Alcohol Related Brain Damage (ARBD) not including Wernicke-Korsakoff syndromes

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This paper outlines alcohol related brain damage by:

i) Introduction, description and generally, what the literature says;
ii) diagnostic issues;
iii) hypotheses of ARBD;
iv) behavioural symptoms;
v) cognitive symptoms;
vi) neural substrates of ARBD;
vii) the case for neuropsychological testing for ARBD;
viii) the expected neuropsychological profile for this syndrome;
ix) neuromedical history;
x) neuropsychological expectations after abstinence;
xi) referring for neuropsychological testing;
xii) treatment strategies;
xiii) conclusion.

Introduction

Sustained, heavy and chronic alcohol abuse harmfully affects certain areas of cognitive functioning and appears to leave unaffected well-established abilities such as language and arithmetic (Lezak,1995). This is alcohol related brain damage (ARBD). It is described by different authors in a variety of ways. Descriptions centre around a theme of alcohol abusers of average intelligence who are 'non-clinical' or non-amnesic in their presentation. This represents 40% – 70% of alcoholics who, if suffering from ARBD, are not obviously impaired either physically or mentally (Rourke & Løberg,1996). In these cases, alcohol abusers often do not walk with the 'staggers' (ataxia), nor do they appear confused or disoriented to the point where their lifestyle is seriously disrupted. Cases of obvious impairment are most often linked to malnutrition and thiamine deficiency and labelled as Wernicke-Korsakoff syndrome (WKS) (Rourke & Løberg,1996). The debate on ARBD includes the suggestion that it is a sub-clinical form of WKS (Bowden, 1992). Thus, ARBD has been viewed as a multidimensional and potentially progressive problem that is comparable to global brain insult, not just frontal lobe damage (Bowden, 1992). This is supported by findings that verbal and visual deficits in heavy users of alcohol have been reported concurrently (Nixon, Kiyawski, Parsons, & Yohman, 1987 as cited in Lezak, 1995). Bowden's (1992) suspicion that the temporal lobes are also involved in ARBD would therefore bear consideration, especially if evidence of aphasia can be found through neuropsychological (NP) test results from detoxified alcoholics.

ARBD often exists concurrently with other factors that cause brain damage, such as toxins other than alcohol, head injury, or age related problems (Rourke & Løberg, 1996). These co-morbid risk factors are likely to exist and play a role in assessment, final evaluation and creation of treatment plans for alcohol related brain/central nervous system (CNS) damage. The common factor for ARBD is a history of heavy drinking, even if only for a relatively short period of time (Canaris & Jurd, 1991; Hage, 1991; Walsh, 1985).

There is a high prevalence in abusers of alcohol of co-morbid mental disorders, including schizophrenia, anxiety, panic, phobia, obsessive-compulsive disorder, bipolar, depression and anti-social personality disorder (Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), American Psychiatric Association, 1994; Glass, 1991). Each co-morbidity may well contribute to the level of NP impairment suffered by a patient. This is essentially a masking effect where the level of impairment attributable to alcohol abuse becomes difficult to distinguish and identify. How this co-morbidity manifests in terms of direction of effect, that is, whether the psychiatric condition led to alcohol abuse or the reverse process occurred, is difficult to ascertain. This issue is beyond the scope of the current discussion, however, it is important to consider the possibility of co-morbidity and dual diagnosis when considering ARBD.

The focus of the present paper is the subtle and variable alcohol related brain insults that may occur following sustained heavy alcohol use. In many cases, ARBD is present in someone who appears remarkably normal with no overtly obvious signs of ill health. This individual can maintain a conversation equivalent to their education level (Canaris & Jurd, 1988). Glass (1991) contends that the effects of alcohol abuse in these individuals can extend to changes in mood, motivation, and decision-making, yet they remain free of gross impairments such as amnesia, staggering gait or dementia induced aphasia. In essence, chronic alcohol abuse over time will affect aspects of cognitive functioning. These problems are sometimes difficult to detect because the established daily functions appear to be untouched (Lezak, 1995).

