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Neuropsychological functioning in Alzheimer's disease and vascular dementia.

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NEUROPSYCHOLOGICAL FUNCTIONING IN ALZHEIMER'S DISEASE AND VASCULAR DEMENTIA

A Thesis Presented

by

PATRICIA A. BOYLE

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ABSTRACT

NEUROPSYCHOLOGICAL FUNCTIONING IN

ALZHEIMER'S DISEASE AND VASCULAR DEMENTIA

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The purpose of the present study was to investigate patterns of neuropsychological functioning in patients with early stage Alzheimer's disease (AD) and vascular dementia (VD). Scores from a battery of neuropsychological tests were obtained from the medical records of thirty-six hospital outpatients (14 AD, 22 VD). Subjects had been diagnosed with AD or VD on the basis of a neuropsychological evaluation and independent radiological exams using CT or MRI. VD patients performed significantly better on the composite of the WAIS than did the AD patients (MANOVA, $p<.01$), although scores were not significantly different between AD and VD patients on any other measure of cognitive functioning. Post-hoc statistical analyses revealed no pattern of cognitive performance that reliably distinguished between AD and VD. The absence of additional differences in the cognitive profiles of AD and VD patients suggests one of two possibilities: first, the currently used battery does not differentiate between AD and VD patients; and second, early stage AD and VD patients are functionally similar in their cognitive abilities. The current findings call into question the relative usefulness of neuropsychological testing in the differential diagnosis of early stage AD and VD.

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CHAPTER 1

INTRODUCTION

More than 4 million Americans currently suffer from dementia, a process of relentless and progressive decline in neuropsychological functioning (Corey-Bloom, Thanl, Galasko, Folstein, Drachman & Lanska, 1995). Estimates rank dementia as the fourth leading cause of death in the United States, and its prevalence is expected to rise as the population ages and life expectancy increases (Folstein, Bassett, Anthony & Romanowski, 1991). Among the many possible etiologies of dementia, Alzheimer's disease (AD) and vascular dementia (VD) account for between 70 and 90% of all dementia cases (American Psychiatric Association, 1994; Corey-Bloom et al., 1995; Cummings, 1990; Folstein et al., 1991; Barr, Benedict, Tune & Brandt, 1992). It is estimated that 50%-70% of dementia cases result from AD and 20-40% from VD, although these estimates may be considered preliminary due to inconsistent diagnostic criteria and the lack of pathological confirmation.

Despite the awareness of dementia as a major health concern in the United States, difficulty discriminating between dementia subtypes remains problematic (Cummings, 1990; Corey-Bloom et al., 1995). Fewer than 50% of VD cases and more than 80% of AD cases were accurately diagnosed in a study that employed standardized diagnostic criteria and confirmed diagnoses by autopsy (Cummings & Benson, 1983), indicating the complexity of AD and VD diagnoses in particular. At

present, no well-defined criteria exist for the diagnosis of VD; some clinicians rely heavily on clinical interviews and neuropsychological test results, while others focus more on neuroimaging studies and medical history reports (Corey-Bloom et al., 1995). Overlapping symptoms and high within-group variability further complicate distinctions between AD and VD. Difficulty discriminating between dementia subtypes poses a significant health risk to the aging population because valid and reliable diagnostic criteria are essential for the development of effective treatment and management strategies for dementia patients.

Significant differences in the neuropathologies associated with AD and VD, however, suggest differences in the patterns of neuropsychological decline between the two disorders (Almkvist, 1994; Gottfries, 1991). Specifically, AD involves a massive degeneration of the cerebral cortex and hippocampus, and the presence of neurofibrillary tangles and neuritic plaques. VD primarily affects subcortical brain regions such that neural pathways that connect cortical and subcortical areas of the brain are disrupted (Caplan, 1995; Cummings, 1994). Focal or diffuse white matter attenuation and ischemic injury characterize VD, and demyelination occurs in variable patterns within subcortical areas of the brain. Cortical areas may be affected during the course of VD, but cortical changes are much less common in VD than in AD, particularly in the early stages of the disorder (Cummings, 1990).

Differences in neuropsychological functioning that appear to reflect differences in neuropathological changes have been reported in AD and VD patients (Almkvist, 1994; Barr et al., 1992; Konliola, Laaksonen, Sulkava & Erkinjutti, 1990). After sudden onset, the cognitive decline in AD progresses gradually, but patients may

experience brief episodes of remission (Corey-Bloom et al., 1995, American Psychiatric Association, 1994; Cummings, 1990). Although VD may also progress gradually, it often begins insidiously and progresses in a stepwise manner (Cummings, 1994). Motor impairments and affective changes are more pronounced among VD patients throughout the course of the dementia (Cummings, 1990; Cummings, 1994; Almkvist, 1994).

Although AD and VD devastate all aspects of neuropsychological functioning over time (Corey-Bloom et al., 1995; American Psychiatric Association, 1994; Cummings, 1990), differences in cognitive functioning between AD and VD patients early in the course of dementia have proven especially difficult to assess (Kertesz and Clydesdale, 1994). Some studies suggest that patients may experience difficulty with delayed free recall and recognition memory early in the course of AD as compared to VD (Barr et al., 1992, Cummings, 1990; Flowers, Pearce, & Peare, 1984), while other studies contest those findings (Degrell, Gyozo, Nagy & Hoyer, 1986). Some studies suggest that aphasia occurs early in the course of AD and later in VD (Konliola et al., 1990; Cummings and Benson, 1983; Kertesz & Clydesdale, 1994; American Psychiatric Association, 1994), but other studies fail to report similar differences between groups or report slight differences on only one out of several verbal or memory tests used (Tierney, Snow, Reid, Zorzitto & Fisher, 1987).

At present, inconsistent findings and methodological limitations make comparisons between studies on the neuropsychological profiles of AD and VD tenuous at best. For example, the use of varying diagnostic criteria and neuropscyhological assessment tools prevent comparisons between samples, and small

sample sizes and differences in patient demographics and dementia severity compromise the reliability and generalizability of results. Moreover, the use of abridged versions of larger tests which have not yet been standardized makes assessments of impairment difficult if not entirely inappropriate (Almkvist, 1994).

The identification of differences in cognitive impairment early in AD and VD will be critical in the development of diagnostic criteria sufficient to distinguish between the two subtypes of dementia. The goal of the current project was to investigate patterns of neuropsychological functioning in patients with early stage AD and VD while trying to minimize methodological concerns that plague previous studies on dementia. To minimize diagnostic imprecision, only those patients who had received a diagnosis consistent with the Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV) guidelines for AD or VD by a licensed neuopsychologist and who also had supporting evidence of the diagnosis via brain imaging studies were included in the sample. Whereas many prior dementia studies used patients diagnosed on the basis of cognitive tests alone, the use of MRI and CT results can help distinguish between AD and VD and systematically improve diagnostic reliability (Tatemichi, 1990; Corey-Bloom et al., 1995). In addition, the use of an extensive battery of standardized tests allowed for specific predictions about differences between groups on a wide range of cognitive functions.

We hypothesized that AD and VD patients would show differences in neuropsychological functioning; specifically, the aim of the project was to aid in the establishment of diagnostic criteria that will accurately distinguish between AD and

VD on the basis of patterns of neuropsychological functioning. Clearly, improved diagnostic criteria will improve treatment options for dementia patients, as proper treatment hinges on proper diagnosis.

CHAPTER 2

METHOD

Subjects

Thirty-six consecutively referred outpatients (14 AD, 22 VD) who met the criteria for inclusion in this study were selected from the Baystate Medical Center (BMC) archives. Subjects were included on the basis of clinical, neuropsychological and neurological data obtained at BMC within the past three years. All patients had received a diagnosis of AD or VD consistent with the DSM-IV criteria and had no other confounding neurologic or psychiatric disorders (i.e. alcohol dependence, primary diagnosis of depression or other head trauma). Diagnoses were made by one of three Baystate neuropsychologists and were independently corroborated by documented reports of brain imaging results (MRI or CT). All patients with cortical degeneration described as excessive of that associated with normal aging were classified as AD. All patients with white matter hyperintensities on MRI or leukoaraiosis on CT were classified as VD.

