase in alcohol consumption) may develop during "crisis periods" when significant changes in an individual's life situation or social role lead to feelings of instability, confusion, and stress (Coleman, 1972). These crisis periods, characterized by the occurrence of one or more major life events that induce heightened stress, include, for example, death of a spouse, divorce, loss of employment, or serious personal illness. During such periods of increased stress, an individual's normal coping methods may prove inadequate, and he may resort to more extreme means of alleviating tension. The actual sedative effect of ethanol further increases the likelihood that some individuals may react to stressful life events through heavy consumption of alcohol.

To examine this notion, we asked our respondents to recall the occurrence of specific stressful life events during the 4 years preceding the interview. The percentage of respondents reporting each of five major stressful life events is shown in Table 4.6. The data are presented only for descriptive purposes, however, since the low base rates for single events and the absence of significant variation among the status categories preclude any formal test of the stressful life events model.

Column (6) of Table 4.6 shows the percentage of respondents who indicated that at least one of the five stressful life events had occurred during the period from 1973 to 1977. Although major stressful events appear to have occurred in the lives of most of our respondents (about 59 percent across the sample), there does not appear to be a systematic relationship between this occurrence and drinking status at 4 years. The lack of a relationship in these data between the objective occurrence of stressful events and problematic drinking behavior is further underscored by the data in Table 4.7. The percentages of respondents who reported symptoms or consequences at 4 years are shown in Table 4.7 according to whether or not they had experienced stressful life events, and as a function of their subjective life satisfaction. As can be seen from the data, the percentage of problematic drinkers increases markedly as a function of subjective dissatisfaction and remains largely unaffected by the occurrence of life stress.

EMOTIONAL ADJUSTMENT AND PERSONALITY

A substantial body of research has focused on identifying a consistent set of characteristics from the domains of personality and psychopathology that correlates with the development of and prognosis for alcoholism. Despite

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90 Psychosocial Functioning at Four Years

Table 4.7 Drinking Problem Rates as a Function of Stress and Life Satisfaction

Item	Subjective Satisfaction ^a		Subjective Dissatisfaction	
	No Stressful Events	One or More Stressful Events	No Stressful Events	One or More Stressful Events
Problem Rate ^b	49	42	82	72
(N)	(167)	(227)	(66)	(85)

^aFrom Question 12, Client Interview form, Appendix E; satisfaction defined as very happy or happy.

^bPercent of group reporting symptoms or consequences at 4 years.

substantial and careful efforts, however, most writers agree that no unique, premorbid alcoholic personality has been discovered. Indeed, given the multitude of factors that impinge on the alcoholism process, few current researchers still expect to find a unitary type of alcoholic personality (Orford, 1976). Moreover, personality research with alcoholic populations is often confounded by ambiguities surrounding the etiological status of observed traits: Do certain personality and/or psychopathological features, in fact, precede the development of alcoholic behavior? Or are they merely a consequence of the addiction that already exists?

A few general findings about the relationship between personality and alcoholism do emerge from the research literature. First, longitudinal studies, although relatively rare, seem to converge on a set of high-risk personality traits that may function as predisposing factors in alcoholic behavior. This "high risk" personality is profiled as unrestrained, impulsive, aggressive, and antiauthoritarian (Williams, 1976). Empirical evidence also suggests that alcoholics show a particular cluster of personality traits once their drinking has become established. Included in this cluster are low stress tolerance (Lisansky, 1960), dependency (Blane, 1968), perceptual dependence (Witkin et al., 1959), negative self-image, and feelings of isolation, insecurity, and depression (Irwin, 1968; Wood and Duffy, 1966). And, consistent with Jessor's (et al., 1968) view of alcoholism as a form of deviant behavior, Cahalan and Room (1974) found intrapunitiveness, impulsivity, and tolerance of deviant behavior other than drinking to be personality trait correlates of problem drinkers. Interestingly, the latter authors demonstrated that personality variables were the major determinants of tangible, adverse consequences from drinking, whereas sociocultural variables were better predictors of actual heavy consumption.

Several measures of psychiatric and emotional adjustment, as well as personality traits, are reviewed in the section below as correlates of the 4-year status categories. Although the causal relationships are ambiguous, certain

consistent features of emotional functioning and mental health do appear to be distinguishable among the 4-year followup groups.

Psychiatric Symptomatology

In a psychiatric sense, alcoholism is viewed as symptomatic behavior indicative of underlying pathological processes. The broad symptom complex of pathological personality traits associated with alcoholic behavior describes people with depressive, neurotic-depressive, sociopathic, and anxiety features (Hoffman, 1976). Because of its sedative effects, beverage alcohol may be used as a form of self-medication for particularly anxious individuals. Again, a mutual cause and effect relationship probably exists between alcohol consumption and psychiatric symptomatology, since prolonged drinking itself is known to produce feelings of anxiety and depression (Davis, 1971) and these feelings, in turn, precipitate further consumption. In advanced cases, heavy and continuous alcohol consumption may even result in chronic brain syndromes reflected by cognitive impairment, including disruptions in concentration and memory deficits.

The percentages of respondents, in each of the seven status categories, who reported relatively frequent occurrences of psychiatric symptoms during the 6-month period preceding the followup contact are given in Columns (1) to (5) of Table 4.8. Two measures of depression were obtained: The first, labeled "Depression" in the table, consists of frequent experiences of feeling "downhearted, blue, or depressed." As can readily be seen, Group 7 respondents were far more likely than others to report frequent depression symptomatology. The second indicator of depression, shown in Column (2) of Table 4.8, reflects "anhedonia," or the inability to experience satisfaction, pleasure, or enjoyment in daily experience; drinkers with symptoms and/or consequences were more likely to report frequent feelings of anhedonia.

Columns (3) and (4) of the table indicate, respectively, measures of general emotional stress or tension, and anxiety. The patterns here are similar, though not fully consistent. Drinkers experiencing adverse consequences (Groups 5 and 7) appear to be most affected by frequent tension or stress. Anxiety symptomatology (frequent occurrence of feeling "anxious, worried, or upset") was reported by 32 percent of Group 7 individuals, compared with more modest rates for the other status groups.

The fifth psychiatric symptom in the table is cognitive impairment, marked by problems in concentrating or remembering. The occurrence pattern is similar to those of the other symptoms, with Group 7 again significantly higher than the other groups. The absolute rates across the sample, however, are somewhat lower than for the other symptoms.

THIS DEPRESSION & the effects it has (CAN HAVE) ON IMPAIRED JUDGEMENT EXTENDS WELL INTO THE RECOVERY PHASE.

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Life Satisfaction

Subjective Dissatisfaction

No Stressful Events	One or More Stressful Events
82	72
(86)	(86)

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Table 4.1 Psychiatric Symptoms^a at Four Years, by Drinking Status at Four Years

Group No.	Drinking Status at 4 Years	Percent of Respondents Reporting Symptom as Occurring "All or Most of the Time"						(N)
		(1) Depression	(2) Anhedonia (lack of enjoyment)	(3) Tension/ Stress	(4) Anxiety	(5) Cognitive Impairment	(6) Frequent ^b "Escape" Drinking	
1	Abstained 1 year	9	14	14	10	4	—	(117)
2	Abstained 6-11 months	5	23	10	10	5	59	(38)
3	Low Q	5	12	10	7	2	10	(42)
4	High Q	12	18	21	9	2	23	(67)
5	Consequences	10	43	47	10	7	47	(28)
6	Symptoms	18	31	25	18	11	49	(67)
7	Symptoms and consequences	31	62	34	32	22	73	(196)
All respondents		24	18	32	11	19	42	(545)
General population ^c		2	4	4	(d)	(d)	(d)	(2235)

^aQuestions 55, 52, 53, 56, 54, respectively, Client Interview form, Appendix E.

^bRefers to "escape" reasons for drinking in past year: "I drink to forget my worries; I drink to relax; a drink helps cheer me up when I'm in a bad mood; a drink helps me when I'm depressed or nervous; I drink when I'm bored and have nothing to do; I drink to increase my self-confidence."

^cFrom Rand's National Health Insurance Study data collected on a general population (N = 2235) of males and females (mean age = 35) in Seattle, Washington.

^dNot available.

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a lcoholism

TREATMENT AND RECOVERY

Edited by Marshall J. Goby, PhD
With a Foreword by Robert M. Morse, MD

The Catholic Health Association of the United States
St. Louis

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Chapter 8

RECOVERY: A WAY OF LIFE

Miles Conway

15 Grapevine,
The A.A. Program
and Services, Inc.,

75), 7-10.

The concept of recovery as a way of life is not generally understood by those outside the alcoholism treatment field. Even some who work in alcoholism treatment and recovery do not totally accept it. Judge Learned Hand, the distinguished New York jurist, stated, "Freedom is most of all apparent when you are really not quite sure what it is." The same idea has its place in trying to fathom recovery in its many stages,

Recovery for the alcoholic's family members is even less understood. They generally must fend for themselves as the alcoholic's illness progresses. Only recently have treatment facilities begun to establish programs that help these individuals make a start on their own recovery. The alcoholic's relatives are given an opportunity to establish their own recovery completely independent of the alcoholic's recovery. Al-Anon represents the treatment of choice for family members, and good treatment centers work hard to prepare them for entrance into this fellowship.

Employers play a key role in the alcoholic's treatment. They can supply pressure that will motivate the alcoholic employee to seek treatment. Of course, no system is foolproof, and the nature of the illness and the many forms of recovery can abort even the best treatment plan.

Attaining sobriety entails abstaining from chemical use, picking up responsibilities, and changing attitudes. Sobriety is not mastered in a day, a month, or even years. That one starts on the journey is the important factor.

The family members and the alcoholic lack balance at the outset of recovery. Certain areas of life might come into focus, and others might remain particularly troublesome. Often there is a "flight into recovery" in which the alcoholic appears to have established himself or herself on firm ground. The alcoholic is then lulled into a false sense of security. At times of particular danger, the individual will need those special supports with which he or she has recently become acquainted. The trouble, however, is that some areas are not necessarily the ones one might expect. They have a way of presenting themselves at exactly the inopportune time.

Mr. Conway is program manager, Parkside Medical Services, Inc., Park Ridge, IL.

alcoholism: Treatment and Recovery

Even the **most** secure people have trouble **keeping in focus** their personal life, job requirements, physical health, and **spiritual** awareness. **The latter factor seems heady business to the sick alcoholic or family member**, Spirituality usually takes practical form In the beginning and matures with time.