In the context of the current paper, ARBD is the toxic effect of alcohol consumption on the brain, which may accumulate with sustained heavy drinking over time (Bowden, 1992; Walsh, 1985). Damage resulting from high levels of alcohol use is believed by some researchers, example,Walsh (1985) to be mainly restricted to the frontal lobes, and is associated with the executive forms of mental activity (Hage, 1991; Walsh, 1983). This interpretation has been criticised by Bates et al. (2002) and Bowden (1992) who have argued that damage to other areas of the brain, such as in the limbic system and hippocampal structures, may lead to poor scores on NP tests such as maze learning. Other researchers support this frontal lobe – limbic system explanation of ARBD and tend to emphasize the examination of executive or adaptive function in planning a battery of NP tests for assessing ARBD (Allen & Landis, 1998; Rourke & Løberg, 1996). A factor analytic study by Bates et al. (2002) of 127 individuals with a history of alcohol abuse found that cognitive deficits fell into four major categories: executive, memory, verbal, and processing speed. This could lead to the conclusion that there is some evidence of temporal lobe dysfunction in alcohol abusers, in addition to frontal lobe damage.

Importantly for the present paper, the sample used by Bates et al. (2002) excluded individuals with WKS from participation in the study. Evert and Oscar-Berman (2001) also found deficits in cognitive functions normally associated with the right cerebral hemisphere in individuals with high levels of alcohol consumption, especially those aged over 50 years. These findings suggest NP deficits associated with alcohol abuse are diffuse and possibly include a global brain spectrum.

It is important to note that references to ARBD in the literature often do not distinguish non-amnesic ARBD from either WKS or alcoholic dementia (Clifford & Maddocks, 1988). WKS is also labelled Alcohol-Induced Persisting Amnestic Disorder in DSM-IV. It often follows a confusional state (Wernicke's encephalopathy) and is characterised by severe short-term memory loss whilst alcoholic dementia resembles WKS in some respects (Abbott, 1984). **Ryan and Butters (1980) state that prior to the emergence of the amnesic syndrome, heavy drinkers have difficulty in accessing neurocognitive strategies associated with concept formation, problem solving and certain memory tasks. Lezak (1995) suggests that the essential ingredient that distinguishes ARBD from WKS is that chronic alcohol abusers do not lose their capacity to be individualistic and seek goal-directed activity.** The alcohol abuser with ARBD is also unlikely to exhibit confabulation (making up stories to mask memory loss) and **is likely to display only a mild and relatively scattered incidence of memory deficits (Lezak,1995).**

Diagnostic issues in ARBD

The patient should be first assessed as a heavy and constant alcohol user before ARBD is considered. The major considerations in forming a diagnosis for alcohol related problems are:

- Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV; American Psychiatric Association, 1994) criteria for an Alcohol Use Disorder;
- Clinical interview/history taking/chronology of alcohol use and alcohol intake to include maximum quantity and frequency per drinking occasion. The maximum quantity and frequency of alcohol use is linked in many studies to ARBD.
- Performance on normative questionnaires. The Alcohol Use Disorders Identification Test Screening Instrument (AUDIT) is a brief (2-minutes) screening instrument to identify an individual with alcohol problems. There are also a number of alternative instruments available for this purpose e.g., in, Review of diagnostic screening instruments for alcohol and other drug use and other psychiatric disorders - National Drug Strategy (Dawe & Mattick, 1997);
- Gender of client. Women who consume 20 to 40 grams of alcohol (ethanol) per day are at comparable risk to men who consume 60 to 80 grams per day (Mann, Batra, Günthner, & Schroth, 1992).
- Biochemical markers. The main pathological test is for liver function, which can be arranged by a physician. Test results can highlight raised levels of blood metabolites, such as gamma-glutamyl transferase and aspartate amino-transferase,

indicating liver damage (Rourke & Loberg,1996). Pathology results showing liver function problems can be used to motivate treatment and abstinence.