Procedure

Patients were administered an extensive battery of neuropsychological tests and participated in an in-depth clinical interview upon their initial visit to BMC. The neuropsychologists examining the patients had access to available prior medical history reports and, in some cases, to interviews with patients and family members or caregivers. Patients' scores on five subtests of the Weschler Adult Intelligence Scale-

Revised (Block Design, Vocabulary, Similarities, Comprehension and Digit Span), the California Verbal Learning Test, the Boston Naming Test, the Fluency Assessment Scale, and the Hooper Visual Organization Test were analyzed to investigate cognitive functioning. This test battery was designed by a team of BMC neuropsychologists to assess short term memory, verbal ability, attention and concentration, verbal fluency, visual reasoning, and visuospatial skills. A brief description of each test follows:

1. WAIS-R: measures multiple aspects of intellectual functioning. The following subtests were administered:

   Digit Span: consists of digit repetition tasks forward and backward, and assesses attention/concentration and immediate memory.

   Similarities: consists of verbal comparisons between items, and measures verbal concept formation and executive functioning.

   Vocabulary: consists of word lists and definitions, and measures language functioning, remote memory, learning, and verbal capacity.

   Block Design: consists of block arrangement tasks, and measures visuospatial organization and manipulation.

2. California Verbal Learning Test: consists of a series of word list learning and cueing, and measures immediate free recall memory, delayed recognition memory, and verbal ability.

3. Boston Naming Test: consists of a series of picture identification cards, and measures recognition memory and language fluency.

4. Fluency Assessment Scale: consists of spontaneous word production according to a set of rules, and measures verbal fluency and production abilities.

5. Hooper Visual Organization Test: consists of a series of visual integration challenges, and measures visual processing and integration, naming, and fluency.

Data Analysis

The AD and VD groups' scores were analyzed using descriptive and inferential statistics. When necessary, analyses of group differences were computed using nonparametric methods. For the purpose of some post-hoc analyses, cognitive test scores were transformed into z-scores and analyzed using descriptive statistics.

CHAPTER 3

RESULTS

Table 1. presents demographic data for the 36 subjects in this sample. Table 2. and Appendix A present descriptive statistics comparing group performances on the battery of neuropsychological tests. The AD and VD groups did not differ significantly with regard to age or education, and all subjects were considered to be in the early stages of dementia because the neuropsychological data was obtained for all subjects at the time of initial diagnosis. Contrary to the hypotheses, t-tests revealed no significant differences between AD and VD groups on any single neuropsychological measure when all tests were analyzed separately. Because the subtests of the WAIS-R are correlated, however, data from the WAIS-R subtests were collapsed by group and analyzed as a composite using a MANOVA. These results revealed a significant main effect of group on subtests of the WAIS-R, indicating that the VD group performed significantly better on the WAIS-R composite than did the AD group ($F [1,34] = 5.03$, $p < .03$).

Given the surprising similarity between the two groups on most measures of neuropsychological functioning, post-hoc analyses were employed to determine whether individual subjects' scores reflected a systematic pattern of impairments consistent with those used for diagnosis as described by the neuropsychologists. For example, the neuropsychologists predicted larger discrepancies between BNT and FAS test scores and greater variability in the general cognitive profile for VD as

compared to AD patients. To investigate whether the data reflected these patterns, difference scores were calculated for the BNT and FAS tests (BNT score - FAS score) and WAIS-R discrepancy scores (maximum WAIS-R subtest score minus minimum subtest score). T-tests revealed no significant differences, although there was slightly more variability among the WAIS-R scores in the VD group. Table 3 and Appendix B present the results of these analyses.

Further theory-driven statistical comparisons were run to determine whether any systematic pattern of impairment differentiated AD from VD patients. Because the neuropathology associated with VD is believed to create a more variable pattern of cognitive impairment, scores on the battery of tests were standardized and several measures of variability were calculated for each individual subject, including variance scores, standard errors of the mean, and standard deviation estimates. No significant differences emerged. Appendix C presents the results of these analyses.

Table 1. Demographic data for AD and VD patients.

Variable	AD Patients	VD Patients
Sample Size	14 (4 male, 10 female)	22 (9 male, 13 female)
Age	75.6 (9.1)	75.7 (7.5)
Education	12.5 (2.4)	11.7 (2.8)

Table 2. AD and VD group means on neuropsychological tests.

Measure	AD		VD	
	Mean	SD	Mean	SD
WAIS-R				
Digit Span	7.6	2.5	8.0	2.7
Vocabulary	8.7	3.0	9.3	2.1
Comprehension	6.1	3.1	8.3	2.7
Similarities	6.8	2.7	7.8	3.0
Block Design	6.8	3.0	7.8	3.0
California Verbal Learning Test	1.9	1.9	2.6	2.0
Boston Naming Test	33.8	14.5	38.4	11.6
Fluency Assessment Scale	18.1	14.3	21.6	11.3
Hooper Visual Organization Test	15.7	7.0	15.8	6.0

Table 3. AD and VD difference score group means.

Measure	AD		VD	
	Mean	SD	Mean	SD
Boston Naming Test-Fluency Assessment Scale	18.9	11.1	16.3	13.2
WAIS-R	4.9	1.7	5.0	2.5

CHAPTER 4

DISCUSSION

The VD group's superior performance on the WAIS-R composite represented the only significant difference between groups. The WAIS-R appears to be a sensitive measure of overall cognitive functioning in AD and VD (Perez, Rivera, Meyer, Taylor & Matthew, 1975; Gainotti, Caltagirone, Masullo & Miceli, 1980; Loring, Meador, Mahurin & Largen, 1986), and the higher scores on the WAIS-R for VD patients in the current sample corroborate previous reports of a more subtle deterioration of intellectual functioning in VD than in AD (Almkvist, 1994). These results suggest that general cognitive functioning may decline more slowly in VD than in AD. However, it may also be the case that VD tends to be diagnosed earlier in the course of the illness than does AD. Pronounced affective and motor changes may accompany VD (Cummings, 1994), and VD patients or their caregivers may therefore be more likely to recognize their symptoms and seek help. Consequently, VD patients' scores on neuropsychological tests may be elevated as a result of the timing of diagnosis.

The absence of differences between groups on the remaining cognitive tests used in the current study contrasts with the findings reported in several prior studies, in which AD and VD groups performed significantly differently on the Boston Naming Test, the Hooper Visual Organization Test and the Fluency Assessment Scale (Barr et al., 1992). However, Almkvist (1994) reported a "slight relative deficit" in VD in executive functions, fluency, attention, and motor functions, and a "slight

relative advantage" in VD in naming after reviewing the dementia literature and taking methodological limitations into consideration. Almkvist concluded that "functional similarity may be the main feature" between AD and VD, and the results of the current study concur with her conclusion. It is possible that the neuropathological changes that occur early in the course of dementia do not produce functionally distinct patterns of cognitive functioning in AD and VD patients.

Two possible hypotheses may account for the striking similarity between AD and VD patients on individual subtests of the WAIS-R and on the remaining cognitive tests in the current battery, including the Hooper Visual Organization Test, the Boston Naming Test, and the California Verbal Learning Test. First, the current test battery may be insufficient to distinguish between patterns of impairment in AD and VD patient groups; second, newly diagnosed AD and VD patients may be functionally similar in their cognitive abilities. Although it is not possible to determine which of these hypotheses accounts for the present results, the stringent criteria used for inclusion in the current study and the inclusiveness of the current battery of cognitive tests favors the latter explanation. Moreover, the implications of these findings call into question traditional methods of differential diagnosis of dementia subtypes which often rely on cognitive profiles.

The absence of differences between groups in the current study are especially surprising given the different neurological profiles observed in AD and VD patients in the present sample. While the WAIS-R composite may be sensitive to the early effects of neurolopathology, the other cognitive measures may not. For example, many of the present tests may rely heavily on prefrontal cortex functioning, which may be

equally compromised in AD and VD. While the cortical degeneration that occurs in AD may compromise prefrontal functioning, similar deficits may occur in VD as a result of the disruption of prefrontal functioning due to severed connections to subcortical brain regions. In addition, the absence of differences between groups on more specific aspects of cognitive functioning, including verbal fluency, naming, and short-term memory also suggests that changes in posterior cortical regions which house language-related functions do not differ between groups at this stage of dementia. Although one would expect that differences in such functions may emerge over the course of the disease, variations in the topographical distribution of brain lesions may prevent the identification of such differences (Snowden, et al., 1997). Recent literature also suggests that AD may involve a vascular component similar to VD, which supports the notion that AD and VD may actually be more similar than different. The brain imaging reports in the current study provided general information about the nature of brain lesions, but did not provide enough anatomical data to systematically examine the topography of brain changes in the current sample.

