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AN ANALYSIS OF MEMORY FUNCTIONS IN PARKINSONISM

by

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the University of Manitoba in partial fulfillment of the requirements
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ABSTRACT

Some researchers have suggested that the memory deficit associated with parkinsonism is a deficit secondary to loss of motor control. Others have asserted that the parkinson memory deficit is a real deficit associated with generalized cerebral cortical impairment. The question of where the memory loss may be occurring has not been explored to date. The primary purpose of the present study was to test the hypothesis that memory loss in parkinsonism results from loss of motor control. A secondary purpose was to attempt to identify whether the parkinson memory loss is mainly an input loss or an output loss.

Four groups of persons served as subjects for this study: those diagnosed as having parkinsonism, those diagnosed as having rheumatoid arthritis, older subjects, and normal subjects. Each group consisted of 14 males and 14 females. Groups were matched for average age (except the older subjects), and average education. Subjects were carefully screened to control for possible hearing loss, language barrier, and other extraneous factors. The independent variables included diagnostic group, position in space (either sitting or reclining), and recall condition (either non-cued recall or cued recall). The dependent variables included Weschler Memory Scale MQ, and number of correctly recalled words from lists of 24 words with 4 words in each of 6

categories. Subjects served as their own controls since each subject was tested in both positions in space and under both recall conditions.

Screening test data analysis revealed that the parkinson group achieved lower WAIS verbal IQ's than the other three groups. Similar results were obtained with the MQ's and the number of correctly recalled words in cued and non-cued recall. Position in space was found to have no effect upon any of the measures. All groups correctly recalled significantly more words under the cued condition than under the non-cued condition.

The above findings supported the hypothesis that parkinson memory loss is real and is produced by generalized cerebral cortical impairment. Such memory loss does not appear to be primarily due to retrieval difficulties, nor to input loss at the perceptual level. Rather, while most new information is received, much of it does not seem to be consolidated into any sort of memory store. The need for further clarification of locus of memory loss was discussed, as were some of the clinical implications of memory loss associated with generalized cerebral cortical impairment.

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CHAPTER I

INTRODUCTION

Overview

Persons diagnosed as having Parkinson's disease often complain of having impaired memory for recent events. They misplace things, forget appointments, and have trouble keeping immediate information available for use. Since parkinsonism is primarily a disturbance of the extrapyramidal system resulting in motor impairment, the cognitive functions have been little investigated. However, recent studies (Reitan & Boll, 1971) indicate that parkinson persons appear to perform relatively below the expected level on intellectual tasks when compared with age- and education-matched non-parkinson peers. These findings seem to be in agreement with recent parkinson autopsy studies which show generalized cerebral impairment in excess of that of age peers (Alvord, Forno, Kusske, Kauffman, Rhodes, & Goetowski, 1974).

Three principal hypotheses have been proposed to account for the observed intellectual and memory deficits found in parkinson persons. Cooper, Riklan, Stellar, Waltz, Levita, Ribera, & Zimmerman (1968) believed the intellectual deficit to be more apparent than real, and hypothesized that this apparent deficit was a result of interference with attention due to impaired automatic

postural reflex function. Reitan & Boll (1971) felt that the intellectual deficit was real, and was most likely due to generalized cerebral impairment. Riklan (1973), noting improved intellectual function following ingestion of L-dopa, hypothesized that the intellectual and memory deficit was a result of a lowered state of arousal.

Most of the studies of parkinsonism have been notable for the lack of adequate controls, incomparability with other studies, and contradictory results. The purpose of this study was to attempt to clarify the nature of some of the memory deficits found in persons with Parkinson's disease, to use adequate controls, to use measures which could be compared with other studies, and to attempt to delineate where the breakdown in memory may be occurring. Each of the three major hypotheses mentioned above was investigated, as well as others which have relevance for conceptualizations of the memory process in general.

Intellectual Functions in Parkinson's Disease

Parkinson's disease is a disorder of the extrapyramidal system, and is usually studied in terms of motor deficit and correction. (For a discussion of the parkinson syndrome, see Appendix A.) Parkinson (1817) felt that the disorder had no effect on the intellect. But parkinson persons themselves complain of reduced ability to think clearly, and physicians over the years have

observed that their parkinson patients gradually deteriorate in intellectual functions as well as motor functions. Only fairly recently have cognitive functions been evaluated as well as motor functions. Such research has been plagued with poor experimental design, lack of control groups, and a lack of replication of standardized test procedures.

The researchers who have done the most work with intellectual functions in parkinsonism have been those working with Cooper's program at St. Barnabas Hospital in New York. Working with parkinson patients referred to Cooper for thalamic surgery, Riklan, Weiner, Levita, Diller, and their co-workers have contributed to the literature consistently for the last 23 years. Ordinarily, their research question has been whether or not thalamic surgery results in any intellectual deficit. When control groups were used, the controls were usually persons with parkinsonism on whom surgery was not performed. It should also be noted that patients used in these studies were private patients of above average education.

In a series of early studies, Diller & Riklan (1956), Riklan, Weiner & Diller (1959), and Riklan, Diller, Weiner, & Cooper (1960) noted that parkinson persons were of about average intelligence, but this average level was somewhat lower than might be expected for their education level. They found that thalamic surgery

appeared to have no effect on intellectual function.

Talland compared parkinson subjects with non-parkinson subjects on the Wechsler-Bellevue (education not matched), and concluded that there appeared to be no cognitive impairment associated with the syndrome. He further felt that parkinsonism did not effect attention or concentration (Talland, 1962). Levita, Riklan, & Cooper (1964), and Riklan, Levita, & Cooper (1966) tested 81 parkinson patients with parts of the WAIS, and concluded that age, motor impairment, and laterality of subcortical involvement did not influence performance based on overt verbal responses, verbal fluency, or visual and visual-spatial discrimination. As in their earlier studies, they concluded that bilateral cryosurgery or chemosurgery had no effect on cognitive functions.

Asso (1969) and Asso, Crown, Russell, & Logue (1969) reported two other studies of parkinsonism patients with the WAIS, and reported no evidence of any pattern of specific intellectual deficit associated with parkinsonism. Yahr, Duvoisin, Shear, Barrett, & Hoehn (1969) reported on the first intellectual studies of parkinson patients taking L-dopa. They concluded that the patients who were intellectually intact but somewhat dulled by anticholinergic medication slowly returned to normal. Others with varying degrees of dementia remained unchanged. In some cases, resolution of the parkinson

symptoms appeared to unmask a dementia that was not previously apparent.

Cooper, Riklan, Stellar, Waltz, Ribera, & Zimmerman (1968) summarized in a lengthy review article much of what was known about parkinsonism at that time. They concluded that parkinsonism has no effect upon the intellect, nor does thalamic surgery. However, Cooper, et al, stated:

A secondary symptom is produced by the necessity for the patient to concentrate on acts which ordinarily do not require attention; e.g., rising from a chair or turning in a close space. The patient has to divert a great deal of attention to the performance of routine motor activities, and this indirectly diverts his attention from the world around him. The effect may imitate intellectual or emotional impairment. Often the patient's apparent intellectual impairment is caused by the urgent need for using all his resources to overcome rigidity in the performance of routine activities; there is no impairment of the intellectual processes (p. 1185).

Selby (1968) also reviewed the literature on parkinsonism up to 1968, and stated that he was unable to contradict Parkinson's original conclusions (no intellectual deficit) with any degree of confidence. He felt that one factor which needed to be isolated was the role that depression might play in suppressing intellectual performance. It is known that depression is very frequently found among parkinson persons. Selby concluded that there was an obvious need for carefully

designed psychometric studies to determine whether or not any intellectual impairment was present in parkinson persons.

The first attempt to include a control group (Meier & Martin, 1970) yielded helpful indications of the possible effect of L-dopa on intellectual functions. The authors compared 39 parkinson patients with 25 non-parkinson controls matched for age and education. While all parkinson patients received L-dopa, none of the controls received L-dopa. Nevertheless, the pre-treatment measures were the first results of comparing intellectual functions between parkinsons and non-parkinson controls matched for age and education. While many details of the procedure were left out of the report and results are difficult to interpret, the authors concluded that L-dopa improved intellectual functioning, and that the improved intellectual function was not due simply to improved motor function.

Reitan & Boll (1971) provided the first definitive, well-controlled evaluation of parkinson intellectual function in the literature. They administered 32 different measures to 25 parkinson patients and 25 non-parkinson controls matched to age, sex, race, and education. The authors found consistent impairment across all measures in the parkinson persons. They concluded that while the parkinson persons appeared to function within normal limits compared with the general population, they

showed clear and significant deficits when compared with age- and education-matched non-parkinson peers. For example, the parkinson patients' means on the WAIS were VIQ=107.6; PIQ=105.2; and FSIQ=105.76. The matched controls obtained means of VIQ=119.6; PIQ=121.96; and FSIQ=122.28. Most of the p-values were $\leq .001$. Reitan & Boll concluded that their results suggested that parkinson patients show impairment which extends far beyond motor function loss, and which implies the presence of widely generalized cerebral impairment.

Loranger, Goodell, McDowell, Lee, & Sweet (1972) was the second study to use a non-parkinson control group. They compared 27 parkinson patients with 27 hospitalized depressed non-parkinson controls, matched for age, sex, and education. The parkinson patients were found to perform consistently poorer on all WAIS subtests than the non-parkinson patients. The authors concluded that depression alone could not account for the observed intellectual deficit in the parkinson patients. To see if motor impairment played a role in the deficit, they allowed subjects to take as long as they wanted to complete the timed tests. Given unlimited time, the parkinson patients did not improve performance at all. To see if the deficit was mainly due to aging, the authors used age-corrected IQ scale scores, and still found the deficit. To see if anticholinergic medications caused the deficit, they compared parkinsons

on anticholinergics with parkinsons not on such medication. There was no difference, and they concluded that there was no empirical support for the role of anticholinergic medication in intellectual deficit in parkinsonism. There was no relationship found between age, age of onset, duration of illness, sex, and intellectual impairment. The authors concluded that the intellectual deficit found in parkinsonism could not be all due to the additive effects of aging, depression, motor impairment, anticholinergics, or thalamic surgery. They felt that probably generalized cortical changes could be implicated in such broad generalized impairment. The authors felt that their investigation supported the first person who differed with Parkinson, Ball, who in 1881 stated, "I would willingly say that a slight degree of intellectual impairment is almost the rule in this disease (Loranger, et al, 1972, p. 412)."

Loranger, Goodell, Lee, & McDowell (1972) reported another study in which they compared WAIS results on 40 parkinson patients before and 5 to 13 months after they were stabilized on L-dopa. The authors found significant improvement in IQ following L-dopa use. Means were:

Pre-L-dopa:	Post-stabilization:
VIQ=116.5	VIQ=123.5
PIQ= 95.4	PIQ=105.8
FSIQ=107.8	FSIQ=116.8

More recent studies by Riklan (1972) and Riklan,

Halgin, Maskin, & Weissman (1973) also show improvements in test scores following L-dopa ingestion, and raise the hypothesis of L-dopa-caused arousal. Riklan, et al, (1973) found that the parkinson patients showed improvements in the binocular critical fusion frequency threshold (CFF) also. The authors described the CFF as a highly sensitive index of cerebral function and efficiency, and concluded that the improved intellectual functions reflected an underlying increase in behavioral arousal. The authors further found that IQ was inversely related to age, directly related to education, and inversely related to degree of bradykinesia. Sex was not a critical factor, nor was length of illness nor dosage of L-dopa.

To summarize, it has been found that parkinson persons show generalized cerebral inefficiency which appears to correlate with extent of observed cerebral cortical impairment. While parkinson persons may appear to be functioning within normal intellectual limits, when they are compared with age- and education-matched non-parkinson peers, they show clear deficit. These persons appear to show some improvement in intellectual function following treatment with L-dopa. This improvement is felt to be due not to placebo effect, practice effect, improved motor ability, nor improved mood. While ventrolateral thalamic surgery usually results in reduced rigidity and tremor, it does not effect akinesia, nor

does intellectual functioning change as a long-term consequence. Substantia nigra degeneration continues uninterrupted whether surgery is done or L-dopa is ingested. (See Appendix A.)

Memory Functions in Parkinson's Disease

A rather detailed review of both the parkinson literature and the memory literature has failed to turn up a single study focused entirely on memory functions of parkinson persons. This is understandable in a sense, because the primary features of the disease occur in the motor sphere. But one of the most consistent complaints of the parkinson persons themselves is that they experience an impairment of recent memory functions. Rehabilitation efforts are focused primarily toward improving motor functions and providing emotional support for a person who has an incurable, progressively debilitating disease. Memory deficits ordinarily are not dealt with.

Most of the above-mentioned studies on intellectual functions also included some memory measures. While the same design problems were replicated in the memory evaluations, the most critical feature was that each study used its own, almost idiosyncratic measure for memory deficit. Furthermore, some of the measures used, such as the Wechsler Memory Scale (WMS) Mental Control and Digit-Span subtests, have been shown to be poor measures of memory (Davis & Swenson, 1970). Recent conceptualizations of short- and long-term memory, or conceptualizations of

input, storage, and retrieval have not yet been applied to the investigation of memory deficits in Parkinson's disease.

Talland (1962) evaluated memory functions by having his parkinson patients and non-parkinson controls learn digits or consonants. While he found no significant differences between parkinsons and non-parkinson controls on these tasks, he did note that the controls performed consistently better than the parkinson persons, with the parkinson persons showing some immediate memory loss.

Levita, et al, (1964) administered the Current Information, Orientation, Mental Control, and Digit-Span parts of the WMS to their parkinson patients. They reported correlations between Digit-Span and Mental Control, and between these tests and degree of rigidity. Such results seem reasonable when seen from the two-factor findings of Davis & Swenson (1970). Davis & Swenson found that Mental Control and Digit-Span represented a non-memory factor best described as freedom from distraction. Such results are in agreement with Cooper's hypothesis (1968) that as rigidity increases, the person's distraction also increases.

Cooper, et al, (1968), and Riklan, et al, (1969) concluded from memory tests given to parkinson patients that such persons show a slight decline in memory functions over time, and such decline occurred independently of thalamic surgery, which itself did not effect memory

functions. Asso (1969) found that her parkinson patients performed less well than her hypothetical normals on the WAIS Digit-Span subtest. Cotzias, et al, (1969) reported that parkinson patients on L-dopa showed an improved memory, but did not mention what measures were used. Meier & Martin (1970) compared parkinsons and non-parkinsons on the Arithmetic and Digit-Span subtests of the WAIS, and found that the parkinsons were slightly lower than normals, and that those parkinsons with the lowest scores improved the most with L-dopa treatment.

On the other hand, Reitan & Boll (1971) found that their parkinson patients scored significantly ($p < .005$) lower than their age- and education-matched non-parkinson peers. The normals recalled an average of 10.16 digits forward and backward, while the parkinsons recalled an average of 7.96. Such results led to the development of the cerebral impairment hypothesis.