- Psychiatric co-morbidity. Although this item is outside the scope of this paper, psychiatric co-morbidity is an important issue in diagnostic evaluation because each possible disorder has its own implications regarding NP impairment;
- NP testing results and profile, which is outlined further in this paper.

Hypotheses of brain models to explain ARBD

Changes in brain structure and function in ARBD are believed to be due to toxic metabolic products, rather than due to poor nutrition, which is linked to WKS (Walsh, 1985). In contrast, Bowden (1992) argues that the available data provides little evidence that ethanol (alcohol for consumption) exerts a neurotoxic effect on the brain resulting in long term deterioration of brain function. **There is, however, a significantly increased "risk of cognitive deficit, particularly of the more adaptive functions" associated with heavy alcohol consumption (Walsh, 1983, p. 84).** Adaptive impairments are then linked to brain damage in the anterior area of the brain. This leads to the hypothesis that **ARBD is associated with frontal lobe damage (Walsh, 1985).** Therefore, neuropsychological tests of the higher adaptive functions could serve as an early warning system for brain impairment involving abuse of alcohol (Walsh, 1985). Losses to adaptive or executive functions refer broadly to the control and feedback systems that support good performance on tests that require abstract thought and flexible information processing (Bates et al., 2002).

Other models of the effects of alcohol abuse on brain functioning include damage to the right cerebral hemisphere and premature aging (Evert & Oscar-Berman, 2001), changes in ventricular and sulcal cerebrospinal fluid (CSF), loss of cerebral white matter, and reductions in brain glucose utilization (Bates et al., 2002). Evert and Oscar-Berman (2001) have posited a link between chronic alcohol use and right hemisphere dysfunction, through comparisons between alcohol abusers and individuals with right hemisphere damage. Even though the alcohol abuse group showed similar deficits to the right hemisphere patients, other cognitive problems were found in the alcohol abuse group that could not be explained by the right hemisphere damage.

Accelerated aging associated with sustained heavy alcohol abuse has been suggested by Ryan and Butters (1980) and Evert and Oscar-Berman (2001). This has received some support in studies focusing on cognitive deficit similarities between younger alcohol abusers and older non-alcoholic control groups. However, other findings suggest that aging and sustained alcohol abuse have independent effects on the brain (Rourke & Løberg, 1996). While this may be the case, Evert and Oscar-Berman (2001) found that aging was significantly correlated with an increased vulnerability to developing cognitive deficits observed in older non-alcoholic individuals.

Behavioural symptoms

Hage (1991) asserts that ARBD patients may present with problems of adaptive behaviour, including difficulties achieving goals. They are also likely to be unable to manage multiple tasks and **may have difficulty in adjusting to change at work or home. This may be concurrent with difficulty in family or social relationships and in general these individuals may have been feeling increasingly frustrated with life (Hage, 1991).** Changes in mood, motivation, and decision-making ability could also be indicative of excessive alcohol use (Glass, 1991), and enthusiasm for both work and social activities can be decreased even while participation continues.

Cognitive symptoms

Between 30% and 80% of individuals entering addictions treatment have been found to have mild to severe neuropsychological deficits (Bates et al., 2002; Canaris & Jurd, 1991; Dikman, Donovan, Løberg, Machamer, & Temkin, 1993). In treatment, such deficits may contribute to poor outcomes if resultant inattention, distractibility and lack of motivation are not addressed (Bates, et al., 2002; Canaris & Jurd,1991). These figures are somewhat difficult to interpret, given that some studies include participants who have co-morbid psychiatric conditions, while these patients have been excluded in others.