SA The entire first year of recovery is mostly devoted to **physical changes**, the second year establishes **some emotional growth**, and during the third year maturity manifests itself. Spiritual recovery, which is **the biggest** part of recovery, surfaces throughout this period. For a time, **spirituality may be an idea clearly grasped**, but its understanding can **lose focus and leave one puzzled** and it is all **one can do to make it through oneday-at-a-time living**.

Acceptance of their own limitations is a new venture for most recovering persons. They **must break down their lives into manageable parts**. The arrogance and fierce independence that have been the lot of **most people die a slow death**, and it takes a program of daily living to **keep them in harmony with the recovering people who have become a part of their new life**. By daily living, one experiences an acceptance of what it is **one needs to do on a daily basis to sustain recovery**.

To offer a road map to people who have just begun the journey of recovery is simple enough, **but they will continuously need to adjust their living patterns**.

Good treatment centers offer lectures on subjects like **honesty, truth, communication, and surrender vs. compliance**. The frustration at being only a neophyte at living is apt to **send the newly recovering person into a type of blue funk**. This is not to say **goals should not be established**. But to **expect the recovering individual to accept all these new ideas as presented in the lectures is naive**, to say the least. Asking **the alcoholic to make drastic, immediate, and lasting changes** shows ignorance of the illness concept of alcoholism. New learning, difficult for everyone, is particularly **troublesome for the person trying to make a fresh new start**,

During recovery, other problems such as family difficulties, financial troubles, and employment adjustments will be identified. Generally, **the first three or four months of recovery** are especially trying. The alcoholic may **spend a great deal of time in the company of other sober alcoholics, and this may perplex or arouse jealousy in some family members**, in time, the alcoholic will return to the family circle.

Family members need to **make a serious commitment to look at their own lives and to start making some changes**. their recovery depends on willingness to see themselves as **profoundly affected by the illness and in need of help**. They will come to **learn a great deal about detachment, that is, the concept of loving the person but not allowing the person's alcoholic behavior to influence one's self**. No doubt their initial reaction to alcoholism will be confusion or outright contempt, but given time they can come to see **the necessity of loving the alcoholic and setting limits on how much they will accept given the reality of sick drinking**.

Recovery: A Way of Life

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The children of alcoholics display the full spectrum of attitudes to recovery. Some take a "wait and see" posture; they have seen the pain and destruction of family life and have withdrawn in an effort to survive. Given time, many will exhibit that spontaneous expression so characteristic of children. Many will extend a full measure of love; others will continue on an emotional roller coaster and will need some support.

Grown children who have already begun to make their own life styles might not return to the family circle. They blame the parents for the pain that has been inflicted and are unable to see that it is the alcoholism that has played the leading role in the disruption of the family unit.

One of the early pioneers in alcoholism treatment was fond of stating, "Alcoholics don't drink, they pathologically, sickly, massively use alcohol." Alcoholism greatly changes the life of each individual it touches. The massive denial experienced by alcoholics and their families renders them virtually powerless. Indeed, acceptance of this powerlessness represents the first step of the spiritual journey toward a new life. Whereas most people have valued independence and freedom, recovery requires a new faith grounded in the reality that doubt exists in all belief. Then an acceptance of self and of the self's limits can surface. Recovery involves focusing on spiritual progress rather than spiritual perfection.

Myths regarding alcoholism make it difficult for most people to see alcoholism for what it is. The anger, rejection, and pity one feels on seeing an intoxicated person represent only the tip of the iceberg of reactions to this state. The alcoholic seems to drink at precisely the wrong moments, and over many years friends and family develop resentment and bitterness. Recovery entails working through these feelings—a lifetime experience taken one day at a time. Possibly we are talking here less of making massive changes than of establishing a more comfortable feeling for oneself. A reasonable acceptance of one's own limitations seems the key. The most effective counselors in alcoholism encourage individuals to change themselves rather than to look for others to change. The latter course of action provides fertile ground for resentments and hard feelings.

Those most skilled at allowing growth have spoken well about the recovering individual doing the same things over and over until this new style becomes a kind of second nature. Harry M. Tiebout, MD, an early pioneer in alcoholism treatment, used to quote a colleague, Adolph Meyer: "Each individual is an experiment in nature, the product of life forces within and without." He viewed each individual as a newly evolved unit from which one could learn.

Learning how to get well represents a large part of the recovery process. This learning includes but is not limited to doing, reading, discussing, observing, thinking, and inspiring.

Doing refers to actions. Recovery requires not only good intentions and statements about change, but following through with the action that is the stuff on which change is built.

alcoholism: Treatment and Recovery

New ideas need to be learned, and setting aside time daily to do some reading can be beneficial. Some of the most practical literature comes from AA and Al-Anon. These accounts are based on millions of hours of experience and are not simply untested theories. The intellect is often given short thrift in a field heavily populated with behavioralists. But there is evidence that reading can be a powerful ally to the recovering alcoholic or family member.

Discussion can also play a major role. Everyone has experienced the urge to share an exciting occurrence with family or friends. Recovery is like a semaphore or Morse code in that one needs to gain familiarity with it. Discussing time-proven recovery formulas allows one to mark progress on the spiritual journey. Learning how to talk about recovery takes time and can have an intense excitement associated with it.

Another area of learning involves observation and imitation of others' behavior, especially those experiencing recovery. It is not so much what others say as it is their demeanor and attitude that is important. A simple smile, a casual remark, and companionship can edify one and instill the simple joy of living. Problems may press on all sides, but being with the "winners" for a time each day can be a profound experience.

Thinking allows one to discipline the mind and prevents the mindless drift that can lead to a "poor little old me" fantasy land. Often, the recovering alcoholic remembers only the good times associated with drinking and tends to forget the more numerous disastrous occasions. Family members also often block out the negative associations and try to grasp for the isolated nearly normal occasions. This "pressing" tips the scales and begins to sour all one's thinking. Feelings are not handled, and resentments begin to grow into lingering hatred of self and of others.

Finally, in the area of new learning, inspiration is a big factor. Not only the lofty religious or philosophical experience, but also the common everyday happening can lift one's spirits. The change of seasons, a small gift (at times purchased for oneself), attending a child's recital or play, going to the library to select a few books, and other commonplace events can inspire one and promote a quiet acceptance of one's new life of recovery. A whole new outlook can begin to emerge from this type of learning, and the newly established recovery begins to take over in every area of one's life.

* The issue of time and its relationship to recovery needs further amplification. The progression into recovery seems to run on a strange timetable. Some areas in a person's life will show improvement, but as one becomes more deeply involved in a life-style adjustment, more seems to be demanded. Without massive support from others, the individual would be virtually helpless.

It appears that the more one is hurting, the better are his or her chances at establishing recovery. Simple logic has a way of running out, and cause-and-effect type thinking represents short-range stopgaps in this area. Veterans at the business of daily living are aware that one is given a daily reprieve. For the

Recovery: A Way of Life

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alcoholic, this means asking for help and not taking that first drink one day at a time. Recovering family members also ask for help and practice detachment in their dally living, These simple concepts are packed wth profound meaning.

Alcoholism should be treated in its uniqueness, The physical and emotional damage can reverse over time, The spiritual component, however, represents the prime factor, and growth in this area will determine one's recovery.

The most natural thing for an alcoholic to do is drink. Indeed, without daily asking for help and seeking out the company of other sober alcoholics, the alcoholic will likely revert to using alcohol. Alcoholics who return to alcohol use need a fresh start. No one blames them. In fact, the relapse can show them that the ground on which they built their sobriety was spongy at best.

The reality of relapse is a built-in teaching factor for sober alcoholics. They can see the results of haphazard living and of not "paying one's dues" with regular attendance at AA meetings and daily attention to one's spiritual life. Massive support is extended to alcoholics who resume drinking if only they seek help—or at times help may be pressed on them, This latter course can have surprising results, but takes careful timing by Its author,

Family members can also experience an emotional lapse, which can be every bit as painful as the "fall from grace" experienced by the alcoholic. At times, family members and friends who are charting a new course for themselves find themselves in their own "Sargasso Sea," bogged down with feelings of anger, resentment, rage, and self-pity. In a sense, these occasions are dry spells, similar to the "dry drunk" experienced by the alcoholic. They call for the individuals to make more careful accounting of their commitment to their awn recovery. Possibly the Higher Power withdraws just far enough to allow the individuals to fall back on their own reserves, which can lead to a disastrous end or to recommitment and growth.

it has been said that alcoholism is a relapse illness. Unquestionably, some recovering alcoholics return to drinking or undergo painful emotional lapses. But thousands have established a life of recovery filled with joy, contentment, and security.

finally, it seems clear that most people view their recovery as a gift. As with most gifts, the recipient has a need to express gratitude. For many, prayer life is a way of demonstrating gratitude. This expression may be informal and Infrequent at the start; intime, it can become profound and will be done on a more regular basis.

These prayers are not necessarily the formal prayers of one's youth. Rather, they may be more expressive of gratitude for recovery or the asking for strength to do that which needs to be done to maintain recovery,

One may also begin to use meditation. This may be foreign in the beginning but, with practice, the recovering individual can reach those plateaus so necessary to sustain the spiritual life.

Alcohol Related Problems

Room for Manoeuvre

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Brain Damage in Chronic Alcoholism

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Alcohol is capable of affecting the brain during acute and chronic stages of abuse. Alcoholic intoxication and losses of memory, the so-called alcoholic blackouts, are commonly observed when alcohol reaches high concentrations in blood. The reverse of this picture is the syndrome of alcohol withdrawal with its multiple manifestations from mild tremulousness to the full syndrome of delirium tremens. In chronic alcoholism, together with those symptoms of intoxication and withdrawal, a number of more or less permanent organic psychosyndromes can appear. The status of and relationship between these syndromes of alcohol related brain damage have been the object of a great deal of interest during the last few years and this paper will be devoted to reviewing some of the more salient points in this area.

Little is known about the frequency of these syndromes in clinical practice. Careful surveys are scarce and it would appear likely that many of those who suffer from alcohol related brain damage may remain in 'skid-row' and never reach the hospital. Horvath (1975) in a study in An Australian centre for treatment and research into alcoholism, found that 9 per cent of 1100 patients seen over the course of five years had a chronic organic brain syndrome. The clinical manifestations of this syndrome were progressive failure of memory, loss of intellectual ability, and personality deterioration. Of these 100 patients, 20 suffered from typical Korsakoff psychosis following Wernicke's encephalopathy; the rest presented with a combination of memory failure, behavioural disturbances and in some cases focal neurological features such as visuo-spatial and language deficits, Horvath did not mention in his study the more subtle and perhaps more frequent abnormalities that can be encountered in the alcoholic who appears to be 'intact' on routine clinical examination.

