IMPAIRMENT OF SELF-MONITORING AND VIGILANCE IN ALCOHOLICS

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Abstract

Self monitoring seems to depend on appropriate memory and attention processes. In chronic alcoholics these processes may be impaired due to neural pathology. Therefore, chronic alcoholics may be impaired in self-monitoring abilities. This hypothesis was tested using 19 chronic alcoholiagnc subjects matched for age, gender (16 males and 3 females), and socio-economic status with 19 non-alcoholic subjects. Two experimental tasks were (a) self-monitoring arm-lifting behaviour and (b) a VDU-based vigilance task, each with two levels of disction. Subjects responded via a hand-held push-button, and all data was automatically collected via computer. All subjects were assessed with the Rey Auditory Verbal Learning Test and the Austin Maze test prior to the experiment. The hypotheses were that alcoholics would be more impaired at both experimental tasks than controls, and that there would be a further impairment due to the effect of the higher level of distraction for the alcoholics. The main hypothesis received some support in that the alcoholics showed impairment on self-monitoring compared to controls, were poorer at self-monitoring than vigilance, and were further impaired during the higher distraction level. The control group however did not find the self-monitoring task more difficult than the vigilance task, although they were poorer on both tasks under the higher level of distraction. The

alcoholic group was significantly impaired in performance of RAVLT and Austin Maze test compared to controls. Results are discussed in relation to possible memory and attention deficits and how these relate to pathology in various diencephalic regions. Methodological weaknesses in the experiment are discussed, an improved design is suggested, and clinical implications relating to remediation and recovery of function are examined in the light of recent research.

CHAPTER 1

Introduction

It is commonly believed by clinicians that alcoholics underestimate the amount of alcohol they consume. One reason for this may be the immediate effects of the alcohol. Another reason may be that they do not want to admit to drinking excessively. However, a third reason for such an underestimation may be that they are impaired in their ability to monitor this consumption. Kanfer (1970) proposed a 3 stage mediational model of self-regulation, the first stage of which is self-monitoring. This thesis examines the hypothesis that alcoholics are unable to adequately self-regulate consumption behaviour because they are impaired in the ability to self-monitor this behaviour.

This thesis also proposes that chronic alcoholics frequently demonstrate cognitive deficits upon which the process of self-monitoring is dependent. Several studies (eg. Walsh, 1985; Walsh, 1987; Luria, 1980; Parsons, 1987; Butters and Granholm, 1987) have suggested that alcoholics frequently present memory impairment at both the coding and decoding stages, and that they often fail to maintain sustained attention for even short periods of time. It is proposed that appropriate memory coding and retrieval and the maintenance of attention are essential to self-monitor

behaviour accurately and because alcoholics may be deficient in one or both of these faculties they are frequently impaired in the ability to self-monitor their consumptive behaviour.

This thesis therefore will attempt to show that chronic alcoholics are impaired in their ability to monitor proprioceptive behaviour as compared to a control group. In addition it will attempt to replicate previous findings that chronic alcoholics present a memory deficit, an attention deficit, and are impaired in their ability to learn new behaviours.

In the next chapter the role of the self-monitoring stage is placed in the context of the overall process of self-regulation. In subsequent chapters, the neuropsychological roles played by certain diencephalic regions is discussed with special regard to the cognitive functions of memory and attention, functions that are proposed to be essential in the process of self-monitoring. Frequent pathology found in chronic alcoholics that affects memory and attention is described, and this forms a premise to the argument that deficits in these cognitive functions may contribute to impairment in self-monitoring ability in alcoholics. Variables that affect memory and attention are discussed, and the rationale for the selection of two instruments that examine memory, attention and learning deficits is outlined. Finally, a study is

described that investigates the correlation of self-monitoring impairment and deficits in memory and attention.

Chapter 2

Self-regulation

An important general process, of which self-monitoring is one component is self-regulation. Kanfer (1970) and Luria (1973, 1980) have suggested that all healthy humans constantly process information from external and proprioceptive sources to evaluate their interaction with the environment. Kanfer(1970) first proposed a model of self-regulation that consists of three stages. These stages are:-

Stage 1. Self-Monitoring. Comparing present conditions to predetermined 'acceptable' conditions.

Stage 2. Self-Evaluation. Evaluating the direction and degree of change to take place.

Stage 3. Self-Correction. Modifying specific behaviour programmes to respond more appropriately to achieve desired goals.

2.1 Self-monitoring

Self-monitoring involves the perception and collation of both internal and external stimuli. Since the effective and proper operation of any information

processing system, including one of self-regulation, depends on precise information input, efficient operation of the first stage, the self-monitoring process is essential for effective self-regulation.

The ability to self-monitor behaviour has been posited by some researchers as playing a critical role in general self-regulatory behaviour (Heilbrun, Cassidy, Diehl, Haas and Heilbrun, 1986; Heilbrun, Tarbox and Madison, 1979; Luria, 1980; Luria, 1973). An alcoholic with an impairment in the ability to self-monitor the rate of consumption of alcohol, or the proprioceptive behaviour associated with drinking, such as arm-lifting, may have difficulty in tracking increasing states of intoxication. Such a difficulty may be expected to result in severe inebriation.

Heilbrun et al, (1986) and Heilbrun et al (1979) have suggested that both acute and chronic alcoholics are less sensitive to internal stimulation and are less able to utilise internally generated information. Lovibond and Caddy (1970) have suggested that alcoholics are impaired in their ability to discriminate their own blood alcohol concentration (BAL), but can be trained to do so. Although interoceptive monitoring is obviously critical in alcohol consumption, this thesis addresses proprioceptive self-monitoring exclusively.

According to the Kanfer (1970) model, in order to recognise the onset of a state of alcohol intoxication, then to self-evaluate, and then finally to self-regulate alcohol consumption, the drinker needs to be able to recognise the signs of that intoxication either through physiological changes (eg. loss of motor coordination, elevated body temperature, reduced muscle tone), or through changes in behaviour (eg. loss of inhibition, agressiveness, etc.), or both, that is, he needs to accurately self-monitor his behavioural and proprioceptive signals.

2.2 Self-evaluation

If motivation exists to self-regulate alcohol consumption, the next stage involves a comparison of coded memory representations relating to the physiochemical state of being sober and of being inebriated. This requires the ability to recall these representations of sober condition and of intoxicated condition. Several researchers have indicated that chronic alcoholics are impaired in their ability to retrieve memory traces. Furthermore, alcoholics may also present a deficit in the memory trace coding mechanism which may then hinder the ability to retrieve what may be poorly coded or incomplete memory traces (Kopelman, 1988; Kopelman, 1985; Cermak, Talbot, Chandler, and Reale Wolbarst, 1985; Butters, Wolfe, Granholm and Martone; 1986).

To maintain such an awareness it is essential that the alcohol abuser hold in memory coded representations of the proprioceptive and behavioural signals relevant to both the states of sobriety and intoxication. Futhermore, he is required to maintain selective attention to detect the occurrence of these cues even when distracting stimuli may be present in a social setting. Butters and Cermak (1976) present evidence to suggest that Korsakoff patients compared to matched alcoholics performed more poorly on a finger maze task (when subjects were blindfolded) and showed a deficit in short-term retention motor movement (which assessed proprioceptive awareness of an arm movement. However, no comparison was made to matched non-alcoholics in this study. It should also be mentioned that several studies and reviews have questioned the validity of a diagnostic distinction between Korsakoff syndrome and chronic alcoholism (see Torvik, Lindboe and Rogde, 1982; Harper, 1983; Harper, Gold, Rodriguez and Perdices, 1989; Bowden, 1990).

2.3 Self-correction

Once an evaluation has been made based on the comparison of the present condition to the desired condition, Kanfer (1970) suggests that the self-correction of the behaviour responsible for the difference between the present state and the ideal

state is addressed according to a behaviourist paradigm. Kanfer suggests that depending on the degree of difference this reinforcement may be of a negative or a positive nature. Although Kanfer (1970) claims that the self-correction stage is critical in the self-modification of behaviour, the primary concern of this thesis is with the self-monitoring stage, thus a comprehensive discussion of the self-correction stage is beyond the scope of the study.

Chapter 3

This chapter describes the cerebral pathology frequently found in alcoholics and the effects that this pathology may have on the cognitive functions of attention and memory. It also shows that deficits in these cognitive functions may impair the ability to self-monitor behaviour.

3.1 Common Cerebral pathology in alcoholics

Victor and Adams (1985), and Harper, Gold, Rodriguez and Percides (1989) propose that the most frequent brain damage seen in alcoholics is associated with the Wernicke-Korsakoff syndrome, and that the pathology usually involves lesions in the thalamus, hypothalamus, brain stem and cerebellum. In addition, the mamillary bodies also are almost always affected (Harper, 1979; Walsh, 1985). Lishman (1986) also, has suggested that neuropathy associated with alcoholism is frequently quite extensive and widespread. Other neuropathology associated with chronic alcoholism may commonly include lesions in gray matter near the third and fourth ventricles, ventricular enlargements, cerebellar atrophy (Wilkinson, 1982), and damage in the diencephalic regions of the hippocampus (Mishkin, 1982; Torvik, Lindboe and Rodge, 1982).

Walsh (1985) has proposed that many of the alcoholics presenting widespread diencephalic pathology show associated cerebral atrophy in the frontal lobes. Courville (1955) reported general cortical atrophy but predominantly in the upper part of the dorsolateral surface of the frontal lobes. Cala, Jones, Mastaglia and Wiley (1979) contend that 73% of patients diagnosed as chronic alcoholic suffered diffuse bilateral atrophy, and most of this was in the frontal lobes. Harper, Kril and Daly (1987) compared the brains of alcoholics and matched controls and found that the alcoholics showed an 18% reduction in neurons in the frontal cortex, and although their data suggested no significant loss in the motor cortex they indicated a reduction in neuron size in both areas.

It should be emphasised that although the frontal lobes may often be affected in alcoholics, other more widespread damage involving the brainstem, the cerebellum and the hippocampus may be more common and often more severe. In fact, the view that alcohol-related neuropathy frequently involves the frontal lobes (eg. Cala et al, 1979) is not without critics. Wilkinson (1987) cautions that much of the evidence cited in the literature associating frontal lobe atrophy with chronic alcoholism is based on Computerised Tomography imaging. In 1982 he highlighted the inaccuracies and weaknesses inherent in

some of the earlier CT imaging procedures and the validity of evidence based on these earlier studies has been thrown into doubt (Wilkinson, 1982). Grant (1987) also, has suggested that improved CT techniques have revealed artifactual contaminations possibly influential in the earlier CT studies. Harper and Kril (1986) have observed that CT scan evidence indicating severe frontal lobe shrinkage appears exaggerated when compared to shrinkage assessed at autopsy. Other researchers such as Torvik, Lindboe and Rogde (1982) and Victor and Adams (1985) have specifically criticised the Courville (1955) findings as of questionable significance.

Allowing for the criticism of the Courville (1955) findings by these authors, Wilkinson (1987) and Ron (1983, 1987) however suggest "two etiologically, functionally, and neuroradiologically distinct types of brain abnormality in alcoholics", Wilkinson (1987, p 88): (i) the Wernicke-Korsakoff syndrome with typical lesions in the mamillary bodies and the thalamus; and (ii) the profile of neuropathy in the cortex, most severe in the frontal lobes, as outlined by Courville (1955). Wilkinson (1987) proposes that these two organic syndromes are not mutually exclusive, and Ron (1987) suggests that they should be considered as part of a continuum. Wilkinson (1987) further contends that these two neuropsychological syndromes may develop in parallel. However, Bowden (1990) has questioned the

validity of the conclusion that there are two separate disease processes. He claims that a nosological distinction is unjustified because it is based, for example, on the observation that the behavioural correlates of one of the proposed separate syndromes are eliminated by the partialling-out of the age factor (Bergman, 1987). Bowden (1990) suggests that the age factor may be more easily separable from the behavioural correlates of the cortical CT indices than from the CT indices associated with the thalamic-ventricular pathology. The proposition by Wilkinson (1987) and Ron (1987) that there may develop two nosologically distinct organic syndromes in alcoholics therefore requires further evidence.