Acute cognitive deficits are often present in ARBD in the first two weeks after cessation of alcohol consumption (Allen & Landis, 1998). However, these deficits can be common to alcohol abusers with and without ARBD during this initial period of abstinence. Improvements in cognitive functioning generally occur in the third and fourth weeks after cessation of drinking, with **marked improvement to around six weeks**. **The prominent problems associated during this stage relate to abstraction and problem-solving, perceptual-motor ability, and both short and long term memory (Allen & Landis, 1998).**

Alcohol abusers with ARBD often have trouble with learning acquisition and selective attention (Evert & Oscar-Berman, 2001; Rourke & Løberg, 1996). They may display retardation in learning from mistakes and show associated deficits in planning (Canaris & Jurd, 1991). This may signal memory deficits. However, **Rourke (1995) has suggested that generally the long-term memory of alcohol abusers is unaffected.** If there are significant cognitive losses in long term memory, it is likely that such deficits are related to a problem other than alcohol abuse (Rourke,1995).

Neural substrates of ARBD

The literature on areas of the brain affected by ARBD has been extensively reviewed by Rourke and Løberg (1996). These authors firstly reviewed studies of computed tomography (CT) scans of the brains of heterogeneous groups of alcoholics and found increases in CSF volume in subarachnoid spaces in heavy alcohol users of all

ages and increases in ventricular dilation (CSF volume in both lateral ventricles and third ventricle) predominately in older and nutritionally compromised alcoholics. Rourke and Løberg (1996) concluded that there was significant evidence to support the contention that increases in the incidence of cerebellar abnormalities has been observed in individuals classified as alcohol abusers. It was also noted by Rourke and Løberg (1996) that much of their analysis reviewed studies that accounted for cerebral changes that occur with normal aging.

Further studies reviewed by Rourke and Løberg (1996) were those that utilised magnetic resonance imaging (MRI) techniques. In recently detoxified alcoholics they found significant evidence of:

- increases in cortical and subcortical CSF volumes over and above aging;
- decreases in cortical grey matter (e.g., mesial temporal, dorsolateral frontal and parieto-occipital cortex);
- decreases in white matter;
- decreases in subcortical brain tissue (e.g., caudate and diencephalon).

A third set of studies reviewed by Rourke and Løberg (1996) were derived from autopsies. These studies indicated that after matching with controls, alcoholic brains were found to have 40% reductions in the cholinergic muscarinic receptors of the frontal cortex, the temporal cortex and the putamen area. These autopsy studies also indicated atrophy of the cerebral cortex and white matter that corresponded to the ventricular enlargements noted above.

Interestingly, Rourke and Løberg (1996) pointed out that physical brain abnormalities apparently caused by alcohol abuse have not correlated with NP testing. An alternative hypothesis is that the brain systems associated with non-amnesic ARBD could possibly be related to breakdowns in connection and communication systems between brain areas, rather than the cortical areas responsible for specific cognitive functions. If this is the case, then this would help explain why apparent atrophy of brain areas found in imaging and autopsy studies is not linked strongly to NP deficits.

The case for neuropsychological testing

The type of ARBD being addressed in this paper is subtle and difficult to detect. The mental state examination (MSE) commonly used in clinical settings has been found to be largely insensitive to this type of ARBD (Canaris & Jurd, 1991). NP tests used after initial MSE evaluations found that 33% of the patients tested had evidence of cognitive impairment that was not diagnosed using the MSE method. A subsequent study by Fals-Stewart (1997, as cited in Bates et al., 2002) also showed very poor concordance between NP test performance and treatment provider's judgements of NP functioning. Based on information from client histories, clinical interviews, physical examinations, brief cognitive screening tests and substance abuse severity evaluations,

clinicians failed to detect cognitive deficits that were later revealed by formal NP testing (Fals-Stewart, 1997, as cited in Bates et al., 2002).

A failure to assess, or even understand, ARBD may result in a poorer prognosis due to treatment failure (Canaris & Jurd, 1991). In order for any treatment to work the patient must be capable of receiving new information and integrating with existing information stores. This process should continue towards a synthesis of information to promote active behaviour change. **Fals-Stewart and Lucente's (1994) study in a residential alcohol treatment setting found that impaired cognitive functioning and anti-social personality disturbances were predictive of low program participation and lower lengths of stay in treatment for clients.** This study was not able to distinguish cognitive impairment from personality problems. However, neurocognitive impairments manifested by poor organization and problem-solving skills were noticeable in the subjects within this study.

