Alcohol dementia: “cortical” or “subcortical” dementia?

Cynthia A. Munro*, Judith Saxton, Meryl A. Butters

University of Pittsburgh Medical Center, Pittsburgh, PA, USA

Accepted 28 March 2000

Abstract

Most dementias are considered to exhibit either a predominantly “cortical” (e.g. Alzheimer’s disease, AD) or “subcortical” (e.g. Parkinson’s disease) pattern. A double dissociation has been reported, such that cortical and subcortical dementias can be differentiated based on performance on tests of declarative and procedural learning. The goal of this study was to determine if subjects with alcohol dementia exhibit a predominantly cortical or subcortical dementia profile. The performance of 10 elderly subjects diagnosed with alcohol dementia, 29 elderly subjects with histories of alcohol dependence but who were not demented, and 11 subjects with AD was compared to 20 elderly control subjects. The results indicated that the procedural learning task did not differentiate among the groups, whereas the discriminability index from the California Learning Test (the declarative learning task) did. Thus, alcohol dementia cannot clearly be ascribed to either dementia classification. © 2001 National Academy of Neuropsychology. Published by Elsevier Science Ltd.

Keywords: Alcohol dementia; Cortical; Subcortical

The term dementia refers to a clinical syndrome of acquired intellectual disturbances produced by brain dysfunction. Dementias may result from a wide variety of disorders, including degenerative (e.g. Alzheimer’s Disease, AD), vascular (e.g. Multi-infarct Dementia), and traumatic (e.g. head injury) and have typically been classified as either “cortical” or “subcortical.” It is important to note that although such anatomical distinctions have been used, these processes rarely, if ever, affect exclusively cortical or subcortical structures, but rather are said to be predominantly one area or the other. Still, the use of these terms to classify dementing processes has been criticized (Gomez-Haro et al., 1999). Because of the

* Corresponding author. Department of Psychiatry, Johns Hopkins Hospital, Meyer 218, 600 N. Wolfe St., Baltimore, MD 21287, USA. Tel.: 4-410-955-2619; fax: +1-410-955-0504.
E-mail address: cmunro@jhmi.edu (C.A. Munro).
ability to distinguish behaviorally between dementias considered to be cortical from those said to be subcortical, however, these terms have provided a useful nomenclature in the literature. Thus, the classification of a dementia as either cortical or subcortical is based primarily on the clinical syndrome rather than the area of anatomical damage, and that is how these terms will be used in the current investigation.

AD is often referred to as the archetypal cortical dementia. Cortical dementias involve neurodegenerative processes primarily in the frontal, temporal, or parietal lobes and are associated with deficits in free recall, recognition, and naming, with sparing of skills involving procedural learning and motor movements (Bondi et al., 1994). Progressive Supranuclear Palsy is the classic example of a subcortical dementia (Albert et al., 1974). Other subcortical dementias include Parkinson’s Disease (Albert, 1978) and Huntington’s Disease (McHugh & Folstein, 1975). Ischemic vascular dementia (Cummings & Benson, 1983; Libon et al., 1998) and the dementia syndrome of depression (Caine, 1981) have also been included under the term subcortical dementia. Subcortical dementias are associated with pathologic changes that involve primarily the thalamus, basal ganglia structures such as the caudate nucleus and the substantia nigra, and related brainstem nuclei. Their cardinal clinical features include forgetfulness, slowing of mental processes, and intellectual deterioration. Neuropsychological deficits associated with subcortical dementias involve disturbance of motor movements, procedural learning, and free recall with relative sparing of recognition memory, verbal abstraction, and naming (Drebing et al., 1994).

Mixed cortical and subcortical dementias have also been identified. Lewy-Body dementia (McKeith et al., 1996) is a classic example of a mixed dementia incorporating features of both AD and Parkinson’s Disease, and therefore displaying both cortical and subcortical characteristics. Other examples of mixed dementias include Corticobasal Degeneration and Frontotemporal dementia (with the amyotrophic form of Motor Neuron Disease).

A number of studies have shown that it is possible to dissociate cortical and subcortical dementias using behavioral measures (Eslinger & Damasio, 1986; Heindel et al., 1988, 1989; Butters et al., 1990; Deweer et al., 1994). A “double dissociation” has been described (Butters et al., 1990) between performance on tests of procedural and declarative learning such that the neuropsychological profiles of patients with cortical and subcortical dementias can be reliably distinguished. Specifically, preservation of procedural learning despite severely impaired free recall and recognition has been associated with patients with AD (i.e. a cortical pattern) and impaired procedural learning and preserved recognition memory has been associated with Parkinson’s Disease and Huntington’s Disease (i.e. a subcortical pattern).