THE SEVERE ALCOHOL RELATED PSYCHOSYNDROMES

Wernicke's encephalopathy was first described in 1881 in two alcoholics and in a patient suffering from persistent vomiting, Wernicke described the classical triad of impaired consciousness, ataxia, and ophthalmoplegia. This combination

of symptoms is not always present and more recent studies (Cravioto *et al.*, 1961) have expressed doubts as to the validity of using such criteria for diagnostic purposes. The syndrome is often clinically undiagnosed and only discovered when a post-mortem examination is performed.

A few years later Korsakoff described the syndrome of amnesia that bears his name. The link between these two conditions is now clearly and firmly established and the clinical manifestations merge. The initial confused state is often followed by the typical amnesic syndrome with occasional confabulation. The neuropathological lesions affecting the periventricular grey matter around the third and fourth ventricles and aqueduct are identical in both conditions, and the role of thiamine deficiency in their causation has been clearly demonstrated. In typical cases of what could be called the Wernicke-Korsakoff syndrome the prognosis is poor. Cutting (1978) found that only 14 per cent were capable of independent existence outside the hospital. His sample contained a greater number of males, but when it occurred in females the drinking history tended to be shorter.

Diagnosis of the full blown picture of the Wernicke-Korsakoff syndrome is not a difficult clinical exercise, but recent studies suggest that in a substantial proportion of cases, perhaps with different or less severe symptoms, the diagnosis is often missed. Cravioto *et al.* (1961) studied the brains of 1600 consecutive patients coming to post-mortem. The typical lesions of Wernicke's encephalopathy were present in 28 cases (just under 2 per cent). In many of them the diagnosis had not been made clinically, although all except one were known to be chronic alcoholics. In a more recent study Harper (1979) found these lesions in 1.7 per cent of another large autopsy series with only a minority correctly diagnosed before death. Of special relevance is the fact that some of the lesions were acute, but others were chronic or acute-on-chronic indicating that repeated insults had occurred in the same patient at different times.

The syndrome of 'alcoholic dementia' is much less clearly defined. It was often used as a diagnostic label before Korsakoff psychosis was widely recognized, and although the term is still in use it is a somewhat elusive concept. Alcoholism has been suspected as the cause of dementia in a number of careful clinical studies in patients diagnosed as suffering from presenile dementia and in whom other possible aetiologies had been carefully excluded (Marsden and Harrison, 1972; Smith *et al.*, 1976). In these reports 'alcoholic dementia' appears to be almost as frequent as multi-infarct dementia. The work of Cutting (1978) has been of particular interest in clarifying some of the nosological problems of 'alcoholic dementia'. Among the patients suffering from alcohol related psychoses admitted to the Maudsley and Bethlem Royal Hospitals he found a group suffering from a clinical syndrome similar to that of Korsakoff psychosis which had appeared insidiously without the preceding Wernicke's encephalopathy. The label of 'alcoholic dementia' had been applied to the majority of these patients. When compared with the typical Wernicke-Korsakoff

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psychosis the slow onset Korsakoff or 'alcoholic dementia' was commoner in older individuals and in women. The drinking histories of these patients were also comparatively longer. The prognosis was better in this group and two-thirds of the patients were capable of independent lives outside the hospital.

In conclusion, given the evidence coming from recent studies, it is tempting to postulate that the extent and severity of the neuropathological lesions have to reach a certain threshold before clinical manifestations become apparent: the mode in which this threshold is crossed, abruptly or insidiously, could determine some of the clinical manifestations of the ensuing psychosyndromes.

THE 'INTACT' ALCOHOLIC

The majority of patients admitted to an alcoholic unit do not present with severe signs of brain damage, and when submitted to a routine clinical examination they appear to be well preserved. After a period of detoxification and treatment such patients are discharged without further investigation. Even in this group of patients the prognosis remains poor. Relapses into drinking are extremely frequent and many of them follow a steady downhill course. The presence of subtle but nevertheless important psychological and morphological brain changes could potentiate addiction and interfere with rehabilitation. This area has been previously reviewed elsewhere (Ron, 1977) and at this point only the more relevant points will be mentioned. These patients tend to have IQ's within the normal range, although this does not rule out the possibility that some degree of deterioration could have taken place. Memory deficits are present during the early stages of withdrawal and often improve with abstinence, but deficits in abstract thinking, problem solving ability, and psychomotor speed can still be detected even after prolonged periods of abstinence.

The clinical manifestations that accompany the cognitive deficits are not immediately obvious in most cases, but it is possible that some of the features traditionally associated with chronic alcoholism, such as inability to abstain and tendency to relapse into drinking, may be closely related to intellectual deficits.

Neuropathological lesions have been described in these patients. Courville (1955) found that cortical atrophy and ventricular enlargement were common. The histological lesions were widespread, but affected the frontal lobes more markedly. Microscopically there was loss of neurones and glial proliferation. Courville considered alcoholism to be the commonest cause of cerebral atrophy in those aged between 40 and 60. Cortical atrophy has also been described in patients with the Wernicke-Korsakoff syndrome (Victor *et al.*, 1971); this illustrates the overlap between the types of neuropathological lesions of chronic alcoholism.

In recent years, with the advent of computerized tomography (CTscan), the study of the morphological changes in the brains of alcoholic patients has received a new impetus. Previous studies using pneumoencephalography had

demonstrated the presence of cerebral atrophy in a substantial proportion of alcoholic subjects. Brewer and Perrett (1971) were able to demonstrate it in 31 of 33 alcoholics. Nevertheless, the morbidity and discomfort of the technique made it impossible to study large, carefully selected groups of alcoholics and the findings could not be extrapolated to the alcoholic population as a whole.

Several studies using CT scans have been published (Bergman *et al.*, 1981; Wilkinson and Carlen, 1981) with approximately similar results. In the last three years a study using CT scans has been under way at the Institute of Psychiatry. The results of this study have been more fully reported elsewhere (Ran *et al.*, 1981), but it is worth mentioning the more salient points here. One hundred consecutive male admissions to an alcoholic unit were investigated. None of the patients suffered from brain damage detectable on routine clinical testing, and in particular clinically diagnosed cases of Wernicke-Korsakoff psychosis were not included. Other possible causes of brain damage were excluded. All patients had been abstinent for at least two weeks before scanning (average 34 days), and their ages ranged from 22 to 62 years (mean 43.5). The average duration of the drinking history, defined as the number of years the subject drank in excess of 150 g of alcohol per day several times a week, was 17.3 years. A group of 80 age-matched normal volunteers who were lifelong abstainers or very light drinkers were used for comparison. A battery of psychological tests was administered to all subjects.

The CT scans were assessed as follows: the degree of sulcal widening was graded on a 4-point scale judging each scan against prearranged criteria. Sylvian fissure and interhemispheric fissure widening were graded in the same way on a 3-point and 2-point scale respectively. Ventricular size was assessed by measuring the area of ventricle within a given slice by means of a planimeter and expressing it as a percentage of the total intracranial area in that slice (ventricle/brain ratio, V/B).

The differences between patients and controls were highly significant for all the indices (see Table 1).

In the alcoholic group all the neuroradiological anomalies were related to age. The duration of the drinking history, age of onset, and quantity of alcohol consumed did not correlate with the severity of the radiological abnormalities.

A more interesting finding was the negative correlation between the length of the period of abstinence prior to scanning and the degree of widening of the sulci and sylvian fissure. A similar trend was also found between ventricular size and the duration of abstinence in the previous year. These correlations suggest that a certain degree of recovery takes place with abstinence and this process of reversibility appears to take longer in older subjects. A follow-up of this initial sample is under way in an attempt to clarify the natural history of these abnormalities. So far these results lend some support to the findings of Carlen *et al.* (1978) who reported improvement of CT scan abnormalities

Alcohol Related Problems

substantial proportion of subjects demonstrate it in 31 percent of the technique groups of alcoholics alcoholic population

Bergman *et al.*, 1981; results. In the last three years at the Institute of Psychiatry. elsewhere (Ron *et al.*, 1981) as here. One hundred subjects were investigated. None of the subjects had a history of routine clinical testing, or Wernicke-Korsakoff psychosis or dementia were excluded. All subjects were scanned (average 34 years of age, range 21-63.5). The average age of years the subject was scanned, was 17.3 years. All subjects were lifelong abstainers or controls. All subjects completed psychological tests

of ventricular widening was assessed using the following criteria. Sylvian fissure was assessed in the same way on each slice. Size was assessed by means of a planimeter measuring the total area in that slice

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ventricular abnormalities were related to the total quantity of alcohol consumed and to radiological abnormalities. There was a positive correlation between the length of alcohol abuse and the degree of widening of the ventricles and between ventricular widening and the degree of brain atrophy. These correlations were also seen in subjects with abstinence and this was confirmed in a follow-up study of 100 subjects. A follow-up study to verify the natural history of alcohol related brain damage and support to the findings of CT scan abnormalities

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Table 1 CT scan Indices in Alcoholics and Controls (The Institute of Psychiatry Study)

	Alcoholics (N = 100)	Controls (N = 50)	Sig
V/B	9.6(3.0)	6.2(2.0)	**
Sulcal width	(N = 99)	(N = 50)	
Grade 0	32	31	
1	24	11	
2	29	2	***
3	14	0	
Sylvian fissure width	(N = 100)		
Grade 0	51	44	
1	35	6	***
2	14	0	
Interhemispheric fissure			
Grade 0	71	46	
1	29	4	**
Cerebellar sulci			
Grade 0	90	50	
1	10	0	*

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$
V/B = Ventricle/brain ratio.

In four of six abstinent alcoholics re-scanned from 33 to 97 weeks after the initial examination,

There were also extensive differences between the two groups in psychological test performance. Alcoholics obtained lower scores than controls in tests of memory and tests of abstracting ability. The correlation between radiological abnormalities and intellectual impairment was not close, but it is worth noting that in those patients with larger ventricular systems the degree of verbal/performance discrepancy in IQ tests was more pronounced and their verbal learning was also more impaired.

SUMMARY AND CONCLUSIONS

Alcohol related brain damage can be detected in a large number of chronic alcoholics. In some cases its presence is clinically obvious, while in others subtle changes can be discovered only after careful search. Even in those cases where the impairment is mild the abnormalities may play an important role and contribute to the poor prognosis.