Specific limitations of the current study suggest caution when making interpretations on the basis of these results. The size of the current sample may limit the possibility of finding significant differences between groups, and uneven male-female ratios may also contribute to the variability in this sample. However, the standard deviations from the mean do not suggest that large differences were masked. Finally, although the use of neurological data added evidence in support of the current diagnoses, the lack of information specific enough to allow for the systematic classification of brain changes prevents an adequate description of the patterns of

neuropathology between groups in this sample. Future studies that describe the nature of brain changes specifically in larger samples may yield differences that are more easily replicated.

Nevertheless, these data call into question the true usefulness of neuropsychological test scores in the diagnosis of dementia subtypes. If groups are functionally similar in the early stages of the disease, cognitive tests alone do not provide useful information. Thus, the traditional reliance on such measures may be misguided. Further, the similarity between groups highlights a logical error in previous studies in which AD and VD patient groups were selected on the basis of cognitive profiles, then reviewed to identify patterns of impairment between groups. Circular reasoning prevents a clear interpretation of results found in studies where diagnoses were decided in this manner, and sampling errors are inevitable. Careful attention must be paid to this error when the dementia literature is reviewed and statements are made about patterns of neuropsychological functioning in AD and VD.

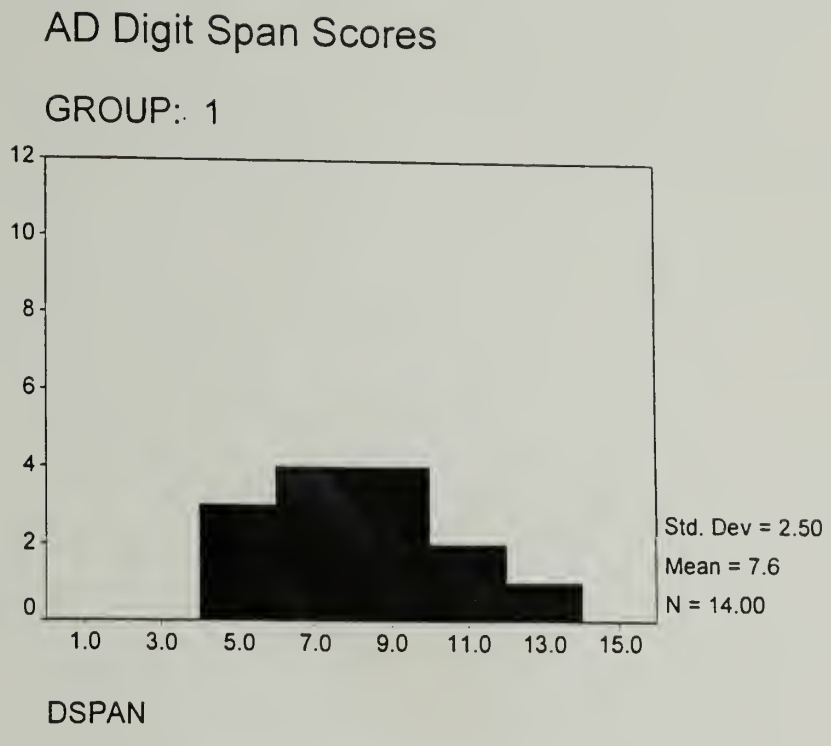
Clearly, neuropsychological test scores have proven successful in discriminating between demented patients and healthy adults, and the importance of this distinction must not be underestimated. However, attempts to differentiate between dementia subtypes on the basis of cognitive profiles have proven more difficult. Tierney et al. (1987) accurately differentiated AD from "other dementias" at a rate of 70% on the basis of cognitive test scores and Parlatto et al. (1990) distinguished AD from VD at a rate of 56-70%; consequently, 30-44% of patients were incorrectly assigned to a dementia group in those studies. Perez et al. (1978) improved on these figures in a series of follow-up studies, although the results of the

Perez studies are confounded because the AD patients were significantly more well educated than the VD patients, and dementia severity was not considered a factor. These inconsistencies illustrate current difficulties discriminating between dementia subtypes.

The present results challenge the use of neuropsychological test scores as a primary source of information used for the differential diagnosis of dementia subtypes. Until patterns of impairment sufficient to distinguish between AD and VD groups are identified and replicated in samples whose diagnoses have been independently supported by brain imaging scans, alternative factors should be weighed more heavily in the differential diagnosis of dementia. For example, neuroimaging findings will elucidate brain changes and medical history reports may indicate pre-existing medical conditions (such as diabetes, heart disease) which are known to play an integral role in the etiology of specific dementia subtypes. In addition, recent literature indicates that affective changes are more prominent in VD than in AD early in the course of dementia, as VD patients appear to remain cognizant of their illness for a longer period of time than do AD patients, although this literature may also be methodologically biased. Depression screens may therefore serve as a useful aid in the differential diagnosis of dementia subtypes. As diagnostic precision improves, so will the current understanding and treatment of dementia. Until then, however, the dementia literature must be approached from a critical perspective and diagnoses must rely on convergent sources of information to improve the likelihood of their accuracy.

APPENDIX A

SUBTEST DISTRIBUTIONS BY GROUP



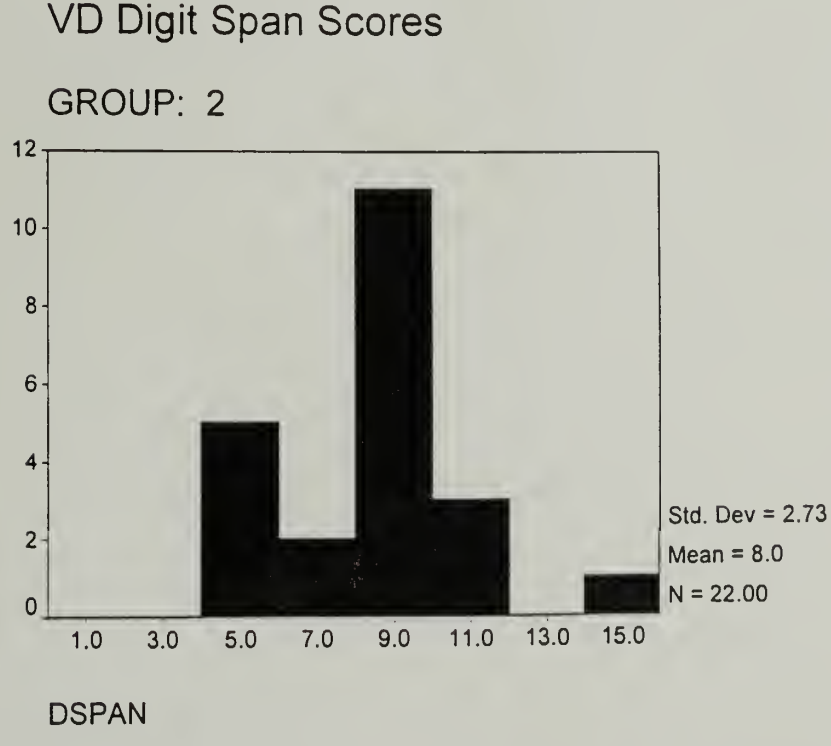
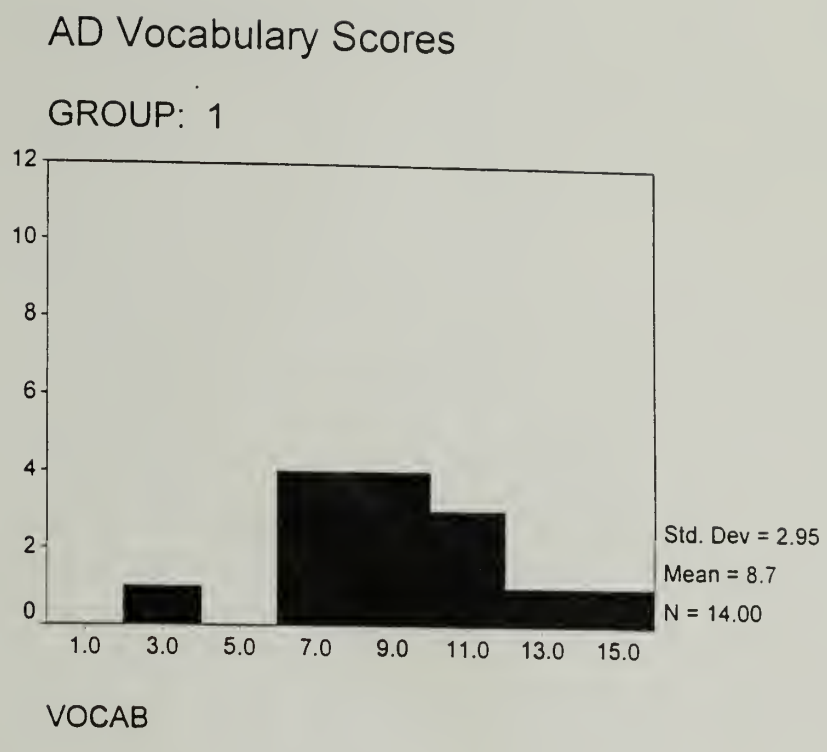