3.2 Neuropsychological deficits associated with alcohol-related organic syndromes.

Parsons and Farr (1981) present a comprehensive review of studies addressing the neuropsychological deficits associated with chronic alcohol abuse as assessed using the Halstead-Reitan Neuropsychological Battery (HRB). Performance by alcoholics, as compared to brain-damaged and control subjects, was impaired on a majority of the subtests of the HRB as cited by most of the studies reviewed. However, the performance of the alcoholics approximated that of the brain-damaged subjects moreso than that of the controls. Further comparison of

scores of the performance and verbal subtests of the Wechsler Adult Intelligence Scale (WAIS) suggests alcoholics are consistently impaired on the performance subtests across the full range of studies.

Oscar-Berman (1987) and Parsons (1987) suggest that the most frequent neuropsychological functions impaired in alcoholics involve problem-solving, abstracting, processing of perceptual-spatial stimuli, episodic and topographical memory, verbal-linguistic learning, and the learning of new skills. Butters and Granholm (1987) propose that the most severe deficit presented by the alcoholic Korsakoff patient is the inability to learn new verbal or nonverbal information (anterograde amnesia). They also indicate that there is frequently a deficit in episodic memory. Butters and Granholm (1987) suggest that the neuropathological basis for anterograde and retrograde amnesia lies in the medial diencephalon, and the visuoperceptive and problem-solving deficits frequent in the alcoholic Korsakoff syndrome are due to pathology in the association cortex. Victor, Adams and Collins (1971), and Butters (1984) proposed that the region of the thalamus is critical to memory and learning. Oscar-Berman and Bonner (1985) and Oscar-Berman (1987) have also contended that selective attention deficits are frequent in patients with Korsakoff's syndrome.

In summary, the consensus of research opinion is that

chronic alcoholics frequently present neuropsychological deficits associated with problem-solving, episodic and topographical memory, learning, and selective attention. The importance of attention and memory in the process of self-monitoring is now examined.

3.3 Attention.

In some of his earlier work, Luria (1966) suggested that there are two states of attention that affect accurate processing of information and execution of action programs. The first and most basic form of attention is Elementary Attention, or the 'waking' state. The second form of attention is selective attention where specific stimuli are monitored in the absence or presence of distractions. Luria (1973), Milner (1965) and Porteus (1965) have also suggested that the speech centres of the frontal lobes can play a contributory role in the maintenance of specific and elementary attention.

3.3.1 Elementary Attention

Evidence by Lindsley et al. (1949) and Luria (1980) suggest that the Ascending Reticular Activating System (ARAS) is one of the most important systems in elementary and generalised forms of attention. In

animals, division of this system induces sleep, and stimulation causes increased vigilance and increased sensation (Lindsley, 1960). Lesions in the upper part of the brainstem cause a drowsy state reducing cortical tone, and severely disturbing the waking, selective state of consciousness. The ARAS and mechanisms of the superior brain stem are responsible only for the generalised state of waking (Luria, 1966, 1980).

Wernicke-Korsakoff patients frequently present pathology in this region of the brain stem (Mayes, Meudell, Mann and Pickering, 1988; Kopelman, 1988; Luria, 1980, 1973), and this is recognisable as a drowsy, inactive predisposition (Victor and Adams, 1985).

3.3.2 Selective Attention.

The second form of attention, selective attention, however, seems to be maintained through co-operation of the limbic system and the frontal lobes (Walsh, 1987, Stuss and Benson, 1986). Boring (1970) defines that this type of attention, requires that specific stimuli be 'selectively recognised', and that irrelevant stimuli be ignored, (and/or that responses to these irrelevant stimuli be inhibited).

The frontal lobes are responsible for the formation of

intentions and programmes, and the regulation and verification of higher mental behaviours (Luria, 1966, 1973; Duncan, 1986; Shallice, 1982; Wilkins, Shallice and McCarthy, 1987). To enable the frontal lobes to do this, they are distinguished by rich systems of connections with virtually all other areas of the diencephalon (including the lower levels such as the medial and ventral nuclei and pulvinar of the thalamus)(Luria, 1966). These connections receive impulses from, and send to, almost all parts of the brain (Luria, 1980). It is therefore suggested that lesions in any regions of the brain cooperating to execute a particular function (such as attention) may to some degree handicap the frontal lobes in their mediation of that function.

Luria (1966) has shown that the animal with frontal lobotomy presents with very limited goal directed behaviour. It is distracted by all irrelevant stimuli upsetting any plans and programmes of its behaviour making them fragmentary and uncontrolled. It's voluntary attention is therefore often disrupted by distracting stimuli. Frontal lobe damage results in disturbance of the ability to inhibit orienting reflexes to distracting stimuli. Malmo (1974) has shown that this leads to inability to perform tasks involving delayed responses under distracting conditions, but this ability is restored if distracting

stimuli are removed. Butters and Cermak (1980) confirm that Korsakoff patients are handicapped severely by distractors when undertaking a task involving learning new material, and that their performance inproves when proactive interference is reduced or eliminated.

Wilkins, Shallice and McCarthy (1987) suggested that lesions in the frontal lobes, as well as atrophy in this region results in increased distractability as well as heightened impulsiveness and a reduced ability to maintain sustained attention to simple vigilance tasks.

Walsh (1985) emphasises that long-term alcohol abusers seem to be particularly susceptible to distraction by irrelevant stimuli, and their poor performance on the Austin Maze test suggests that they experience severe difficulties with formation and execution of intentions. However an alternative explanation for this poor performance on maze tests has been proposed by De Renzi (1982) and Milner (1965) who suggest that the damage to the right hippocampus and amygdala results in a deficit in topographical memory. Such pathology is commonly associated with the Wernicke-Korsakoff syndrome.

3.3.3 Effects of the speech centre on attention.

Luria (1973) has concluded from extensive study of patients with injury to the posterio-lateral sections of the frontal lobes that the speech centres assume a major role in the functioning of the frontal lobes.

He emphasises that a spoken instruction will result in an orienting response in the non-damaged human brain. The orienting response in this case can be seen as a means of reestablishing sustained attention, after distraction has occured, or as a means of maintaining attention while performing a task. Porteus (1965) and Milner (1965, 1969) have suggested that self-talk may be a useful strategy in maitaining attention while non-damaged persons are traversing a maze. However, if pathology exists in or near the medial zones of the frontal cortex (of the left hemisphere) attention is frequently not sustained by means of self-talk (Porteus, 1965).

Luria (1966, 1980) has suggested that if the medial zones of the frontal cortex are damaged, this orienting response becomes either very unstable, or fails to appear at all. This further suggests that the frontal lobes participate in the regulation of activation processes lying at the basis of voluntary attention (Luria, 1973).

The finding that spoken commands also arouse the orienting response is further evidence that the speech centres play a crucial interactive role with the frontal lobes, since this does not happen when there is frontal lobe damage, although it does happen when there is damage to the posterior zones (Luria, 1980).

A person with this type of problem may be given some mental task, but be continually distracted by irrelevant and vicarious stimuli, so that they cannot complete (by being unable to attend to) the task (Milner, 1964; Luria, 1966; Stuss and Benson, 1984).

3.4 Memory.

The direct connections of the frontal lobes to the medial sections of the thalamus may directly involve the executive functions of the frontal lobes in the processing, ie. coding, retention and retrieval, of memory traces (Stuss and Benson, 1984).

In the study by Cala et al (1979) almost three quarters of the patients diagnosed as chronic alcoholics presented considerable bilateral cortical atrophy, and although some of these were free of amnesia, most presented the General Amnesic Syndrome typified by a total inability to learn new material, whether presented verbally or nonverbally, and irrespective of

the modality of presentation. If something appeared to be learnt it was soon forgotten. Most subjects also showed cognitive deficits that were revealed best by digit symbol substitution, block design, and object assembly subtests of the WAIS. There were no significant correlations between cortical atrophy and the other subtests of the WAIS suggesting ambiguity regarding the relationship between this condition of pathology and intelligence.

As Luria (1980) has suggested, one fundamental condition for the imprinting of memory traces is the maintenance of sufficient cortical tone, ie. elementary attention. If this cortical tone is lowered, according to Stuss and Benson (1984) the retention and recall of traces is affected.

Luria (1966) has proposed that the efficiency of recall from memory depends on:-

- the degree of cortical tone or a state of vigilance,

- the degree of intention of what is required to be recalled,

- the integrity of the cortical zones that carry the analysers.

He goes on to say that these analysers must be able to categorise the incoming information into the modally-specific cues (auditory, visual, tactile), select the relevant cues, and then assemble them into dynamic structures. Many of these analysers are concerned with coding through the use of the language functions (Stuss and Benson, 1986; Walsh, 1987). Since many different cortical and subcortical zones are involved with the processing of traces, any pathology in any of these zones may give rise to defects in the processes of retention and recall in different ways.

Since some chronic alcoholics may suffer considerable widespread neuropathology (whether diagnosed as Wernicke-Korsakoff patients or not, see Harper et al, 1986; Bowden, 1990) they may be unable to satisfactorily meet some or any of the conditions mentioned above. Their ability to code and retrieve memory traces may as a result often be hindered.

It has been found that lesions of the medial zones of the temporal lobes and the mammilary bodies (the relay nuclei for fibres running from the hippocampus to the 'circle of Papez') (Grunthal, 1939), result in severe disturbances of memory by disturbing the patient's general ability to imprint traces of current experience. This is typically the case in the Wernicke-Korsakoff syndrome. Because all of these regions are involved in the control and modulation of

cortical tone, disturbance of this control via lesions may affect the selective imprinting of traces. Walsh (1987) has stated that a large percentage of patients suffering from alcoholism that present neural pathology exhibit damage to the mammilary bodies, to regions around the third and fourth ventricle, and perhaps less relevant to the issue of cortical tone, to areas involving the hippocampus and amygdala.

Luria (1966, 1973, 1980) presents much clinical data to support the contention that persons with deep lesions that affect the primary elements of trace formation are unable to recall information not because of rapid and spontaneous decay of traces, but because of mutual inhibition of traces by other non-modality specific information input. He thus claims that the traces in these subjects do not decay more rapidly than in other (normal) people but new stimuli that follow form new traces that then inhibit the recall of the old traces (retroactive interference).

Patients with neural damage limited to the frontal lobes are unlikely to suffer damage in memory mechanisms that form and code traces (Luria, 1980; Stuss and Benson, 1984). However, Luria (1973, 1980) and Jetter, Poser, Freeman and Markowitch (1986) suggest that lesions here often lead to gross disturbances in the ability to form intentions and plans, disturbance of the formation of behaviour

programs, and an inability to focus attention on stimuli to be remembered. Therefore, patients cannot form stable intentions to memorise information, nor can they devise means of assisting in the process of memorising (for example, by devising mnemonic aids (Moscovitch, 1982)). If concurrent with such neural damage the patient also suffers neuropathy in brainstem and other subcortical regions such as the hippocampal system, memory deficits are likely to be further exaggerated.

Several researchers (eg. Luria, 1973; Walsh, 1985; Stuss and Benson, 1983, 1984) have proposed that it is essential to the process of self-regulation that certain cortical regions function cooperatively. These authors suggest that because the frontal lobes are responsible for the generation of action programs and the maintenance of selective attention they need to operate in harmony with the limbic system and the brain stem. It is important to emphasise that lesions in either cortical or subcortical regions may result in malfunction and therefore deficits in the processes subsumed by the integration of these regions.