O'Brien, DiGiacomo, Fahn, & Schwarz (1971) administered the WMS to 15 parkinson patients placed on high doses of L-dopa. No non-parkinson controls were used. The patients were tested before and during the administration of L-dopa, but no information was presented about when the second testing occurred. The pre-L-dopa average Memory Quotient (MQ) was found to be 122. The post-L-dopa average MQ was 124.4. While some patients' MQ's decreased with L-dopa, most improved. One male's MQ increased from a pre-L-dopa MQ of 59 to a post-L-dopa

MQ of 86.

Briefly, in other studies, Loranger, et al, (1972) found that parkinson patients increased their memory-attention IQ scores an average of 5.1 points with L-dopa treatment. Loranger, et al, (1972) found a consistent and significant deficit in parkinson patients on the Arithmetic and Digit-Span subtests of the WAIS when compared with non-parkinson controls. Again, the memory-attention deficit was not found to be due to depression, aging, or motor problems. Riklan, et al, (1973) found his parkinson patients performed poorer on the WMS Mental Control subtest than age- and education-matched non-parkinson controls. The parkinsons showed a significant improvement on this task after being stabilized on L-dopa. Riklan, et al, concluded that this type of impairment is probably a reflection of reduced arousal level in the parkinson person.

In summary, studies on memory functions in Parkinson's disease have been even less organized or unified than in the area of general intellectual functions. While the parkinson persons appear to show some kind of deficit on memory tasks, it is not clear whether the deficit is due to cerebral inefficiency, distractibility, reduced arousal, or other factors. Surgery does not appear to facilitate memory functions, while L-dopa does. L-dopa does not in any way stop or correct cerebral cortical degeneration.

Memory

No one to this point has conceptualized or investigated the parkinson memory deficit in terms of the different stages of the memory process, so the question of whether the parkinson deficit reflects mainly input, storage, or retrieval difficulties has not been asked. As with the case of intellectual functions as a whole, the three major hypotheses of parkinson memory deficit (Cooper: distraction; Riklan: arousal; Reitan: cerebral impairment) each seems able to account for some of the research findings. Clarification of the above-mentioned issues seemed desirable. The strategy was to design a study which would use adequate controls, use measures used by others, and which would manipulate some of the likely variables.

Theoretical Position. For this study, Kesner's (1973) definition of memory was used. Memory is defined as the process of encoding information by comparing and combining sensory inputs with innate and previously acquired knowledge, storing the information, and decoding the stored information into motor out-puts (Kesner, 1973). The model for memory presented by Kesner seemed to be the most coherent and useful one for this study because it appears to integrate memory conceptualizations so well with neurological conceptualizations. Kesner makes use of several features of memory models by Shiffrin & Atkinson (1969), Waugh & Norman (1965), and Patterson

(1966); and the influence of Hebb (1949) is apparent throughout most of the models of short-term and long-term memory.

Kesner's model assumes that registration, encoding, and storage of new information is processed by an initial pre-perceptual or cue-access store characterized by a match-mismatch process between sensory input and the long-term store. After input, the information is transferred in parallel to a short-term memory system and a long-term system. The short-term system includes a short-term store, and a decay process. The long-term system includes a long-term store and a consolidation process. Both the decay process of the short-term system and the consolidation process of the long-term system are affected by the initial input, the state of the organism's arousal, selective attention, and rehearsal. The short-term system has a limited capacity store, with decay occurring over time.

The amount of information processed in the short-term system is a function of the characteristics of the input, such as intensity, quality, quantity, and a number of sense modalities utilized. A neurophysiological mechanism which could serve the short-term memory (STM) system is the recovery cycle of evoked responses in the association cortex. The recovery time varies from 40-140 seconds, depending upon the nature of the stimulus. The amount of information that is retrievable from the

STM would be a function of the amount of information remaining in the decaying STM. Failure to retrieve information from STM can be attributed to the decay of electrical activity in those structures serving STM. Thus, retrieval from STM is seen as trace-dependent retrieval. Other events which would facilitate decay would include the presentation of interfering items of information (proactive and retroactive interference), and any event which would interfere with the electrical or biochemical activity of those structures serving STM.

While others raise the same or similar points (Barbizet, 1969; Luria, 1973; Guyton, 1972; Brierly, 1966; Stepien & Sierpinski, 1960; Brooks, 1972; Talland, 1968; Angelerques, 1969; John, 1967), Kesner (1973) most clearly proposed that the midbrain reticular formation and the association cortex make up the critical neurological substrata for the operation of STM and its decay process. Kesner stated that the reticular formation directly effects STM through its control of arousal and its control of selective attention. The cerebral cortex, on the other hand, constitutes the field upon which the STM traces are laid in coded form.

Long-term memory (LTM) is served primarily through regions of the hippocampus and the rest of the limbic system. It is possible to interfere with STM without interfering with LTM, and is also possible to affect both parallel systems at once. For example, persons with

hippocampal lesions have good STM, and can adequately recall words, digits, and sentences. But beyond 60" delay, their recall performance drops to chance, and they may not even recall that there was a task at all. Even with cues provided, such persons cannot recall an LTM task, and thus have been observed to read the same article over and over, each time thinking it is new. Kesner's explanation is that the information is getting in, is perceived, registered, and coded in STM, but decays out of STM and never gets consolidated in LTM.

Neurological Aspects. While most of the neurological and biochemical aspects of memory remain a mystery, there is increasing experimental and clinical evidence to implicate some brain structures more than others for certain aspects of memory. Stempien & Sierpinski (1960) reported a surgical case which clarifies the role of cortical traces and interference with decay. The patient, a 15-year old female, had seizures from the age of 6 months. She was of average intelligence, showed no impairment of LTM, attention, concentration, reasoning ability, or verbal recall. However, she had great difficulty with STM. EEG's revealed continuous abnormal discharges over the right posterior frontal and temporal areas. Surgical removal of the anterior temporal lobe and part of the posterior frontal cortex resulted in a loss of all abnormal EEG patterns, and a consequent disappearance of all disturbance of STM. The authors

felt that the abnormal electrical impulses interfered with the STM traces, in a manner akin to what Kesner (1973) proposed. Such results were similar to those of Penfield & Milner (1958), who found that similar subjects could keep in mind a short sentence or series of numbers if they were permitted to keep their attention directed to the task; but if someone spoke to them or they turned their attention to other matters, the to-be-remembered items were lost.

Luria (1973) emphasized that STM "...requires optimal cortical tone or a state of total vigilance, without which any selective mental process would be impossible (p. 287)." He went on to point out that the complex process of receipt and coding of incoming information requires the complete integrity of the cortex. Any form of disturbance or pathological state will result in a memory impairment the nature of which will depend upon the site of the disturbance. Cortical disturbance particularly seems to accent the role of irrelevant interfering events upon the input and STM process.

Brooks (1972) compared known cortical-damaged persons with age- and education-matched hospitalized non-cortical-damaged persons on the Logical Memory and Associate Learning subtests of the WMS. Subjects were retested 30 minutes after the first test to measure percent of forgetting. He found that those with head injuries performed significantly poorer than normals on

the first test, and forgot over six times more than the normals on the follow-up test. The author compared older (over 30) with younger (under 30) head-injured persons, and found that the older patients had greater trouble with Logical Memory (1 trial) than with Associate Learning (3 trials) compared with the younger persons. He concluded that cortical-injured were handicapped doubly, because they acquired much less information than the normals initially, and they forgot much more of what they did acquire than the normals did. Brooks felt that the increase in forgetting in his head-injured patients was probably due in part to a less efficient initial learning resulting in a memory trace that is less resistant to distortion and decay. He added that the obtained deficits could not be due to strictly input problems, and that further work was needed to reveal the relative importance of input deficits, STM and LTM deficits, and retrieval deficits.

Ojemann, Blick & Ward (1971) tested 25 parkinson patients who were about to undergo surgery. While the patients were on the operating table and awake, the authors inserted an electrode through a burr-hole to the ventrolateral thalamus, and provided below-patient-threshold electrical stimulation while administering a 60-trial STM test. The authors found that stimulation during presentation of items did not significantly differ from non-stimulation. Stimulation during recall

alone significantly increased recall errors of the omission type. Stimulation during any part of the test did not affect misnaming errors on the recognition phase. The authors hypothesized that stimulation during presentation improved recall because it directed the person's attention to the stimulus. Stimulation during recall increased errors because it interfered with the search of memory prior to retrieval and output. The interference of search accounts for the omission errors. Had more misnaming errors occurred, the interference with the decision system between search and output would more likely have been implicated. Thus, ventrolateral thalamic stimulation served both to interfere with the retrieval process, and to facilitate the input process by directing attention to the external environment. The authors concluded that left ventrolateral thalamic stimulation has an effect on what gets into and what comes out of STM at any given time.

While animal research has limitations in being extrapolated to account for human conditions, recent studies do appear to have some relevance to the neurological implications of memory deficit in parkinsonism. Phillips (1974) found that rats lesioned in the zona compacta of the substantia nigra could not learn an avoidance response. When given L-dopa, they were able to learn the response as well as sham-operated controls. On the other hand, rats overlearned to an avoidance response

then lesioned were still able to continue making such a response. The implications that substantia nigra lesions do not directly affect memory, but appear more to affect input seem clear. Here, the role of attention in input processes is implicated, and some indirect support for Riklan's hypothesis of arousal is provided.

Aging Aspects. As people age, they report a reduced ability to recall recent events, while some childhood events remain vivid. Most experimenters agree that STM is less efficient for older people than for younger people (Chown, 1972). Attempts to identify where the difficulty lies have so far seemed to consistently implicate a breakdown in storage (McNulty & Caird, 1967) or retrieval (Schonfield, 1967).

Wimer & Wigdor (1958) found that if they allowed older persons to learn a paired-associate list to criterion, they recalled as well as much younger persons. However, it took the older persons twice as many trials to reach criterion as compared with the younger persons. The authors concluded that in the aged, the memory was intact, but learning skills were impaired. Such a study is subject to confounding by original learning, as pointed out by Underwood (1966), and such results are difficult to interpret.

Schonfield (1967) found that older persons made more recall errors than recognition errors, and raised the issue of a possible retrieval problem. Laurence (1967)

provided older subjects with category cues of to-be-recalled words, and found a significant improvement under cued recall conditions. While young persons obtained means of 14.9 and 16.6 correctly recalled items under non-cued and cued conditions, older persons obtained recall means of 11.6 and 15.7 for non-cued and cued conditions. The cued recall conditions resulted in the loss of differences between age groups... This finding suggested that the memory loss experienced by older people was a retrieval loss.

Talland (1968) noted that other factors which might play a role in older persons' STM include reduced motivation, negative attitudes toward the test situation, reduced state of arousal, and susceptibility to external interference. Talland also pointed out that older persons make more errors of omission than errors of misnaming. Such a pattern is similar to the results of Ojemann, et al, (1971) who suggested a difficulty in the search process, which Talland also mentioned. Other factors which affect STM in older persons include such problems as perceptual acuity in the different perceptual modes. McGhie, et al, (1965) found that STM for visual information begins to deteriorate after the age of 60, while auditory information can be handled much better (if the auditory system is functioning). The authors also noted that older persons' STM performance declines when interference is introduced; for example, by slowing down the

rate of presentation of digits to recall.

Input-Retrieval Aspects. The above review of neurologically impaired and older persons' performance on memory tasks suggests that such persons do not take in as much information as normals, nor do they retrieve proportionately as much as normals. Events which affect or interfere with the hypothesized trace speed its decay and consequently reduce or eliminate the memory store. When cues are provided, retrieval is enhanced and memory functions improve. Such results are in agreement with Tulving and Thomson's (1973) conceptualization of the "encoding specificity hypothesis" which states that specific encoding operations performed on what is perceived determine what is stored; and what is stored determines what retrieval cues are effective in providing access to what is stored. The importance of such a principle lies in its further elaboration of the role of cues in differentiating what is "available" in the memory store and what is "accessible". The distinction between availability and accessibility was made by Tulving & Pearlstone (1966) when they provided cued or non-cued recall tasks for 948 high school students. The superiority of cued over non-cued recall suggested that specific information about words was available in storage in form sufficient for the reproduction with cues. But at least some of this information was not accessible under a non-cued condition. Such a finding implies support for

the breakdown of memory into input, storage, and retrieval stages, and has been replicated by many others.

Barker (1974) replicated parts of the Tulving & Pearlstone (1966) experiment, and included a carefully matched group of schizophrenic persons. One of the most interesting results was that while the schizophrenics showed a recall deficit compared to normals on the non-cued lists of words with 4 items per category (IPC=4), the schizophrenics did not differ significantly from the normals on the IPC=4, cued condition. This finding suggested that the schizophrenics were able to utilize the cues to increase their recall to a level comparable with that of the normals. Such a finding was also able to put to question the idea that input or attentional factors alone caused the schizophrenic STM deficit. Barker's study was therefore a critical evaluation of input or attentional problems versus retrieval problems, with a clear-cut indication of some retrieval problems. The finding that under IPC=1 schizophrenics improved under cued as compared with non-cued conditions, but not to the same extent as normals was accounted for in terms of the observation that the memory span appears to be limited to about 7 ± 2 bits of information (Miller, 1956). The IPC=1 condition contained 24 categories, while the IPC=4 condition contained only 6. Barker raised the question that possibly one of the reasons attentional difficulties played such a minor role in the study was

that all subjects were young (mean age = 25).

Problem

People with parkinsonism complain of impaired recent memory which appears to exceed that of their age-matched non-parkinson peers. Such a deficit is slightly improved with L-dopa, but not to the level of non-parkinson peers (Loranger, et al, 1972). Such memory deficit is not affected by surgery. The source of the deficit could be due to cerebral impairment (Reitan & Boll, 1971), to reduced input due to reduced arousal (Riklan, 1973), to attentional interference from motor movement defects (Cooper, et al, 1968), or to retrieval problems similar to those of schizophrenics (Barker, 1974). Such a deficit does not appear to be due to aging, sex, depression, or motor deficit per se (Loranger, et al, 1972).

The purpose of this study was to investigate how much of a memory deficit may exist, and to investigate where such a deficit may be occurring. Such information was seen to be of practical importance because it was hoped to provide some clear cues for rehabilitation efforts with such individuals, who number in the thousands. Such a study was also seen to be of theoretical importance because the results would provide further tests of the Tulving & Thomson (1973) conceptualization and the Kesner (1973) model with an entirely different clinical population. The information obtained from such a study would also contribute to the general

field of Parkinson's disease research by clarifying the nature of one of the clinical aspects of the syndrome about which parkinson patients often complain.

The study had two broad questions. The first was, "Does position in space affect memory functions?" Cooper's hypothesis would predict that position in space would affect memory functions. His prediction would be in the direction that if parkinson patients were tested horizontally, they would perform better than they would if tested vertically, because in the horizontal position they would be subject to less interference due to loss of motor control.