Neuropsychological profile of impaired person recently detoxified

Below is a summary list of NP tests that have been used to assess the presence or absence of neuropsychological deficits in severe alcohol abusing populations. The NP profile below is drawn from Rourke (1995) and Rourke and Løberg (1996). They refer to this profile representing an alcoholic with intact verbal skills and an IQ within the normal range. Some clients with ARBD will show neurocognitive strengths and weaknesses based on their pre-morbid level of functioning which can be derived from their level of education and occupation. These factors must be considered whenever tests of cognitive ability are administered and analysed (Lezak, 1995; Dikmen et al.,1993).

<u>WAIS-III:</u>

Deficits on: Digit Span, Arithmetic, Similarities

Digit Symbol, Block Design, Object Assembly

<u>Attention and Concentration measures:</u>

Deficits on: Digit Span and Arithmetic

Speech Sounds Perception Test, Seashore Rhythm

<u>Abstracting and Reasoning Ability/Cognitive Flexibility Tests:</u>

Deficits on: Category Test

Trail Making Test (in particular, Part B)

Levine Hypothesis Testing Task

Shipley Institute of Living Abstraction Scale

Raven Progressive Matrices

Wisconsin Card Sorting Test

Complex Perceptual Motor Integration/Visual Spatial Tests:

Deficits on: Tactual Performance Test (total time)

Embedded Figure Tests

Visual Search

Block Design & Object Assembly (WAIS-III/WAIS-III-R)

Simple Motor Skills:

Deficits on: Finger Tapping

Grooved Pegboard

Hand Dynamometer

Learning and Memory

Deficits on: Verbal and Visual learning (Wechsler Memory Scales; WMS/WMS-R)

Learning ability and recognition memory on AVLT (with intrusions and false positives increased.

Neuromedical history

A major issue in assessing a patient for ARBD is that those who have a history of heavy drinking are likely to have concurrent neuromedical risk factors. These may interact with drinking practices to shape the neuropsychological profile (Rourke, 1995; Rourke & Løberg, 1996). Rourke (1995) has put forward the following risk factors that increase with ongoing and heavy alcohol consumption:

- risk for head trauma
- head injuries (falling over; fights; motor vehicle accidents);
- alcohol overdoses;
- alcohol withdrawal seizures;

- use of other drugs (illicit and licit);
- liver dysfunction (elevated enzymes);
- alcohol hepatitis (fatty liver) and cirrhosis;
- nutritional deficiencies (alcohol often replaces the need to eat);
- vascular disease;
- chronic obstructive pulmonary disease;
- sleep apnea (hypoxia).

The medical problems listed above are associated with reduced blood flow and reduced oxygen supply to the brain as well as lesions through trauma or toxins.

Neuropsychological expectations after abstinence

As discussed, if a heavy drinker ceases alcohol consumption there is initially some rapid NP improvement in the first few weeks of abstinence and more gradual improvement with increasing abstinence (Allen & Landis, 1998; Lezak, 1995). In addition, long term abstinence (4 – 7 years) is associated with normal NP findings (Rourke, 1995). **If there is a resumption of drinking this is likely to result in continued NP deficits and further deterioration (Rourke, 1995). Age becomes a factor in that increasing age is associated with incomplete NP recovery despite two years of abstinence. Furthermore, increasing age is associated with more NP deficits in alcohol abusers who resume drinking (Evert & Oscar-Berman, 2001; Rourke, 1995).**

Referral for neuropsychological testing

The role of the clinician is to assess whether a patient should be referred for NP testing if ARBD is suspected. Prior to a NP referral the clinician should encourage the patient to become abstinent from alcohol and mood changing drugs for at least several weeks. Rourke (1995) suggests that the clinician should then:

- evaluate whether cognitive complaints change and parallel a decrease in affective symptomatology with abstinence;
- assess the presence of persistent problems with attention and concentration, memory and reasoning skills;
- consider patient's age, past neuromedical risk factors (e.g., fighting, head injury history) and concurrent medical co-morbidity.