Long-term abuse of alcohol is related to deficits in neuropsychological functioning (Rourke & Løberg, 1996) and has been associated with both cortical and subcortical damage (Cala & Mastaglia, 1980; Lishman, 1981; Walker et al., 1981; Jernigan et al., 1991). Although the alcohol literature has focused on the disorder of Wernicke–Korsakoff’s Syndrome (WKS), abnormalities at the structural and functional level have been identified in people meeting criteria for alcohol dependence who are cognitively impaired (i.e. alcohol dementia) (Pfefferbaum et al., 1992). Diagnosed under the term alcohol-induced persisting dementia (American Psychiatric Association, 1994), the dementia of alcoholism is not typically considered as predominantly cortical or subcortical. Alcohol dementia is associated
with various neuropsychological deficits including diminished motor skills and decline in higher cortical function such as verbal abstraction and problem-solving (Delin & Lee, 1992). The literature does not provide adequate data to conclude whether alcohol dementia would be more appropriately classified as a cortical or subcortical dementia. Elderly individuals who meet criteria for histories of alcohol dependence and dementia present with mild cognitive deficits in multiple cognitive domains together with possible motor and/or visual disturbance.

The findings from neuropathological and neuroimaging research also suggest both cortical and subcortical pathology. However, on the whole, the evidence leads one to believe that alcohol dementia may more suitably be described as a subcortical dementia. Pathological studies of animal models have shown reduction in hippocampal neurons (Walker et al., 1981) and cerebellar Purkinje cells (Pentney, 1982; Cragg & Phillips, 1983). Human neuropathological studies have revealed significantly decreased brain weight and reduction in white matter volume among individuals diagnosed with alcohol dependence (Harper & Kril, 1991). Reduction in the volume of the cerebral cortex in individuals who meet criteria for alcohol dependence has been demonstrated by both pathological studies (De la Monte, 1988) and using magnetic resonance imaging (MRI) (Jernigan et al., 1991). However, as Harper and Kril (1991) point out, atrophy of the cerebral cortex could be accounted for by reduction in white matter volume rather than loss of cortical tissue. Bergman et al. (1980) also suggest that older individuals diagnosed with alcohol dependence are particularly vulnerable to subcortical rather than cortical damage. In addition, a study using MRI reported significant correlations between subcortical changes and cognitive measures rather than cortical fluid volumes and cognitive measures among this group (Jernigan et al., 1991).

An association has also been reported between cerebellar damage and alcohol use in a number of investigations (Tavares & Paula-Barbosa, 1982; Pentney, 1993) and the cerebellum has been implicated in the acquisition of one type of procedural learning (classical conditioning) (Thompson et al., 1983). To the extent that subjects with alcohol dementia have cerebellar damage, their performance on a test of procedural learning could be expected to be impaired. Finally, performance on a different test of procedural learning, the Pursuit Rotor Learning Test (PRLT), has repeatedly been shown to be vulnerable to the acute effects of alcohol (Dougherty et al., 1998; Hiltunen, 1997). Thus, subjects with alcohol dementia may demonstrate deficits in procedural learning for a number of reasons, but the resulting neuropsychological pattern would resemble the subcortical pattern.

In the current study, we compared four groups of subjects: patients with AD, patients with alcohol dementia, non-demented elderly subjects who met criteria for alcohol dependence, and normal elderly control subjects. Using the “double dissociation” protocol described by Libon et al. (1998), we investigated whether based on performance on these tests, alcohol dementia could best be described as a cortical or a subcortical dementia. We hypothesized that patients with alcohol dementia would exhibit the test pattern typical of subcortical dementias. Thus, patients with alcohol dementia would be impaired on the free recall portion of the California Verbal Learning Test (a test of declarative memory) but unimpaired on the recognition portion of the test. Furthermore, alcohol dementia patients would exhibit impaired performance on a test of procedural learning.
1. Method