The second broad question was, "Where in the memory process is memory breaking down?" The hypothesis of Cooper, et al, (1968) would be that parkinson persons have trouble with memory because the information is not getting in. Thus, Cooper would predict that in a NC-C test situation, the parkinson individual might show the usual cue effect, but even under cued conditions he would continue to show a deficit. Luria (1973) and Reitan & Boll (1971) would predict that people with parkinsonism would show registration or storage deficits, because of impaired cerebral cortical function. On the other hand, if parkinson memory loss was a retrieval deficit, the prediction would be that parkinson subjects would recall as many words correctly as the normal subjects, when both are tested under cued-recall conditions.

Hypotheses:

The purpose of the present study was to test the known hypotheses in response to the two basic questions outlined above. The operational hypothesis were:

1. Parkinson subjects will show superior scores on the WMS MQ, and will recall more words correctly when reclining, as compared with their scores while sitting. (This is a direct test of Cooper's hypothesis quoted on p. 5.)
2. Arthritics will show superior scores on the WMS MQ, and will recall more words correctly when reclining, as compared with their scores while sitting.
3. Older and normal people will show no significant differences in WMS MQ scores or number of correctly recalled words between reclining and sitting positions.
4. There will be no significant differences between parkinson and normal subjects on number of correctly recalled words in cued recall conditions. (This hypothesis tests the notion that parkinson memory loss is a retrieval loss.)

CHAPTER II

METHOD

Design

For the part of the study which focused on the first question of effect of position in space upon memory function, a 4 x 2 repeated measures factorial design was used. The first factor was the diagnosis factor. The four levels were: (1) diagnosis of parkinsonism, (2) diagnosis of rheumatoid arthritis, (3) advanced age, and (4) normal. The second factor, a repeated measures factor, was position in space, either sitting or reclining. All subjects were tested in both positions, balanced for order of occurrence, and given alternate forms of the Wechsler Memory Scale (WMS), Forms I and II (Wechsler, 1945). In this part of the study, the gross dependent measure was the Memory Quotient (MQ). The individual subtest scores were also retained and used as dependent measures.

For the part of the study which focused on the second question of where the memory may be breaking down, a 4 x 2 x 2 repeated measures factorial design was used. Again, the first factor was the diagnostic factor with the four levels as described above. The second factor was the repeated measures factor of position in space, as described above. Thus, the impact of position in space upon memory function could be assessed by a second

measure in addition to the MQ mentioned above. The third factor was also a repeated measures factor. Here, parts of the Tulving & Pearlstone (1966) and Barker (1974) procedure were replicated in the sense that non-cued and cued recall tasks were given to all subjects in both positions in space. Immediately after the presentation of a list of words, two recall tests were given, either non-cued recall followed by cued recall (NCR-CR), or cued recall followed by cued recall (CR-CR). In this part of the study the dependent measure was #C, or number of correctly recalled words out of 24.

Subjects

A total of 112 people served as subjects for this study. There were 28 subjects in each of the four diagnostic groups. The group of individuals diagnosed as having parkinsonism was designated as the experimental group. Three control groups were used: arthritis, older, normal. Those with arthritis were assumed to be likely to have motor impairment due to peripheral pain and stiffness while at the same time having an intact central nervous system. Those older persons were assumed to be likely to have the beginnings of cerebral impairment normally found in aging people, while at the same time having no motor impairment. The normal subjects were assumed to be likely to have intact central nervous systems and no motor impairment. See Table 1 for a description of subjects.

TABLE 1, Part 1

Description of Subjects (Means and Standard Deviations) on Age, Education
and Screening Test Scores

Variable	Group							
	Parkinson		Arthritis		Older		Normal	
	\bar{X}	s	\bar{X}	s	\bar{X}	s	\bar{X}	s
Age	60.04	8.53	58.39	9.42	76.29	6.91	57.07	9.55
Education	9.21	3.26	10.11	2.51	8.93	2.52	9.79	3.66
Right Finger Tap	22.24	9.14	19.32	19.04	32.14	8.12	43.57	11.56
Left Finger Tap	22.67	7.70	18.21	17.46	30.46	10.67	42.79	10.67
Right Ruler Tap	51.61	23.68	45.0	35.49	64.89	23.56	86.21	25.11
Left Ruler Tap	49.64	17.47	42.86	34.04	63.18	24.03	81.21	24.07
Chair time ^a	13.74	5.51	10.22	2.46	10.43	3.24	9.14	2.63

^aChair time means the time in seconds it took the subject to rise from a chair, walk 310 cm, turn around, and return to the chair.

TABLE 1, Part 2

Description of Subjects (Mean and Standard Deviations) on Age, Education
and Screening Test Scores

Variable	Group							
	Parkinson		Arthritis		Older		Normal	
	\bar{X}	s	\bar{X}	s	\bar{X}	s	\bar{X}	s
WAIS VIQ	97.46	13.33	106.36	15.12	105.18	10.31	108.50	13.05
Information	9.86	2.03	10.96	2.22	9.68	2.11	11.29	3.15
Comprehension	8.53	3.33	11.14	3.46	8.57	2.39	11.50	3.27
Arithmetic	8.93	3.53	10.50	3.27	8.61	2.18	11.25	3.41
Similarities	7.64	2.92	8.14	3.58	7.21	2.78	9.21	3.74
Digits	8.36	3.41	9.39	2.67	8.14	2.49	8.86	2.80
Vocabulary	10.04	2.74	11.82	3.22	10.29	2.34	11.92	3.49
MMPI Depression Scale T-score	69.82	14.40	67.71	10.43	59.36	12.73	52.57	8.70

TABLE 1, Part 3

Description of Subjects (Means and Standard Deviations) on Age, Education
and Screening Test Scores

Variable	Group							
	Parkinson		Arthritis		Older		Normal	
	\bar{X}	s	\bar{X}	s	\bar{X}	s	\bar{X}	s
Category Practice Time	302.0	139.17	211.07	54.0	267.89	144.56	180.71	82.99
Category errors ^b	1.39	1.83	0.71	1.41	1.11	1.91	0.46	0.96
#correct/4 ^c	3.32	1.12	3.82	0.39	3.25	0.93	3.93	0.26

^bCategory errors means the number of errors in placement of words under correct category.

^c#correct means the number of correct words recalled out of 4 in one category.

Parkinson subjects. Eight males and four females were recruited from the Health Sciences Centre in Winnipeg, Manitoba. Six males and ten females were recruited from the Winnipeg Clinic in Winnipeg. The parkinson patients ranged in age from 40-72 years, with a mean of 60 years, and a standard deviation of 8.53 years. They ranged in education from 3-16 years, with a mean of 9.2 years, and a standard deviation of 3.26 years. Their WAIS Verbal IQ's (VIQ's) ranged from 78-136, with a mean VIQ of 97.46, and a standard deviation of 13.33. Other screening test results are shown in Table 1, and Appendix B.

Arthritis subjects. Fourteen males and fourteen females were recruited from the Rheumatic Disease Unit of the Rehabilitation Centre in Winnipeg. These patients all had rheumatoid arthritis of a fairly severely crippling nature. They ranged in age from 35-74 years, with a mean age of 58.39 years, and a standard deviation of 9.42 years. They ranged in years of education from 6-15 years, with a mean of 10.1 years, and a standard deviation of 2.51 years. Their VIQ's ranged from 81-134, with a mean VIQ of 106.36, and a standard deviation of 15.12. Other screening results are shown in Table 1 and Appendix B.

Older subjects. Four males and nine females were recruited from Lion's Manor, a retirement home in Winnipeg. Three males and five females were recruited from Donwood

Manor, a retirement home in Winnipeg. Three males were recruited from the Stradbrook Senior Citizen Centre, one male was recruited from the Smith Street Senior Citizen Centre, and four males were recruited from the Notre Dame Senior Citizen Centre, all in Winnipeg. These subjects ranged in age from 65-92, with a mean of 76.29 years, and a standard deviation of 6.91 years. Their years of education ranged from 5-16 years, with a mean of 8.93 years, and a standard deviation of 2.52 years. Their WAIS VIQ's ranged from 83-125, with a mean of 105.18, and a standard deviation of 10.31.

Normal subjects. Six males and twelve females were recruited from Our Saviour's Lutheran Church in Winnipeg. Two females were recruited from Donwood Manor. One male was recruited from Lion's Manor, and four males were recruited from the Smith Street Senior Citizen Centre, all in Winnipeg. These subjects were "normal" for the purposes of this study in the sense that they had full functioning ability in all areas tapped directly by the screening test tasks. These subjects ranged in age from 41-75 years, with a mean age of 57.07 years, and a standard deviation of 9.55 years. They ranged in years of education from 1-17 years, with a mean of 9.79 years, and a standard deviation of 3.66 years. Their WAIS VIQ's ranged from 88-137, with a mean of 108.50, and a standard deviation of 13.05.

Generally, the four groups of persons were selected

in such a way that they were matched for average education, average age (except for the older group), sex, and handedness (all subjects except two were right-handed). The parkinson and arthritis patients were approximately matched on degree of motor impairment, while the older and normal subjects were approximately matched on lack of motor impairment. The scarcity of parkinson and arthritis patients made it necessary to use groups based on average age rather than absolute age. Thus, there was some overlap across the age groups.

The original plan was to select only parkinson persons and arthritis persons stabilized on their medications. The purpose of such a plan was to compare the effects of the medications on the different groups. It became apparent during the screening procedure, however, that ten of the parkinson persons and six of the arthritis persons had discontinued their medications. A series of t-tests between the parkinson persons on medication (L-dopa) and the parkinson persons not on medication revealed no significant differences on any of the screening measures. A similar set of t-tests between the arthritis persons on medication (Entrophen) and the arthritis persons not on medication also revealed no significant differences. (See Appendix B for t-test results.) For this reason, as well as the great difficulty in getting additional parkinson and arthritis subjects, the groups were maintained intact. Table 2

summarizes the amount and type of medication used by parkinson and arthritis subjects.

Apparatus

All testing was done on an individual basis where the person lived. In some cases, persons were tested in their own homes; in some cases, persons were tested in small apartments; and in some cases, persons were tested in hospital rooms. While furniture varied from place to place, all screening testing was done with the subject in the sitting position in an armless chair next to a table. The same was true for the memory testing done in a sitting position. Memory testing in the reclining position was done with the subject in a full horizontal position upon a bed or a couch.

Screening test materials and equipment. Screening test equipment included the following:

1. A Counselor model bathroom scale, manufactured by the Brearley Company in Rockford, Illinois. The scale measured in pounds, which were then converted to kilograms.
2. The finger-tapping test used as part of the Halstead-Reitan Test Battery (Reitan, 1971). The finger-tapping test consists of a manually operated trigger connected to a small counter mounted on a 20 cm x 15 cm wooden board. The number of finger taps is read directly off the counter.

TABLE 2

Amount and Type of Medication by Diagnostic Group^a

Measure	Group (Medication)			
	Parkinson (L-Dopa)		Arthritis (Entrophen)	
	\bar{X}	s	\bar{X}	s
Weight (KG) ^b	69.14	12.97	67.10	11.52
Medication (MG)	1739.29	1501.69	2578.57	1787.48
Ratio (MG/KG)	25.32	21.86	32.93	24.82

^aOnly two older subjects took medication (aspirin), and none of the normal subjects took medication. See Appendix B for details.

^bThe mean weight for older subjects was 69.21, and for normal subjects was 71.71. Weight did not differ significantly across diagnostic groups. See Appendix B for details.

3. A standard 12-inch (31 cm) ruler was used as a stimulus. The subject's task was to tap alternately each end of the ruler as rapidly as possible.
4. A 10-foot (310 cm) marking string was used to mark a standard distance for each subject to walk after rising from a chair.

Screening test materials included a biographical information form, a permission form, the verbal subtests of the WAIS (Wechsler, 1955, 1958; Doppelt & Wallace, 1955), the Depression Scale of the Minnesota Multiphasic Personality Inventory (MMPI) (Hathaway & McKinley, 1940; Dahlstrom & Welsh, 1960), and the set of category practice cards used by Barker (1974). Copies of all screening test materials are presented in Appendix C.

Memory test materials. Copies of all memory test materials are presented in Appendix D. Materials used included:

1. Wechsler Memory Scale (WMS), Form I (Wechsler, 1945).
2. WMS, Form II. The visual reproduction subtest was omitted from both forms of the WMS to eliminate possible contamination due to lack of control of visual orientation responses or "attentiveness". MQ's were computed on pro-rated totals based on the remaining six subtests.
3. For the NCR - CR, CR - CR memory tasks, the three lists of 24 words with 6 categories and 4 items per

category used by Tulving & Pearlstone (1966) and Barker (1974) were used. Since Barker (1974) used only three such lists, the original source of the lists (Battig & Montague, 1969) was consulted to generate a fourth list of 24 words with 6 categories and 4 items per category. The rationale for selecting the lists of 24 words with 6 categories and 4 items per category was that these were the lists which most effectively picked up the retrieval deficit in schizophrenic persons in Barker's 1974 study. The four lists of words appears in Appendix D.

All arrangements for order of position in space, order of presentation of word lists, order of category words within lists, order of items within categories, and order of presentation of WMS, Form I and Form II were balanced to control for possible effects of sequence of material. In this case, "balance" means that each sequence occurred equally often within the limit of sequences given.

Correspondence materials. Copies of each type of correspondence with the subjects are presented in Appendix E.

Procedure

Recruiting procedure. Arthritis and parkinson subjects were recruited from lists of inpatients and

outpatients as described above. Older and normal subjects were recruited from the various sources described above. All persons were informed of what would be expected of them, and were informed that each would be paid \$5.00 cash for each testing session. It should be pointed out that the physicians provided an initial screening as they offered names of arthritis and parkinson patients who were likely to participate fully in the study. Thus, many parkinson and arthritis patients who were too ill or too incapacitated to participate were not bothered with recruitment letters. Parkinson and arthritis patients were informed in their first letter from their physician that participation was voluntary, that results would be confidential, and that they had the right to refuse to participate without this refusal having any effect upon their continued care by their physician.

Approximately three days after the parkinson and arthritis patients received the initial letter from their physician, the experimenter called, introduced himself, and asked if they wished to participate in the study. Those who wished to were given an appointment to be given the screening tests at their living quarters at their convenience. Those who did not wish to participate were thanked for their time, and were not contacted again. The refusal rate for this study was

6/136, or .04.

The older and normal subjects were recruited in a variety of ways, but generally, the experimenter gave a brief presentation at a retirement centre business meeting, answered questions about the project, and signed up volunteers afterwards. Many of the normal persons were recruited from a local church after the project was explained to the pastor and he took the entire series of screening and memory tests himself.

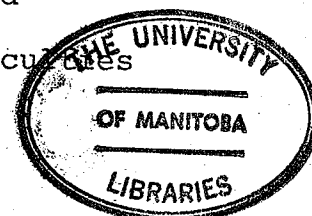
Screening procedure. The experimenter personally administered all screening tests. Prior to starting the screening tests, the experimenter described the general purpose of the project and answered any questions the persons might have had. See Appendix E for the introductory comments which were given in a paraphrased fashion to all persons serving as subjects. All subjects were informed that the project consisted of two testing sessions. The first session was to select four groups of people who were about alike in age, education, sex, and motor ability. Not all taking the screening tests would be given memory tests.