The different syndromes of alcohol related brain damage are probably part of a continuum which encompasses the acute Wernicke-Korsakoff syndrome,

the so-called 'alcoholic dementia', and the more subtle deficits encountered in a large proportion of chronic alcoholics, The aetiology of many of these deficits is likely to be multifactorial so that in a given patient a variety of factors could be at play, Among such factors, besides the action of alcohol and its metabolites and the accompanying dietary deficiencies, it is worth mentioning the possible role of head injury, and of metabolic disturbances, including hypoglycaemia, often present during the acute stages of intoxication,

Of special interest is the fact that some of the abnormalities detected appear to be, at least in part, reversible with abstinence. Improvement takes place over long periods of time, and it is unlikely to be exclusively due to deranged water and electrolysis balance. Other mechanisms, still uncertain, need to be invoked.

Our knowledge of the effects of alcohol abuse has increased in the last few years, but there remain more questions to be answered. The paper has dealt exclusively with subjects who suffered from alcohol related deficits but it is a well-known fact that many alcoholics with similar histories appear to escape damage. The study of protective factors is a potentially rewarding area. Finally, little is known about the comparative susceptibility of women to alcohol related brain damage. The possibility that a greater vulnerability could be present, as appears to be the case for liver damage, is worth further consideration.

ACKNOWLEDGEMENTS

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REFERENCES

- Bergman, H., Borg, S., Hindmarsh, T., Idestrom, C-M., and Mutzell, S. (1981). Computed tomography of the brain: a neuropsychological assessment of male alcoholic patients and a random sample from the general male population, *Acta Psychiat. Scand.* Suppl. 286, 62, 47-56.
- Brewer, C. and Perrett, L. (1971). Brain damage due to alcohol consumption: an alicephalographic, psychometric and electroencephalographic study. *Br. J. Addict.*, 66, 170-182.
- Carlen, P. L., Wortzman, G., Holgate, R. C., Wilkinson, D. A., and Rankin, J. G. (1978). Reversible cerebral atrophy in recently abstinent chronic alcoholics measured by computed tomographic scans. *Science*, 200, 1076-1078.
- Courville, C. B. (1955). *The Effects of Alcohol on the Nervous System of Man*. San Lucas Press, Los Angeles.
- Cravito, H., Korein, J. and Silberman, J. (1961). Wernicke's encephalopathy: a clinical and pathological study of 28 autopsied cases. *Arch. Neurol.*, 4, 510-519.
- Cutting, J. (1978). The relationship between Korsakoff's syndrome and 'Alcoholic dementia'. *Br. J. Psychiatry*, 132, 240-251.
- Harper, C. (1979). Wernicke's encephalopathy: a more common disease than realised. A neuropathological study of 51 cases. *J. Neurol., Neurosurg. Psychiat.*, 42, 226-231,

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Brain Damage in Chronic Alcoholism

249

- Horvath, T. B. (1975). Clinical spectrum and epidemiological features of alcoholic dementia. In: J. G. Rankin (ed.), *Alcohol, Drugs and Brain Damage*. Addiction Research Foundation of Ontario, Toronto.
- Marsden, C. D. and Harrison, M. T. G. (1972). Outcome of investigation of patients with presenile dementia. *Br. Med. J.*, 2, 249-252.
- Ron, M. A. (1977). Brain damage in chronic alcoholism: a neuropathological, neuroradiological and psychological review. *Psycholog. Med.*, 7, 103-112.
- Ron, M. A., Acker, W. and Lishman, W. A. (1981). Morphological abnormalities in the brains of chronic alcoholics—a clinical, psychological and computerized axial tomographic study. *Acta Psychiat. Scand.*, Suppl. 286, 62, 41-46.
- Smith, S. J., Kiloh, L. G., Ratnavale, G. S. and Grant, D. A. (1976). The investigation of dementia. The results in 100 consecutive admissions. *Med. J. Australia*, 2, 403-405.
- Victor, M., Adams, R. C., and Collins, G. H. (1971). *The Wernicke-Korsakoff Syndrome*. Blackwell's Scientific Publications, Oxford.
- Wernicke, C. (1881). *Lehrbuch der Gehirnkrankheiten, Part 2*. Kassel, Berlin. p.229.
- Wilkinson, D. A. and Carlen, P. L. (1981). Relationship of neuropsychological test performance to brain morphology in amnesic and non-amnesic chronic alcoholics. *Acta Psychiat. Scand.*, Suppl. 286, 62, 89-101.

Cognitive Loss and Recovery in Long-term Alcohol Abusers

Isaac Brandt, PhD; Nelson Butkrr, PhD; Christopher Ryan, PhD; Roger Bayog, MD

The nature of the memory and visuospatial defects associated with chronic alcoholism, and the recovery of these functions, were investigated in a large group of alcoholic men and well-matched nonalcoholic controls. Both young and old alcoholics displayed significant impairments on tasks requiring the learning of novel associations and the holding of information in memory over longer delay intervals. The recovery of cognitive skills was found to depend on the length of abstinence and the particular behavioral functions examined. Whereas psychomotor skills and short-term memory improved significantly with prolonged abstinence, long-term memory was impaired even after seven years of continuous sobriety. We propose that recovery of short-term memory reflects reestablishment of cortical functioning, while the persistent long-term memory defect indicates more permanent damage to diencephalic structures.

(Arch Gen Psychiatry 1983;40:435-442)

The information-processing deficits associated with alcoholic Korsakoff's syndrome have been extensively studied and are well established. These patients display profound anterograde and retrograde amnesia as well as a variety of visuospatial and abstract-conceptual difficulties, all in the face of normal performance on intelligence tests.¹ Traditional neurological teaching maintains that the memory disorder of Korsakoff's syndrome emerges only after the acute Wernicke's stage of the illness has been resolved with the administration of thiamine.^{2,3} It has recently been suggested, however, that the cognitive and neuropathological changes associated with Korsakoff's disease develop gradually during many years of alcohol abuse and are related to the neurotoxic effects of alcohol as well as to nutritional deficiencies. Ryback,⁴ in particular, has sug-

gested that a "continuum of cognitive impairment" may exist in which the long-term alcohol abuser lies somewhere between the patient with Korsakoff's syndrome and the social drinker in memory, perceptual, and problem-solving capacity. As a consequence of this proposal, several groups of investigators have examined whether cognitive defects similar to those seen in patients with Korsakoff's syndrome exist in an attenuated form in the long-term alcoholics without Korsakoff's syndrome.

Many of the initial studies assessing the long-term alcoholic's learning and retentive capacities were unable to detect significant differences between abusive drinkers and their nonalcoholic peers.^{5,6} Since several of these studies relied heavily on standard clinical tests that may not have been sensitive enough to the subtle but real memory defects of "intact" alcoholics, Ryan and his collaborators⁷ developed a battery of challenging memory tests specifically designed to uncover mild to moderate impairments. In their initial investigation, Ryan et al. administered their tests to a group of long-term alcoholic men (with at least ten-year histories of alcohol abuse and a minimum of four weeks of abstinence) and to a carefully matched group of nonalcoholic controls. The results indicated that alcoholics perform significantly more poorly on verbal and nonverbal paired-associate learning tasks and on a verbal short-term memory test. (Throughout this article, "short-term memory" (STM) refers to the system that can maintain information for only a few seconds in the absence of rehearsal. In neuropsychological research, it is often assessed by interposing distractor activity between stimulus presentation and recall. Short-term memory is thus distinguished from both "immediate memory span," (which requires continuous focused attention and is rarely impaired even in frankly amnesic patients,) and "long-term memory," a more permanent store that requires the establishment of associative links between items. The alcoholics were also impaired on timed digit-symbol and symbol-digit substitution tests, which require the integrity of visuosperceptual, associative, and psychomotor functions, and are therefore very sensitive indicators of impaired cognitive functioning.^{8,9} Subsequent studies by Ryan and Butters¹⁰ have confirmed the existence of memory impairments in detoxified alcoholics and

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From the Psychology Service, Boston Veterans Administration Medical Center (Drs Brandt, Butters, and Ryan); the Department of Neurology, Boston University School of Medicine (Drs Brandt, Butters, and Ryan); and the Department of Psychiatry, Brockton (Mass) VA Medical Center (Dr Bayog). Dr Brandt is now with the Department of Psychology, The Johns Hopkins University, Baltimore.

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have suggested that alcoholics, depending on their patterns of alcohol abuse, may occupy different positions along the continuum of cognitive impairment proposed by Ryback.⁴ The recent reports^{11,12} that conceptual and mnemonic performance is also correlated with specific patterns of social drinking further strengthens the proposal that alcohol's effect on the nervous system is insidious and progressive.

It has been noted that the cognitive deficits associated with long-term alcohol abuse appear similar, in some respects, to those impairments commonly associated with normal and abnormal aging.^{13,14} That is, declines in memory, visuosperceptual, and conceptual processes often accompany normal senescence and are very conspicuous in the senile form of Alzheimer's disease. These behavioral parallels have led to several research efforts to specify the exact nature of the interaction between alcohol abuse and aging with regard to various aspects of intellectual functioning.¹⁵ Several studies have demonstrated that while older alcoholics (>50 years of age) are impaired relative to their nondrinking peers, no such relationship exists for younger alcoholics.^{16,17} Jones and Parsons¹⁶ and Klisz and Parsons¹⁷ have interpreted this interaction between age and alcoholism as indicating that the aging brain is more vulnerable to the toxic effects of alcohol. An alternative explanation, that chronic abuse of alcohol accelerates normal age-related declines in cognitive functioning,¹⁸ found support in a study by Ryan and Butters.¹⁹ They reported that both young and old alcoholics display poorer memory abilities than normal alcoholics of the same ages. Furthermore, the performance of the 84- to 49-year-old alcoholics in their study was virtually identical to that of 50- to 59-year-old non-alcoholics. Likewise, 50- to 59-year-old alcoholics performed as did 60- to 65-year-old normal subjects. While acknowledging alternative explanations of their results Ryan and Butters¹⁹ suggested that alcohol abusers display age-related cognitive losses five to ten years earlier than normal individuals.

The present investigation was undertaken to answer two questions. The first issue (study 1) is whether both young and old alcoholics display significant memory and perceptual deficits when compared with nonalcoholics. Ryan and Butters¹⁹ results were based on a relatively small sample of patients; subsequent to the publication of that article, many additional alcoholic patients have been evaluated with their test battery. The second question (study 2) concerns the effect of prolonged abstinence on the recovery of cognitive functioning in alcoholics. Previous studies that have demonstrated significant recovery^{20,21} have done so over a relatively short time course, usually less than one year of continuous abstinence. The typical finding in these studies is that neuropsychological status improves dramatically in the first few weeks of detoxification, but following this initial improvement, recovery is modest, if it occurs at all.²² For example, Ryan and his colleagues,²³ using a cross-sectional experimental design to guard against practice effects due to repeated testing, assessed the memory and visuosperceptual abilities of short-term (one to three months) and long-term (one to five years) abstinent alcoholics with equivalent drinking histories. While partial recovery was found on digit-symbol substitution tasks, the two abstinent groups did not differ on paired-associate or STM tasks. In the present study, a group of alcoholics abstinent for five years or more was examined to determine whether any recovery of learning and memory capacities can be detected after extensive alcohol-free periods. Any evidence that prolonged abstinence is rewarded by substantial cognitive recovery has obvious clinical significance.