The referral should specify the risk factors involved with the individual, such as specific cognitive complaints, noting abilities that appear impaired. Evidence of organicity or tissue damage (brain lesions) should be eliminated based on depending on whether the patient has had a recent brain scan.

Treatment issues associated with NP impairments

When a patient is treated for alcohol abuse, a principle recommendation is for the clinician to carry out a functional analysis of the antecedents and consequences of the alcohol use (Bates et al.,2002). The analysis should include an assessment of the existing skills and resources of the patient. This is where NP deficits contributing to inattention, distractibility and motivation towards treatment may be deduced from patients' self reports (Bates et al, 2002; Rourke,1995).

The clinician should be aware that NP deficits will affect treatment approaches and outcomes (Rourke,1995). NP impairments may increase if the individual being treated has had relapses to alcohol use during the course of treatment (Rourke and Løberg 1996). As well as previously mentioned neuromedical risk factors, there are other variables such as personality dynamics and mood states, which could mediate treatment outcome and cognitive recovery. The process of treatment and recovery must include expectations that relapses could occur and cognitive function is likely to be affected.

Conclusion

A body of evidence has been reviewed to show that alcohol abuse relates to deficits in neuropsychological status. Dikmen et al. (1993) summarise this position by asserting that the degree of alcohol use is also related to the degree of neuropsychological impairment. However, the simplicity of this statement fails to account for factors that work concurrently with alcohol to cause brain damage. **Research so far has not been able to untangle the independent contributions of the harmful effects of heavy alcohol use from the effects of risk factors that often present as co-morbid problems. Risk factors such as head injuries, mental health status, poor education, medical conditions, and exposure to other toxic substances commonly exist alongside alcohol abuse**. Therefore, alcohol induced NP impairment should be considered as part of the total assessment regime of clients showing cognitive deficits consistent with ARBD. Assessment for ARBD should be cautious and objective and inclusive of co-morbid risk factors for other causes of NP impairment.

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Annotated Bibliography

Abbott, M.W. (1984). The early diagnosis of alcohol-related brain damage. *Australian Alcohol/Drug Review, 3*(2), 121-126.

Allen, D. N., & Landis, R. K. B. (1998). Neuropsychological correlates of substance use disorders. In P. J. Snyder & P. D. Nussbaum (Eds.), *Clinical neuropsychology* (pp. 591-612). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Bates, M. E., Labouvie, E. W., & Voelbel, G. T. (2002). Individual differences in latent neurological abilities at addictions treatment entry. *Psychology of Addictive Behaviours, 16*(1), 35-46.

Studied 197 individuals entering a treatment program for substance abuse. For the 127 individuals diagnosed with alcoholism, performance deficits were observed on various tests of executive function including Tower of Hanoi, Product Recall Task, Active Passive Voice Task. Confirmatory factor analysis found a four-factor solution that included executive functioning, memory, verbal ability, and processing speed. The authors conclude that the study provided evidence that alcohol abuse affects multiple brain regions. It was also found that risk factors of childhood behaviour problems, familial alcoholism, and co-morbid psychopathology accounted for 34% – 57% of the true variance in neuropsychological test abilities.

Bowden, S. (1992, June). Changing concepts of alcohol related brain injury. *Think*, 17-20.

Review of myths of alcohol related brain damage referring to specific effects of alcohol on the frontal lobes. Also addresses amnesic syndromes related to alcohol use.

Canaris, C., & Jurd, S. (1991). The diagnosis of alcohol-related brain damage: A retrospective study in alcoholics undergoing in-patient rehabilitation. *Drug & Alcohol Review, 10*, 85-88.

From a participant pool of 98 referrals, the authors selected 32 adults referred to Macquarie Hospital in New South Wales. Selected participants had no obvious co-morbid conditions or dependencies. Short term memory was found to be a better indicator of ARBD than MSE. Several patients referred to the authors by medical practitioners for non-ARBD assessment were found to have some degree of ARBD on examination.