1.1. Subjects

Thirty-nine elderly individuals meeting criteria for alcohol dependence, who had been abstinent for at least 4 weeks prior to testing, were recruited from area Veterans Administration Medical Centers. They were given a structured clinical interview to determine that they met DSM-IV diagnoses of alcohol dependence, and all had a history of at least 10 years of heavy drinking. All subjects were given a breathalyzer test (Alco-Sensor IV) to ensure that they were sober at the time of testing. A dementia screening battery was administered to determine whether subjects met criteria for “Alcohol Dementia” or “Alcohol — Not Demented” (see below). Two of the authors (JS and MB), both neuropsychologists, blind to subject’s performances on the procedural and declarative memory tests, determined this classification on the basis of the dementia screening profile and the clinical history. In addition, 11 subjects with a diagnosis of probable ADe were recruited from the University of Pittsburgh’s Alzheimer’s Disease Research Center, where they were diagnosed according to ADRDA/NINCDS criteria. Finally, 20 elderly control subjects underwent the same procedures. Subjects were excluded if they had any history of events or conditions known to affect cognitive functioning, such as stroke, WKS, loss of consciousness for more than 1 h, coma, or significant psychiatric history (e.g., schizophrenia or bipolar disorder), or other substance dependence history. The age range for all subjects was 55 to 83 years. Demographic information is included in Table 1.

1.2. Procedure

Subjects were administered a clinical interview, including assessments of medical, psychiatric, and drinking histories, current mood, and a brief battery of tests designed to diagnose dementia according to DSM-IV criteria (American Psychiatric Association, 1994). This battery consisted of tests from the dementia assessment developed by the Consortium to Establish a Registry for Alzheimer’s Disease (Morris et al., 1988) as well as additional tests sensitive to dementing illnesses. The battery included the following tests: Mini Mental State Examination (MMSE) (Folstein et al., 1975); Trailmaking Test (Reitan & Wolfson, 1993); Clock Drawing (Rouleau et al., 1992); Modified Boston Naming Test (Kaplan et al., 1983); Word List Memory and Recall (Atkinson & Shiffrin, 1971); and Constructional Praxis

<table>
<thead>
<tr>
<th></th>
<th>(A) AD</th>
<th>(B) Alcohol dementia</th>
<th>(C) Alcohol — not demented</th>
<th>(D) Control</th>
<th>Significant differences</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>11</td>
<td>10</td>
<td>29</td>
<td>20</td>
<td>–</td>
</tr>
<tr>
<td>Age</td>
<td>73.6 (5.4)</td>
<td>69.8 (9.3)</td>
<td>64.5 (6.1)</td>
<td>70.3 (7.8)</td>
<td>C &lt; AD</td>
</tr>
<tr>
<td>Education</td>
<td>11.9 (2.3)</td>
<td>12.1 (3.5)</td>
<td>12.2 (2.2)</td>
<td>13.7 (2.6)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Gender (m/f)</td>
<td>4/7</td>
<td>9/1</td>
<td>26/3</td>
<td>9/11</td>
<td>–</td>
</tr>
</tbody>
</table>
(Rosen et al., 1984). In addition to the dementia screening battery, all subjects completed a second, more comprehensive battery of tests designed to measure different aspects of neuropsychological functioning. Among these tests were the PRLT and the CVLT.

1.2.1. Procedural memory

Procedural memory was measured using the PRLT (model number 30010, distributed by the Lafayette Instrument Co.). The PRLT is an electrical device used to measure motor skill by requiring subjects to keep a stylus within a circle measuring 2 cm in diameter. The circle is off-center on a rotating disk, which measures 25.5 cm in diameter. The disk rotates and stops in 20-s intervals. The procedure used for the PRLT in this study was identical to that used in previous investigations (Heindel et al., 1989). Subjects were allowed four practice trials of 15, 30, 45, and 60 rpm in order to determine which of the four speeds yielded time on target closest to 5 s and that speed was used throughout the protocol. Subjects completed six blocks of four trials. After the first four trials, subjects rested for 1 min before proceeding to the next four trials. After the second and fourth blocks, subjects were administered other tasks such that approximately 1 h separated the second and third blocks and the fourth and fifth blocks. Total time on target for the four trials in each of the six blocks was recorded.