Table 1.—Characteristics of Subject Groups in Study 1 (Mean ± SD)

	Alcoholics (N=194)	Controls (N=78)
Age, yr	48.30 ± 7.93	49.75 ± 10.48
Education, yr	11.94 ± 2.22	12.12 ± 1.24
Wechsler Adult Intelligence Scale vocabulary score	11.89 ± 2.18	12.84 ± 1.97*
Alcoholism, yr	21.12 ± 9.24	...
Abstinence, mo	7.89 ± 10.23	...

STUDY 1

Subjects and Methods

A group of 194 male alcoholics and an age- and education-matched group of 76 nonalcoholic men participated in this study. All subjects were white, working-class men between the ages of 80 and 60. Extensive medical histories were taken before testing; potential subjects were excluded if they had histories of hepatic cirrhosis, severe head trauma, psychosis, neurological disease, polydrug abuse, or learning disabilities. Individuals with histories of severe affective illness, necessitating psychiatric hospitalization and/or electroconvulsive therapy, were likewise excluded. No subject was included who, at the time of testing, was taking psychotropic medication other than minor tranquilizers. All the subjects in this study had scaled scores of at least seven on the vocabulary subtest of the Wechsler Adult Intelligence Scale (WAIS).

The alcoholic subjects were selected from among the outpatient populations of local Veterans Administration treatment facilities. All had at least ten-year histories of daily alcohol consumption and had been abstinent at least one month (but no more than 59 months) at the time of testing. Drinking histories disclosed that all alcoholic subjects consumed a minimum of six drinks (approximately 85 mL of ethanol) and an average of 12 drinks (approximately 170 mL of ethanol) per day. The nonalcoholic control subjects were recruited primarily from newspaper advertisements. Drinking histories indicated that all of these individuals were light social drinkers. None had been treated for alcoholism or alcohol-related medical disorders. The alcoholic and non-alcoholic subjects did not differ in age or number of years of education (Table 1). There was, however, a small but statistically significant difference in WAIS vocabulary scaled scores. Since the vocabulary score correlates highly with performance on many memory and perceptual tasks, this factor was statistically partialled out in all analyses of the data.

The two subject groups (alcoholics and nonalcoholics) were further divided into two age groups. "Young" subjects were those under age 50, while those 60 years old and over were classified as "old" subjects. This breakdown resulted in 71 young alcoholics (mean age, 42.15 years), 29 young controls (mean age, 39.56 years), 53 old alcoholics (mean age, 62.11 years), and 47 old controls (mean age, 57.75 years). The two young subgroups did not differ from the two old subgroups in educational background or WAIS vocabulary score. While the young alcoholics did not differ from the old alcoholics in education, vocabulary score, number of drinks per day, or length of abstinence at time of testing, they did have significantly shorter histories of alcohol abuse (20.36 years for the young alcoholics, 45.81 years for the old alcoholics; $t=4.48$, $df=182$, $P<.001$).

Tests.—**Four-Word STM Test.**—This verbal STM test uses the Brown-Peterson distractor technique.²⁴ The subject is read four unrelated monosyllabic or bisyllabic words at the rate of one word per second. Following presentation of the fourth word the examiner reads aloud a three-digit number, and the subject must count backwards by threes from that number until the examiner tells him to stop. At that time, the subject attempts recall of the four words. For ten trials, the subject counts backwards for 15 s before attempting recall; for ten trials, the counting procedure

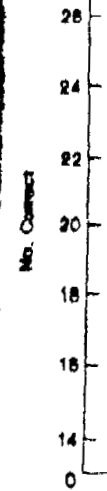


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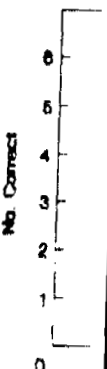


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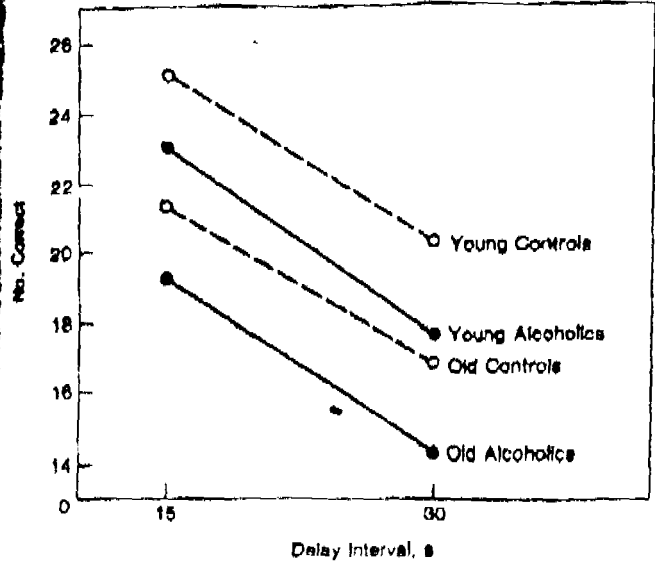


Fig 1.—Performance of "young" and "old" alcoholic and non-alcoholic subjects on Four-Word Short-term Memory Test.

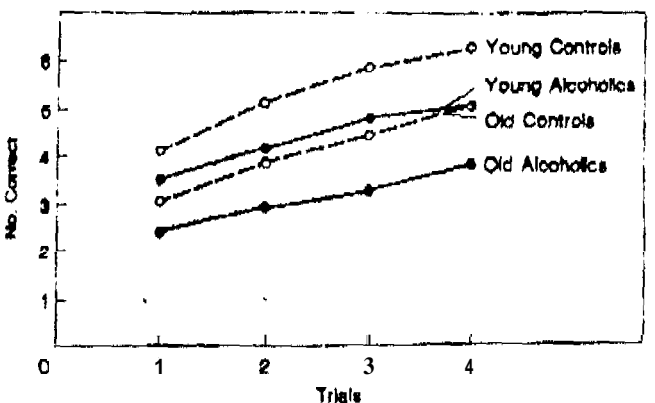


Fig 2.—Learning curves of "young" and "old" alcoholic and nonalcoholic on Symbol-Digit Paired-Associate Learning Test.

continuer for 30 s. The 15- and 30-s delay trials are presented in unpredictable order.

Symbol-Digit Paired-Associate Learning Test.—This test of new learning was first used by Kapur and Butters.¹¹ It requires the subject to learn associations between visually presented, abstract, meaningless symbols and single digits. The seven symbol-digit pairs are each printed on a separate card. The seven cards are initially exposed for study for 8 s each. Then, each symbol alone is shown to the subject, whose task is to recall the number that was paired with it. Each response is followed by presentation of the original symbol-digit card. The seven-item tort is reported four times to assess the extent to which subjects are able to benefit from feedback and practice.

Substitution Tests.—These two timed coding tests are modeled after the digit-symbol subtest of the WAIS. In the Digit Symbol Substitution Test (DSST),¹² the subject is presented with nine digit-symbol pairs and rows of digits with empty boxes beneath them. His task is to draw the appropriate symbol in each box by referring to the code. The subject's score is the number of symbols correctly drawn in 90 s. In the Symbol-Digit Substitution Test (SDST), the relationship between the digits and symbols is reversed, and a new set of symbols is used. The number of digits correctly written in 90 s yields the subject's score.

Benton Visual Retention Test (BVRT).—The multiple-choice

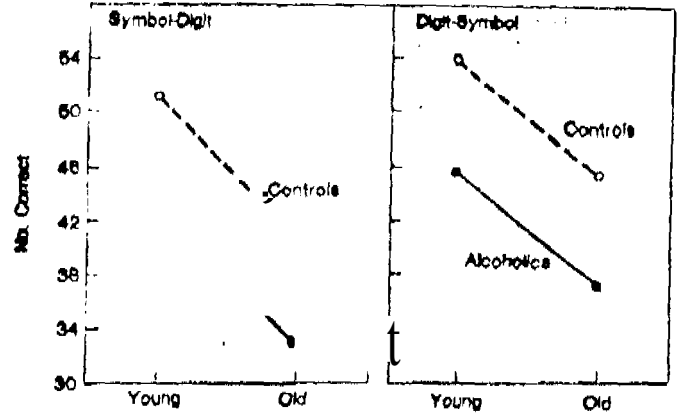


Fig 3.—Performance of "young" and "old" alcoholics and non-alcoholics on two substitution tests.

version (forms F and G) of the BVRT¹³ was used to examine nonverbal STM. Each of the 15 stimulus cards of form F is presented for 5 s and is immediately followed by a four-choice response card. For form G, the stimulus exposure is increased to 10 s, but presentation of the response card is delayed for 15 s. The subject simply sits quietly during the delay interval.

Embedded Figures.—In this multiple-choice test, developed by Kapur and Butters,¹⁴ the subject is shown 20 cards, each containing four complex line configurations and a simpler geometric nonsense figure that is "hidden" in one of the four designs. The subject's task is to point to the design that contains the hidden figure. Sixty seconds are allowed for each response. The number of correct responses yields the subject's score.

Procedure.—Subjects were tested individually in a quiet room. Testing was typically conducted in one session with rest periods interspersed as needed. The order of the tests was the same for all subjects. Because of time constraints and patient fatigue, some subjects were administered abbreviated batteries. The resulting changes in sample size are reflected in the *d*'s for the statistical tests.

Results

Test results were analyzed with 2 x 2 analyses of covariance (group by age), with WAIS vocabulary scaled scores (age corrected) as the covariate. In all of the figures, kit scores are corrected for the covariate.

For the Four-Word STM Test (Fig 1), separate analyses were performed for the 15-s delay, the 30-s delay, and the total score. The main effect for *p* was not significant at the 15-s delay, but was significant at the 30-s delay ($F = 4.19; df = 1/199; P < .05$) and for the total score ($F = 4.89; df = 1/199; P < .05$). The age factor for all three scores was highly significant ($P < .005$), and there were no significant group-by-age interactions. Newman-Keuls Multiple Range Tests were performed on the total scores to determine whether both young and old alcoholics were impaired on this task relative to age-matched controls. Both comparisons yielded significant results ($P < .05$).