Figure A.1. Digit Span Distribution by Group



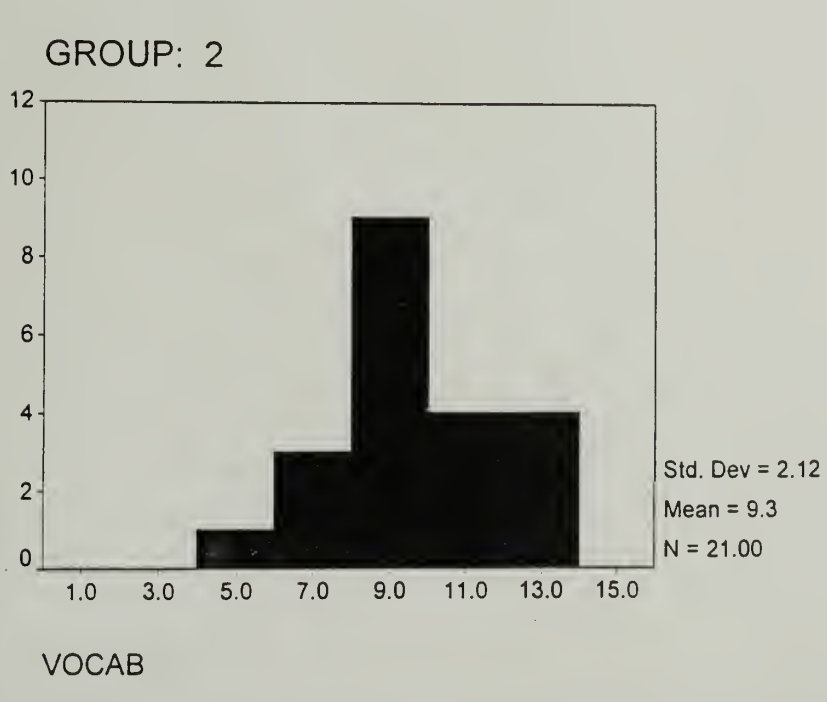
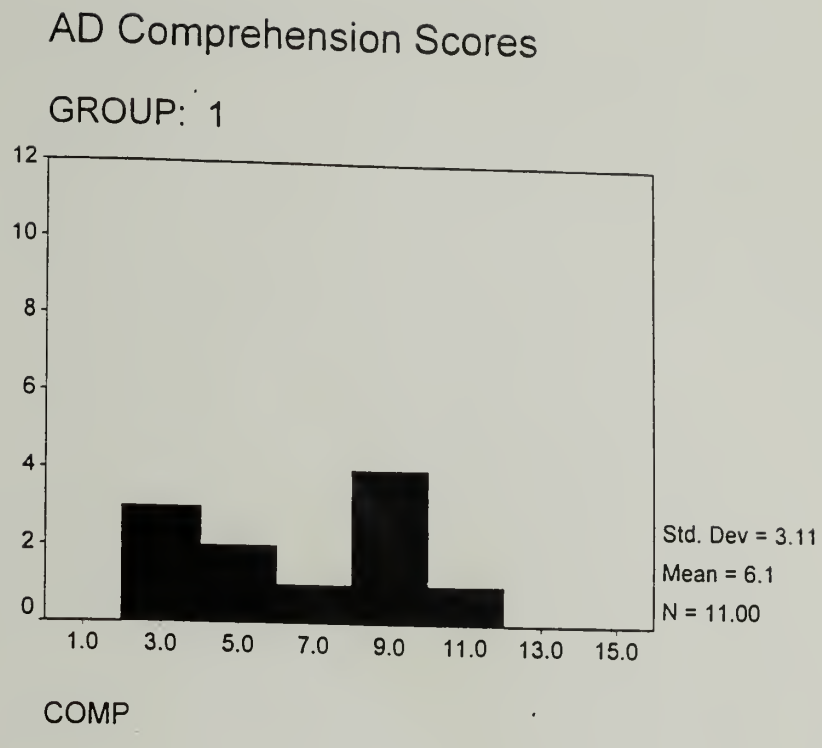


Figure A.2. Vocabulary Distribution by Group



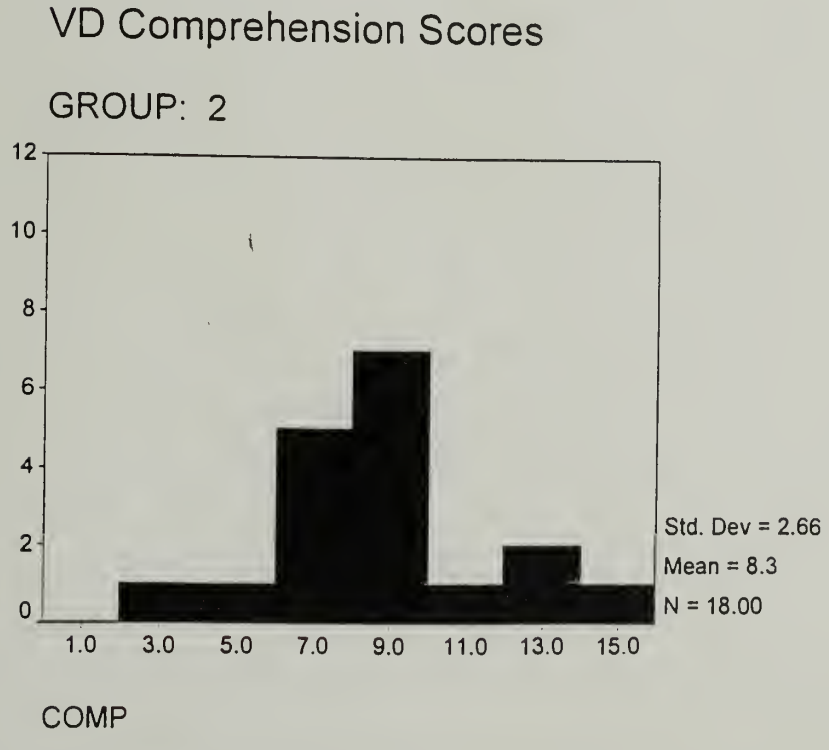
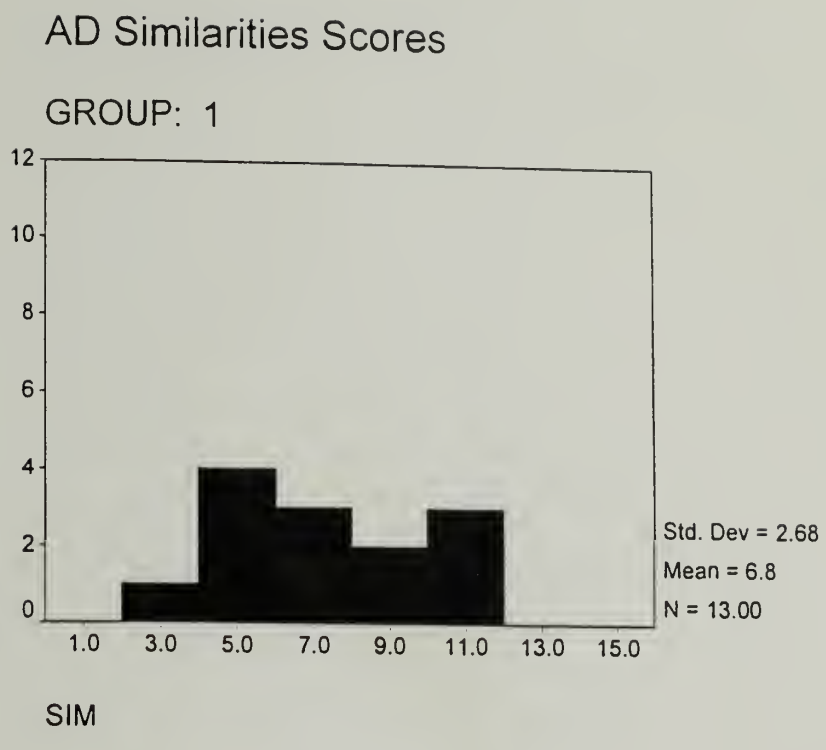