Deficits in memory or attention may therefore result from pathology in either the brainstem, the limbic system or the frontal lobes, or damage to all three of these regions (Walsh, 1985, 1987). Memory coding deficits may result from inadequate elementary

attention due to lesions involving the ARAS in the brainstem (Luria, 1980). Other memory deficits may occur due to damage to the hippocampus and/or amygdala (De Renzi, 1982; Mishkin, 1982). Selective attention, to ensure adequate coding and transportation to storage, or the use of mnemonic techniques in the attempt to memorise material, may be affected by loss in the frontal cortex (Stuss and Benson, 1984; Moscovitch, 1982).

The evidence presented in this section indicates at least three explanations for the memory difficulties of chronic alcoholics. Neural damage that involves (1) the brainstem, (2) the hippocampus and/or amygdala, and (3) the frontal cortex, may affect the ability to code and retrieve memory traces. Indeed, diffuse damage to the brainstem and the limbic system is more frequently cited as typical pathology associated with alcoholism than damage to the frontal lobes. There are therefore several explanations that endeavour to account for the neuropsychological deficits associated with alcoholism.

3.5 Cognitive facilities required to self-monitor.

Kazdin (1974) has suggested that there are two cognitive facilities that are required to self-monitor behaviour: the maintenance of sustained attention, and accurate coding and decoding of memory traces. Despite distractions, the subject must be able to sustain selective attention to enable accurate tracking of the stimulus and must, of course, remember what the expected stimulus is, and how to respond to indicate that he has detected the stimulus. This requires that complete memory traces have been formed and that retention and retrieval mechanisms are intact. House, Manelis and Kinscherf (1983) have suggested that these functions are employed during a vigilance task, and that self-monitoring therefore functionally resembles experimental vigilance paradigms. McCarthy (1989) has suggested that vigilance tasks are often less cognitively demanding than self-monitoring tasks perhaps because the stereotypic nature of proprioceptive behaviour often renders it less noticable than an externally generated stimulus, eg. a display on a VDU screen.

The control of attention and memory, therefore appear to play an important role in the process of

self-monitoring. Because of the frequent cortical and subcortical pathology presented by chronic alcoholics of both the diagnosed and the 'sub-clinical' Wernicke-Korsakoff types, deficits in memory and attention may arise due to pathology in a number of locations. The ability to self-monitor behaviour, requiring adequate maintenance of attention and appropriate utilisation of memory processes, may therefore be impaired in chronic alcoholics.

The next chapter examines methodological issues that need to be considered in the assessment of attention and memory. Variables such as age, gender, diet and intellectual ability have been proposed as qualitatively affecting either memory or attention processes, and sometimes both. These variables are briefly examined since their control may be an essential element in a study that investigates deficits in such cognitive functions. Also, since it is desirable to assess memory and attention abilities independently of the assessment of self-monitoring abilities the rationales of two tests that have been proposed as qualitatively investigating these functions are discussed.

CHAPTER 4

Methodological and assessment issues

A number of studies have examined the effects of age, diet, intelligence, and gender on cognitive functions. Some of the investigations that have addressed the effects of these variables on memory and attention are briefly discussed in this chapter.

4.1 Age

Lezak (1983) has suggested that the severity of brain damage on cognitive functions tends to increase as age increases. Contrary to popular belief, normal aging does not affect the immediate memory span (Goldstein and Shelly, 1973), nor the primary, working memory capacity (Erickson, 1978). However, storage and retrieval problems tend to occur with advancing age (Botwinick, 1981). Lezak (1983) has proposed that the main reason why elderly individuals often perform poorly on memory tasks is because of the speed factor involved in these tests.

Deficits in selective attention have been demonstrated in alcoholic patients with Korsakoff's disease, in non-amnesic alcoholics, and in normal aging individuals (Oscar-Berman, 1980; Oscar-Berman, 1984). It has be suggested that certain neuropsychological deficits

associated with alcoholism share a similarity with those of aging (Wood and Elias, 1982) and Ryan (1982) has suggested that this similarity in cognitive decline may be attributable to a premature aging of the central nervous system in chronic alcoholics. These researchers have demonstrated increases in both memory and attention deficits with increasing ages of non-clinical non-alcoholic individuals and similar impairments with non-amnesic chronic alcoholics of younger ages than the non-alcoholic subjects. However, not all studies have replicated this finding (eg. Oscar-Berman and Bonner, 1985).

4.2 Intelligence

Since memory and attention are cognitive functions that are intrinsically assessed with conventional intelligence tests, defects in either of these functions may depress the overall performance on these tests. Although alcoholics who have been abstinent for as short a period as one month often perform at a considerably improved rate compared to their performance immediately after alcohol withdrawal (Loberg, 1986), and sometimes within the normal range (Goldman, 1983), the effects of attention deficits and memory impairment due to alcohol related pathology cannot be easily determined (Lezak, 1983).

Walsh (1985) suggests that the subtests of the WAIS-R often fail to elicit the subtle deteriorations of intellectual functions associated with frontal lobe pathology found in chronic alcoholics. Performance of alcoholics on the verbal subtests of the WAIS is usually not significantly different to that of controls (Loberg, 1986). The most frequently found difference in WAIS measures is that of a reduction in scores of the performance subtests as compared to those of the verbal subtests (Walsh, 1985; Parsons and Farr, 1981; Loberg, 1986; Grant, 1987). The collection of the results from various studies by Parsons and Farr (1981) seems to indicate that the scores on the performance subtests of the WAIS-R such as Block Design, Object Assembly, Digit Symbol and Picture Arrangement are the most frequently affected in alcoholics, perhaps representing deficits in the visuo-spatial, visuo-perceptive processing domains (Brandt and Butters, 1986). Jarho (1973) has suggested that these deficits, like the memory disorders, are due to damage in the brainstem, specifically near the third ventricle, whereas Parsons (1975) has pointed to atrophied cortical areas as the loci of these deficits.

Although performance scores on the WAIS are usually found to be poorer than the verbal scores, reports that have failed to replicate this pattern have been published. For example, Kopelman (1985) found that

alcoholic Korsakoff patients' performance on the WAIS was comparable to that of age-matched controls, as did Butters et al (1985). When this differential is not found in a group of alcoholic subjects (eg. Fitzhugh, Fitzhugh and Reitan, 1965) it may indicate normal performance on the WAIS, suggesting no decline in intellectual functioning (Butters and Cermak, 1980).

4.3 Gender

Various studies have considered gender differences in response to alcohol. Some report notable performance differences for chronic alcoholics. For example, Fabian et al (1981) found that women, both alcoholic and control, performed better on the memory component of the Tactual Performance Test (Reitan, 1964). A study by Niaura, Nathan, Frankenstein, Shapiro and Brick (1987) investigated gender differences in acute response to alcohol. They found that when relative dose of alcohol was controlled (absolute alcohol/ kg of body weight) only the recovery rate of memory functioning favoured men over women. These authors found no significant differences on measures of attention, pursuit tracking ability and subjective level of intoxication. However, Jones and Jones (1976) found females impaired in comparison to males on an immediate recall memory task. Burns and Moskowitz (1978) found gender differences in favour of males in

acute response to alcohol on measures of delayed memory, subjective impairment and divided attention.

Inglis and Lawson (1981) drew together the results of 14 studies involving men and women with left and right-sided lesions (not alcoholics) and concluded from these studies that while men presented the expected deficits in performance tests compared to verbal tests, women did not present this pattern of impairment in performance tests. Silberstein and Parsons (1979) proposed that the lateralisation in brain organisation seems to be less pronounced in women than in men since the alcoholic women were less impaired in visuo-spatial type tests than the alcoholic men. The evidence for gender differences in the cognitive domain during intoxication therefore appears to be tentative and the issue warrants further investigation. The question of gender differences in neuropsychological performance after abstinence also needs to be further addressed (Inglis and Lawson, 1981).

4.4 Diet

Lishman (1981) has suggested that the psychotic stage of Korsakoff-Wernicke syndrome may be due to a nutritional deficiency of vitamin B1 (thiamine). Lezak (1983) points out that the diet of chronic alcoholics during periods of binge drinking is often insufficient to sustain the body's thiamine needs. Freund (1982)

and Butters (1981) link a thiamine deficiency in alcoholics directly with alcohol toxicity. Lishman (1981) and Victor and Adams (1985) also suggest that the depletion of another vitamin (nicotinic acid) may be involved in the confusional disorder that alcoholics frequently experience. However, Victor and Adams (1985) suggest that this condition (pellagra) is far less common than Wernicke-Korsakoff disease and can occur in non-alcoholics (because of its specific dietary aetiology). Guthrie and Eliot (1980) have observed a high incidence of malnutrition in chronic alcoholics and that the more malnourished the subject the more cognitive deficits such as memory difficulties and inability to maintain attention appear to be displayed. Ryan and Butters (1982) in a review of the literature have concluded that three weeks after a balanced diet has been administered, and the subject has been abstinent, there is a recovery of many of these cognitive functions, some of which return to normal.

4.5 Assessment of memory and attention deficits

It has so far been argued that both voluntary attention to, and memory of, the target stimulus is necessary for the process of self-monitoring a specific behaviour. It has been shown that with specific types of pathology

a deficit in any of the many roles of voluntary attention (eg. the maintenance of elevated cortical tone, or the formation of intentions) will indirectly affect the quality of coding and retrieval of memory traces. This may be the case even if the mechanisms used in coding and retrieval are still intact. But because of the widespread pathology (in other zones as well as the frontal lobes) often found with chronic alcoholism, these primary mechanisms of memory may also be considerably damaged.

4.5.1 The Rey Auditory Verbal Learning Task (RAVLT)

The Rey Auditory-Verbal Learning Test (RAVLT) (Rey, 1964) has been suggested by Lezak (1983) as a useful indicator of memory deficits when applied to a task of learning a short word list. The RAVLT is not denoted as a memory assessment instrument specifically for patients with frontal-lobe pathology although the formation of strategies of recall and maintenance of attention are assumed to be frontal lobe functions. Lezak suggests that this test measures immediate memory span, provides a learning curve, elicits retroactive and proactive interference tendencies and tendencies toward confusion and confabulation. Many chronic alcoholics, either diagnosed as Wernicke-Korsakoff patients or suspected of presenting a subclinical Wernicke-Korsakoff syndrome present memory deficits of

this nature. This test therefore appears to be a suitable indicator of memory deficits frequently found in subclinical Wernicke Korsakoff syndromes (Bowden, 1990). There are therefore three reasons why this test was incorporated in this study. Firstly, the finding that the RAVLT seems to reveal these memory deficts adequately. Secondly, Lezak (1983) suggests that subjects that repeat several of the stimuli words (words to be recalled) a number of times during the recall phase of each of the five trials may reflect a problem in self-monitoring and tracking of their responses during a learning task. Thirdly, the RAVLT is also believed to be able to elicit retention and retrieval problems, because a recognition task is also included. Lezak cites her own norms and those of O'Brien and Lezak (1981) as indicators of retention and retrieval problems of various non-damaged groups and traumatically brain injured patients.

4.5.2 The Austin Maze

Walsh (1985) has suggested that the Austin Maze is an adequate indicator of frontal lobe damage and of deficits in various executive functions. The inability to erradicate errors while traversing the maze despite frequent verbalisation of instructions, and the inability to observe instructional rules (to the extent that these rules are constantly broken, even though the subject can verbalise these rules) are frequent, almost

typical, deficits presented by chronic alcoholics. This claim is supported by previous literature (Konow and Pribram, 1970; Tarter, 1973; Hunt, 1979).

However, it should be cautioned that other researchers have found that maze learning deficits occur with lesions in almost all areas of the right hemisphere (Milner, 1965; De Renzi, 1982). Even if the executive functions mentioned by Walsh (1985) are intact, it appears that pathology in other contributory regions may give rise to poor performance on maze tests. Milner (1965) for example found that patients with lesions in the right temporal lobes, and with ablations of this lobe when it included the hippocampus performed much more poorly than patients who had similar operations on the left temporal lobe (these did not differ from normals). When one considers that a body of evidence (see De Renzi, 1982) confidently identifies the hippocampus-amygdala axis as responsible for spatial recognition (especially on the right side) it is easily seen why damage in this region, which is frequently seen in subclinical as well as diagnosed Wernicke-Korsakoff patients, is usually associated with poor performance on maze tests, and with topographical memory disorders.