The performance of the four subject groups on the Symbol-Digit Paired-Associate Learning Test is shown in Fig 2. As in previous studies with this test, the difference in performance between alcoholic and nonalcoholic subjects was highly significant ($F = 18.05; df = 1/205; P < .001$). There was also a large difference between the performance of young and old subjects ($F = 81.94; df = 1/205; P < .001$). The age-by-group interaction was not significant. Post hoc comparisons with the Newman-Keuls test demonstrated that both the young and old alcoholics were impaired relative to their nonalcoholic peers ($P < .01$).

A significant main effect for group was also obtained on the two substitution tests (Fig 3). Alcoholics consistently performed more poorly than nonalcoholics ($F = 29.17; df = 1/205; P < .001$ for SDST; $F = 27.69; df = 1/205; P < .001$ for DSST). These tasks were also

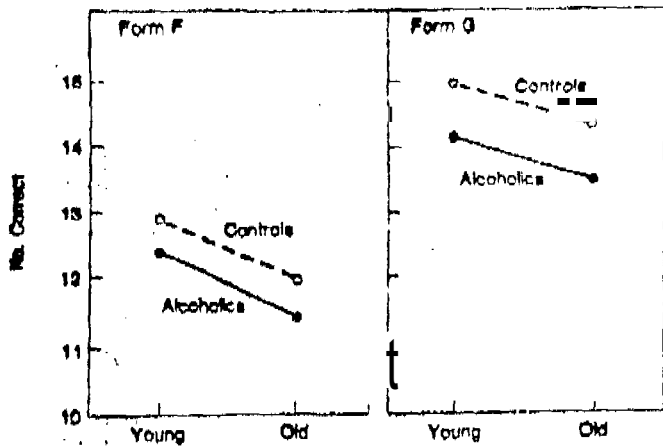


Fig 4.—Performance of "young" and "old" alcoholics and control subjects on Benton Visual Retention Test.

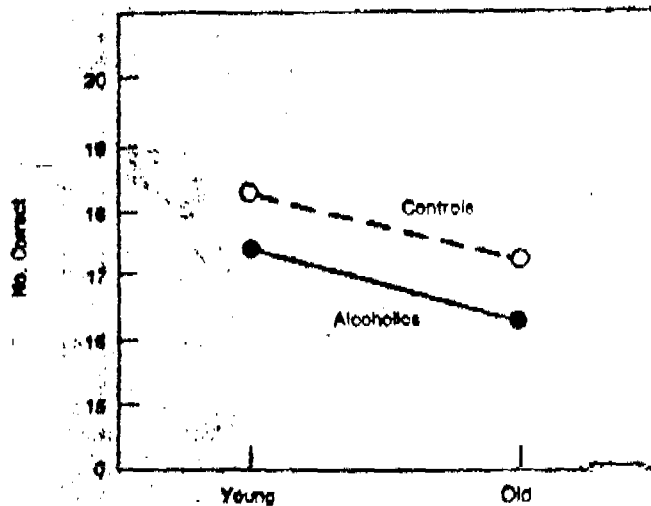


Fig 5.—Mean number of embedded figures correctly identified by "young" and "old" alcoholics and matched controls.

extremely sensitive to age-related declines. The age effect for the SDST was significant at $P < .001$ ($F = 51.85$; $df = 1, 205$), as was the age effect for the DSST ($F = 51.540$; $df = 1, 205$). For both tests, the group-by-group interaction was not significant. Newman-Keuls tests showed that both young and old alcoholics were impaired on these two tasks. On the SDST, the comparison between old alcoholics and old controls was significant at $P < .05$, while the comparison between young alcoholics and young controls was significant at $P < .01$. On the DSST, both post hoc comparisons were significant at $P < .01$.

The results for the BVRT are seen in Fig 4. On the immediate recognition form F, the group factor only approached significance ($P < .07$). However, a highly significant difference between alcoholics and controls was obtained on the delayed recognition form G ($F = 8.78$; $df = 1/129$; $P < .005$). On both forms, old subjects performed more poorly than young subjects ($F = 12.06$; $df = 1/129$; $P < .001$ for form F; $F = 6.70$; $df = 1/129$; $P < .02$ for form G). Neither of the group-by-age interactions was significant. For both forms F and G, Newman-Keuls comparisons between young alcoholics and their controls and old alcoholics and their controls failed to detect any significant differences.

The Embedded Figures Test also effectively discriminated between alcoholic and nonalcoholic groups and between young and old subjects (Fig 5). Alcoholics detected fewer of the hidden figures than did nonalcoholics ($F = 4.18$; $df = 1/129$; $P < .05$), while older

subjects performed more poorly than younger subjects ($F = 8.20$, $df = 1/129$; $P < .02$). As in all the previous analyses, there was no significant interaction between group and age. Post hoc analyses did not show significant differences between young alcoholics and their nonalcoholic peers, or between old alcoholics and their nonalcoholic peers.

Comment

As have other recent reports from this laboratory,^{1,2,11} the present findings support the proposition that alcohol-related memory and visuosperceptual disorders are not limited to patients with Korsakoff's syndrome. Detoxified alcoholics who had no history of Wernicke's encephalopathy or other organic disorders that might produce cognitive deficits were significantly impaired in their ability to acquire and retain new information and to analyze complex visual stimuli. Subjects with histories of prolonged, excessive alcohol consumption displayed poorer verbal and nonverbal STM, paired-associate learning, digit-symbol substitution, and detection of embedded figures than matched, non-alcoholic subjects. The smallest and least reliable differences between alcoholics and nonalcoholics emerged on tests that place minimal demands on the subject's ability to efficiently encode and store information: the Four-Word STM Test at 15-s delay and the BVRT form F. More robust differences were obtained on tasks demanding psychomotor speed (SDST and DSST), as well as those requiring the learning of novel associations (Symbol-Digit Paired-Associates) and the holding of information in memory for longer periods of time (Four-Word STM Test at 80-s delay and the BVRT form G).

A second major finding of this study was that young men were as vulnerable to the damaging effects of alcohol as were older men. On none of the measures examined was there a significant group-by-age interaction. Furthermore, on every test where old alcoholics were impaired relative to their controls, young alcoholics were impaired relative to their controls. Perhaps the strongest statement about alcohol and aging that we can make from these data is that the memory and perceptual performances of the alcoholic men in their early 40s mimicked those of nonalcoholic men in their late 50s. Thus, on a purely descriptive level, it appears as if excessive alcohol consumption results in a functional aging of the nervous system. Of course, the quantitative data available do not allow us to logically argue that the same psychological or neuronal processes underlie the poor performances of young alcoholics and older non-alcoholics. What are clearly needed are more refined examinations of the quality of the processing breakdowns seen in aging and alcoholism, and more telling biochemical, electrophysiological, and neuropathological studies to determine whether normal senescence and ethanol consumption affect the same brain systems in the same ways. In fact, the most recent neurophysiological evidence suggests they do not. Porjesz and Begleiter,¹² for example, reported that several components of the cortical evoked response are aberrant in long-term alcoholics but not in older normal individuals. In both young and old normal subjects, the late components of the event-related potential differ when processing task-relevant and task-irrelevant information. No such differentiation is seen in the waked responses of long-term alcoholics.

Our findings of impaired brain functioning in young as well as old alcoholics obviously stand in contrast to several studies in the recent literature. Jones and Parsons¹³ reported that older long-term alcoholics (mean age, 48 years) were impaired on Halstead's Category Test while younger

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Table 2.—Characteristics of Abstinent Alcoholic Groups in Study 2 (Mean \pm SD)

	Abstinence		
	Short-term (1-2 mo) (N=59)	Long-term (13-36 mo) (N=88)	Prolonged (\geq 60 mo) (N=30)
Age, yr	47.32 \pm 9.26	48.74 \pm 7.17	52.80 \pm 7.15*
Education, yr	11.73 \pm 2.26	11.89 \pm 1.56	11.46 \pm 2.24
Wechsler Adult Intelligence Scale vocabulary score	11.71 \pm 8.41	12.03 \pm 1.76	10.83 \pm 1.76
Alcoholism, yr	21.89 \pm 7.13	23.61 \pm 8.07	17.83 \pm 7.38†
Abstinence, mo	1.29 \pm 0.46	20.69 \pm 7.31	80.36 \pm 19.13‡

* $P < .02$.
† $P < .009$.
‡ $P < .001$.

alcoholics (mean age, 36 years) were not. Klisz and Parsons²² found a large interaction between age and duration of alcoholism on a hypotheses-testing and concept-formation task. The older alcoholics in their study were more impaired when compared with age-matched controls than were the younger alcoholics. In both of these studies, younger individuals displayed a relative immunity to the deleterious effects of alcohol. The present study, similar to that of Ryan and Butters,²³ found no such sparing. A possible source of this difference is that Parsons' studies involved tests of conceptual thinking and abstract reasoning, while the present research examined memory and visuoperceptual functioning. It is certainly conceivable that the different neural structures mediating these various aspects of cognitive functioning are differentially affected by the long-term ingestion of alcohol. Thus, the ability to efficiently store and retrieve new information may be compromised in young alcoholics, while their ability to formulate and test hypotheses is relatively intact.

Grant and associates²⁴ have also reported that young (mean age, 37 years), recently detoxified alcoholics perform normally on a wide variety of neuropsychological tasks, including immediate and delayed recall of verbal and non-verbal information. Perhaps the most likely source of the discrepancy between Grant and colleagues' results and those reported here is in the subjects' drinking histories. The subjects in the Grant et al study had abused alcohol for an average of 6.1 years, while the young alcoholics in the present study had histories of abuse in excess of 20 years. It is almost certainly the case, as Jones and Parsons²⁵ and Riege et al²⁶ have indicated, that short-term alcoholics are typically less cognitively impaired than long-term alcoholics.

Having firmly established that alcohol consumption impairs human information processing, we next turned our attention to the issue of recovery.

STUDY 2 Subjects and Methods

From our entire population of alcoholic men, three subject groups were constructed, based on length of continuous abstinence. The "short-term abstinence" group comprised 59 men who had been sober between one and two months at the time of testing. The 86 subjects in the "long-term abstinence" group were abstinent between one and three years. Subjects in the "prolonged abstinence" group (N = 30) had remained abstinent for at least five years.