First, the subjects were interviewed to complete the biographical information form. They were then weighed on a scale, fully-dressed, but with shoes removed. Next, each person completed an approximation of the diary form on which medications and hours of sleep were reported. This information was used to facilitate the scheduling

of memory tests to coincide with when the individual felt physically and mentally most alert. It has been observed that people with arthritis are often sore and stiff in the mornings and feel better in the afternoons, while people with parkinsonism often feel best in the mornings after a good rest.

All persons who were able to use their hands were then given the Finger-tapping test from the Halstead-Reitan Test Battery (Reitan, 1971). Each person was tested three times with each hand. Each trial was for 10 seconds. The results were totaled, and a mean for each hand was computed. The same procedure was followed with the ruler-tap test, except each trial lasted 30 seconds. Next, those persons who were able to walk were asked to rise unaided from a chair, walk to the 310 cm. marker, turn around, and return to sit in their chair. Each person was timed by a stop watch from the time of rising to the time of sitting.

The above three tasks were administered to assess each person's degree of motor control and balance. Parkinson persons had great difficulty with all of the above tasks. The triad of symptoms, tremor, rigidity, and bradykinesia, were noticeable on all three tasks. Arthritis persons also had difficulty on these three motor tasks, but mainly due to peripheral pain and stiffness. Normal and older persons had no difficulties with any of the motor tasks.



Next, all persons were asked to read and sign a permission form. The permission form stated that they understood the project, knew they would be paid for their work, and gave permission for the experimenter to check their medical records for medications. They also gave permission for the experimenter and his assistant to come into their homes and administer the tests.

The Verbal subtests of the WAIS were then administered in the standardized manner. After the WAIS, the persons then completed the MMPI Depression scale. They were then given the practice list of 30 nouns and 12 categories, the same practice list as used by Barker (1974). Following the completion of the category practice test, each person was given a further rehearsal of the actual memory task by being presented with the category cue, "parts of a boat", then being given verbally the four items, "oar, cabin, sail and anchor." The person was then asked to recall as many of the four words as possible.

Screening tests were evaluated to eliminate those older and normal persons with motor and postural defects, and to eliminate any persons with WAIS VIQ of less than 80, or MMPI Depression scale t-score value of greater than 100.

Table 3 indicates the attrition rate, or loss of subjects throughout this study. A total of 18 persons were lost, or 14%. Such a low attrition rate facilitates

TABLE 3
Cause and Attrition Rate of Subjects

Cause of Attrition	Group			
	Parkinson	Arthritis	Older	Normal
WAIS VIQ <80	2	-	-	-
MMPI D >100	4	-	-	-
Hearing loss	-	1	-	-
Experimenter's Screening error	1	4	4	2
Total	7	5	4	2

the generalizability of the obtained results. Nevertheless, the attrition rate combined with the difficulty in obtaining parkinson and arthritis persons made it necessary not to extend the study to the point in time where all 28 parkinson and all 28 arthritis persons serving as subjects were actually and verifiably stabilized on their appropriate medications. Generally, nearly all the persons who were screened were kept in the study with the exception of those few noted in Table 3. No normals or older subjects had motor defects.

Once it was known that the person was to be included in the study, the name was randomly assigned to a memory test protocol which specified the exact arrangement of position in space, WMS Form I or II, order of lists, order of category words, and order of items. Randomization was accomplished by placing numbers 1 - 28 in a bowl and drawing one number to assign each subject to a protocol.

Memory testing procedure. Once a subject's protocol was assigned, the experimenter gave the subject's name, address, phone number, and protocol to the assistant. The assistant was not informed of the age, education, diagnosis, or medications of any subject. Furthermore, the assistant was not informed of the hypotheses of the study. The assistant then sent a letter to the person, and followed the letter up three days later with a telephone call during which he introduced himself and

made an appointment with the person. At the time of the appointment, the assistant explained the general sequence of events, and asked the subject to specify where the two parts of the testing (sitting and reclining) would be done. Since each administration of the memory tests varied according to the protocol assigned to it, a typical sequence of events is described below.

First, the assistant informed the persons which position would be used for the first half of the memory tests. The individual assumed the appropriate position, and the assistant read the instructions for the first list of words to be remembered. The subject was told that he would be presented with a list of 24 words with 6 categories and 4 items per category. He was told that each group of 4 words would be preceded by another word or phrase which described the words to be remembered, but which itself did not have to be remembered. The subject was encouraged to listen carefully and repeat each word (not the categories) after the assistant said it.

The assistant then read through the list of words at the rate of approximately one word each two seconds (approximately equal to Barker's slide presentation time of one cue and 4 words for each 10 seconds). Following the presentation of the list, the assistant then asked the person to recall as many words as he could in three minutes (NCR condition). Following the above event, the

assistant then read off one category cue each 30 seconds, and again asked the person to recall as many words as possible (CR condition). Again, the total recall time was three minutes. The assistant wrote down verbatim what the subject said, including comments and misnaming errors.

Next, the assistant administered the WMS, Form I. The procedure was standard, except that the Visual Reproduction subtest was omitted to eliminate any confounding due to poor visual acuity or lack of control of visual orienting motor behavior. After the WMS, the assistant administered List 2 using the CR - CR sequence.

After the completion of the second recall task, the assistant and subject took a brief break, then moved into the other testing position. In this position, the assistant administered List 3 in CR - CR sequence, WMS II, and List 4 in NCR - CR sequence. The total testing time for the memory tests was usually an hour.

At the completion of all testing, the assistant gave the subject a typed page which explained the project in more detail. The assistant then paid the person \$5.00 in cash, and had him sign a receipt. Within a day after receiving the completed memory test data, the experimenter mailed the subject a letter thanking him for his participation in the project.

CHAPTER III

RESULTS

Overview

The results of this study can be divided into those which were found from the analysis of the screening tests (preliminary data analyses), and those which were found from the analyses of the memory tests (primary data analyses). The most important finding of the preliminary analyses was that the parkinson patients as a group had a lower WAIS VIQ than the other three diagnostic groups, in spite of being matched for sex, average age, and average education.

The results of the primary data analyses indicated that position in space had no effect upon memory function, while diagnostic group had a significant effect upon memory function. Parkinson and older subjects performed less well on the memory tasks than did arthritic and normal subjects. Condition of recall had a significant effect which transcended all other factors: All subjects recalled more items correctly in the cued-recall condition than in the non-cued recall condition. Most of the above results were clear-cut in the sense that the main effects were significant, while there were very few interactions. In addition, intellectual and memory functions were not significantly related to age, medication (within diagnostic group), MMPI D-score, nor reported hours of

sleep.

Preliminary Data Analyses

The main purpose of the screening tests was to assure that subjects in each diagnostic group were matched on average age, sex, average years of education, and general motor ability. Except for the planned exception of the older group, the remaining three groups did not differ significantly on age or education. See Appendix B, Tables 9 and 10, for summaries of one-way analyses of variance and summaries of the appropriate post-hoc comparisons (Nie, Hull, Jenkins, & Bent, 1975).

While all parkinson patients were selected because they were reported to be stabilized on L-dopa, it was found that ten patients were not taking their medication. Table 11 in Appendix B indicates that for all purposes of this particular study, there were no significant differences between parkinson patients on medication and those patients not on medication.

While all subjects with arthritis were selected because hospital records indicated that they were stabilized on Entrophen, it was found that six patients were not taking their medications. Table 12 in Appendix B indicates that generally there were no significant differences between arthritic patients on medication as compared with such patients not on medication. It should be noted, however, that those arthritic patients on medication were found to have a

higher WAIS VIQ than those patients not on medication ($t = 2.58$, $df = 26$, $p < .05$).

The subjects with arthritis showed significant motor impairment when compared with the older and normal subjects on the motor tasks, and showed no significant differences between themselves and parkinson subjects on such tasks. Parkinson subjects also showed significant impairment on the motor tasks as compared with the older and normal subjects. Table 12 in Appendix B presents the important data analyses to support the above comments.

The parkinson patients, while matched for average age and education, obtained WAIS VIQ's considerably lower than the normal subjects ($F = 12.75$, $df = 3$, 108 , $p < .01$; $t = 3.13$, $df = 54$, $p < .008$). Tables 13 and 14 in Appendix B present further data analyses.

Parkinson and arthritic subjects obtained significantly higher MMPI Depression-scores than older and normal subjects (see Tables 15 and 16 in Appendix B). To see if the high MMPI D-score was a function of not taking the prescribed medication, parkinson patients not on L-dopa were compared with those on L-dopa in terms of MMPI D-score. No significant differences were noted (see Table 11, Appendix B). Similarly, persons with arthritis were compared across conditions of medication versus no medication on the MMPI D-scale, and again no significant differences were noted (See Table 12, Appendix B). It was also noted that there was no significant correlation

(across groups) between WAIS VIQ and MMPI D-score. A Pearson product-moment correlation of $-.15$ was obtained.

Other studies have related loss of sleep to impaired memory functions (Ekstrand, 1972; Kales, Ansel, Markham, Schart, & Tan, 1971). The reason for including the sleep diaries mentioned in Chapter II was to get at least a crude indication of whether or not hours of sleep had an effect on the type of measures taken in this study. Results obtained from the sleep diaries indicated that while both parkinson and arthritis subjects reported waking up more times in the night than older and normal subjects, all groups reported about the same number of hours of sleep (see Tables 17 and 18 in Appendix B).

In summary, it was clear that the four diagnostic groups were appropriately matched on sex, average age, and average education, and that they also differed on the desired measures. The finding that the arthritic subjects did not differ from the normal subjects on the WAIS VIQ in a sense already pointed toward the non-support of Cooper's hypothesis (motor impairment results in a measurable but specious intellectual deficit). Raw data from the screening tests are presented in Tables 19, 20, 21, and 22 in Appendix B.

Primary Data Analyses

Effect of position in space on WMS MQ. The first task of this study was to see if position in space was a significant factor in parkinson memory deficit as measured

by the Wechsler Memory Scale MQ. Obtained mean MQ's and standard deviations (s.d.'s) for each diagnostic group tested in each position are presented in Table 4, with the prior obtained WAIS VIQ's included as a reference point. The parkinson group, which had a mean WAIS VIQ of 97.46, obtained a mean MQ of 97.39 while sitting, and 98.82 while reclining. The arthritis group, which had a mean WAIS VIQ of 106.36, obtained a mean MQ of 108.71 while sitting, and 108.04 while reclining. The older group, which had a mean VIQ of 105.18, obtained a mean MQ of 102.61 while sitting, and 100.11 while reclining. The normal group, which had a mean VIQ of 108.50, obtained a mean MQ of 112.79 while sitting, and 113.21 while reclining. The above results are presented in graphic form in Figure 1. Means and s.d.'s of each of the WMS subtest scores are shown in Table 23 in Appendix F.

Utilizing the 4 x 2 factorial design described in Chapter II, a repeated measures analysis of variance (ANOVA) was done on the MQ measures, as well as on the subtest scores of the WMS. Table 24 in Appendix F presents the summary of the ANOVA on the MQ data, which indicates that the main effect of groups was significant ($F = 6.39$; $df = 3, 108$; $p < .001$). The main effect of position in space was non-significant ($F = 0.67$; $df = 3, 108$; $p > .6$). There was no significant group x position interaction.

Since position in space was found to be a non-

TABLE 4
Means and Standard Deviations of
Wechsler Memory Scale MQ, by Group and Position

Variable	Group							
	PD		A		O		N	
	\bar{X}	s	\bar{X}	s	\bar{X}	s	\bar{X}	s
WAIS VIQ, Sit	97.46	13.33	106.36	15.82	105.18	10.31	108.50	13.05
WMS MQ, Sit	97.39	14.64	108.71	11.44	102.61	12.27	112.79	17.60
WMS MQ, Recline	98.82	14.44	108.04	14.97	100.11	12.80	113.21	20.60

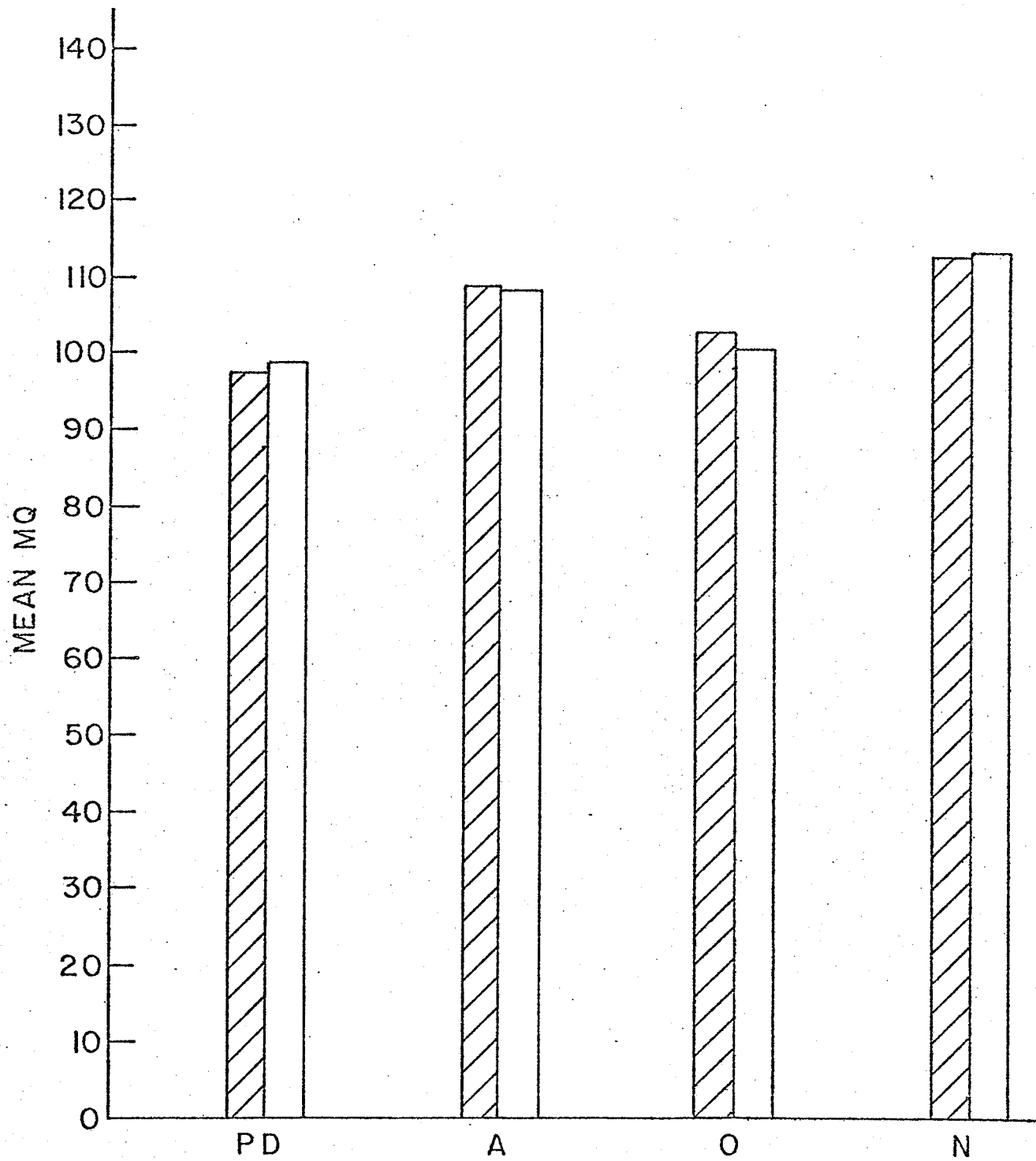


Figure. I. Mean MQ by Group and Position in Space.