Poor performance on the Austin Maze test therefore does not neccessarily indicate frontal lobe damage since a much more likely explanation might be the commonly

found pathology in the hippocampal memory system (Harper et al, 1986; De Renzi, 1982). However, inattention, distractibility, and refusal or inability to follow frequently repeated instructions might be behaviours indicative of frontal lobe damage (Walsh, 1985) and contribute to the poor performance of many acoholics on the Austin Maze test. Thus, the evidence suggests that the overall poor performance by alcoholics on this test may be due to the cumulative effect of diverse neuropsychological deficits resulting from widespead pathology not limited to the frontal lobes.

CHAPTER 5

Hypotheses

The evidence reviewed in Chapter 3 indicates that chronic alcoholics may be impaired in their ability to self-monitor their alcohol-drinking behaviour compared to non-alcoholics. It is expected that this impairment is the result of deficits in memory and attention, and the vigilance task, the RAVLT, and the Austin Maze test are means of testing hypotheses that such deficits exist in chronic alcoholics.

The hypotheses posed are:-

1. That in comparison to non-alcoholics, chronic alcoholics will be impaired in the ability to self-monitor arm lifting behaviour and that chronic alcoholics will show more impairment in their performance of the self-monitoring task as compared to their performance on the vigilance task. This hypothesis is expected to support the House et al (1984) and the McCarthy (1989) claim that the vigilance task is less demanding than the self-monitoring task, although both these studies used non-clinical groups.

2. That the control group will also perform better during the vigilance task than during the self-monitoring task. This hypothesis is posed primarily to support the finding by House et al, and McCarthy that vigilance of external stimuli is less demanding than that of internal or proprioceptive stimuli.

3. That the alcoholic group will be more impaired on the self-monitoring task compared to the non-alcoholic group, than on the vigilance task, ie. that there will be an interaction of Groups X Detection task.

4. That when a high and low distraction condition is presented the chronic alcoholics will exhibit a greater deficit in Self-monitoring performance than in Vigilance performance, ie. that there will be a Detection task X Level of distraction interaction for the alcoholic group.

5. That for the non-alcoholics there will be a similar Detection task X Level of distraction interaction.

6. That at the higher level of distraction there will be a greater impairment in the performance of the self-monitoring task by the alcoholic group than by the non-alcoholic group, ie. that there will be

a (Groups X Detection task X Level of distraction) interaction.

In relation to the hypotheses posed to investigate memory deficits it is further proposed that the chronic alcoholic group will:-

7. Present memory deficits as elicited by the RAVLT such as depressed learning curve, retrieval and retention problems, and that these deficits will reflect the norms of the alcoholic Korsakoff patients of Butters et al (1986) and Lezak (1983) and O'Brien and Lezak (1981).

8. Present behaviour and poorer performance during the administration of the Austin Maze test that suggests a deficit in topographical memory as is frequently found in Wernicke-Korsakoff patients.

CHAPTER 6

Method

6.1 Design

A factorial design was chosen which was a 2(groups) X 2(vigilance tasks) X 2(distraction levels) with repeated measures of the vigilance tasks and distraction level.

The two groups were:-

a) chronic alcoholics diagnosed by mental health professionals according to DSM-III-R (APA, 1986) categories of Alcohol abuse and Alcohol dependence, and,

b) non-drinkers or social drinkers that did not meet the criteria for alcohol abuse according to DSM-III-R.

The two vigilance tasks were:-

a) a task in which the subject was to indicate via a button press whenever he had lifted his arm.

b) a task in which the subject was to respond via a button press whenever a computer-controlled stimulus

was presented on a VDU positioned in front of the subject.

The two distraction levels were administered via audio cassette tapes and played to the subject via stereophonic headphones. The two distraction levels were selected to present high and low distraction properties. These were:-

a) LOW - instrumental (melodic but non-lyrical) piano, flute, and acoustic guitar music played at tempos between larghetto (60 beats/min) and moderato (96 beats/min).

b) HIGH - a monologue comedy tape by a well-known comedian.

The dependent variables of interest were:-

a) accuracy of vigilance, ie. (no. of responses/no. of stimuli),

b) subjective estimate of number of responses per condition.

The subjects in each group were matched according to age, socio-economic background, and gender. The vigilance tasks were counterbalanced so that half the subjects were administered the visual task first and

the other half were presented the self-monitoring task first. The distraction tasks were also counterbalanced so that half of these received high distraction level and half received low distraction level first.

Each condition consisted of one block of 300 trials, each trial being of 3 seconds duration. Together with breaks between conditions, the experimental session lasted approximately 75 minutes.

6.2 Subjects

19 alcoholic subjects were matched for age (+5 years), sex (16 males and 3 females), and socio-economic status with 19 control subjects.

Experimental.

The experimental group (chronic alcoholic) consisted of 19 regular patients at a social centre for chronic alcoholics provided by the Tasmanian Mental Health Services Commission (TMHSC). All had been diagnosed as chronic alcoholics on admission by psychiatrists from the centre and had at some stage been interned in one of the facilities of the TMHSC. A condition of selection for this group was that all subjects had been abstinent for a period of at least one month and were receiving an appropriately balanced diet. They ranged in age from 38 to 72. The mean group age was 56.11 (SD

= 10.07).

Controls.

The control group (non-drinkers and social drinkers) consisted of 19 members of various temperance groups such as the Salvation Army, church groups, who had no history of alcoholism. The remainder were obtained by private contact from the general community or by referral from subjects. The mean group age was 54.58 (SD = 8.99), with an age range of 37 to 67.

6.3 Equipment

The test equipment included:-

a) the Austin Maze (electronic),
b) list of stimulus words/response sheets for the Rey Auditory-Verbal Learning Test.

The experimental apparatus included:-

a) an Apple II computer which collected all responses for both vigilance tasks and stored all data on floppy disk. This computer also generated the stimuli for the detection task (this stimulus was a '.').
b) two VDUs (one on which the stimulus was presented for the detection task), and one used by the experimenter to set up each condition.
c) a gravity-activated switch embedded in a towelling wrist band,

d) a hand-held push button response switch,

e) a Sanyo tape recorder with headphone outlet,

f) a set of stereophonic headphones,

g) two 60 minute cassette tapes carrying selected recordings for the low and high distraction levels.

6.4 Assessment - The Austin Maze (Walsh, 1985)

This is an electrically activated 'stepping stone' maze that was developed to study self-correcting behaviour, the ability to follow instructions, and the ability to recall visuo-spatial information. Walsh believed the first two functions to be under mediational control of the frontal lobes (as did Luria, 1973, 1980).

The ability to code visuo-spatial information into working memory is also indicated by this test ie. the length and directions of the elements of the pathway, and it therefore appears to be a valid indicator of topographical memory deficits as found frequently in diagnosed Wernicke-Korsakoff patients and/or subclinical Wernicke-Korsakoff alcoholics.

It requires the subject to learn a long pathway through a 10 X 10 grid of button switches that light up in either of two colours (green and red) to indicate a correct or incorrect movement along the path. The subject needs to learn this pathway by trial and error

initially, but normally with each succeeding trial the number of errors reduces until the subject is able to traverse the pathway twice successively without errors (criterion). Scores are normally both the number of errors per trial and the number of trials to criterion.

6.5 Assessment - The Rey Auditory-Verbal Learning Test (RAVLT)

Lezak (1983) claims that this test measures immmediate memory span, indicates the learning strategies that are being utilised, provides a learning curve, as well as eliciting retroactive and proactive interference tendencies. Any tendencies to confabulate on memory tasks are also revealed. The RAVLT also allows the differential diagnc subjects matched for age, gender (16 males and 3 females), and socio-economic status with 19 non-alcoholic subjects. Two experimental tasks were (a) self-monitoring arm-lifting behaviour and (b) a VDU-based vigilance task, each with two levels of distras that he can after each trial. This procedure allows the assessment of learning taking place after each trial. A second list of 15 words (list B) is then presented (once only) to allow an assessment of the degree of intrusion of memory traces from the first list into the recall of the second list (proactive

interference). A sixth presentation of list A is then made to establish the degree of interference of 'list B' words on the recall of the previously learnt 'list A' words. All words are concrete nouns. The words are presented at the rate of one word per second. The test is initially a measure of immediate memory span for words. For each of the 5 trials the number of words recalled are recorded in the order mentioned by the subject. The second word list is then read to the subject and he is instructed to recall as many of these words as possible. To determine whether the subject presents a coding deficit and therefore forms poor or incomplete memory traces, or a retrieval deficit signifying complete traces but the inability to access these traces, Rey (1964) suggested that a paragraph that includes all of the stimulus words from the first word list plus some distractor words be read to the subject. The measure of interest being the number of words from the stimulus list that are recognised.

Rey (1964) gives norms for trials I through V for adults according to age and social class. O' Brien and Lezak (1981) provide norms for mixed brain damaged patients and age-matched controls for trial V, trial VI, and the score on trial V minus trial VI.

6.6 Procedure

The subject's age, gender and socioeconomic status were recorded before commencement of testing. The Austin Maze test was administered first to all subjects and the number of trials to criterion (2 error-free trials in succession) were recorded as well as number of errors per trial until the criterion was reached. Comments regarding types of errors were also recorded. The Rey Auditory-Verbal Learning test was then administered and results recorded before commencement of the experiment proper.

The subject was seated in a comfortable armchair beside a coffee table carrying an ashtray, peanuts, biscuits and tea or coffee. The headphones were then placed on the subject and adjusted for comfort. The tape recorder was then switched on and the volume was adjusted by the experimenter until the subject could hear it comfortably. The tape recorder was then rewound and switched off.

The appropriate apparatus was prepared according to which condition was being run first. If a self-monitoring condition was first the gravity-activated switch was then fastened to his/her preferred arm, and the response button was placed in the subject's other hand. If a detection task was

first, the subject was shown the stimulus and it was adjusted for contrast and brightness until the subject could see it clearly. The response button was then placed in the subject's non-preferred hand. The subject was given instructions as in Appendix 3 or Appendix 4 and the tape recorder was switched on. The experimenter then left the room, returned after 15 minutes, and switched off the tape recorder (without altering the volume setting).

As the next condition as per the subject's counterbalanced order was being set up, and the subject was asked casually how many times he believed he lifted his arm or saw the stimulus, whether he wanted tea or coffee (if the next condition was a self-monitoring one), or if he/she needed to leave the room. The instructions for the next condition were then given. The appropriate recording was placed in the player and the next condition was then commenced. This procedure was repeated until all conditions were completed.

6.7 Scoring

Self-monitoring.

Since the software recorded the occurence of the stimulus (arm-lifting) and of the response (button press) in discrete 3 second intervals, it was neccessary to decide what stimulus/response latency was

to be regarded as signifying awareness of the stimulus. For this purpose, it was decided that if the subject failed to respond to the stimulus before the end of the next interval, ie. minimum of 3 seconds and maximum of 6 seconds, this was scored as a failure to self-monitor. The total number of button presses within the criterion time allowance was then calculated as a percentage of the total number of arm-raises.

Detection task.

Similarly, this task was also divided into 3-second time intervals. The same criterion for detection of the stimulus was adopted for this task. The subject was scored as having detected the stimulus if he/she pressed the response button within the stimulus interval or within the subsequent 3-second interval. The dependent variable was the percent detected within the criterion time allowance.

Subjective estimates of frequency of arm-raising and stimulus occurrence.