Of the 184 alcoholic men in the first study, 85 had abstinence

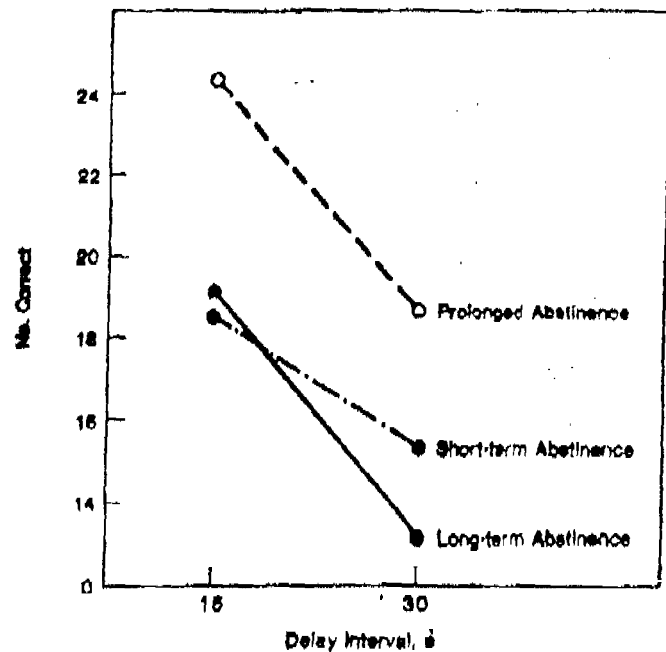


Fig 6.—Recovery of short-term memory. Performance of three groups of abstinent alcoholics on Four-Word Short-term Memory RBI.

histories corresponding to these categories, and their data are included here. The additional 49 subjects were not included in the first study because of constraints imposed by the use of matched samples and by the upper limit of five years of abstinence.

Table 2 presents the relevant characteristics of the subject groups. The groups were matched for years of education, but differed significantly in mean age and duration of alcoholism. Therefore, age and years of drinking were used as covariates in analyses of the data. The WAIS vocabulary score did not differ significantly among the three groups ($P = .30$), but since this variable consistently accounted for a large proportion of the variance in test scores, it too was used as a covariate in statistical analyses.

Tests and Procedures.—The same battery of neuropsychological tests and the same procedures used in the first study were employed here.

Results

Differences between abstinence groups were analyzed with the one-way analysis of covariance. Age, duration of alcohol abuse, and age-corrected WAIS vocabulary scaled score were the covariates.

Significant recovery of verbal STM was found on the Four-Word STM Test (Fig 6). At the 15-s delay, the groups differed in number of words recalled ($F = 8.92$; $df = 2/84$; $P < .001$). At the 30-s delay, the difference among groups was also significant ($F = 8.92$; $df = 2/84$; $P < .025$). Post hoc tests, using the Newman-Keuls procedure, disclosed that the short- and long-term groups did not differ from each other, but that both groups performed more poorly ($P < .01$) than did the prolonged group. To determine whether the STM of the prolonged abstainers had fully recovered, their performance was compared with that of a group of 50 nonalcoholic men (matched for age, education, and WAIS vocabulary score). No differences were found at either delay interval or for the total score ($t = .19$; $df = 78$; P value was not significant).

Rapid transcription of symbols to numbers (SDST) and numbers to symbols (DSST) improved with continued abstinence (Fig 7). The three groups differed in their mean scores on both the DSST ($F = 7.65$; $df = 2/119$; $P < .001$) and the SDST ($F = 4.55$; $df = 2/119$; $P < .02$). In both cases, the short-term and prolonged abstinence groups differed from each other at $P < .01$, while the long-term group did not differ from either of them. Recovery on the DSST

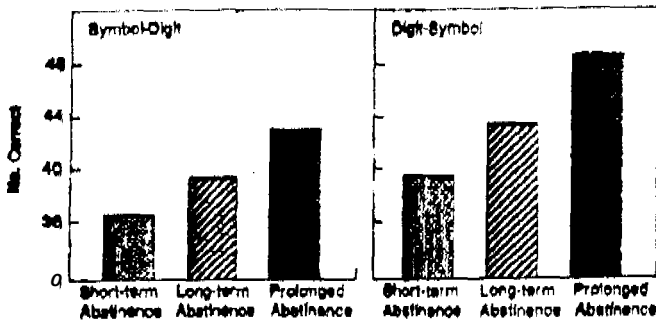


Fig 7.—Substitution Test performance of short-term, long-term, and prolonged abstinent alcoholics.

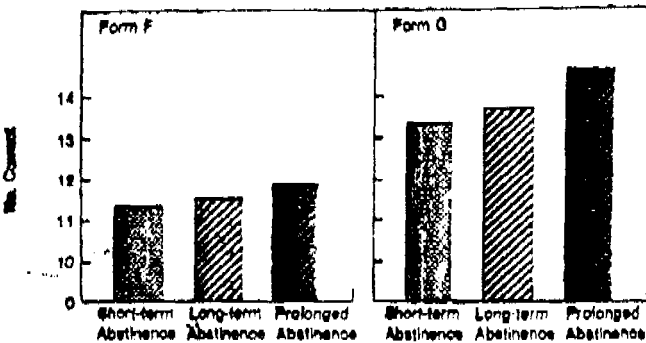


Fig 8.—Performance of three groups of abstinent alcoholics on forms F and G of Benton Visual Retention Test.

appears to be complete in the prolonged group, as these subjects performed as well as a group of 50 matched nonalcoholics ($t = -1.34$; $df = 78$; P value was not significant). On the SDST, the fact that the prolonged abstainers were still impaired in comparison with a matched group of 50 nonalcoholic men ($t = 2.40$; $df = 78$; $P < .02$) indicates that recovery was not complete on this task.

Figure 8 shows the results for forms F and G of the BVRT. On form F, with 5-s stimulus exposure and immediate recognition, a very small and nonsignificant improvement was found with longer sobriety ($F = 1.12$). Since alcoholics as a group are only minimally impaired on this form, this is hardly a surprising finding. On the delayed recognition condition (form G), however, alcoholics as a group were markedly impaired and significant recovery related to length of abstinence was found ($F = 3.87$; $df = 2/84$; $P < .05$). Comparisons among the three groups disclosed that the score of the short-term group was significantly lower than that of the prolonged group ($P < .05$). An additional comparison between the performance of the prolonged group and a group of 50 nonalcoholic controls failed to demonstrate a significant difference ($t = -1.17$; $df = 71$), a finding that indicates complete recovery of these nonverbal memory skills seven years after cessation of drinking.

Performance on two of the tests in our battery was unrelated to length of abstinence. No significant recovery was found on either the Symbol-Digit Paired-Associate Learning Test ($F = 1.50$; $df = 2/110$) or the Embedded Figures Test ($F = 1.80$; $df = 2/84$). Note that detoxified alcoholics were found to be severely impaired on both of these tasks in study 1 of this report.

Comment

The obvious conclusion to be drawn from these data is that recovery of cognitive function after cessation of drinking is not an all-or-none phenomenon. Improvement with prolonged abstinence was very notable on tasks requiring short-term retention of nonverbal and verbal information. In fact, alcoholics who remained sober for a minimum of five

years performed, as a group, as well as nonalcoholics on our measures of STM. Dramatic improvements were also seen on tests of rapid symbol-to-number and number-to-symbol transcription, which may reflect recovery of motor regulation and/or increased psychomotor speed.

On the other hand, no significant improvement was seen on the Symbol-Digit Paired-Associate Learning Test. Even after almost seven years of continuous abstinence, long-term alcoholics displayed a profound deficit in the learning of novel associations. Diminished capacity for complex visual figure-ground analysis, as measured by the Embedded Figures Test, also persisted over the time course examined.

Several previous studies, such as those by Page and Linden²¹ and Guthrie,²² have demonstrated significant recovery of cognitive abilities during the first year of abstinence. However, virtually all of this improvement appears within the first few weeks after cessation of drinking. This probably reflects the resolution of the acute brain syndrome that initially brings the patient to the attention of physicians. Ryan and associates²³ examined recovery after extended periods of abstinence and did not find restitution of any memory skills with abstinence periods of one to five years. Berglund and co-workers,²⁴ in one of the few long-term follow-up studies of cognitive and social functioning in alcoholics, examined subjects an average of 3.7 years after inpatient alcoholism treatment. They found that subjects who had remained abstinent or whose alcohol consumption had significantly declined displayed slight improvements on several measures of intellectual functioning. However, the initial evaluation of these patients took place after only one or two weeks of sobriety, at a time when the acute effects of alcohol are still present. Thus, the improvement seen at 3.7 years may have all taken place very early in the abstinence period (ie, one to four weeks). The same criticism may be levied against the study by Long and McLachlan²⁵ and several others in the literature. Since in the present study we examined patients who had been abstinent for at least four weeks, the recovery that was found could not be attributed to simple detoxification.

An unexpected highlight of this study was that short- and long-term memory differed in recovery rates. Long-term alcohol abusers were moderately impaired in their ability to hold small amounts of information in temporary storage, but this faculty seemed to recover totally with extended periods of abstinence. On the other hand, the ability to form new associations between ostensibly unrelated stimuli was more severely compromised in alcoholic men, and this defect appeared to be persistent. These findings have important implications both for psychological theories of normal memory and for the neurobiological bases of memory dysfunction. The distinction between immediate memory, a limited-capacity STM, and a relatively permanent long-term memory has been a major postulate of experimental psychology for several decades.^{26,27} While this separation has been proposed largely on the basis of the performance characteristics of normal individuals, the study of brain-disordered patients has contributed substantially to the dissociation and has provided important clues as to the neuroanatomical structures involved in short- and long-term memory.

Perhaps the strongest evidence in favor of separate memory stores is provided by the well-known case of patient H.M.^{28,29} and by systematic study of patients with left- or right-hemisphere cortical lesions. Following bilateral resection of the mesial portion of the temporal lobes, patient H.M. displays a profound inability to learn new

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associations, although his immediate memory (as assessed by digit span) was within normal limits. Subsequent studies of patients with unilateral mesial temporal resections have implicated the hippocampus as the likely source of amnesia in bilateral temporal lobectomy.⁴⁰

In contrast to patient H.M., patients with damage to the posterior association cortex have been reported to have modality-specific deficits in immediate and short-term, but not long-term, memory. Warrington and Scoville's⁴¹ patients with left-hemisphere damage had abbreviated digit spans and showed rapid forgetting of orally presented verbal materials on a Peterson distractor task. Despite this severe inability to hold orally presented information in STM, these patients acquired lists of paired associates in normal fashion. A parallel finding was reported by Butters and Samuels and their collaborators⁴² in patients with lesions in posterior sectors of the right hemisphere. Their patients were impaired on Peterson distractor tasks in which verbal and nonverbal materials were presented visually.