1.2.2. Declarative memory

Declarative memory was measured using the CVLT (Delis et al., 1987). The CVLT was administered in the standard fashion. List A, which consists of 16 grocery items, is administered over five trials with subjects naming as many of the items on the list as they can remember after each trial. List B, a list of 16 different grocery items used as distractor items, is read to subjects only once after the fifth trial of List A and subjects are asked to report as many of the items from List B as they can recall. Immediately afterward, subjects are then asked to recall as many of the items from List A as they can remember. A cued recall condition is then administered wherein subjects are given four categories (semantic cues) and asked to recall, after each category, as many of the words in List A that were in each category. After approximately 20 min, a delayed free recall condition is administered in which subjects are again asked to generate as many words from List A as they can recall (free recall score). They are then given semantic cues as before and asked to recall words from the list (cued recall score). The final portion of the test consists of a recognition trial during which subjects hear 44 grocery items (the 16 from List A, 16 from List B, and 12 items from neither list) and are required to report if each word had been part of List A (recognition score). Scores were also obtained regarding the total number of items recalled over the five trials of List A (Trials 1–5); discriminability index; and number of intrusion errors following semantic cues (cued recall intrusions). The first three of these factors were chosen consistent with Libon et al. (1998), who determined that these three measures loaded on separate factors on the nine-item CVLT, and appear to assess different types of declarative memory. Because this investigation included the 16-item CVLT, results from this investigation may not necessarily generalize to the shorter version of the test. We chose the more difficult version, however, to eliminate potential ceiling effects among our control group. The fourth factor was chosen due to its face validity in assessing recall after a delay.
2. Results

The demographic information presented in Table 1 was submitted to analyses of variance (ANOVAs) to determine if the groups differed on age or education. Results indicated that the groups did not differ with regard to education, $F(3,69) = 1.92, p > 0.05$. Age, however, did differ significantly among the groups, $F(3,69) = 5.61, p < 0.01$. Post-hoc tests (Tukey’s test) revealed that those who met criteria for alcohol dependence but who were not demented were younger than both the AD group ($p < 0.01$) and the control group ($p < 0.05$).

CVLT and PRLT results are presented in Table 2. The variables listed in the table were also analyzed with ANOVA and post-hoc tests. Age was not correlated with any of the dependent variables, so it was not entered as a covariate in any analyses. Tukey’s tests were again used, but the homogeneity of variance assumption was violated for the cued recall intrusion variable from the CVLT and the total time (blocks 1 through 6) on the Pursuit Rotor variable. Therefore, for those variables, the Dunnett T3 test was employed. The CVLT measures revealed differences among groups, depending upon which measure was compared. On free recall of all five trials of List A, the two groups who were diagnosed with dementia (AD and alcohol dementia) performed worse than both the control group and the subjects meeting criteria for alcohol dependence but who did not meet criteria for dementia. The mean scores for each group indicate that the performance of the non-demented group with histories of alcohol dependence (group C in Table 2) was virtually identical to that of the control group. On the discriminability measure, the AD subjects, as expected, performed worse than all other

<table>
<thead>
<tr>
<th></th>
<th>(A) Alzheimer's disease (n = 11)</th>
<th>(B) Alcohol dementia (n = 10)</th>
<th>(C) Alcohol — not demented (n = 29)</th>
<th>(D) Normal controls (n = 20)</th>
<th>Specific comparisons (see below)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mini Mental State Exam</td>
<td>20.91 (5.06)</td>
<td>25.10 (3.04)</td>
<td>27.83 (1.95)</td>
<td>28.40 (1.76)</td>
<td>A &lt; CD, B &lt; D</td>
</tr>
<tr>
<td>15-item BNT</td>
<td>12.00 (2.45)</td>
<td>13.60 (1.35)</td>
<td>14.17 (1.54)</td>
<td>14.85 (0.37)</td>
<td>A &lt; D</td>
</tr>
<tr>
<td>Category fluency</td>
<td>11.18 (2.56)</td>
<td>15.78 (5.91)</td>
<td>17.21 (4.48)</td>
<td>20.00 (4.29)</td>
<td>A &lt; CD</td>
</tr>
<tr>
<td>WLLT</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>10.27 (3.72)</td>
<td>12.50 (3.37)</td>
<td>17.48 (3.30)</td>
<td>21.35 (3.86)</td>
<td>AB &lt; CD, C &lt; D</td>
</tr>
<tr>
<td>DR</td>
<td>1.18 (1.89)</td>
<td>2.70 (1.49)</td>
<td>5.55 (1.80)</td>
<td>7.10 (1.41)</td>
<td>AB &lt; CD, C &lt; D</td>
</tr>
<tr>
<td>CERAD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Figure copy</td>
<td>8.72 (2.76)</td>
<td>7.90 (1.79)</td>
<td>8.93 (1.83)</td>
<td>10.30 (1.78)</td>
<td>(1.78) B &lt; D</td>
</tr>
<tr>
<td>Figure recall</td>
<td>3.00 (2.72)</td>
<td>4.20 (2.49)</td>
<td>7.17 (2.80)</td>
<td>8.68 (2.50)</td>
<td>AB &lt; CD</td>
</tr>
<tr>
<td>Trails A time</td>
<td>74.90 (32.09)</td>
<td>45.70 (9.70)</td>
<td>44.38 (24.05)</td>
<td>32.15 (8.95)</td>
<td>AB &gt; D</td>
</tr>
<tr>
<td>Errors</td>
<td>0.20 (0.42)</td>
<td>0.00 (0.00)</td>
<td>0.06 (0.26)</td>
<td>0.25 (0.55)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Trails B time</td>
<td>183.86 (114.96)</td>
<td>158.00 (88.34)</td>
<td>120.00 (79.19)</td>
<td>91.15 (26.79)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Errors</td>
<td>2.57 (2.64)</td>
<td>1.10 (1.73)</td>
<td>1.07 (1.78)</td>
<td>0.65 (0.99)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Clock</td>
<td>5.82 (2.68)</td>
<td>5.70 (1.64)</td>
<td>6.93 (1.33)</td>
<td>7.75 (0.97)</td>
<td>B &lt; D</td>
</tr>
</tbody>
</table>