Each subject was asked how often he/she raised the stimulus arm during the self-monitoring sessions, or how often they saw the stimulus during the detection task. This dependent variable was again scored as a percentage of the actual number of stimulus occurrences (whether it was self-monitoring or the detection task).

Austin Maze.

The number of errors per trial were recorded for each traverse of the pathway, and the number of trials required to achieve criterion performance (2 successive error-free traverses) were also recorded. It should be noted that some of the experimental subjects showed no apparent learning during this task (ie. no reduction of errors across trials) and began to show considerable agitation, impatience and distress. When this was the case the subject was requested to complete at least 10 trials and to try to remember the path and rules as well as he could.

RAVLT.

The number of words recalled per trial were recorded for each of the 5 presentations of the first word list, the single presentation of the second word list and the 6th presentation of the first word list. Also recorded were the number of words identified as stimulus words from the recognition paragraph, as were the number of words repeated on trial V. A record was also take of words from the first list that were mentioned (transposed) as part of the second list.

CHAPTER 7

Results

The dependent variables in this study were the actual percentage correct of responses made per stimuli presented, and the subjective estimate by each subject of this accuracy or number presented. This section examines the interactions and main effects that relate to the hypotheses outlined previously.

The results of the chronic alcoholic group and the controls are then presented for the performances on the RAVLT and Austin Maze respectively, and the results of paired between group t-tests are presented for the comparison of various performances on the RAVLT.

The F statistics presented in this section are always independent univariate, either ANOVA or simple effects (Keppel, 1982, P 212). Since the present study tests multiple hypotheses it was decided to reduce the Type 1 error rate by adopting a significance level of p = .01 (two-tailed, equal to 0.005 one-tailed). Since the ANOVA yielded p values based on the hypotheses being non-directional, and the hypotheses in this study were all directional, this value of p is a further control of the Type 1 error rate. If the value of p for a particular comparison fell between p= .05 and p = .01 it was decided to follow the suggestion by Keppel

(1982, P 162-164) to suspend judgement regarding the significance of a difference, for the purpose of drawing attention to a possible effect, rather than dismissing the experimental hypothesis being tested by that particular comparison. This strategy was adopted for all analyses made in this study.

7.1 Actual scores - interactions and main effects.

The summary of the three way ANOVA of groups (alcoholics and non-alcoholics), type of detection task (self-monitoring and vigilance), and level of distraction (low and high) for the dependent variable % accuracy of responses made/ stimuli provided is presented in Table 1. There is a significant interaction between groups and task, which is graphed in Figure 1.

An analysis of simple effects showed that the chronic alcoholic group performed less accurately than the control group during the self-monitoring task, $F(1,36)=15.28$, $p < .001$. Furthermore, the simple effect indicating that the alcoholic group performed less accurately during the self-monitoring task than during the vigilance task is also significant, $F(1,36)=38.14$, $p<.001$. These findings lend support to the first hypothesis that the alcoholic group would be impaired at self-monitoring compared to the non-alcoholic group,

Table 1

Summary of ANOVA of Groups (alcoholics, non alcoholics), Type of Detection Task (Self-Monitoring, Vigilance) and Level of Distraction (High, low) for % accuracy of stimulus detection.

Source of variation	df	Sum of Squares	Mean Square	F	P
Groups	1	4221.059	4221.06	6.757	.0135
Error	36	22488.289	624.675		
Type of Task	1	6025.322	6025.32	28.34	.0000
Groups x Tasks	1	2472.164	2472.16	11.628	.0016
Error	36	7653.763	212.60		
Distraction Level	1	1717.901	1717.90	18.763	.0001
Groups x Dist'n	1	220.322	220.32	2.406	.1296
Error	36	3296.026	91.556		
Task x Dist'n	1	64.480	64.480	1.293	.2629
Groups x Tasks x Distraction	1	37.007	37.007	.742	.3946
Error	36	1794.763	49.855		

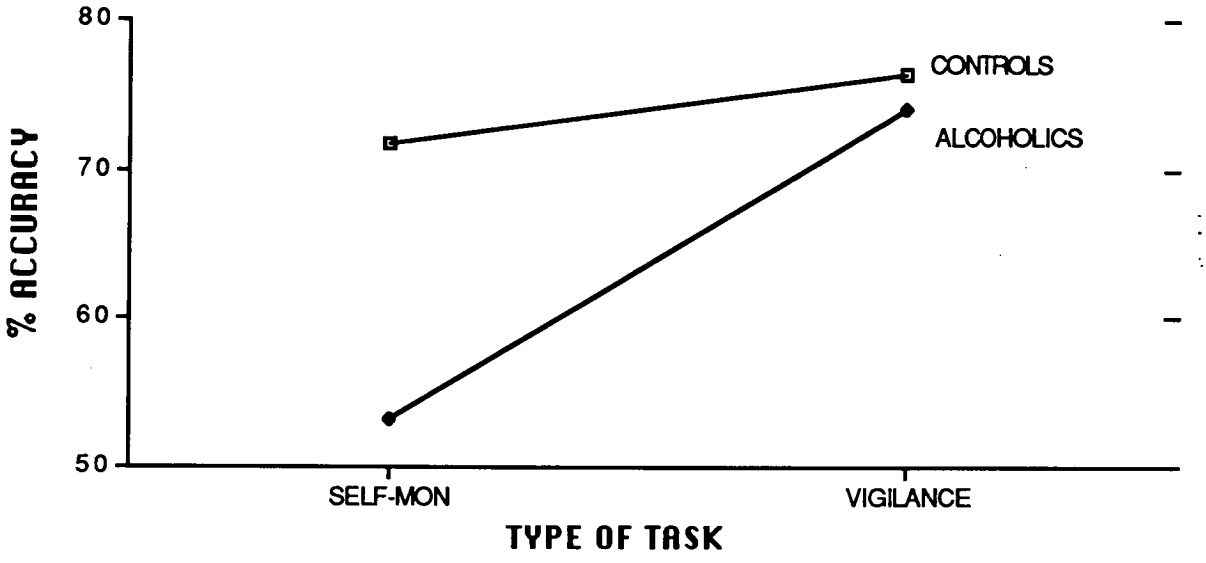

Figure 1. % accuracy of detection for alcoholic and non alcoholic subjects during self-monitoring and vigilance.

and that the alcoholic group would find the vigilance task less cognitively demanding than the self-monitoring task.

The second hypothesis that the control group would also find the self-monitoring task more difficult than the vigilance task is however not supported in that the simple effect of this comparison was not significant ($F(1,36)=1.83$, $p=.18$).

Figure 1 suggests that across both tasks and both levels of distraction there is a tendency towards a main effect that suggests that the alcoholic group's performance was worse than that of the control group $F(1,36)=6.76$, $p=.014$. According to the decision to suspend judgment, this main effect was regarded as a potential effect. From Figure 1 it also seems that this difference in performance was primarily due to the poorer performance of the alcoholic group during the self-monitoring task since there was an interaction of Groups x Tasks ($F(1,36) = 11.63$, $p<.01$; see Table 1) and the difference between the performances of the alcoholics and controls during the vigilance task was not significant (simple effect $F(1,36)=.29$, $p=.59$). This supports the third hypothesis that there would be a Group X Detection task interaction that favoured the performance of the controls at the self-monitoring task.

Figure 2 shows the effects of distraction level for alcoholics and controls across both detection tasks. There was a significant main effect of distraction ($F(1,36)=18.76$, $p<.01$) showing that the high distraction condition was effective in reducing performance.

The simple effect, reflecting the difference in performances by the alcoholic group during the high distraction level as compared to the low distraction level was significant, $F(1,36)=17.30$, $p<.01$. However, the difference in performances between high and low distraction level for the control group was not significant.

Neither hypothesis four nor five was supported. The interaction of groups x distraction level was not significant. Neither the performance of the alcoholic group nor that of the control group was impaired significantly more by the high distraction level during the self monitoring task than during the vigilance task.

Table 1 also indicates that the Groups x Detection task x Level of distraction interaction was also not significant thereby failing to support the sixth hypothesis.

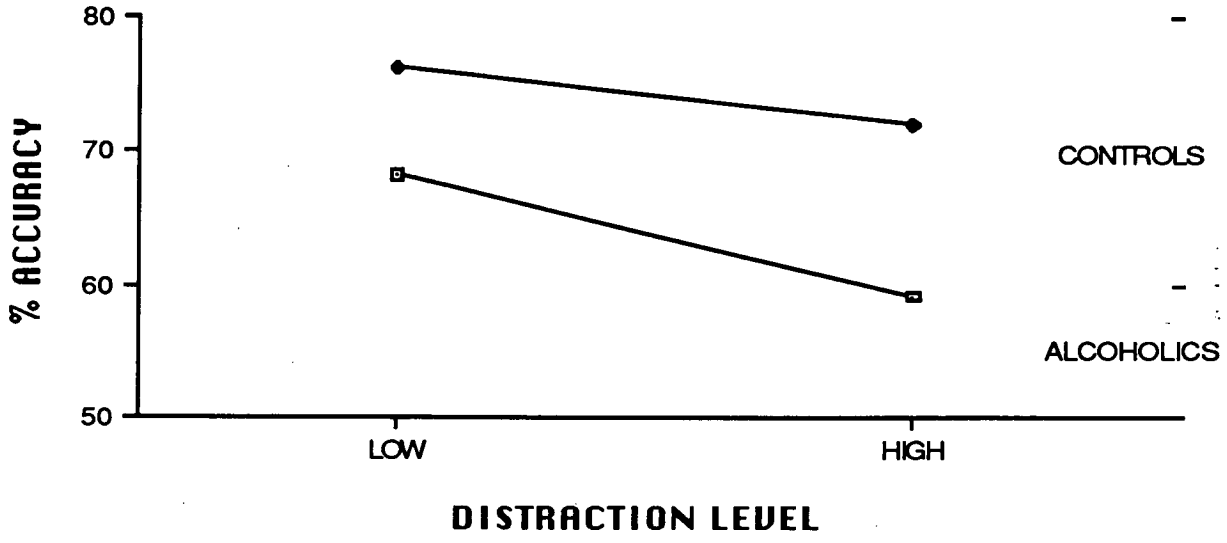

Figure 2. % Accuracy of detection by alcoholics and controls during performance of detection tasks at high and low levels of distraction.

7.2 Subjective estimates of actual performance - interactions and effects.

The summmary of the ANOVA of the subjective estimates of performance for the above design is presented in Table 2. There were no significant interactions but the main effect of distraction level was significant, $F(1,36)=21.15$, $p<.01$, and the main effects of groups, $F(1,36)=6.86$, $p=0.013$, and type of task, $F(1,36)=5.45$, $p=0.025$, approached significance. Figure 3 shows the mean estimated performance scores for the alcoholic and control groups for both levels of the Detection task. The actual performances for the same conditions are superimposed in this figure for purposes of comparison. It can be seen that both groups' subjective estimates of performance underestimated their actual performance at both detection tasks.

It can be seen from Table 2 and Figure 3 that the alcoholic group's subjective estimate of their performance tended to be lower than the control group's estimate when these estimates were collapsed across both tasks. An analysis of simple effects indicates that the alcoholic group made significantly lower estimates of their performance on the self-monitoring task than the control group, $F(1,36)=9.17$, $p<.01$. Although a trend in this direction also applied for the vigilance task, this difference was not significant.

Table 2

Summary of ANOVA of Groups (alcoholics, non alcoholics), Type of Detection Task (Self-Monitoring, Vigilance) and Level of Distraction (High, low) for subjective estimate of% accuracy of stimulus detection.