Clifford, C. A., & Maddocks, D. (1988). Alcohol-related brain damage: The neurospsychological deficits and their implication for independent living. *Australian Drug & Alcohol Review, 7*, 79-81.

Discusses the more subtle neuropsychological impairments found in individuals who abuse alcohol but show no physical symptoms. Cognitive deficits may leave an individual "intellectually blunted", unreceptive to change, and lacking in cognitive flexibility. Implications of these cognitive deficits are discussed in terms of treatment and outcomes.

Dawe, S., & Mattick, R. (1997). Review of diagnostic screening instruments for alcohol and other drug use and other psychiatric disorders - National Drug Strategy. Australian Government Publishing Service.

Dikmen, S. S., Donovan, D. M., Løberg, T., Machamer, J. E., & Temkin, N. R. (1993). Alcohol use and its effects on neuropsychological outcome in head injury. *Neuropsychology, 7*(3), 296-305.

Examined neuropsychological outcomes of head injury with respect to alcohol consumption levels. The authors had difficulty with confounding variables, including demographic, lifestyle, and cognitive factors.

Evert, D. L., & Oscar-Berman, M. (2001). Selective attentional processing and the right hemisphere: Effects of aging and alcoholism. *Neuropsychology, 15*(4), 452-461.

The authors suggest that the pattern of cognitive deficits observed in alcohol abuse resembles that of right cerebral hemisphere damaged patients. This argument received only limited support, as it could not account for some cognitive deficits observed in the alcohol abuse group. The authors conclude that there is support for a premature aging of the brain hypothesis, and that an increased vulnerability to the effects of alcohol occurs with aging.

Fals-Stewart, W., & Lucente, S. (1994). Effect of neurocognitive status and personality functioning on length of stay in residential substance abuse treatment: An integrative study. *Psychology of Addictive Behaviours, 8*(3), 179-190.

Glass, I. B. (1991). Alcoholic brain damage: What does it mean to patients? *British Journal of Addiction, 86*, 819-821.

Hage, M. (1991, September). Alcohol and brain damage. *Substance*, 3-5.

Lezak, M. D. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford University Press.

Mann, K., Batra, A., Günthner, A, & Schroth, G. (1992). Do women develop alcoholic brani damage more readily than men? *Alcoholism: Clinical & Experimental Research, 16*(6), 1052-1056.

This study of 51 males and 14 females who were alcoholics found no significant difference in liver damage between sexes. Degree of brain shrinkage was also not significantly different between sexes, however, the females in this study had a history of consuming significantly less alcohol than the male group. The authors conclude that female have a greater vulnerability to brain damage due to alcohol abuse.

Neiman, J. (1998). Alcohol as a risk factor for brain damage: Neurologic aspects. *Alcoholism: Clinical & Experimental Research, 22*(7), 346S-351S.

Discusses immediate and long term effects of alcohol on the brain. Effects of ethanol, withdrawal, nutritional deficits, electrolyte disturbances, and liver damage are reviewed in terms of potential contribution to brain damage. Interaction with illicit drugs is also discussed.

Rourke, S. B. (1995, June). The natural history of alcoholism: Diagnostic issues, neuropsychological assessment, treatment implications and clinical case presentations. Paper presented at the Alcohol Research Seminar, University of New England, Armidale, Australia.

Rourke, S. B., & Løberg, T. (1996). The neurobehavioural correlates of alcoholism. In I. Grant & K. M. Adams (Eds.), *Neurospychological assessment of neuropsychiatric disorders* (2nd ed, pp. 423-485). New York: Oxford University Press.

Ryan, C., & Butters, N. (1980). Learning and memory impairments in young and old alcoholics: Evidence for the premature-aging hypothesis. *Alcoholism: Clinical & Experimental Research, 4*, 288-293.

Walsh, K. (1983). Alcohol-related rain damage: An hypothesis. *Australian Alcohol & Drug Review, 2*(1), 84.

Walsh, K. W. (1985). *Understanding brain damage: A primer of neuropsychological evaluation.* Edinburgh: Churchill Livingston.