Figure A.3. Comprehension Distribution by Group



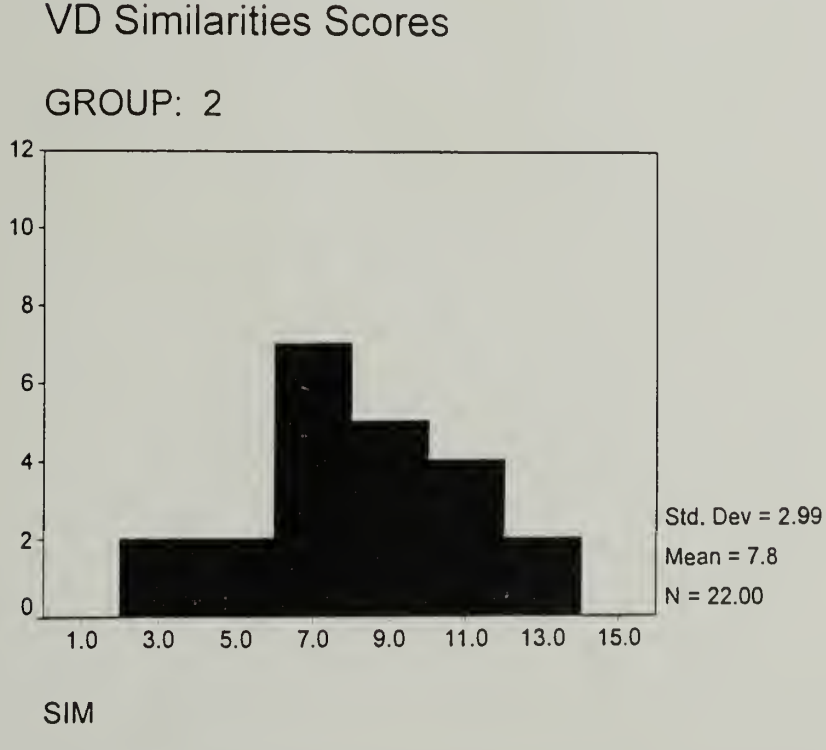
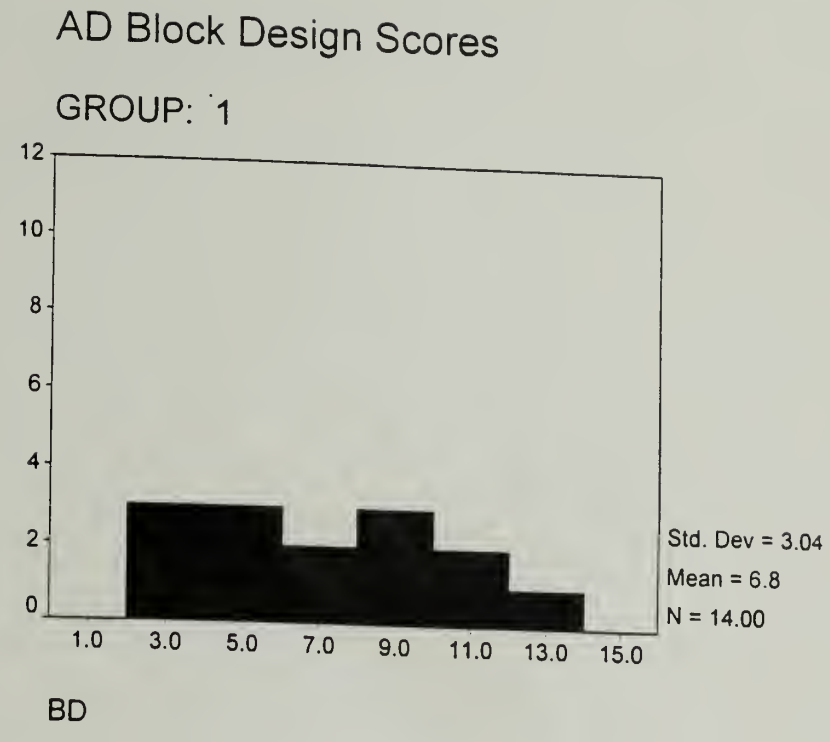


Figure A.4. Similarities Distribution by Group



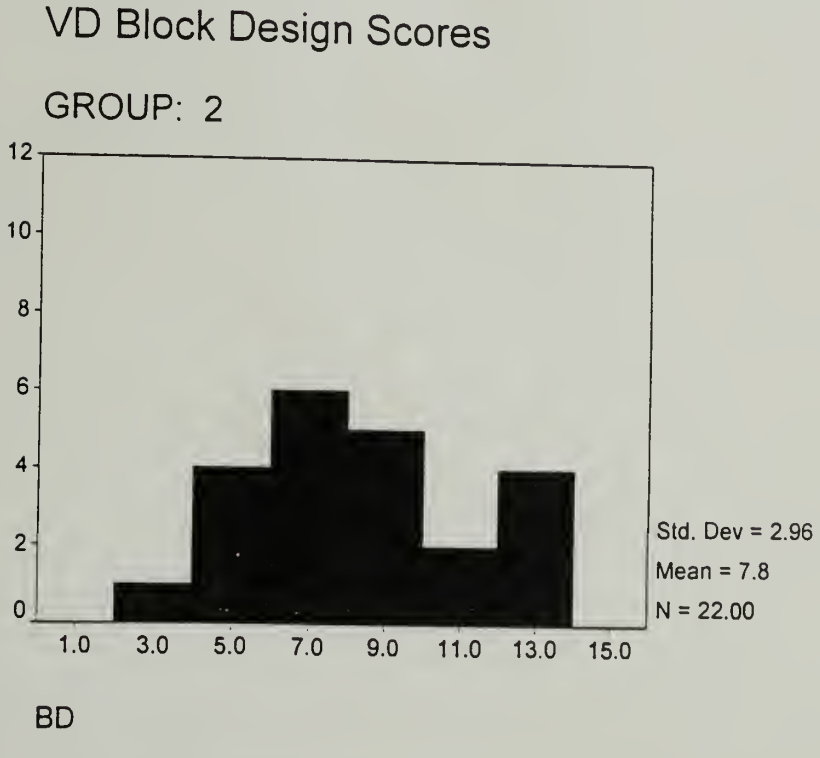
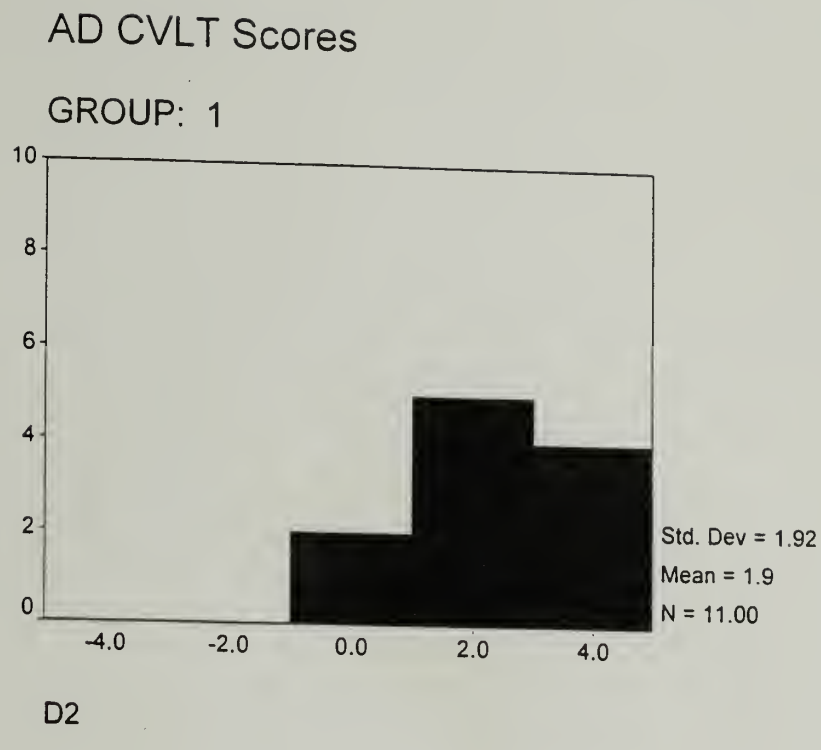