Source of variation	df	Sum of Squares	Mean Square	F	P
Groups	1	5400.24	5400.24	6.86	.013
Error	36	28346.737	787.41		
Type of Task	1	1765.29	1765.29	5.45	.025
Groups x Tasks	1	881.28	881.28	2.723	.1076
Error	36	11652.42	323.68		
Distraction Level	1	2661.16	2661.16	21.15	.0001
Groups x Dist'n	1	306.94	306.94	2.44	.127
Error	36	4528.89	125.80		
Task x Dist'n	1	42.11	42.11	.623	.435
Groups x Task x Distraction	1	17.79	17.79	.263	.611
Error	36	2433.11	67.59		

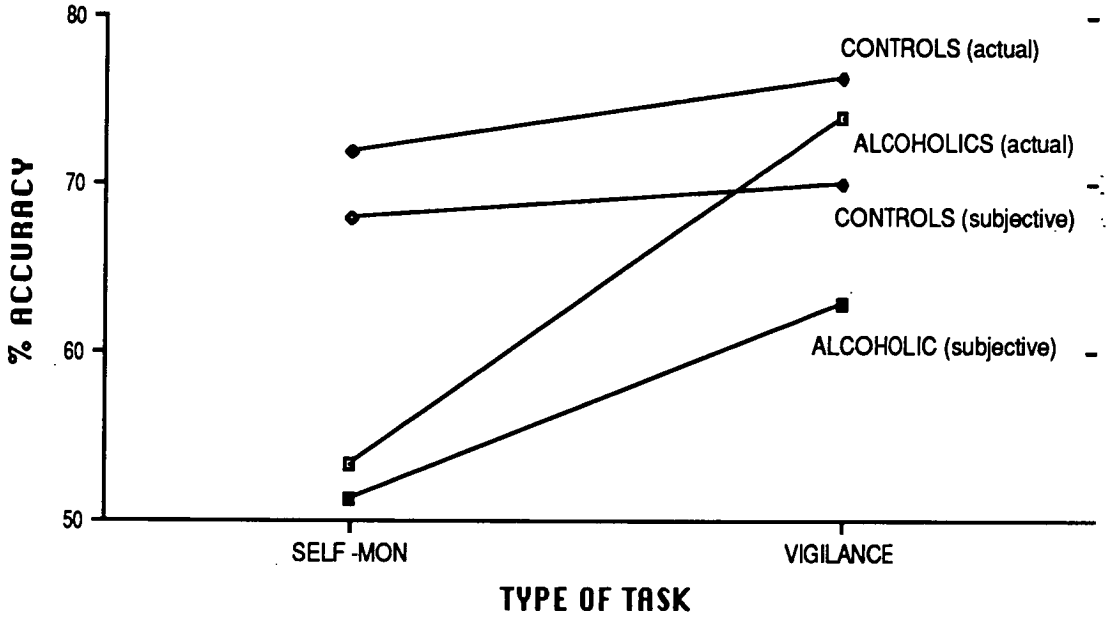

Figure 3. Actual % accuracy and subjective estimated accuracy of alcoholics and controls for performance on self-monitoring task and vigilance task.

Table 3 shows the mean actual % accuracy scores of the alcoholic group and the controls groups and the mean subjective estimates of both these groups together with the difference between the actual and subjective scores. In all conditions both groups underestimated their number of responses and this underestimation appears greatest for the alcoholic group in regard to the vigilance task.

These measures are considered to be two separate dependent variables since the actual score reflects processing of a physical stimulus (either on the VDU or arm-lifting behaviour) and the subjective score reflects an attempt to recall the rate of responding. It was considered that an inferential statistical comparison between these variables would be inappropriate.

7.3 Performance on the Austin Maze.

Walsh (1985) has suggested that some chronic alcoholic subjects continually repeat many of the errors when traversing the test path of the maze, and exhibit no apparent learning even after many trials. Some of the subjects in this study failed to show any apparently consistent reduction in errors after ten trials and became increasingly agitated. It was decided that to have continued trials until the criterion was reached would be excessively discouraging and stressful to the

Table 3

Actual and subjective estimates of % accuracy and differences of performance of alcoholics and controls for both self-monitoring and vigilance tasks at low and high levels of distraction.

Alcoholics

	Self-mon		Vigilance	
	Low	High	Low	High
Actual	59.00	47.58	77.37	70.52
SD	20.34	17.58	13.93	22.13
Subjective	57.68	44.74	67.58	58.11
SD	24.40	21.56	16.65	22.65
Difference	1.32	2.84	9.79	12.41

Controls

	Self-mon		Vigilance	
	Low	High	Low	High
Actual	74.21	69.58	78.42	74.42
SD	10.16	9.35	13.45	8.70
Subjective	70.68	65.00	72.53	67.37
SD	11.26	12.83	16.10	14.85
Difference	3.53	4.58	5.89	7.05

subject as well as time-consuming. In such cases the test was terminated after 10 trials.

There were seven subjects for whom the test was terminated for this reason. These subjects were also distracted frequently and failed to observe the rules of the test despite the fact that they could verbalise these rules accurately. Appendices 1(a) and 1(b) present the raw data of performance on the Austin Maze of the control subjects and the alcoholic subjects respectively.

7.4 Performances on the Rey Auditory Verbal Learning Task.

Figure 4 shows the mean number of words recalled during trials 1 to trial 5 of the first 15-word list (list A), upon the single presentation of the second 15-word list (list B), and Trial 6 (the re-presentation of list A after list B was presented) for the alcoholic group and the control group. The number of words retrieved when the recognition paragraph was presented is also shown.

The summary of an ANOVA of a 2(groups) x 8(trials) with trials as repeated measures design is shown in Table 4. There was a significant groups X task interaction,

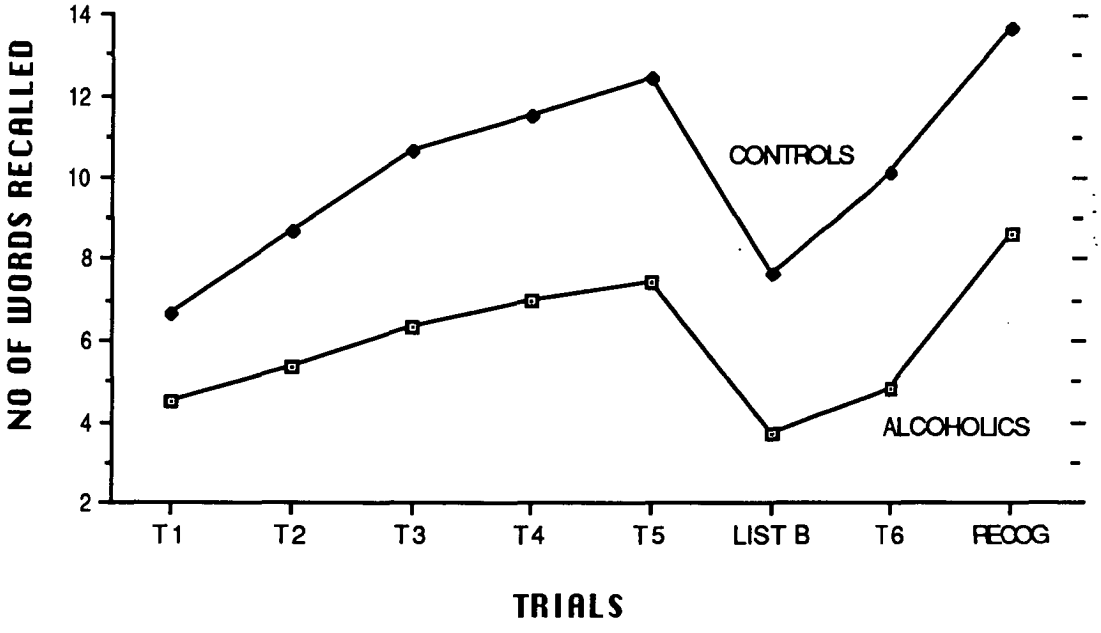

Figure 4. Number of words recalled on the trials of the RAVLT by alcoholics and controls.

Table 4. Summary of ANOVA of Groups and Trials (on RAVLT) for number of words recalled during the RAVLT.

Source	df	SS	MS	F	p
Groups	1	1347.368	1347.368	70.915	.0000
Error	36	683.987	19.000		
Trials	7	1068.000	152.571	81.839	.0000
Groups x Trials	7	70.947	10.135	5.437	.0000
Error	252	469.803	1.864		

$F(7,252)=5.44$, $p<.01$. As shown in Figure 4, the number of words recalled were increasingly greater for the control group than for the alcoholic group. An analysis of simple effects suggested that the control group recalled significantly more words at all trials compared to the alcoholics.

To examine whether there was a learning effect between trials, four planned comparisons using two-tailed paired t-tests were made to examine the difference in words recalled between a number of trials within each group. This is further discussed in the next section.

7.5.1 Inter-trial analysis for Alcoholic group.

For this group the improvement in words recalled from trial 1 to trial 5 was significant, $t(18)=10.88$, $p<.01$. This first comparison suggests that the alcoholic group appeared to experience some learning over the 5 trials. However, a second comparison of performance on trial 1 and trial 6 suggests that there was no significant difference between the performance on these trials, $t(18) = 1.10$, $p>.05$) suggesting that no real long-term learning had occurred. The comparison to determine the effect of interference of the second list (list B) on recall of the first list (list A) revealed that the reduction in words recalled from trial 5 to trial 6 (after the second word list was presented) was also significant, $t(18)=6.99$, $p<.01$. The comparison of the

performance at trial 6 and during the recognition trial was also significant, $t(18)=3.87$, $p<.01$.

7.5.2 Inter-trial analysis for Control group.

This group also appeared to learn from trial 1 to trial 5, $t(18)=18.84$, $p<.01$. Performance dropped significantly from trial 5 to trial 6, $t(18)=10.57$, $p<.01$, although long-term learning appeared to occur from trial 1 to trial 6, $t(18)=10.07$, $p<.01$. Also, recognition performance was significantly higher than recall performance during trial 6, $t(18)=9.34$, $p<.01$.

7.6 Comparisons of performances to those of other studies.

In order to compare the performances of the control group and the alcoholic group on the RAVLT to the norms for controls of Rey (1964) (cited in Lezak (1983)) and Butters et al (1986), and the alcoholic Korsakoff patients of Butters et al (1986), single group t-tests were done. Rey's (1964) norms for controls included manual labourers, professionals, elderly professionals and elderly labourers. Since the subjects of this study did not fit into one of these categories but included some of each, these norms were averaged. The norms of controls and alcoholic Korsakoff patients from

the Butters et al (1986) study are also included and all of these norms are shown in Table 5.

In comparing the performance of this study's controls to those of the Butters et al (1986) group there was no significant difference in the means of any of the corresponding trials. When comparing the study's controls to those of the averaged Rey (1964) controls only on trial 1 and trial 6 was there a significant difference ($t(18)=2.98$, $p<.01$ (in favour of this study's controls) for trial 1, and $t(18)=4.474$, $p<.01$ (in favour of the Rey controls) for trial 6. This can be seen in Table 5.

The differences in performance of this study's alcoholic group and the Butters et al (1986) and Rey(1964) control groups was also significant at least at the $p<.01$ level.

When comparing the performance of this study's alcoholic group to the performance of the Butters et al (1986) alcoholic Korsakoff patients, it was found that only on the words recalled on trial 1 was the difference not significant. On trial 2 through to trial 6 the Butters Korsakoff patients recalled significantly fewer words (at least at $p<.01$) than the alcoholics of the present study.

This suggests that the alcoholic subjects serving in

Table 5. Mean number of words recalled for trials T1 to T6 by this study's controls and experimentals (alcoholics), and those of other studies.

Control Means

	Trials					
	1	2	3	4	5	6
This Study	6.68	8.68	10.68	11.52	12.42	10.16
SDs	1.25	1.45	1.77	1.47	1.39	1.42
Butters (1986)*	6.4	8.8	10.7	11.8	12.3	10.9
Rey (1964)*	5.83	9.03	10.3	11.48	12.08	11.62

Experimental Means

	Trials					
	1	2	3	4	5	6
Alcoholics	4.47	5.37	6.32	7.0	7.47	4.84
SDs	1.74	1.95	2.06	2.13	2.06	1.86
Butters (1986) alcoholic Korsakoff*	4.0	4.0	3.9	5.1	5.6	.8

* SDs not available from these studies.

this study were not as impaired in their learning ability as the alcoholic Korsakoff patients tested by Butters et al (1986). It seems possible therefore that memory and/or attention deficits in this alcoholic group may not have been as severe as typically found in alcoholic Korsakoff patients.