All group differences which were significant beyond the 0.05 level are reported.
groups ($p < 0.001$ for all comparisons). Again, the mean scores for the groups indicate that the two alcohol groups performed very similarly to one another and to the control group. Finally, a comparison of cued recall intrusions among the groups revealed that all three patient groups fared worse than the control group on this measure ($p < 0.05$ for all significant comparisons). The two alcohol groups performed similarly to each other, whereas the AD group had more intrusions than the other three groups, despite this difference not reaching significance (Table 3).

Contrary to our prediction, the PRLT variables did not discriminate among the groups. An ANOVA performed on the total time (in seconds) on target across the six blocks indicated no significant differences among the groups. Fig. 1 illustrates the performance for each group across all six blocks. In contrast to previous work with subcortical dementias (e.g. Heindel et al., 1988), none of the groups appeared to have difficulty on this task compared to the control group. A related measure, the time on target for the first block subtracted from the

Table 3
Means for each of the groups on various neuropsychological measures

<table>
<thead>
<tr>
<th></th>
<th>(A) AD</th>
<th>(B) Alcohol dementia</th>
<th>(C) Alcohol — not demented</th>
<th>(D) Control</th>
<th>Significant differences</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVLT</td>
<td>(M) (SD)</td>
<td>(M) (SD)</td>
<td>(M) (SD)</td>
<td>(M) (SD)</td>
<td></td>
</tr>
<tr>
<td>List A: Trials 1–5</td>
<td>25.3 (13.1)</td>
<td>33.9 (10.9)</td>
<td>44.9 (9.6)</td>
<td>45.9 (9.1)</td>
<td>AB &lt; CD</td>
</tr>
<tr>
<td>Discriminability index</td>
<td>63.6 (13.6)</td>
<td>88.3 (7.5)</td>
<td>89.0 (7.5)</td>
<td>92.7 (6.5)</td>
<td>A &lt; BCD</td>
</tr>
<tr>
<td>Cued recall intrusions</td>
<td>16.9 (10.1)</td>
<td>4.2 (2.6)</td>
<td>3.5 (3.4)</td>
<td>1.3 (1.5)</td>
<td>ABC &gt; D</td>
</tr>
<tr>
<td>PRLT</td>
<td>Trials 1–6 total time</td>
<td>272.2 (89.8)</td>
<td>263.1 (125.1)</td>
<td>251.5 (56.7)</td>
<td>251.1 (63.2)</td>
</tr>
<tr>
<td>Block 6–Block 1</td>
<td>26.5 (11.2)</td>
<td>20.4 (13.4)</td>
<td>22.1 (15.6)</td>
<td>30.6 (16.4)</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

Fig. 1. Mean time on target (in seconds) for the four subject groups.
time on target for the last block again revealed no differences when submitted to an ANOVA. The mean scores among the groups suggest that there is a trend toward our prediction that the alcohol dementia subjects exhibited the least amount of learning on this task, but this trend must be interpreted with caution, as it was not statistically significant. One interesting note is that the minimum scores for the groups on this variable (Block 6–Block 1) were all positive scores (i.e. indicating improvement in performance across trials), except for the group with alcohol dependence histories without dementia (Group C in Table 2), where the lowest score was \(-2.78\), indicating that at least one subject demonstrated no improvement on the task.