Figure A.5. Block Design Distribution by Group



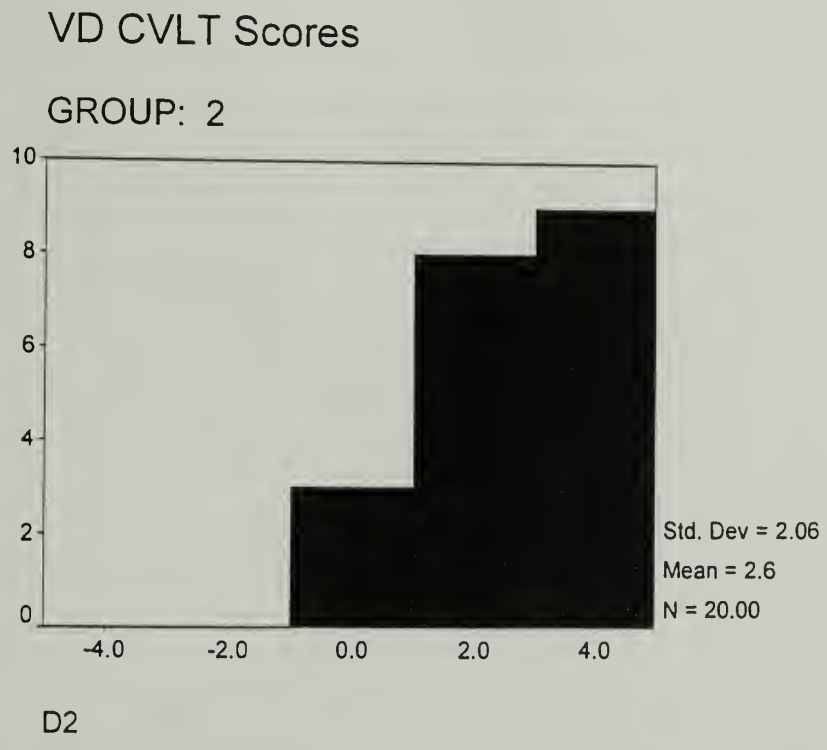


Figure A.6. California Verbal Learning Test Distribution

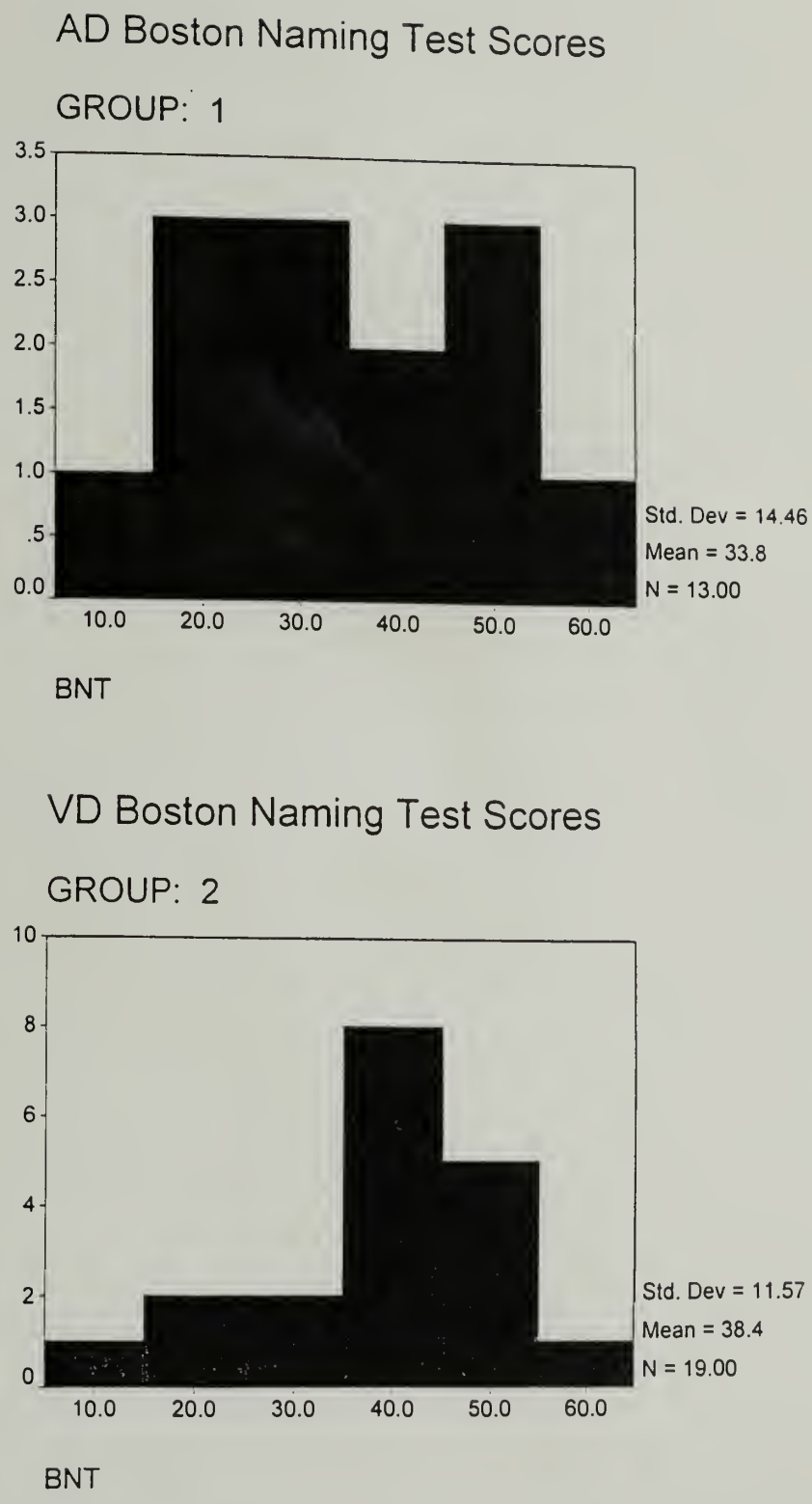
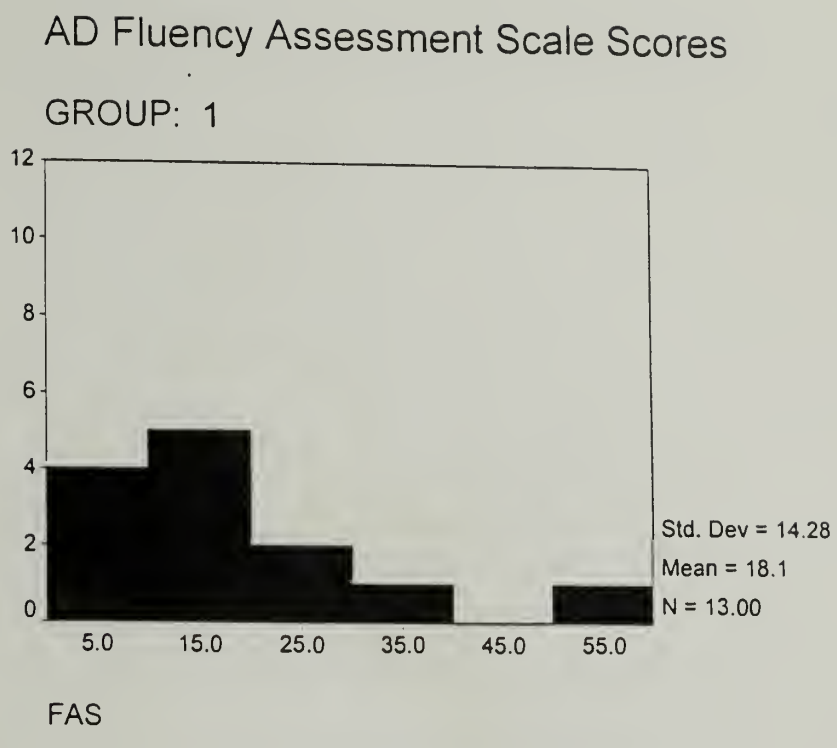