CHAPTER 8

Discussion

The results tend to support the main experimental hypothesis that alcoholics are impaired in the ability to self-monitor proprioceptive behaviour when compared to non-alcoholics. The significantly depressed performance of the alcoholics on the RAVLT and on Austin Maze test suggested that this group presented with memory and attention deficits. This supports the argument that the poorer performance of the alcoholic group on the self-monitoring task may be associated with memory and attention deficits.

The finding that the alcoholic group was also more impaired during the performance of the self-monitoring task than during the vigilance task may indicate support for the McCarthy (1989) and House et al (1986) conclusion that such a vigilance task was cognitively less demanding than a task that required monitoring of proprioceptive behaviour. At first glance this appears to suggest that memory and attention processes are less critical during the vigilance task than during the self-monitoring task, or at least are used less during the vigilance task. However, the finding that the control group was not significantly impaired in performing the self-monitoring task as compared to the vigilance task does not support the McCarthy and House

et al conclusion that there is a difference in cognitive demand by the two tasks. In fact, both of these studies used a non-clinical group that resembled this study's control group. There were however differences between McCarthy's experimental group and this study's control group. McCarthy's experimental group consisted predominantly of university students and they were therefore likely to be of a higher educational level/socio-economic status than the present control group. The McCarthy controls were also younger (mean age 24.3 compared to mean age of 54.58 of the present study's control group). It is however unclear how these variables could have a differential effect on the two tasks.

Although the fourth hypothesis that there would be a main effect of groups across both levels of distraction and both tasks was supported, it should be noted that most of this effect was due to the considerable impairment of the alcoholic group during the self-monitoring task when compared to the control group, rather than an impairment of performance by the alcoholic group on both tasks. As the results show, the alcoholic group was not significantly worse than the control group on the vigilance task.

The hypothesis that there would be a Groups x Detection Task interaction was also supported by the fact that the alcoholic group was better at the vigilance task

than at the self-monitoring task, compared to the control group which managed these two tasks equally well. As noted before the control group did not appear to find the self-monitoring task more difficult than the vigilance task.

Chronic alcoholics occasionally present with signs indicative of frontal lobe damage. One of these signs is a deficit in the ability to maintain attention when performing a task (Luria, 1966, 1973; Walsh, 1985, 1987). The assessment of attention deficits in the alcoholic group was planned by including two levels of distraction to assess whether the alcoholic group would be more affected by increased distraction than the control group.

The higher level of distraction did have a significant inhibiting effect on the performance of the alcoholic group. The performance by the control group was apparently also affected more by the higher distraction level than the lower level. There was therefore no significant group x distraction level interaction.

However, the alcoholics were no more distracted during the self-monitoring task than during the vigilance task. There were two reasons to expect that the alcoholic group would be more affected during the self-monitoring task. Firstly, is the suggestion by previous researchers (House et al, 1984; McCarthy,

1989) that the self-monitoring task is more cognitively demanding of attention capacity than vigilance tasks. Secondly, it was expected that a deficient attention maintaining mechanism, that may be present in alcoholics, would be more taxed during this high distraction level of the more difficult task (the self-monitoring) than during the vigilance task. It was therefore expected that the alcoholics would find it more difficult to maintain attention during the high distraction level as compared to the low level of the self-monitoring task than during these respective levels of the vigilance task. Such an interaction of detection task x level of distraction did not occur for the alcoholic group.

To a lesser degree, it was expected that the control group would be more impaired by the higher distraction level during the self-monitoring task than during the vigilance task, not because an attention deficit existed for this group but solely because the vigilance task was expected to be less cognitively demanding. This was not evident.

8.1 Subjective estimates of performance.

The purpose of asking each subject for a spontaneous subjective estimate of the number of responses made during each task was to test the hypothesis that the alcoholic group, in comparison to the controls, would

underestimate this response rate. It was expected that the alcoholic group would present an attention deficit, and because of this deficit even less attention would have been available to attend to the response rate. If as suspected there existed also a memory deficit in the alcoholic subjects then the expected underestimation of the response rate could have been seen as a cumulative effect of poor trace coding due to insufficient attending and poor retention or retrieval due to a memory deficit. Luria (1966, 1973) and Olton (1989) have suggested that poorly coded memory traces are formed when insufficient attention is maintained during the presentation of a stimulus.

Because some of the attention was directed by the experimental instructions to the detection tasks, both groups would have theoretically had only the remaining attention available to note the response rate. As noted before, it was expected that the alcoholics might have an attention deficit and perhaps a memory deficit and that therefore they would underestimate their response rate more than the controls. However, given that all subjects were exposed to some distraction, either high or low in both of the detection tasks, it was expected that even the control subjects would present some loss of awareness of their response rate.

Because it was assumed that the self-monitoring task required more attention it was expected that both

groups would underestimate their performance more during this task than during the vigilance task. Less attention would be available to process and code into memory the response rate during the self-monitoring task. The results however show that although the alcoholic group underestimated their response rate more than the controls, there was no further reduction in estimated response rate after the self-monitoring task compared to the response rate after the vigilance task. This overall reduction in awareness of their response rate might have been expected of the alcoholics if it had been assumed that this group was handicapped by memory and attention deficits, and would therefore suffer a greater additional memory loss regarding response occurence. Although not supported by the evidence, it was also expected that the greater additional load imposed by the self-monitoring task would result in a greater difference between the actual response rate and subjective estimates of response rate for the self-monitoring task than that for the vigilance task. However, both groups seemed to be more aware of how often they responded during the self-monitoring task than during the vigilance task, the alcoholic group, in fact, appearing to be less aware of their response rate during the vigilance task than during the self-monitoring task.

It should be mentioned that this response rate was elicited from the subjects in a very casual manner to

project the impression that this was not important information. It is assumed therefore that the subjects may not have intentionally directed much attention to remembering how often they responded.

It may be methodologically questionable to pose comparisons of an actual response rate and a subjective estimate of response rate since these measures may be interpreted as different dependent variables. Another point that may have a bearing on the interpretation of this result relates to the casual nature of the way in which the subjects were asked for their estimate of response rate. Because it appeared as a casual question there seemed to be a tendency to guess in multiples of 10, eg. "40" or "20", and in terms of dozens, eg. "a dozen and a half" or "two dozen". Although such vagueness may be assumed to cancelled out across 19 subjects per group it remains as a possible source of error as well as a possible reason for this result.

8.2 Indications of attention and memory deficits.

Although no statistical analysis was made of the performances of the alcoholic subjects on the Austin Maze test and therefore no interpretation was possible, some of these subjects performed at a level that Walsh (1985) regards as normal. Walsh (1985) has stated that

the decision as to whether a subject is brain-damaged or presents with a sub-normal performance due to a learning deficit should be largely based on a qualitative assessment of the subject's behaviour during the test rather than on quantitative measures of performance. He concedes that unless such quantitative measures are obviously sub-normal, such decisions always need be made cautiously.

Walsh (1985, 1987) suggests that normal subjects require less than 15 traverses to achieve two error-free trials, and that between 15 and 20 traverses may indicate a learning deficit.

The fact that 3 of the alcoholic subjects performed within the normal range (13, 14, and 15 trials to criteria) on this test, and another 6 performed at a level indicating a learning disability (slow learners) suggests that the effects of chronic alcoholism may be varied considerably within the group. It may indicate an absence of cognitive impairment, or indeed a recovery of function from a previously impaired condition. Most of the alcoholic subjects had undergone varying periods of abstinence, and several researchers have addressed the question of reversibility of pathology and recovery of cognitive function. Brandt and Butters (1986) for example, claim that long-term recovery of memory and neuropsychological function may occur within the first

year of abstinence. Parsons (1986) extensively reviews the evidence that suggests that abstinence may yield significant improvement in cognitive functions in a considerable percentage of chronic alcoholics and this evidence is supported by other authors (eg. Parsons et al. 1987; Victor and Adams, 1985).

Although the abstinence of this study's alcoholic group was not strictly controlled or monitored, all subjects had availed themselves of a regular well-balanced diet in a custodial/supervised setting for at least the previous month, and in some cases, for longer than twelve months.

The performance of the alcoholic group on the RAVLT however does not support the indication of recovery of function that might be drawn from the performance on the Austin Maze test. The RAVLT performance suggests that the alcoholic group did indeed present with a memory deficit in that they recalled fewer words per trial between trials 1 and trials 5 than the control group. Although this group appeared to experience some learning during this part of the test, there was no lasting increase in the number of words learnt from trial 1 to trial 5 since there was no difference in the number of words recalled on trial 1 and those on trial 6. The fact that this group was severely effected by the interference list (list B), and recognised only slightly more than half of the words (of list A) during

the recognition trial suggests that they experienced considerable proactive interference and a coding problem. Rey (1964) and Lezak (1983) suggest that when the memory deficit is a coding deficit, then because the stimuli are not coded adequately no more of them will be recognised than recalled on trial 5. They further conclude that if the problem is one of retrieval but the stimuli are coded appropriately then all of the stimuli words should be recognised during this section of the RAVLT.

When the performance of the alcoholic group was examined it was seen that the degree of memory loss within the group varied considerably. Some subjects recalled substantially fewer words on all trials and during the recognition trial than some of the apparently less impaired. The fact that these more impaired subjects recognised no more words during the recognition trial than the number of words recalled on trial 5 suggests a severe coding problem of the nature described by Walsh (1985) and Cala et al (1978) as typical of Korsakoff syndrome patients. This apparent inability to code memory traces appropriately may be due to an attention deficit which may have played a part in the poor performance of these subjects during the Austin Maze test, but it could be argued that a memory deficit of this nature may be the result of a combination of attention deficit and some memory deficit (eg. Luria, 1973).

The fact that these apparently less impaired subjects performed significantly better at all trials than the other alcoholic subjects, but significantly poorer than the control group, suggests that the memory deficit of this group is either of a lesser degree or of a different nature. These more 'normal' alcoholic subjects recalled as many words on trial 1 as the control group, suggesting that both groups were able to adequately code an equal number of words to the initial memory store, but nonetheless, these better performers from the alcoholic group appeared to learn at a significantly slower rate. The fact that the performance during the recognition trial appeared to be quite normal for some of these alcoholic subjects (see Appendix 1(b)) suggests that the deficit was one of retrieval rather than one of retention due to a coding deficit. It can therefore be argued that these few alcoholic subjects adequately coded the stimulus words but were less able than the control group to retrieve these codings from memory. The finding that recognition performance was normal suggests that the degree of learning was also normal, that is, the words had been appropriately coded.

It might therefore be surmised that the alcoholic group, in general, presented with primarily a coding deficit and perhaps also a retrieval deficit (although this cannot be determined if the stimuli are

inadequately coded), and that the better performers in the alcoholic group presented with only a retrieval problem (which gives the impression of a learning deficit). The poorer performance (than that of the control group) of these less impaired alcoholic subjects on the Austin Maze test supports this impression of a learning deficit in this group.

Because previous studies had specifically used alcoholic Korsakoff patients that had been comprehensively assessed and qualified (eg. Butters et al, 1986) the performances of the present study's control group and experimental group on the RAVLT were compared to those of Rey (1964) and Butters et al (1986). The subjects selected as the experimental group for the present study were all diagnosed as chronic alcoholics, but only 7 were diagnosed as alcoholic Korsakoff patients and the period since diagnosis for these 7 subjects varied from 3 years to 15 years. There was some uncertainty therefore regarding the similarity of this study's experimental group and those of other studies.