3. Discussion

This study attempted to demonstrate a double dissociation between subjects considered to have alcohol dementia and subjects diagnosed with AD on tasks of procedural and declarative learning. The performance of four groups of subjects (AD, alcohol dementia, alcohol—not demented, and an elderly control group) on the CVLT and the PRLT were compared. We hypothesized that subjects with alcohol dementia would perform similarly to subjects with other subcortical dementias who have been tested in previous studies. That is, they would perform worse than the control group on the PRLT and the free recall measure of the CVLT, but similar to the control group on the discriminability (recognition) measure of the CVLT. We hypothesized that the AD group would demonstrate the opposite pattern: normal performance on the PRLT and impaired performance relative to controls on all measures of the CVLT.

Contrary to our prediction, we did not identify a double dissociation between the performance of the AD and alcohol dementia groups. Whereas results from the CVLT were consistent with our predictions, subjects’ performances on the PRLT were not. Specifically, on the CVLT, subjects with alcohol dementia performed worse than the control group on free recall of List A, and cued recall intrusions, but not on the measure of discriminability. Subjects with AD, however, performed as predicted; that is, worse than the control group on all three measures of the CVLT. In the case of declarative learning, the fact that the alcohol dementia subjects demonstrated free recall skills that were significantly worse than those of the not-demented alcohol and control groups, whereas their recognition scores were not impaired, suggests that for declarative learning measures, the alcohol dementia subjects performed like patients with subcortical dementias in previous studies.

Performance on the PRLT was not worst among the subjects meeting criteria for alcohol dependence in addition to dementia, and thus did not differentiate among the groups. There are several possible explanations for this finding. One explanation is that our subjects may have had AD in addition to histories of heavy alcohol use, rather than alcohol dementia. While this explanation cannot be entirely discounted, we believe that it is unlikely based on the different neuropsychological profiles seen in the dementia screening battery among the subjects with AD and those with alcohol dementia. Subjects diagnosed with AD were rarely fully oriented based on their performance on the MMSE, whereas alcohol dementia subjects did not exhibit disorientation to time or place. Additionally, the difficulty with naming and access to semantic information typical of AD was not observed among the alcohol dementia
subjects, whose performances on tests of naming and category fluency were not significantly different from those of the control group.

Alternatively, perhaps the subcortical dementias examined in previous investigations have included only those dementias that have been associated with deficits in procedural learning. Alcohol dementia, however, may be a type of dementia that, although subcortical, affects only one subcortical system (i.e. that underlying free recall) as opposed to those involved in other prototypical subcortical dementias (i.e. those involved in procedural learning in addition to free recall). Or, perhaps, most of the motor effects associated with alcohol dementia occur by way of the cerebellum, which affects coordination, as opposed to motor movements seen in subcortical dementias such as Parkinson’s Disease or Huntington’s Disease, which may have as bases for motor deficits, damage in the striatum or basal ganglia.

Another consideration is that perhaps the difficulty of the PRLT task was not sufficient to reveal differences among the groups. There is some suggestion that not all procedural learning tasks are susceptible to impaired performances by those with subcortical dementias (Haaland & Harrington, 1997) and that if the task is not of sufficient difficulty, impaired performance may not be seen. In a study of patients with Parkinson’s Disease, Haaland and Harrington (1997) found that only when the speeds were varied on a PRLT tasks did patients with Parkinson’s Disease exhibit impairment. Thus, subjects with alcohol dementia in the current study may have demonstrated impaired performances on the PRLT under a more difficult condition.

Still another explanation is that alcohol dementia does not fit the classical understanding of a “subcortical dementia.” A substantial body of research indicates that long-term alcohol use affects the brain both cortically and subcortically. That these effects are perhaps not significant enough to cause impairment typically indicative of a subcortical dementia is certainly possible. It could be that the damage associated with the diagnosis of alcohol dementia results from more diffuse brain damage, perhaps rendering alcohol dementia as one of the “mixed” dementias, incorporating features of both cortical and subcortical dementias.

4. Conclusions

Alcohol dementia continues to distinguish itself as one of the more difficult types of dementia to characterize. Results of the current study suggest that the neuropsychological performance of those with alcohol dementia resembles that of a subcortical dementia in terms of impaired recall with intact recognition memory, but without the marked impairment in motor skill learning noted among other types of subcortical dementias. It appears, therefore, that alcohol dementia may be considered to be a “mixed” dementia, having characteristics seen in both cortical and subcortical dementias. Future investigation of procedural learning directly comparing alcohol dementia and other subcortical dementia subjects (e.g. Huntington’s Disease, Parkinson’s Disease) is needed in order to discern more clearly the nature of procedural learning ability associated with alcohol dementia.
References