Sit = ▨

Recline = □

significant factor in the MQ data, the data was submitted to a one-way analysis of variance with the data summed across the two positions in space. With this ANOVA, there was a significant groups effect ($F = 6.39$; $df = 3$, 108 ; $p < .01$), as shown in Table 25, Appendix F. Planned comparisons using the Dunn Multiple t (Kirk, 1968) revealed that the parkinson patients obtained significantly lower MQ's than arthritic and normal subjects, and did not differ significantly from older subjects. Older subjects, who obtained VIQ's approximately the same as normal subjects, obtained MQ's significantly lower than the normals. Table 5 summarizes this data analysis. Tables 26 and 27 in Appendix F present additional findings from one-way ANOVA's on the WMS subtests, and summaries of the appropriate post-hoc comparisons of the subtests between groups summed across positions in space.

While it is generally known that the WAIS VIQ and the WMS MQ are highly correlated, it seemed worthwhile to specify that relationship obtained for this particular study. Combining data from all four groups, a Pearson product-moment correlation of $+ .56$ ($p < .001$) was found between WAIS VIQ and WMS MQ. For the parkinson group, a correlation of $+ .41$ ($p < .01$) was found. For the arthritis group, it was $+ .51$ ($p < .01$), and for the older group, it was $+ .54$ ($p < .01$). For the normal group, the correlation between VIQ and MQ was $+ .61$ ($p < .001$).

TABLE 5

Planned Comparisons (Dunn Multiple t)
between Diagnostic Groups summed across
Positions in Space on MQ

Comparison	df	t-value
PD - A	108	-2.73*
PD - O	108	-0.86
PD - N	108	-3.96*
A - O	108	1.86
A - N	108	-1.23
O - N	108	-3.09*

* $p < .008$

The raw data for the WMS are presented in Appendix F, Tables 28, 29, 30, and 31.

Effect of position in space and cued versus non-cued recall on number of correctly recalled items. The second part of this study was to see if the memory deficit noted in parkinsonism (and demonstrated with the MQ data) could be determined to be either an input or an output (retrieval) deficit, and to see what effect position in space might have on this aspect of the memory deficit. The idea was to test Cooper's (1968) hypothesis (balance problems cause specious intellectual deficit) while at the same time replicating parts of the Barker (1974) study and the Tulving & Pearlstone (1966) study on a new clinical population. The dependent variable for the recall task in this part of the study was #C, or the number of items correctly recalled out of 24 possible items. Table 6 presents the obtained means and s.d.'s for #C by group, position in space, and recall condition.

Generally, what is noted in an inspection of means is that there is a strong non-cued versus cued effect across all diagnostic groups regardless of position in space. On the one hand, there is no particular gain in #C from trial 1 to trial 2 when both conditions are cued (C - C). On the other hand, there is a marked improvement in #C from trial 1 to trial 2 when conditions shift from non-cued to cued recall (NC - C). This observed lack of change from trial 1 to trial 2 in the cued condition

TABLE 6
Means and Standard Deviations of #C by Group, Position,
and Recall Condition

		Group							
		PD		A		O		N	
Variable		\bar{X}	s	\bar{X}	s	\bar{X}	s	\bar{X}	s
<u>Sit:</u>									
1	NC	7.04	3.82	9.14	3.82	6.93	4.31	10.00	5.11
	C	12.71	3.70	14.11	3.24	13.36	3.88	15.64	4.97
2	C	12.64	4.59	15.00	3.78	12.93	3.08	16.29	4.04
	C	12.32	4.21	14.46	3.94	12.61	3.40	15.96	4.38
<u>Recline:</u>									
1	NC	6.25	4.54	10.14	3.95	5.07	3.03	10.00	5.42
	C	13.21	3.94	15.04	3.49	11.79	3.40	14.57	5.24
2	C	13.29	3.15	15.54	3.72	13.82	3.77	16.36	3.69
	C	12.64	3.51	14.93	3.76	13.32	4.05	15.86	3.99

(C - C) was consistent across all groups and both positions in space. Because of this consistency, and because of similar findings reported by Barker (1974) and Tulving & Pearlstone (1966), no further data analyses were performed on the second recall trial. As Barker (1974) pointed out, the only really interpretable data are found in the first recall trial, under either cued or non-cued conditions.

The summary of the ANOVA on #C (Table 32, Appendix F) indicates that there were two significant main effects: groups ($F = 8.21$; $df = 3, 108$; $p < .001$), and recall condition ($F = 276.63$; $df = 3, 324$; $p < .001$). The main effect of position in space was non-significant ($F = 0.03$; $df = 1, 108$; $p > .6$). There were no significant two-way or three-way interactions.

Since position in space was found to be a non-significant factor in #C, and since there were no interactions, the data was re-analyzed with positions in space summed across groups. A one-way ANOVA with the non-cued recall data revealed that the group effects were significant ($F = 3.57$; $df = 3, 108$; $p < .05$). Post-hoc comparisons using the Dunn Multiple t revealed that there were no significant differences between groups in the non-cued recall condition. See Tables 33 and 34 in Appendix F. A similar one-way ANOVA with the cued recall data indicated significant group differences ($F = 5.52$; $df = 3, 108$; $p < .01$). Similar post-hoc comparisons

revealed that the parkinson subjects recalled significantly fewer correct items than the normals ($t = -3.49$, $df = 108$, $p < .008$), while the older subjects also recalled significantly fewer correct items than the normals ($t = -3.21$, $df = 108$, $p < .008$). See Tables 35 and 36 in Appendix F. Figure 2 and Table 7 show the means of #C by group and recall condition, summed across positions in space.

To summarize all the above findings, position in space had no effect on memory function as measured by MQ and #C. Recall condition had a significant effect upon the #C, with all subjects correctly recalling more items under cued recall than non-cued recall. There were no significant 2-way and 3-way interactions. Diagnostic group was a significant factor in that parkinson and older subjects recalled fewer correct items than arthritic and normal subjects.

Tables 37, 38, 39, and 40 in Appendix F present the raw data for all the above memory tests.

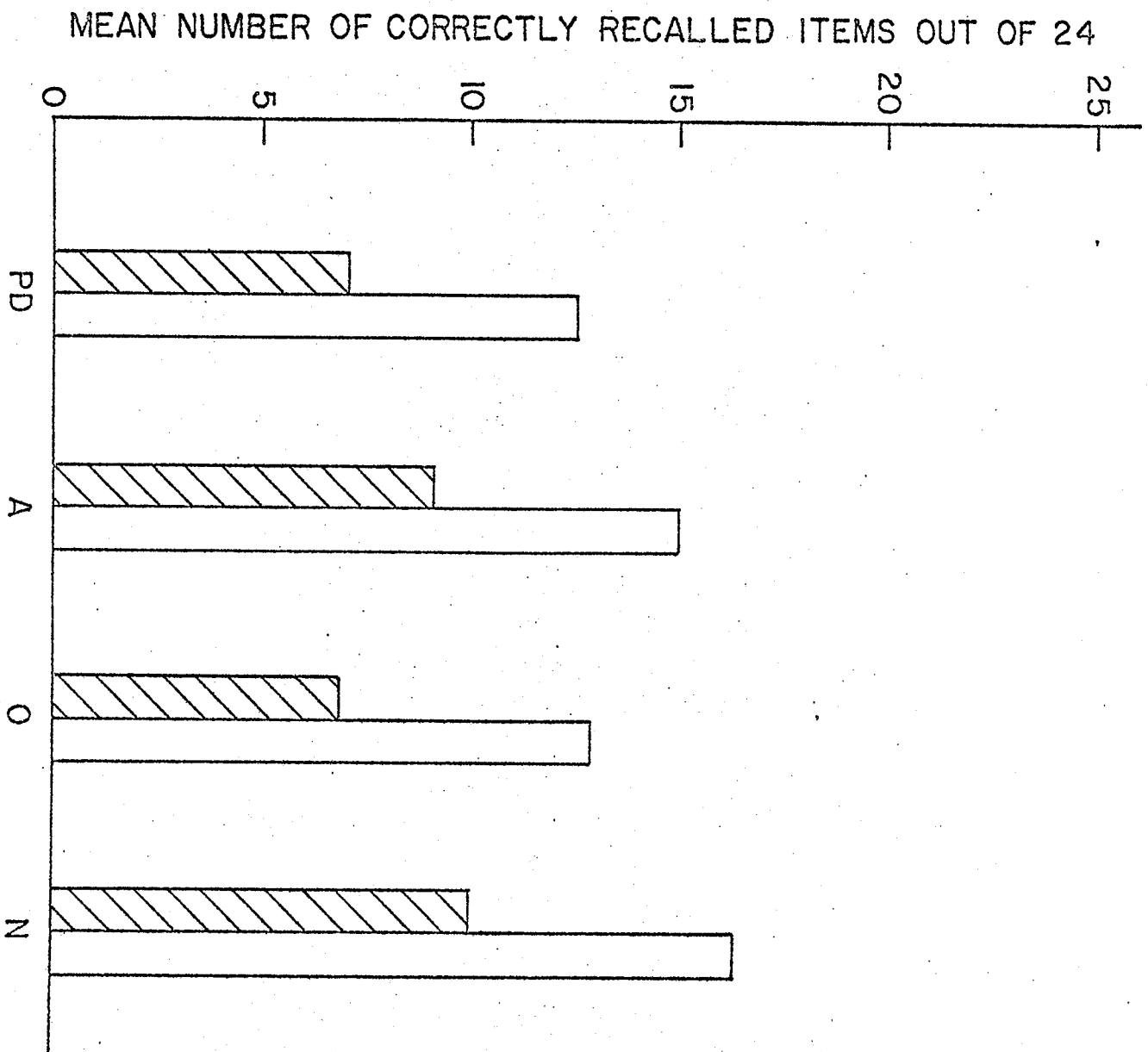


Figure 2.

Mean Number of Correctly Recalled Items
by Group and Recall Condition, Summed
Across Positions in Space.

Non-Cued Recall = ▨, Cued Recall = □.

TABLE 7

Means and Standard Deviations of #C by Group and Recall
Condition, Summed Across Positions in Space

Condition	Group							
	PD		A		O		N	
	X	s	X	s	X	s	X	s
NC	7.04	3.82	9.14	3.82	6.93	4.31	10.00	5.11
C	12.64	4.59	15.00	3.78	12.93	3.08	16.29	4.04

CHAPTER IV

DISCUSSION

Evaluation of Hypotheses

Both broad questions asked by this study were answered in a fairly detailed statistical manner. The first question, "Does position in space effect parkinson memory function?", received the fairly clear-cut answer of "No." The second broad question, "Where in the memory process is parkinson memory breaking down?", received the less clear-cut answer, "Probably after input, either in registration or storage, but at least not entirely in output."

Hypothesis 1, which predicted that parkinson subjects would obtain higher MQ's and #C scores while reclining than while sitting, was not supported. Position in space did not appear to effect memory functions as measured by the Wechsler Memory Scale MQ or #C.

Hypothesis 2, which predicted that arthritis subjects would obtain higher MQ's and #C scores while reclining than while sitting, was also not supported. This finding in this control group means that in spite of demonstrated motor impairment, position in space did not effect memory functions as measured by MQ or #C.

Hypothesis 3, which predicted that older and normal subjects would show no differences in MQ or #C across the two positions in space, was supported. There was none.

The evaluation of these three hypotheses, associated

with the use of these four diagnostic groups, failed to support Cooper's (1968) notion that parkinson intellectual deficit is simply an apparent loss due to interference from competing postural responses. A rather low-level inference made in this study was that when a person is resting horizontally on a safe, firm surface, he does not have to devote a significant amount of auditory attention to maintaining balance. On the other hand, testing in a normal sitting position demands at least some degree of motor control, which was indeed measurably impaired in many of the arthritis and parkinson patients. But regardless of the state of motor control, age, and intellectual function, position in space was demonstrated to be not significantly related to memory function.

The lack of interactions facilitated the interpretation of the results. Diagnostic group did effect measured performance on the memory tasks. Parkinson and older subjects demonstrated poorer memory functions than arthritis and normal subjects. These results suggest that memory deficit is more a function of generalized cerebral cortical impairment than a function of attention deficit due to motor impairment.

Hypothesis 4, which predicted that there would be no significant differences between parkinson and normal subjects on cued recall #C, was not supported. Parkinson and normal subjects differed significantly on #C under

cued recall conditions. Again, position in space was a non-significant factor. The implication here is that the parkinson memory deficit is not primarily a retrieval deficit. Since all subjects immediately repeated back the words, an input problem was not noted. Thus, most words were getting in, but some were not being registered or stored. Those words which were stored were retrieved adequately with cues. But providing cues did not eliminate the parkinson deficit.

While not related to the purpose of the present study, the finding that the control group of older subjects continued to show a recall deficit even under cued conditions is interesting because it contradicts the finding of Laurence in 1967. Laurence noted that her older subjects improved from 11.6 to 15.7 correctly recalled words under cued recall. The present study's older group improved from 6.93 to 12.93. Thus, both groups showed retrieval deficits, but the present study's older subjects did not reach the level of normals while Laurence's older group did. Laurence could conclude from her data that memory loss in older people is primarily a retrieval loss. Such a conclusion can not be substantiated by the results of the present study. While the older person benefits from cues, a deficit still remains which may be a result of cerebral cortical changes.

Relationship of This Study to Other Studies

The screening test results of this study do not support Parkinson's original impression that in Paralysis Agitans "the senses and intellect" are uneffected (Parkinson, 1817, p. 1). The screening test results do support Ball's claim in 1881 that "a slight degree of intellectual impairment is almost the rule in this disease (Loranger, et al, 1972, p. 412)".

Results from this study are also in agreement with Reitan & Boll (1971). Reitan & Boll found their parkinson subjects to be within the normal range of measured intelligence, but found them to be significantly inferior to age- and education-matched non-parkinson controls. This study found similar results, but the differences between parkinson and non-parkinson controls were smaller. Also noteworthy is that Reitan & Boll's subjects were an average of 50 years old, and had an average of 12 years of education, while in this study, the average age was 56, and the average education was 9 years. The difference in obtained VIQ's (Reitan & Boll: PD = 107.6, N = 119.6; this study: PD = 97.6, N = 108.6) may reflect cultural, age, education, and possibly procedural differences. The interpretation of the results of this study also parallels Reitan & Boll: the parkinson intellectual deficit reflects a generalized cerebral deficit, and does not reflect a deficit due to depression, aging, motor impairment, or other peripheral factors.