Butters et al (1986) compared the performances of alcoholic Korsakoff patients, early and advanced Huntington's disease patients and normal controls on the RAVLT. As far as the controls of this study and those of the Butters et al study is concerned there was no significant difference on the performances of any of

the trials. The controls of this study compared to those of Rey (1964) differed only on the performance of trials 1 and 6. As explained in Chapter 7 there is some doubt as to whether the performances of Rey's (1964) four sub-groups can be validly averaged to provide an adequate age-of-subject match for this study's control group. The difference in performance of trials 1 and 6 may therefore have been due to inadequate matching.

8.3 Methodological considerations.

A number of questions could be raised regarding whether the vigilance task and the self-monitoring task employed similar cognitive functions. Were the dependent variables identical for the two tasks despite the fact that one was a detection task where the stimulus was generated by the subject himself and the other where the stimulus was generated by a computer? It could be argued as McCarthy (1989) has claimed, that this difference is the reason why the two tasks are of varying cognitive load. McCarthy argues that the spontaneous movement of an arm is less likely to be brought into awareness because of its stereotypic nature. Whereas the stimulus during the vigilance task was presented on the VDU screen, represented an external visual stimulus.

The fact that it is difficult to control for stimulus frequency with this type of self-monitoring task can be seen as another possible weakness of this design. The instructions can only encourage the arm movement at the beginning of each condition, and then no further control is available to the experimenter. This relies on the memory and attention of the subject during the instruction phase. During this experiment, subjects who responded fewer than 15 times during any one of the 4 conditions were disqualified. Indeed, two experimental subjects and one control subject were discarded for this reason. For the vigilance task however, 50 stimuli were programmed to be generated during each condition. It was not possible to match the number of stimuli generated during the vigilance task to the number of arm-lifts because (due to the counter-balancing) at least half the subjects were given the vigilance task first.

The appropriateness of the distraction modes might also be questioned. The low distraction level consisted of a mixture of peaceful instrumental background music with all other auditory stimulation excluded by the headphones. It seems possible that with the experimental subjects who as a group were inactive and generally more relaxed, that this distraction level served to lower their state of arousal rather than act as a competitor for attention availability. The fact that the high distraction level was of a verbal nature

rather than a musical one, may have had some effect although the nature of this effect is not clear.

A possible solution to this problem might be to require the subjects to perform some other vigilance task simultaneously with the two outlined in this study. This concurrent vigilance task could then be of different levels of difficulty to represent the two levels of distraction. In this way the two levels of difficulty may represent more appropriately varied demands for attention.

Another possible methodological flaw involves the subjective estimates of response rate. Perhaps because of the casual manner in which the subjects were asked for this estimate, the response was also casual, and therefore may have been poorly considered. It must also be conceded that some subjects may have been inclined to count the stimuli once the question had been asked, thereby priming the monitoring and vigilance process. In this way, for some of the subjects, the validity of the actual measure may have been jeopardised.

8.4 Clinical implications.

The finding that some of the chronic alcoholics seemed to present only minimal cognitive impairment suggests

that appropriate training may prove therapeutically effective. Indeed, of the 19 chronic alcoholic subjects 3 performed within the normal range of performance during the Austin Maze test and the RAVLT. According to Walsh (1985), the 6 others in the alcoholic group that performed the Austin Maze test almost 'normally' may have been only slow learners, may not have incurred any pathology to a debilitating degree prior to diagnosis, or may have recovered cognitive functioning as a result of alcohol abstinence and/or dietary compensation. Given the assumption that this chronic alcoholic group is typical, these findings seem to indicate that memory training and teaching alcoholic patients to utilise cues to monitor drinking rate may prove to be beneficial.

Some researchers have reported some success in training alcoholics to drink in moderation rather than yielding to the widespread assumption that the only effective treatment for alcoholism is total abstinence. Lovibond and Caddy (1970) for example, trained alcoholics to discriminate their own level of blood alcohol concentration and used an aversive conditioning paradigm to cue subjects to stop drinking once they had reached a certain blood alcohol concentration. Sobell and Sobell (1973) used individualised behaviour therapy to retrain alcoholics to become controlled drinkers by becoming aware of the cues of intoxication, and by controlling both the rate of alcohol intake and the

concentration of alcohol intake. In all of these cases it might be concluded that attention focusing cues had been established in subjects' memories, and these cues served as indications to curtail or initiate behaviours.

8.5 SUMMARY

In conclusion, it appears that the hypothesis that the alcoholic group would find the self-monitoring task more difficult than the control group was supported, but the alcoholics were no more affected by the two levels of distraction than the control group. If the alcoholic group did indeed present with an attention deficit, the performance during the higher distraction condition should have been worse. If it is assumed that the self-monitoring task required more cognitive processing, then the self-monitoring task should have been more difficult for both groups. The performance on this task should have been more adversely affected by the higher distraction level than the vigilance task was affected by this level. This suggests that there may be some doubt as to whether these distraction levels did represent varying degrees of attention diversion. The need for more carefully qualified measures of distraction may be required if this experiment is to be replicated.

The alcoholic group did perform at a lower level during the RAVLT and the Austin Maze test suggesting that the alcoholic group presented a memory deficit and an attention deficit.

The reports from various researchers seem to indicate that it is possible to retrain alcoholics to become controlled drinkers if they can be taught to self-monitor proprioceptive and interoceptive cues as well as attending to overt behaviour that indicates the onset of intoxication. These other reports therefore suggest that alcoholic patients who appear to be presenting with only minor memory and attention deficits are capable of responding to retraining.

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Appendix 1 (a). Individual performances of control subjects on the RAVLT and Austin Maze test. Scores from T1 to RECOG are words recalled, and for Austin Maze (AM) are trials to criterion.

Controls

Sub	T1	T2	T3	T4	T5	T6	RT5	PI	B	RECOG	AM
1	6	9	9	10	12	10	2	0	8	14	13
2	7	10	12	13	13	9	3	3	7	11	11
3	5	8	8	9	9	7	2	3	6	14	14
4	6	7	9	9	11	9	3	2	10	15	15
5	9	10	14	13	13	11	2	0	8	13	12
6	8	10	14	14	15	12	1	2	6	15	10
7	7	7	9	11	12	10	2	0	8	14	11
8	6	9	11	10	11	11	3	1	8	12	16
9	5	7	9	12	13	12	1	1	6	14	14
10	7	8	10	13	14	11	2	0	8	15	12
11	8	10	12	12	12	10	3	2	7	13	14
12	8	11	12	12	14	12	2	3	8	15	13
13	6	8	11	12	13	10	0	0	9	13	11
14	7	8	10	10	12	8	2	2	7	14	15
15	5	7	11	12	12	9	2	3	9	14	13
16	8	10	12	13	13	11	1	2	9	15	13
17	6	10	11	12	14	12	3	2	7	13	11
18	8	10	11	12	12	10	3	3	8	14	13
19	5	6	8	10	11	9	3	2	6	12	16
X	6.7	8.7	10.7	11.5	12.4	10.2	2.1	1.6	7.6	13.7	13
SD	1.3	1.5	1.8	1.5	1.4	1.4	.89	1.2	1.2	1.2	1.8

Appendix 1 (b) Individual performances of alcoholic subjects on the RAVLT and Austin Maze test. Scores from T1 to RECOG are words recalled, and for Austin Maze (AM) are trials to criterion.

Alcoholics

Sub	T1	T2	T3	T4	T5	T6	RT5	PI	B	RECOG	AM
1	3	4	4	5	5	3	4	2	4	3	25
2	4	6	6	5	7	5	2	0	4	14	19
3	5	6	6	8	9	5	2	1	6	12	17
4	4	3	7	8	9	4	2	4	3	14	16
5	3	3	4	5	7	3	5	2	4	6	25
6	4	5	6	6	6	3	3	0	3	2	20
7	2	4	4	4	4	2	5	3	1	5	25
8	6	8	9	10	10	5	3	2	4	14	14
9	7	7	8	9	9	6	2	1	5	15	16
10	5	6	7	7	8	4	0	1	4	12	22
11	3	3	4	5	6	3	4	2	4	5	23
12	7	9	10	12	12	7	3	3	5	15	13
13	6	6	7	6	7	5	4	2	2	4	25
14	4	6	6	7	8	5	2	0	4	10	16
15	7	6	8	8	8	6	0	2	5	9	17
16	2	4	5	6	5	4	3	2	1	2	25
17	3	3	3	5	5	5	6	0	3	5	25
18	7	9	10	10	10	10	2	4	5	13	15
19	3	4	6	7	7	7	5	0	3	4	25
X	4.5	5.4	6.3	7.0	7.5	4.8	3.0	1.6	3.7	8.6	17.3
SD	1.7	1.9	2.1	2.1	2.1	1.9	1.6	1.3	1.3	4.8	3.1

Appendix 2 (a) Means, SDs and % accuracy of stimulus/response detection of individual alcoholic subjects during the self-monitoring and vigilance tasks at two levels of distraction.

Alcoholics

	SELF-MONITOR		DETECTION TASK	
	LOW	HIGH	LOW	HIGH
S1	53	42	44	48
S2	69	63	92	90
S3	83	63	94	84
S4	71	54	94	82
S5	10	13	80	70
S6	62	41	70	42
S7	41	21	58	20
S8	71	46	64	60
S9	90	71	88	86
S10	64	66	70	90
S11	33	43	74	86
S12	74	54	86	90
S13	54	47	66	44
S14	81	78	96	92
S15	59	53	92	90
S16	48	41	88	80
S17	48	33	68	46
S18	82	60	76	94
S19	28	15	70	46
MEAN	59.00	47.58	77.37	70.53
SD	20.34	17.58	13.95	22.13

Appendix 2 (b) Means, SDs and % accuracy of stimulus/response detection of individual control subjects during the self-monitoring and vigilance tasks at two levels of distraction.

Controls

	SELF-MONITOR		DETECTION TASK	
	LOW	HIGH	LOW	HIGH
C1	57	63	84	80
C2	84	72	92	84
C3	90	76	78	64
C4	81	71	90	76
C5	67	72	84	86
C6	82	71	58	64
C7	77	80	72	80
C8	81	69	88	72
C9	63	74	48	60
C10	70	55	48	68
C11	56	63	78	68
C12	61	74	84	90
C13	70	68	76	70
C14	71	42	72	68
C15	83	75	94	76
C16	90	81	88	64
C17	83	84	78	76
C18	75	67	92	88
C19	69	65	86	80
MEAN	74.21	69.58	78.42	74.42
SD	10.16	9.35	13.45	8.70

Appendix 3. Instructions for self-monitoring level.

In this session I want you to feel free to smoke, drink your tea or coffee, and help yourself to the biscuits, peanuts etc, on the table.

But I want you to do this only with this hand (point to preferred hand), the one that you have the wrist band on. So, when you smoke, drink from your cup, or pick from the table, or just scratch your face, please do it only with the wrist-band hand.

In your other hand, you have the button-press. Whenever you raise your wrist band arm for any reason at all please press the button that you are holding in your other hand.

The computer gets a signal from the wrist-band whenever it is raised, and from the push-button whenever you press it, so there is no need to count anything.

Do you understand that ? Explain again if necessary.

Do you have any questions ? Explain and answer.

Enjoy the tape ! I'll be back in 15 minutes.

Appendix 4. Instructions for Vigilance level.

In this session your going to see a dot appear every now and then in the middle of the screen.

This is what it looks like. You tell me when you can see a dot clearly in the middle of the screen. [Adjust the contrast and brightness (from lowest to highest) until the subject can see it clearly].

Whenever you see this dot on the screen, I want you to press the button that you are holding in your preferred hand as soon after you see the dot as possible.

I will tell you when you are finished. The dots may be some time apart or sometimes in a row.

Do you understand ? Explain again or answer questions.

Do you have any questions ? Answer questions.