Figure A.7. Boston Naming Test Distribution by Group



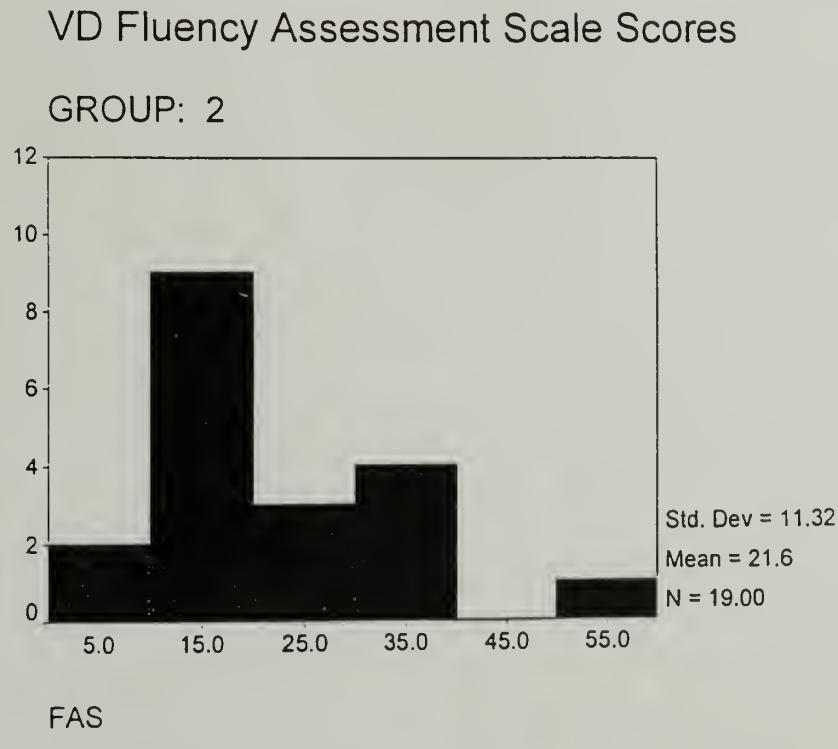


Figure A.8. Fluency Assessment Scale Distribution by Group

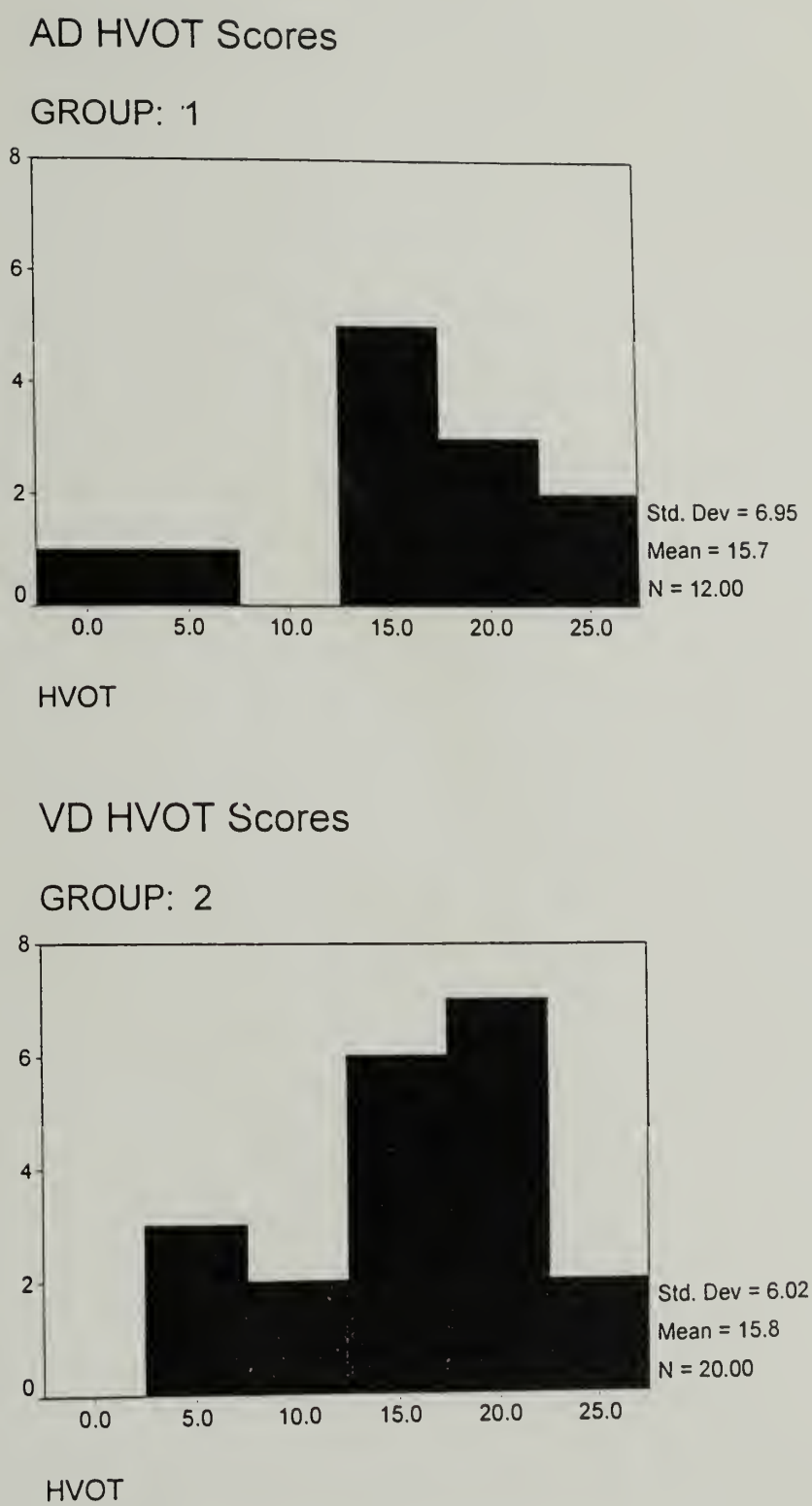
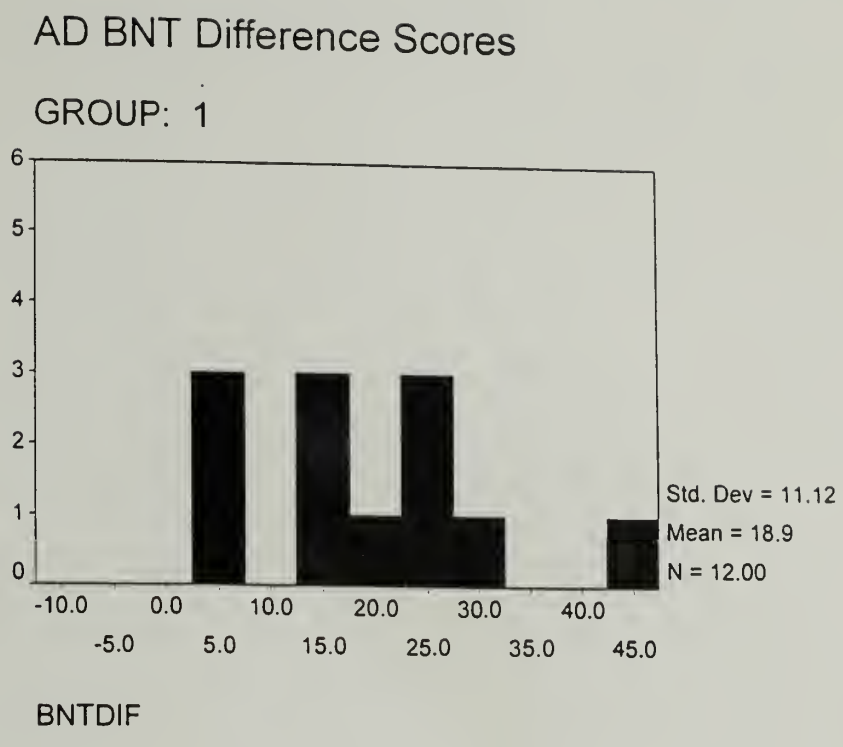