Combined with Reitan & Boll (1971), and Loranger, et al, (1972), this study correlates with the autopsy studies and histological findings reported by Alvord, et al, (1974). The generalized cerebral deterioration noted on autopsy of deceased parkinson persons has been strongly documented in a range of psychological test results accumulated in a fairly sophisticated fashion. As Alvord, et al, reported, degree of intellectual loss was not so much a function of degree of motor impairment, but was more a function of degree of generalized cerebral change, which was greater than anticipated for the age of the parkinson person.

The results of this study also support Loranger, et al, (1972) in the sense that motor loss did not account for the memory deficit. Arthritic patients had similar motor loss, but in spite of being matched on age, sex, and education, had significantly superior VIQ's, MQ's, and other memory scores when compared with parkinson patients. As Loranger, et al, also reported, depression was also not the causal factor in parkinson intellectual and memory loss. Arthritic patients were equally as depressed as the parkinson patients, and still the parkinson patients were significantly lower than the arthritis patients on VIQ, MQ, and the other measures. Intellectual loss could also not be attributed to aging, as Loranger, et al, had speculated. The older subjects had, in fact, higher WAIS VIQ's than the

parkinson subjects, even though the parkinson subjects had more education than the older subjects.

It is more difficult to relate the results of this study to the general findings and positions of Cooper, et al, (1968), and Riklan (1973). Riklan's notion that parkinson intellectual loss is due to a decrease in arousal does not appear to be supported by the results of this study. This lack of support occurs mainly in the comparison of those parkinson patients on L-dopa with those parkinson patients not on L-dopa. While there appeared to be no significant differences between these two groups of parkinson persons, the interpretation of such results is open to many pitfalls. Perhaps those patients who refused to take the L-dopa were also the ones who were less deteriorated and therefore less willing to suffer the nausea which was generally the reason for discontinuing the L-dopa. Perhaps those who took the L-dopa were more deteriorated, and correspondingly improved a great deal. But when these two groups are compared, no difference would be noted.

This study was at least in part stimulated by the finding of Barker (1974) that memory deficits noted in schizophrenic subjects could be demonstrated to be at least partly related to output or retrieval difficulties. It was hoped that if parkinson subjects demonstrated a similar retrieval loss, some fairly clear-cut suggestions

for rehabilitation efforts could be derived. It was specifically hoped that the difference between parkinson and non-parkinson subjects found in the non-cued recall condition would disappear in the cued recall condition. Unfortunately, that hope was not realized, and the differences between parkinson and non-parkinson subjects were found to be significant in both non-cued and cued recall conditions.

Table 8 summarizes some of the key measures found in Barker's (1974) study, Tulving & Pearlstone's (1966) study, and this present one. The most unexpected difference found was that between Barker's group of normals and the present study's group of normals on non-cued recall. While Barker's normals recalled about 15/24 correctly in the non-cued recall condition, the normals of this study correctly recalled only 10/24 in the non-cued recall condition. Both groups correctly recalled about 16/24 in the cued recall condition. Such a difference in the non-cued recall condition suggests that the normals of the present study may have more retrieval difficulties than Barker's normals who were on the average about 32 years younger. But these differences disappear under cued recall conditions. Similar results were noted with the arthritic subjects. But both older and parkinson subjects show a continued deficit when compared with Barker's normals under the cued recall condition.

TABLE 8

Means of Certain Measures in Barker's (1974) Study,
Tulving & Pearlstone's (1966) Study, and This Study

Group	Dependent Measures			
	Age	Education	NC, #C	C, #C
<u>Barker:</u>				
Schizophrenic	25	11	9 ^a	15
Normal	25	11	15	16
<u>Tulving, et al:</u>				
Normal	17	11	14	16
<u>This Study:</u>				
Parkinson	60	9	7	12
Arthritis	58	10	9	15
Older	76	9	7	13
Normal	57	10	10	16

^aAll numbers in the last two columns refer to the number of correctly recalled items only from lists of 24 items with 4 items in each of 6 categories.

Theoretical Implications

The Encoding specificity hypothesis. While the Tulving & Pearlstone (1966) and Tulving & Thomson (1973) formulation of the memory process contributes a great deal to the clarification of procedures to isolate likely factors in memory deficit, much further clarification is still left to be desired. Their procedure of using non-cued and cued recall was replicated in this study, and results similar to theirs were obtained. On the other hand, the parkinson and older subjects' display of a deficit under the cued condition becomes difficult to interpret, and in fact, perhaps the Tulving procedure was not designed to elucidate features of registration or storage loss.

The Kesner Model. The Kesner (1973) model of the memory process was selected from a wide range of models because it seemed to have the greatest explanatory power for this particular study. Kesner's notion that retrieval could be interfered with by competing stimulation, such as Cooper, et al, (1968) hypothesized; by control of selective attention due to state of arousal, such as Riklan (1973) hypothesized; or by cortical tone, such as Luria (1973) hypothesized; seemed to offer the greatest integration of the various conceptualizations of what was happening during the memory process.

The results of this study offer support primarily to the "cortical tone" aspect of the Kesner model.

According to Kesner, while localization of memory function is not absolutely known, it appears that input into long-term memory is primarily through regions of the hippocampus and the rest of the limbic system. Input into short-term memory is primarily through the reticular formation into the cerebral cortex. In this study, Wechsler Memory Scale subtest scores showed that there were no significant differences across groups on the subtests of Information and Orientation. These subtests primarily tap long-term memory. On the other hand, those subtests which tapped cortical functions of memory, such as Logical Memory and Associate Learning, showed significant impairment in parkinson and older groups as compared with arthritis and normal groups (see Table 27, Appendix F).

The general implication of the results of this study as expressed through the Kesner model is that the memory deficit noted in parkinsonism is of a similar nature to the memory deficit noted in older persons. Such a memory deficit is subject to the state of health of the individual's cerebral cortex. Thus, when parkinson and older persons complain of experienced memory deficit, they are simply describing a symptom of cerebral cortical impairment. The symptom demonstrated by the present study is that information gets into the central nervous system, but is not sufficiently registered or stored. Hippocampal and limbic system integrity

appears to be little compromised by age or by parkinsonism.

Clinical Implications

While it is true that parkinsonism is primarily a disease of the extrapyramidal system, the individual who suffers the disease also experiences a decline in intellectual power. This decline is not just apparent; it is measurable, and is quite real. Reitan & Boll's (1971) conclusions that parkinson patients' emotional upsets are secondary to their experienced cognitive impairment need to be given serious consideration from both a clinical and rehabilitative standpoint. Rather than simply give superficial encouragement to support the parkinson patient in the light of his motor losses, the treatment team should routinely evaluate for intellectual function, and should listen carefully and with trust to a parkinson patient's concern about loss of memory and other subtle cortical functions. When a parkinson patient reports that he cannot think as clearly as before, it does not mean that he is distracted by motor impairment; it more likely means that he really cannot think as clearly as before. When the above complaint is noted, then it becomes the responsibility of the treatment team to accurately assess the level of intellectual function, and to institute whatever rehabilitation or coping enhancement procedures are available for this very real loss of function.

Since the findings of this study indicate that the

parkinson memory deficit is mainly a registration or storage loss probably secondary to cortical deterioration, efforts to support or enhance remaining memory functions should focus upon facilitating the input and retrieval processes. Efforts should be made to maximize the clarity of the information to be processed. Presenting information in small chunks of considerable intensity may help. The use of cues has also been demonstrated to be helpful.

Directions for Further Research

To clarify at least one procedural ambiguity in this area of research, a study should be done in which the category cue is repeated by the subject. Demonstrating the encoding of the cue would thus set the stage for clarification of the issue of input versus output loss. The above should be done in an appropriate factorial design.

Another study needed is one in which the non-cued recall condition is given in Trial 2. The difficulty in interpreting the results of the second cued recall trial exists primarily because the recall conditions have never been properly factored out. For example, four groups of matched subjects could be given the four sets of words in balanced order of the following arrangements of first and second recall: (1) NC - NC, (2) NC - C, (3) C-NC, (4) C - C. So far, only the arrangements of (2) and (4) have been used, so the interpretation of the results

remains in doubt.

The clinical area of depression in relation to incurable disease, particularly as it relates to experienced loss of intellectual function, could be explored in further detail. The realization of gradual loss of intellectual function could in fact have a greater effect on an individual than the awareness of gradual loss of motor function. Such issues could be elucidated through further team research projects similar to those done by Cooper's group in New York.

REFERENCES

- Alvord, E. C., Forno, L. S., Kusske, J. A., Kauffman, R. J., Rhodes, J. S., & Goetowski, C. R. The pathology of parkinsonism: A comparison of degenerations in cerebral cortex and brainstem. In F. H. McDowell & A. Barbeau (Eds.), Advances in neurology. Vol. 5. New York: Raven, 1974.
- Angel, R. W., Alston, W., & Higgins, J. R. Control of movement in Parkinson's disease. Brain, 1970, 93, 1-14.
- Angelerques, R. Memory disorders in neurological disease. In P. J. Vinken & G. W. Bruyn (Eds.), Handbook of clinical neurology, Vol. 3: Disorders of higher nervous activity. New York: American Elsevier, 1969.
- Aring, C. D. The riddle of the Parkinson syndrome. Archives of Neurology, 1962, 6, 1-5.
- Asso, D. WAIS scores in a group of parkinson patients. British Journal of Psychiatry, 1969, 115, 555-556.
- Asso, D., Crown, S., Russell, J. A., & Logue, V. Psychological effects of the stereotactic treatment of parkinsonism. British Journal of Psychiatry, 1969, 115, 541-553.
- Barbeau, A. The clinical physiology of side effects in long-term L-dopa therapy. In F. H. McDowell & A. Barbeau (Eds.), Advances in neurology. Vol. 5. New York: Raven, 1974.
- Barbizet, J. Psychophysiological mechanisms of memory. In P. J. Vinken & G. W. Bruyn (Eds.), Handbook of clinical neurology, Vol. 3: Disorders of higher nervous activity. New York: American Elsevier, 1969.
- Barker, W. J. Interference and retrieval deficit in schizophrenic memory dysfunction. Doctoral dissertation. University of Manitoba, 1974.
- Bartholini, G., Stadler, H., & Lloyd, K. G. Cholinergic-dopaminergic interaction in the extrapyramidal system. In D. B. Calne (Ed.), Advances in neurology. Vol. 3. New York: Raven, 1973.

- Bartholini, G., Lloyd, K. G., & Stadler, H. Dopaminergic regulation of cholinergic neurons in the striatum: Relation to parkinsonism. In F. H. McDowell & A. Barbeau (Eds.), Advances in Neurology, Vol. 5. New York: Raven, 1974.
- Battig, G., & Montague, W. E. Category norms for verbal items in 56 categories. Journal of Experimental Psychology, 1969, 80, (3, pt.2).
- Brierly, J. B. Some aspects of the disorders of memory due to brain damage. In D. Richter (Ed.), Aspects of learning and memory. London: Heinemann, 1966.
- Brooks, D. N. Memory and head injury. The Journal of Nervous and Mental Disease, 1972, 155, 350-355.
- Bucy, P. C., & Case, T. J. Tremor, physiological mechanism and abolition by surgical means. Archives of Neurology and Psychiatry, 1939, 41, 721-746.
- Bunney, W. E. Present status of psychological reactions to L-dopa. American Journal of Psychiatry, 1970, 127, 361-362.
- Chown, S. M. Human aging. Baltimore: Penguin, 1972.
- Chusid, J. G. Correlative neuroanatomy and functional neurology. 15th Ed. Los Altos, California: Lange, 1973.
- Cooper, I. S., Riklan, M., Stellar, S., Waltz, J.M., Levita, E., Ribera, V. A., & Zimmerman, J. A multidisciplinary investigation of neurological rehabilitation in bilateral parkinsonism. Journal of the American Geriatric Society, 1968, 16, 1177-1306.
- Cooper, I. S. Involuntary movement disorders. New York: Harper & Row, 1969.
- Cotzias, G. C., Van Woert, M. H., & Shiffer, L.M. Aromatic amino acids and modification of parkinsonism. New England Journal of Medicine, 1967, 276, 374.
- Cotzias, G. C., Papavasiliou, P. S., & Gellene, R. Modification of parkinsonism: Chronic treatment with L-dopa. New England Journal of Medicine, 1969, 280, 337-345.

- Cotzias, G. C., & McDowell, F. H. (Eds.), Parkinson's disease: Recent advances in therapy. New York: MEDCOM, 1971.
- Dahlstrom, W. G., & Welsch, G. S. An MMPI handbook: A guide to use in clinical practice and research. Minneapolis: University of Minnesota Press, 1960.
- Davis, L. J., & Swenson, W. M. Factor analysis of the Wechsler Memory Scale. Journal of Consulting and Clinical Psychology, 1970, 35, 430.
- Denny-Brown, D. Diseases of the basal ganglia: Their relations to disorders of movement. Lancet, 1960, 2, 1099.
- Diller, L., & Riklan, M. Psychosocial factors in Parkinson's disease. Journal of the American Geriatric Society, 1956, 4, 1291-1300.
- Doppelt, J. E., & Wallace, W. L. Standardization of the Wechsler Adult Intelligence Scale for older Persons. Journal of Abnormal and Social Psychology, 1955, 51, 312-330.
- Doshay, L. J. Parkinson's disease: Its meaning and management. New York: Lippincott, 1960.
- Duvoisin, R. C., Yahr, M. D., Schweitzer, M. D., & Merritt, K. I. Parkinsonism before and since the epidemic of encephalitis lethargica. Archives of Neurology, 1963, 9, 38-42.
- Duvoisin, R. C. A review of drug therapy in parkinsonism. Bulletin of the New York Academy of Medicine, 1965, 41, 898-910.
- Earle, K. M. Studies on parkinson's disease including x-ray fluorescent spectroscopy of formalin fixed brain tissue. Journal of Neuropathology and Experimental Neurology, 1968, 27, 1.
- Ekstrand, B. R. To sleep, perchance to dream. In C. P. Duncan, L. Sechrest, & A. W. Melton (Eds.), Human memory: Festschrift in honor of Benton J. Underwood. New York: Appleton-Century-Crofts, 1972. Pp. 59-82.
- Ericsson, A. D., & McCann, D. S. Dihydroxyphenylacetic acid in the treatment of parkinsonism. In F. H. McDowell & A. Barbeau (Eds.), Advances in neurology. Vol. 5. New York: Raven, 1974.

Flynn, W. R. On the psychology of the shaking palsy. Psychiatric Quarterly, 1962, 26, 203-222.

Forno, L. S., & Alvord, E. C. Depigmentation in the nerve cells of the substantia nigra and locus ceruleus in parkinsonism. In F. H. McDowell & A. Barbeau (Eds.), Advances in neurology. Vol. 5. New York: Raven, 1974.

Freeman, W. Pathology of paralysis agitans. Annals of Clinical Medicine, 1925, 4, 106-116.