If it is indeed the case that STM is highly dependent on cortical mechanisms and long-term memory requires intact limbic-diencephalic structures (eg. hippocampus, dorsomedial nucleus of the thalamus, mammillary bodies), one might expect long-term alcoholics, who are impaired in both of these functions, to display both cortical and subcortical pathology. This appears to be the case. Recent studies examining the computed tomographic (CT) scans of long-term alcoholics have found atrophy of the cerebral cortex, especially in the frontal lobes, to be the most conspicuous abnormality.⁴³ In addition, Carlsson et al⁴⁴ found marked dilation of the third ventricle, suggesting that alcohol induces a degeneration of midline thalamic nuclei. A recent study by Gebhardt⁴⁵ provided evidence that this subcortical periventricular area is, in fact, related to long-term memory. Using computerized analysis of the CT scans of alcoholics, she discovered that the density of brain tissue around the ventricular system was highly correlated with performance on our Symbol-Digit Paired-Associate Learning Test. No such correlation was obtained with the Four-Word STM Test.

Finally, the present finding of marked improvement in short-term, but not long-term, memory in prolonged abstinent alcoholics may indicate that cortical tissue is more prone to reconstitution than is subcortical tissue. Carlsson and associates⁴⁶ have already provided evidence that short-term abstinence from alcohol results in a reversal of cortical atrophy seen on CT scans. The data reported here are consistent with that finding and raise the intriguing possibility that this mending of cortical tissue and the reinstatement of cortical functioning may continue for many years after cessation of drinking.

CONCLUSIONS

It is widely accepted that ingestion of ethanol has acute detrimental effects on most cognitive processes. Studies of intoxicated individuals⁴⁷ have indicated that the presence of alcohol in the blood stream interferes significantly with the acquisition and recall of new information. When inebriated, alcoholic subjects appear to process incoming information in idiosyncratic ways and fail to employ the encoding strategies that favor superior performance on memory tests.⁴⁸

During alcohol detoxification, there is typically a rapid improvement in mental status.⁴⁹ In fact, the recovery of cognitive functioning with the metabolism and elimination of alcohol is so dramatic that the existence of any residual

deficits has been questioned. The results of the present study clearly indicate that there are significant deficits in short-term retention, new learning and memory, and visuo-perceptual analysis in thoroughly detoxified long-term alcoholics. These deficits are likely the result of both cortical and limbic/diencephalic pathology and are seen in alcoholics regardless of age.

The existence of such pronounced cognitive impairments, especially in the realm of verbal short-term and paired-associate learning, lead us to question the common practice of immersing alcoholic patients in lectures, workshops, and psychotherapy during the detoxification period. Given the memory defects and the often-reported impairments in conceptual thinking in alcoholics,^{50,51} it is unlikely that the large amounts of complex, mostly verbal material presented in therapy programs is being adequately processed. It may be more appropriate, therefore, to allocate the limited resources of alcohol treatment programs to early intervention and education rather than dynamic psychotherapy during or shortly after detoxification.

On a more optimistic note, it appears that recovery of cognitive functioning after alcoholism is not limited to the period immediately after detoxification. Improvement in some functions may, in fact, continue for many years. In particular, the skills involved in STM and psychomotor performance appear to return with extended periods of sobriety. On the other hand, impairments in long-term memory appear to be refractory to spontaneous recovery. The question remains however, as to whether explicit training or practice in the use of mnemonic aids can improve the memory performance of alcoholics. At least one recent study⁵² has suggested that the paired-associate learning of cognitively impaired alcoholics is improved by teaching them visual imagery and verbal mediation strategies. Whether such instruction is helpful to older, longer-term alcoholics, and whether it might actually hasten the course of recovery, are important questions certainly in need of further study.

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References

1. Butters N, Cermak LS: *Alcoholic Korsakoff's Syndrome: An Information-Processing Approach to Amnesia*. New York, Academic Press Inc, 1969.
2. Tulland GA: *Deranged Memory*. New York, Academic Press Inc, 1965.
3. Victor M, Adams RD, Collins GE: *The Wernicke-Korsakoff Syndrome*. Philadelphia, FA Davis Co, 1971.
4. Ryback RS: The continuum and specificity of the effects of alcohol use on memory: A review. *Q J Stud Alcohol* 1971;32:985-1016.
5. Jonason C-O, Cronholm B, Litzkowitz B: Intellectual changes in alcoholics: Psychometric studies on mental sequelae of prolonged intensive abuse of alcohol. *Q J Stud Alcohol* 1962;23:221-242.
6. Weingartner H, Fallace LA, Markley HG: Verbal information retention in alcoholics. *Q J Stud Alcohol* 1971;32:269-308.
7. Parsons OA, Prigatano GP: Memory functioning in alcoholics. In Birnbaum IM, Parker ES (eds): *Alcohol and Human Memory*. Hillsdale, NJ, Lawrence Erlbaum Associates Inc, 1977.
8. Butters N, Cermak LS, Montgomery K, et al: Some comparisons of the memory and visuo-perceptive deficits of chronic alcoholics and patients with Korsakoff's disease. *Alcoholism* 1977;1:78-80.
9. Ryan C, Butters N, Montgomery K, et al: Memory deficits in chronic alcoholics: Continuities between the "intact" alcoholic and the alcoholic Korsakoff patients. In Begleiter H (ed): *Biological Effects of Alcohol*. New York, Plenum Publishing Co, 1980.
10. Glosser G, Butters N, Kaplan E: Visuo-perceptual processes in brain-damaged patients on the digit-symbol substitution test. *Int J Neurosci* 1977;7:69-86.

associated with prolonged alcohol abuse. *Alcoholism: Clin Experimental Res* 1981;5:378-385.

35. Berglund M, Laljonquist H, Horlen M: Prognostic significance and reversibility of cerebral dysfunction in alcoholics. *J Stud Alcohol* 1977;38:1761-1770.

36. Waugh NC, Norman DA: Primary memory. *Psychol Rev* 1968;75:89-104.

37. Atkinson RC, Shiffrin RM: Human memory: A proposed system and its control processes, in Spence KW (ed): *The Psychology of Learning and Motivation: Advances in Research and Theory*. New York, Academic Press Inc, 1968.

38. Scoville WB, Milner B: Loss of recent memory after bilateral hippocampal lesions. *J Neurol Neurosurg Psychiatry* 1957;20:11-21.

39. Milner B: Psychological defects produced by temporal-lobe excision. *Res Publ Assoc Res Nerv Ment Dis* 1958;36:244-257.

40. Milner B: Memory and the medial temporal regions of the brain, in Pribram KH, Broadbent DE (eds): *Biology of Memory*. New York, Academic Press Inc, 1970.

41. Warrington EK, Scoville R: The selective impairment of auditory-verbal short-term memory. *Brain* 1969;92:885-895.

42. Scoville T, Warrington EK: Independent functioning of the verbal memory stores: A neuropsychological study. *Q J Exp Psychol* 1970;22:261-278.

43. Butters N, Samuels I, Goodglass H, et al: Short-term visual and auditory memory disorders after parietal and frontal lobe damage. *Cortex* 1970;6:440-459.

44. Samuels I, Butters N, Goodglass H, et al: A comparison of subcortical and cortical damage on short-term visual and auditory memory. *Neuropsychologia* 1971;9:298-306.

45. Cala LA, Jones B, Mastaglia FL, et al: Brain atrophy and intellectual impairment in heavy drinkers: A clinical, psychometric and computerized tomography study. *Aust NZ J Med* 1978;8:147-153.

46. Lee K, Miller L, Hardt F, et al: Alcohol-induced brain damage and liver damage in young males. *Lancet* 1979;2:759-761.

47. Kroll P, Selge R, O'Neill B, et al: Cerebral cortical atrophy in alcoholic men. *J Clin Psychiatry* 1980;41:417-421.

48. Carlsson C, Claesson L-E, Karlson K-T, et al: Clinical psychometric and radiological signs of brain damage in chronic alcoholism. *Acta Neurol Scand* 1979;60:85-92.

49. Gebhardt C: *Computer Analyses of CT Scans of Chronic Alcoholics: The Relation of Third Ventricular Region to Memory and Frontal/Parietal Regions to Perception*, thesis. Boston University, Boston, 1981.

50. Carlen PL, Worsman G, Holgate TC, et al: Reversible cerebral atrophy in recently abstinent chronic alcoholics measured by computed tomography scans. *Science* 1978;200:1076-1078.

51. Tamerin JA, Weiner S, Poppen R, et al: Alcohol and memory: Amnesia and short-term memory function during experimentally induced intoxication. *Am J Psychiatry* 1971;127:1659-1664.

52. Weingartner H, Fallace LA: Alcohol state-dependent learning in man. *J Nerv Ment Dis* 1971;158:395-406.

53. Parker ES, Aikana RL, Birnbaum IM, et al: Alcohol and the disruption of cognitive processes. *Arch Gen Psychiatry* 1974;31:824-825.

54. Rosen LJ, Lee CL: Acute and chronic effects of alcohol use on organizational processes in memory. *J Abnorm Psychol* 1976;85:806-817.

55. Burdick JA, Johnson LC, Smith JW: Measurements of change during alcohol withdrawal in chronic alcoholics. *Br J Addict* 1970;65:273-280.

56. Tarter RE, Parsons OA: Conceptual shifting in chronic alcoholics. *J Abnorm Psychol* 1971;77:71-75.

57. Binder LM, Schreiber V: Visual imagery and verbal mediation as memory aids in recovering alcoholics. *J Clin Neuropsychol* 1980;2:71-74.

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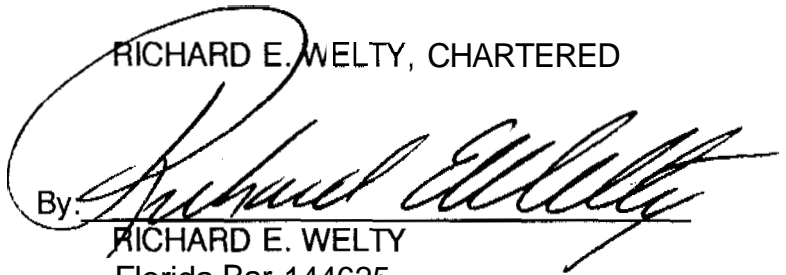
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CERTIFICATE OF SERVICE

I CERTIFY that two copies of the foregoing initial brief of Respondent, **MYRON C. PREVATT JR.**, were mailed to MIMI DAIGLE, The Florida Bar, 650 Apalachee Parkway, Tallahassee, FL 32399-2300, on 21 January 1992 by United States Post Office Express Mail.

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