Figure A.9. Hooper Visual Organization Distribution by Group

APPENDIX B

DIFFERENCE SCORE DISTRIBUTIONS BY GROUP



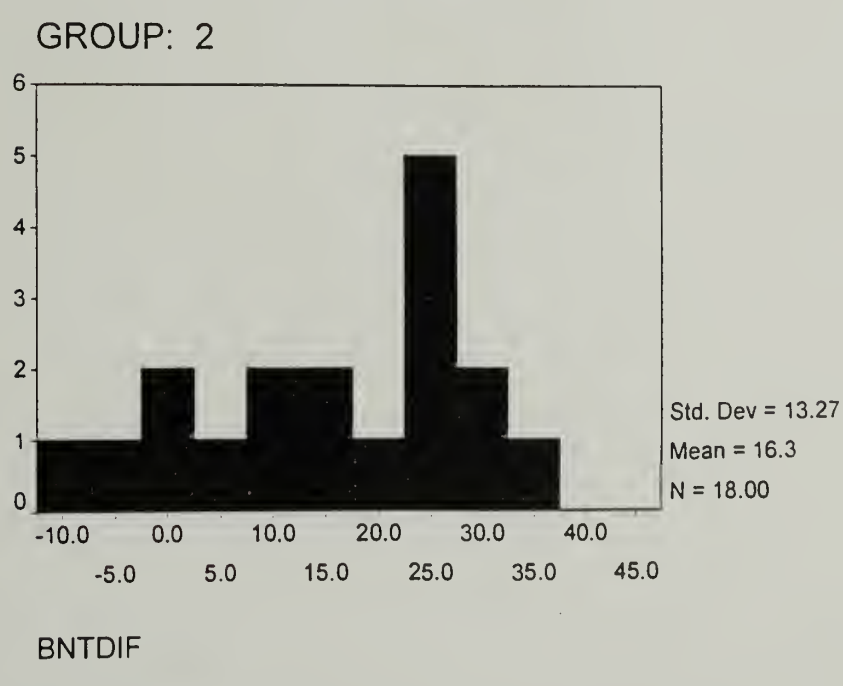
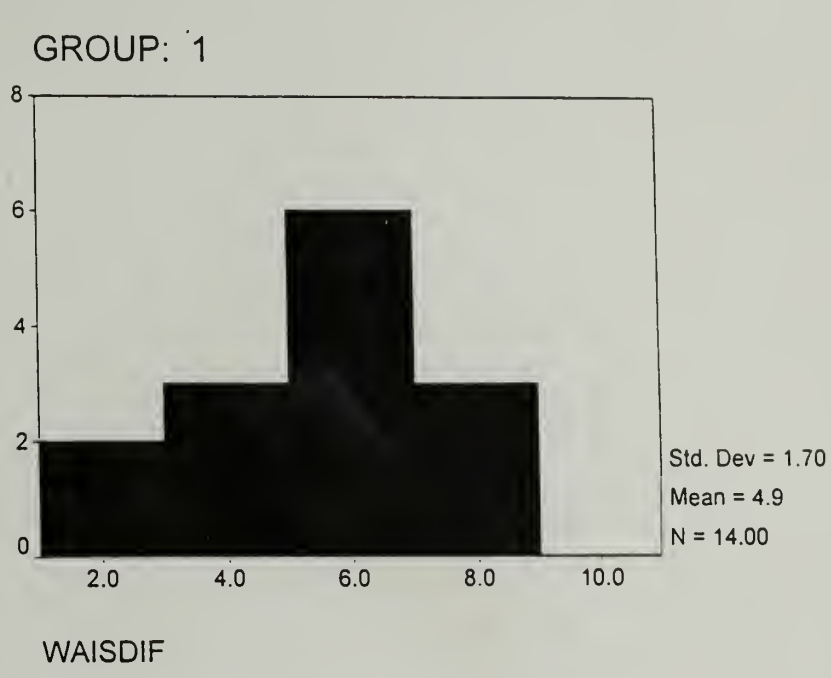


Figure B.1. BNT-FAS Difference Score Distribution by Group



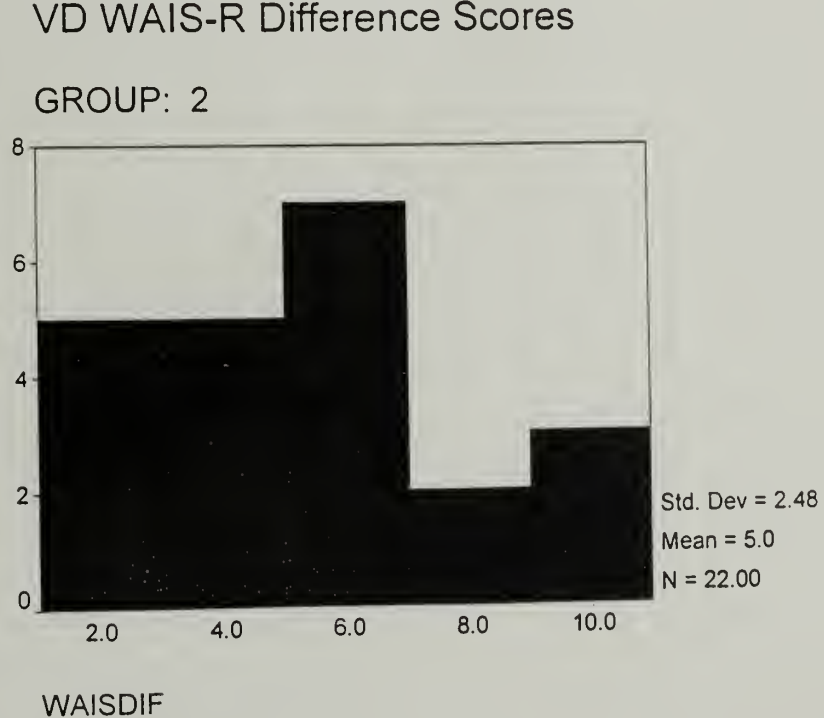


Figure B.2. WAIS-R Difference Scores Distribution by Group

APPENDIX C

DESCRIPTIVE STATISTICS FOR AD AND VD

Descriptive Statistics

GROUP		N	Mean		Std.	Variance
		Statistic	Statistic	Std. Error	Statistic	Statistic
1	Zscore(A)	14	-1.17E-02	.3385653	1.2667952	1.605
	Zscore(A1)	14	2.79E-02	.3218979	1.2044316	1.451
	Zscore(A5)	14	-4.73E-02	.3768911	1.4101973	1.989
	Zscore(B)	14	-.1661247	.2950220	1.1038711	1.219
	Zscore(B1)	14	6.83E-02	.3833220	1.4342596	2.057
	Zscore(B2)	14	-.2581362	.3795106	1.4199987	2.016
	Zscore(B3)	14	-.2457635	.2649074	.9911928	.982
	Zscore(B4)	14	-.1195229	.3554907	1.3301243	1.769
	Zscore(BD)	14	-.2018429	.2721264	1.0182037	1.037
	Zscore(BNT)	13	-.2098779	.3133876	1.1299350	1.277
	Zscore(C1)	14	.3935689	.3599278	1.3467265	1.814
	Zscore(C2)	14	.1855666	.2698608	1.0097267	1.020
	Zscore(C3)	14	.1029293	.3214285	1.2026753	1.446
	Zscore(COMP)	11	-.4646498	.3133425	1.0392396	1.080
	Zscore(D1)	12	1.63E-02	.2643996	.9159071	.839
	Zscore(D2)	11	-.2060812	.2886981	.9575032	.917
	Zscore(DSPAN)	14	-8.36E-02	.2560044	.9578808	.918
	Zscore(FAS)	13	-.1687221	.3166057	1.1415379	1.303
	Zscore(HVOT)	12	-9.14E-03	.3199148	1.1082175	1.228
	Zscore(SIM)	13	-.2188657	.2580890	.9305533	.866
	Zscore(VOCAB)	14	-.1393284	.3200038	1.1973447	1.434
	Valid N (listwise)	7				
2	Zscore(A)	21	7.80E-03	.1768426	.8103948	.657
	Zscore(A1)	21	-1.86E-02	.1897571	.8695763	.756
	Zscore(A5)	21	3.15E-02	.1388322	.6362090	.405
	Zscore(B)	21	.1107498	.2042125	.9358192	.876
	Zscore(B1)	21	-4.56E-02	.1304636	.5978594	.357
	Zscore(B2)	21	.1720908	.1218122	.5582136	.312
	Zscore(B3)	21	.1638423	.2172263	.9954560	.991
	Zscore(B4)	21	7.97E-02	.1593638	.7302967	.533
	Zscore(BD)	22	.1284455	.2111044	.9901675	.980
	Zscore(BNT)	19	.1436007	.2074245	.9041425	.817
	Zscore(C1)	21	-.2623793	.1273167	.5834385	.340
	Zscore(C2)	21	-.1237111	.2179034	.9985588	.997
	Zscore(C3)	21	-6.86E-02	.1886622	.8645587	.747
	Zscore(COMP)	18	.2839526	.2090563	.8869509	.787
	Zscore(D1)	21	-9.30E-03	.2328256	1.0669410	1.138
	Zscore(D2)	20	.1133446	.2300330	1.0287389	1.058
	Zscore(DSPAN)	22	5.32E-02	.2226939	1.0445268	1.091
	Zscore(FAS)	19	.1154414	.2075999	.9049068	.819
	Zscore(HVOT)	20	5.48E-03	.2145300	.9594075	.920
	Zscore(SIM)	22	.1293297	.2212623	1.0378123	1.077
	Zscore(VOCAB)	21	9.29E-02	.1884132	.8634176	.745
	Valid N (listwise)	13				

Figure C.1. Descriptive Statistics for AD and VD: Measures of Variability

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