Gatz, A. J. Manter's essentials of clinical neuroanatomy and neurophysiology. 4th Ed. Philadelphia: Davis, 1972.

Greenfield, J. G., & Bosanquet, F. D. The brain-stem lesions in parkinsonism. Journal of Neurology, Neurosurgery, and Psychiatry, 1953, 16, 213.

Greenfield, J. G. The pathology of Parkinson's disease. In M. Critchley (Ed.), James Parkinsonism (1755-1824). London: MacMillan, 1955.

Guyton, A. C. Organ physiology: Structure and function of the nervous system. Philadelphia: W. B. Saunders, 1972.

Hathaway, S. R., & McKinley, J. C. The measurement of symptomatic depression with the Minnesota Multiphasic Personality Schedule. Psychological Bulletin, 1940, 37, 425.

Hebb, D. O. The organization of behavior. New York: Wiley, 1949.

Hoehn, M. H., & Yahr, M. D. Parkinsonism: Onset, progression, and mortality. Neurology, 1967, 17, 427-442.

Hyde, J. B. Some short notes on the nervous system. Winnipeg: University of Manitoba, 1971.

John, R. Mechanisms of memory. New York: Academic Press, 1967.

Kales, A., Ansel, R. D., Markham, C. H., Scharf, M. B., & Tan, T. L. Sleep in patients with Parkinson's disease and normal subjects prior to and following levodopa administration. Clinical Pharmacology and Therapeutics, 1971, 12, 397-406.

- Kesner, R. A neural system analysis of memory storage and retrieval. Psychological Bulletin, 1973, 80, 177-203.
- Kirk, R. Experimental design: Procedures for the behavioral sciences. Belmont, California: Brooks/Cole, 1968.
- Laurence, M. W. Memory loss with age: A test of two strategies for its retardation. Psychonomic Science, 1967, 9, 209-210.
- Levita, E., Riklan, M., & Cooper, I. S. Verbal and perceptual functions after surgery of subcortical structures. Perceptual and Motor Skills, 1964, 18, 195-202.
- Levita, E., Riklan M., & Cooper, I. S. Cognitive and perceptual performance in parkinsonism as a function of age and neurological impairment. The Journal of Nervous and Mental Disease, 1964, 139, 516-520.
- Loranger, A. W., Goodell, H., Lee, J. E., & McDowell, F. Levodopa treatment of Parkinson's syndrome. Archives of General Psychiatry, 1972, 26, 163-168.
- Loranger, A. W., Goodell, H., McDowell, F. H., Lee, J. E., & Sweet, R. D. Intellectual impairment in Parkinson's syndrome. Brain, 1972, 95, 405-412.
- Luria, A. R. The working brain: An introduction to neuropsychology. London: Penguin, 1973.
- Martinez, A. J., & Utterback, R. A. Unilateral Parkinson's disease, clinical and neuropathologic findings. Neurology, 1973, 23, 164-170.
- McGhie, A., Chapman, J., & Lawson, J. S. Changes in immediate memory with age. British Journal of Psychology, 1965, 56, 69-75.
- McNulty, J. A., & Caird, W. K. Memory loss with age: An unsolved problem. Psychological Reports, 1967, 20, 283-288.
- Meier, M. J., & Martin, W. E. Intellectual changes associated with levodopa therapy. Journal of the American Medical Association, 1970, 213, 465-466.
- Miller, G. H. The magical number seven, plus or minus two: some limits on our capacity for processing information. Psychological Review, 1956, 63, 81-97.

- Miller, H. Parkinson's disease. British Medical Journal, 1954, 2, 158.
- Nie, N. H., Hull, C. H., Jenkins, J. G., & Bent, D. H. Statistical package for the social sciences (Second Ed.). New York: McGraw-Hill, 1975.
- O'Brien, C. P., DiGiacomo, J. N., Fahn, S., & Schwarz, G. A. Mental effects of high-range levodopa. Archives of General Psychiatry, 1971, 24, 61-64.
- Ojemann, G. A., Blick, K. I., & Ward, A. D. Improvement and disturbance of short-term verbal memory with human ventrolateral thalamic stimulation. Brain, 1971, 94, 225-240.
- Onuagulchi, G. Drug treatment of parkinsonism and its assessment. In P. J. Vinken & G. W. Bruyn (Eds.), Handbook of clinical neurology, Vol. 6: Diseases of the basal ganglia. New York: Wiley, 1968.
- Parkinson, J. An essay on the shaking palsy. London: Whittingham & Rowland, 1817.
- Penfield, W., & Milner, B. The role of the hippocampal complex in memory. Archives of Neurology and Psychiatry, 1958, 79, 475.
- Peterson, L. R. Short-term verbal memory and learning. Psychological Review, 1966, 73, 193-207.
- Phillips, A. G. Reversal by L-dopa of impaired learning due to destruction of the dopaminergic nigro-striatal projection. Science, 1974, 185, 960-962.
- Pletscher, A. Effect of inhibitors of extracerebral decarboxylase on levodopa metabolism. In D. B. Calne (Ed.), Advances in neurology, Vol. 3. New York: Raven, 1973.
- Poskanzer, D. C., & Schwab, R. S. Studies in the epidemiology of Parkinson's disease predicting its disappearance as a major clinical entity by 1980. Transactions of the American Neurological Association, 1961, 86, 234-240.
- Ranson, S. W., & Clark, S. L. The anatomy of the nervous system: Its development and function. Philadelphia: W. B. Saunders, 1964.
- Reitan, R. Manual for administration of neuropsychological test batteries for adults and children. Indianapolis: Indiana University Medical Center, Neuropsychological Laboratory, 1971.

- Reitan, R. M., & Boll, T. J. Intellectual and cognitive functions in Parkinson's disease. Journal of Consulting and Clinical Psychology, 1971, 37, 364-369.
- Richardson, E. P. Remarks on the pathology of Parkinson's disease. In A. Barbeau, L. J. Doshay, & E. A. Spiegel (Eds.), Parkinson's disease: Trends in research and treatment. New York: Grune & Stratton, 1965.
- Riklan, M., Weiner, H., & Diller, L. Somato-psychologic studies in Parkinson's disease. The Journal of Nervous and Mental Disease, 1959, 129, 263-272.
- Riklan, M., Diller, L., Weiner, H., & Cooper, I. S. Psychological studies on the effects of chemosurgery of the basal ganglia in parkinsonism. I: Intellectual functioning. Archives of General Psychiatry, 1960, 2, 22-31.
- Riklan, M., Levita, E., & Cooper, I. S. Psychological effects of bilateral subcortical surgery for parkinson's disease. The Journal of Nervous and Mental Disease, 1966, 141, 403-409.
- Riklan, M. Levodopa and behavior. Neurology, 1972, 22, 43-55.
- Riklan, M., Halgin, R., Maskin, M., & Weissman, D. Psychological studies of longer range L-dopa therapy in parkinsonism. The Journal of Nervous and Mental Disease, 1973, 157, 452-464.
- Roche Labs. Larodopa: A significant advance in the management of Parkinson's disease. Montreal: Roche, 1973a.
- Roche Labs. Parkinson's disease and the parkinson syndrome. Montreal: Roche, 1973b.
- Schonfield, D. Memory loss with age: Acquisition and retrieval. Psychological Reports, 1967, 20, 223-226.
- Schwab, R. S. Progression and prognosis in Parkinson's disease. The Journal of Nervous and Mental Disease, 1960, 130, 556-561.
- Schwarz, G. A., & Fahn, S. Newer medical treatments in parkinsonism. Medical Clinics of North America, 1970, 54, 773-785.

- Selby, G. Parkinson's disease. In P. J. Vinken & G. W. Bruyn (Eds.), Handbook of clinical neurology, Vol. 6: Diseases of the basal ganglia. New York: Wiley, 1968.
- Shiffrin, R. M., & Atkinson, R. C. Storage and retrieval processes in long-term memory. Psychological Review, 1969, 76, 179-193.
- Singer, C. A short history of anatomy from the Greeks to Harvey. New York: Dover, 1957.
- Stepien, L., & Sierpinski, S. The effect of focal lesions of the brain upon auditory and visual recent memory in man. Journal of Neurology, Neurosurgery, and Psychiatry, 1960, 23, 334-340.
- Talland, G. A. Cognitive functions in Parkinson's disease. The Journal of Nervous and Mental Disease, 1962, 135, 196-205.
- Talland, G. A. Disorders of memory and learning. Baltimore: Penguin, 1968.
- Tellegen, A., & Briggs, P. F. Old wine in new skins: Grouping Wechsler subtests into new scales. Journal of Consulting Psychology, 1967, 31, 499-506.
- Tulving, E., & Pearlstone, Z. Availability versus accessibility of information in memory for words. Journal of Verbal Learning and Verbal Behavior, 1966, 5, 381-391.
- Tulving, E., & Thomson, D. M. Encoding specificity and retrieval processes in episodic memory. Psychological Review, 1973, 80, 352-373.
- Turner, B. Pathology of paralysis agitans. In P. J. Vinken & G. W. Bruyn (Eds.), Handbook of clinical neurology, Vol. 6: Diseases of the basal ganglia. New York: Wiley, 1968.
- Underwood, B. J. Experimental psychology. New York: Appleton-Century-Crofts, 1966.
- Van Allen, M. Pictorial manual of neurological tests. Taipei, Taiwan: (Pirated edition), 1969.

- Vogt, M. The relationship between cholinergic and monoaminergic activity in the caudate nucleus. In F. H. McDowell & A. Barbeau (Eds.), Advances in neurology, Vol. 5. New York: Raven, 1974.
- Walsh, L. S. Surgical treatment of parkinsonism. In D. B. Calne (Ed.), Advances in Neurology. Vol. 3. New York: Raven, 1973.
- Waugh, N. C., & Norman, D. A. Primary memory. Psychological Review, 1965, 72, 89-104.
- Webster, D. D. Critical analysis of the disability in Parkinson's disease. Modern Treatment, 1968, 5, 257-282.
- Wechsler, D. A standardized memory scale for clinical use. Journal of Psychology, 1945, 19, 87-95.
- Wechsler, D. Manual for the Wechsler Adult Intelligence Scale. New York: Psychological Corporation, 1955.
- Wechsler, D. The measurement and appraisal of adult intelligence. (4th ed.) Baltimore: Williams & Wilkins, 1958.
- Wimer, R. E., & Wigdor, B. T. Age differences in retention of learning. Journal of Gerontology, 1958, 13, 291-295.
- Yahr, M. D., & Duvoisin, R. C. Medical therapy of parkinsonism. Modern Treatment, 1968, 5, 283-300.
- Yahr, M. D., Duvoisin, R. C., Schear, M. J., Barrett, R. E., & Hoehn, M. M. Treatment of parkinsonism with levodopa. Archives of Neurology, 1969, 21, 343 - 354.
- Yahr, M. D., Wolf, A. Antunes, J. L., Miyoshi, K., & Duffy, P. Autopsy findings in parkinsonism following treatment with L-dopa. Neurology, 1972, 22, 56-65.

APPENDIX A

A REVIEW OF PARKINSON'S DISEASE

History

While most historians of parkinsonism point to the year of 1817 as the beginning of the investigation of parkinsonism, it should be pointed out that Galen of Pergamum (129-199 A.D.) described tremors in his practice as a surgeon to the gladiators in Rome (Singer, 1957). Franciscus (Sylvius) de la Boe (1614-1672) first discriminated between a resting tremor and an action tremor. He also noted an association between tremor and paralysis (Roche, 1973b). Glaudius (1705-1780), and Boisser de Sauvages in 1795, both described a disorder of gait associated with tremors, and both were used as references by James Parkinson in his classic work, An Essay on the Shaking Palsy (1817).

Parkinson's 66-page description of the syndrome was based on six cases, three of which he examined in detail, and three of which he observed casually on the streets or in public places in London. Parkinson defined the syndrome in this manner:

Shaking Palsy. (Paralysis Agitans.)
Involuntary tremulous motion, with
lessened muscular power, in parts not
in action and even when supported; with
a propensity to bend the trunk forwards,
and to pass from a walking to a running
pace: the senses and intellect being
uninjured (Parkinson, 1817, p. 1).

Parkinson believed that the syndrome was caused by "a disordered state of that part of the medulla which is contained in the cervical vertebrae (p. 56)." For treatment, he recommended that "blood should be taken from the upper part of the neck (p. 58)."

Parkinson's essay was little noted at the time, and no progress was made until Ordenstein, a student of Charcot's, first prescribed belladonna as a treatment for the symptoms in the 1870's. There was some benefit, and belladonna alkaloids were prescribed up until the 1940's. By 1888, Gowers felt that the tremor of parkinsonism was caused by a diffuse cerebral disease involving the cortex, hypothalamus, and internal capsule (Roche, 1973a). In 1895, Brissaud deduced from clinical evidence that the localization was at least sub-thalamic. In 1917, Tretiakoff demonstrated in an autopsy study that there was a reduction in the number of pigmented cells in the zona compacta of the substantia nigra. From 1918-1927, a world epidemic of encephalitis lethargica (Von Economo's disease) aroused further interest in the parkinson syndrome, because many sufferers of encephalitis showed then and years later the signs of parkinsonism. Real progress in the biochemistry and histology of the syndrome has only come in the last 20 years. At this time, the cause of the syndrome is still unknown, and no cure exists.

Symptoms

The three primary symptoms of the parkinson syndrome are tremor, rigidity, and akinesia. Tremor (regular alternating contractions of opposing muscle groups) in parkinsonism is distinctive because it occurs at a fairly uniform frequency of 4-8 cycles per second, and because it is a resting tremor rather than an intentional tremor. The tremor tends to disappear during sleep, and can be abolished with extreme effort on the patient's part for brief periods of time. The tremor is likely to increase under periods of stress. Parkinson patients feel more relaxed and tremor less when laying down.

Rigidity, usually a later appearing symptom, refers to the resistance felt by an examiner when moving a person's limb through its range of motion. The resistance, described as "plastic", may be either constant or variable. The variable resistance occurs most frequently, and is called "cogwheel rigidity". Rigidity is experienced by the patient as a slowness of the limbs to respond to his own desires.

Akinesia refers to an impairment of the ability to initiate voluntary and spontaneous movement. This deficit is expressed clinically both as a slowness and poverty of movement (Angel, Alstrom, & Higgins, 1970).

Bradykinesia refers to a slowness or sluggishness of movement on request. Akinesia and bradykinesia may be

elicited when a person is asked to walk across a room or touch his nose with his finger. The typical posture, facial expression, hand position, and shuffling gait of a person with clear parkinsonism may be seen in Figure 3.

Demographic Data

Incidence. The incidence of parkinsonism is about 1/1000 for the general population, but increases to about 1/100 for persons 65 years old and older (Roche, 1973a). The occurrence of the syndrome does not appear to be related to sex, family history of parkinsonism, other neurological disorders, nationality, cultural background, nor premorbid intellectual level (Hoehn & Yahr, 1967).

Etiology. The distinction of the disease processes within the parkinson syndrome is made by attempting to determine the etiology. When any one or more of the triad of symptoms appears and no known cause can be established, the disorder is referred to as Parkinson's disease, or idiopathic paralysis agitans (a shaking paralysis of unknown etiology). The two primary known etiological factors are encephalitis lethargica, or Von Economo's disease, and toxic conditions caused by excess manganese, carbon monoxide, reserpine, or phenothiazines. The commonest variety of parkinsonism is the idiopathic type. Over 80% of new cases carry this diagnosis (Roche, 1973b).

Progression. At the time of Parkinson's

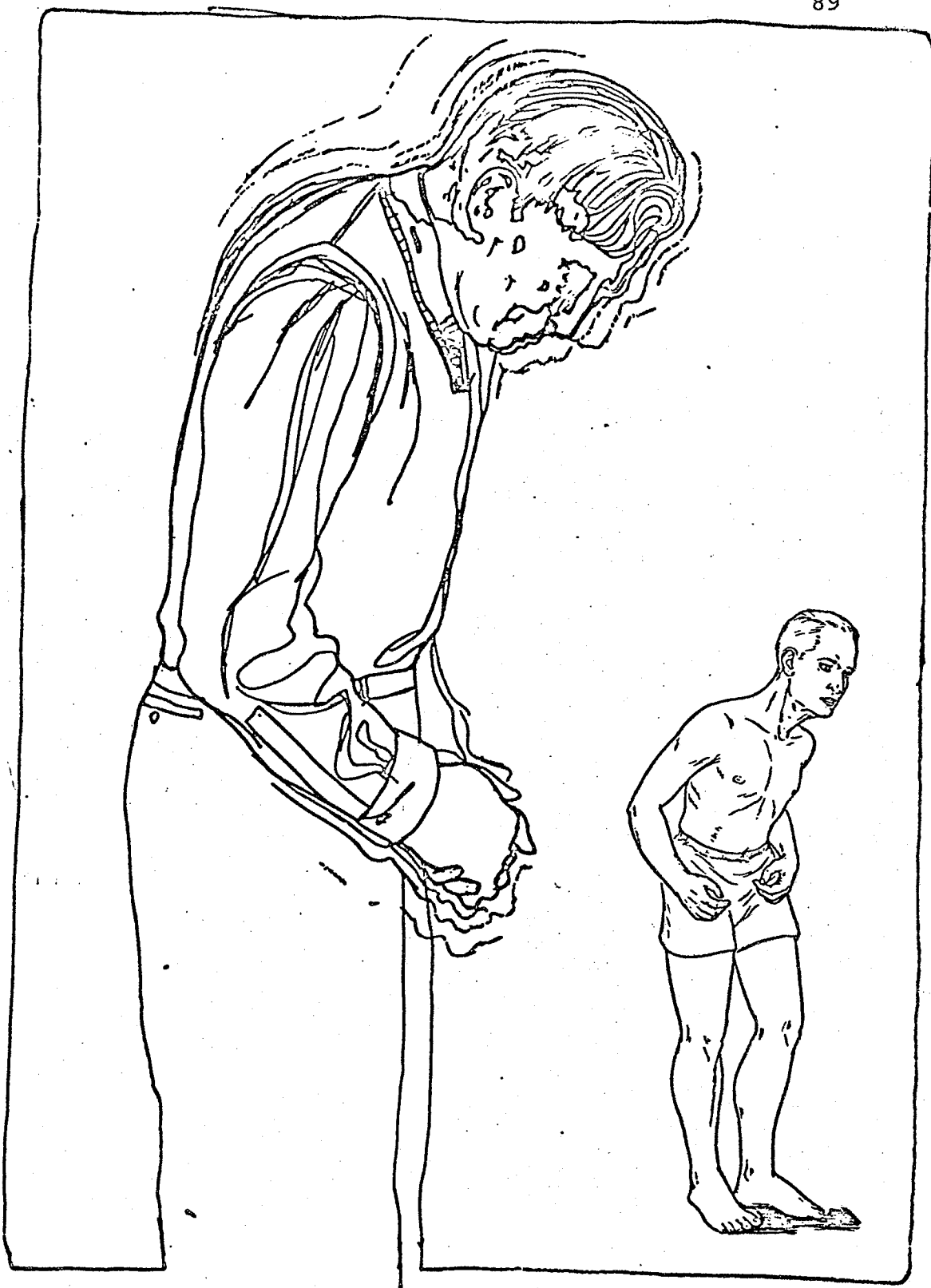


FIGURE 3. Typical posture of parkinsonism (Van Allen, 1969; Roche, 1973b).

publication and during the 1800's, the average age of onset of the syndrome was 56. Following the outbreak of encephalitis, the average age of onset dropped to 54 in the 1920's, to 37 in the 1930's, and rose back up to 45 in the 1940's. As the persons exposed to the 1918-1927 epidemic died, the average age of onset returned to its present level of 56. Many researchers hoped that Parkinson's disease was entirely due to post-encephalitic factors, and predicted in the 1960's that parkinsonism would completely disappear by the 1980's (Poskanzer & Schwab 1961). Unfortunately, while new cases of post-encephalitic parkinsonism are becoming rare, the syndrome continues to have about the same incidence rate as always, and continues to occur at about the average age of 56 (Duvoisin, Yahr, Schnitzer, & Merritt, 1963).

Parkinsonism is a progressive disorder, and the progression of symptoms today follows remarkably close to Parkinson's original descriptions of his three detailed cases. Real remissions of the syndrome are unknown. Neither Doshay's (1960) account of the disease as the "friendly disease" because it does not shorten life, is not contagious, is not inherited, is painless, and does not impair intelligence, nor Miller's (1954) account of inevitable progression of symptoms of hopeless dependency or death in seven or eight years are quite

accurate. Many persons develop the symptoms very slowly over a period of twenty years, and experience minimal impairment. Others show a fairly gradual but persistent decline from onset to helplessness in ten to twelve years. A few others deteriorate rapidly after onset, and die within two or three years (Schwab, 1960).

Early signs of onset of parkinsonism are often misdiagnosed as anxiety reaction, depression, or conversion hysteria (Cooper, 1969; Flynn, 1962; Webster, 1968). In about 75% of the cases, the first sign is a resting tremor in a single finger or limb when the person is under stress. Slowly developing reduction of arm swing and reduced facial spontaneity are often early signs noticed by relatives or friends, and are often passed off as due to anger or depression. Frequently, the affected person is not aware of his reduced ability to move until a friend who has not seen him for a year or so comments on how differently he seems to move or talk. Handwriting skills show a characteristic deterioration: as a person writes, his handwriting becomes smaller and smaller until it becomes illegible. The person's voice often shows the same pattern: as he talks, his voice fades out into an inaudible whisper. The parkinson person gradually develops postural and gait changes, so that he

progresses from normal mobility to impaired mobility to immobility to complete dependence. As his gait and balance become impaired, the person becomes realistically concerned about falling, and does indeed fall until he no longer risks being mobile. Such persons feel safer while laying down, and sometimes feel more at ease while in water or in the dark. It becomes very difficult for such persons to concentrate upon ordinary tasks when they must constantly consciously maintain balance to keep from falling over or off something. Gradual loss of the automatic saliva-swallowing response results in the person drooling. Other difficulties include trouble swallowing, trouble with elimination of wastes, impaired recent memory, and in some cases gradually developing dementia.

Mortality. Parkinson's disease in itself does not appear to shorten life. When persons with parkinsonism die, the cause is seldom attributed to Parkinson's disease, because other more recognizable causal factors are present. The major causes of death among parkinson persons are, in order, heart trouble, pneumonia, cancer, and cerebrovascular accidents (Hoehn & Yahr, 1967).

Physiology

Anatomy. The symptoms of parkinsonism arise from disturbances within the extrapyramidal system.

The extrapyramidal system is composed of extrapyramidal portions of the cerebral cortex, the thalamic nuclei connected with the striatum, the corpus striatum, the subthalamus, and the rubral and reticular systems. The extrapyramidal system is conceptualized as a functional system with three layers of integration: cortical, striatal (basal ganglia), and tegmental (midbrain). This system is functionally concerned with associated movements, postural adjustments, and automatic integration. Lesions at any level within the system may obscure or abolish voluntary movements, or replace them with involuntary movements.

The corpus striatum includes the caudate nucleus, the lenticular nucleus, and the tracts of the internal capsule. The caudate nucleus lies adjacent to the floor of the lateral ventricle, and is an elongated mass of grey matter bent back on itself like a horseshoe. The anterior, pear-shaped head lies adjacent to the inferior border of the anterior horn of the lateral ventricle. The slender end continues backward and downward as the tail, entering the roof of the temporal horn of the lateral ventricle and ending near the amygdala. The lenticular nucleus consists of the putamen and globus pallidus, and is located between the insula (Island of Reil), the caudate nucleus, and the thalamus. The putamen is the larger, convex peach-pit-shaped grey

mass lying lateral and just beneath the insular cortex. The globus pallidus is medially adjacent to the putamen, and is separated from the thalamus by the internal capsule. The internal capsule is a fan-shaped radiation of white fibers passing from the motor and sensory cortex down between the caudate nucleus and lentiform nucleus, and between the globus pallidus and the thalamus, extending beneath these structures to form the cerebral peduncle. Beneath and more medial than the globus pallidus, the substantia nigra forms a flattened plate of cells extending from the subthalamic region down into the midbrain. The substantia nigra is divided into the zona compacta and the zona reticularis. The substantia nigra (black substance) gets its characteristic black color from its melanin-containing cells. See Figure 4 for visual aid.

Numerous loop circuits exist between these nuclei. The caudate nucleus sends many fibers to the putamen, which in turn sends fibers to the globus pallidus. The putamen and globus pallidus receive some fibers from the substantia nigra. The thalamus sends fibers to the caudate nucleus. The above nuclei and tracts are bilateral, and effect the body in a contralateral fashion. The above information has been summarized from Chusid (1973), Cooper (1969), Ranson & Clark (1964), Gatz (1972), and Hyde (1971).

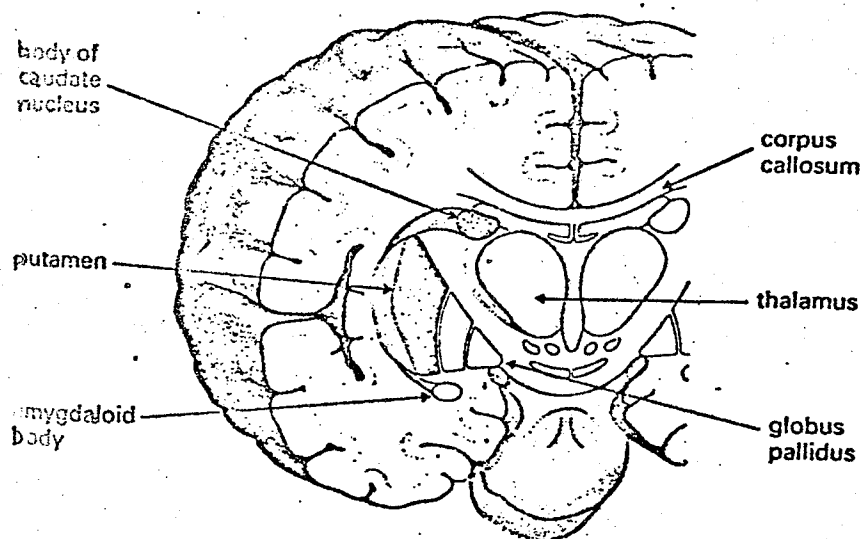
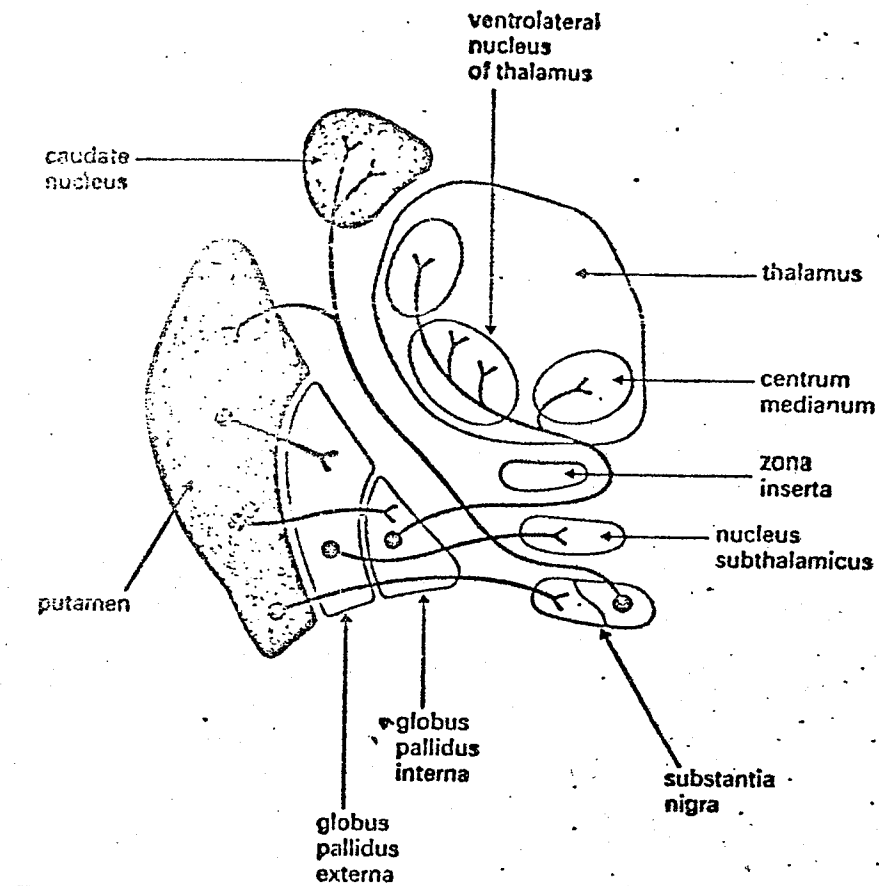


FIGURE 4. The basal ganglia (Roche, 1973 a).

Biochemistry. In the central nervous system there are a number of biogenic amines, or neurotransmitting substances, including: Acetylcholine; 5, hydroxytryptamine (serotonin); and 3, 4, dihydroxyphenethylamine (dopamine). These substances are located and are active at synaptic junctions, and have the capacity to transmit impulses across synaptic gaps or modify the sensitivity of synaptic membranes. Nerve cells and their synaptic contacts are defined by the chemical nature of their neurotransmitters. Thus dopaminergic systems use dopamine as a neurotransmitters, while cholinergic systems use acetylcholine. See Figure 5.

Nearly all the dopamine in the normal human brain is located in the corpus striatum, or basal ganglia, and the substantia nigra. Dopamine is a metabolite of dopa, which itself is formed from tyrosine. Tyrosine is an amino acid derived from the essential amino acid, phenylalanine. Both tyrosine and dopa can cross the blood-brain barrier, while dopamine cannot. Thus, any dopamine in the brain must be formed there. Dopamine is possibly synthesized by neurons in the nigrostriatal tract (the bundle of neurons running from the substantia nigra to the caudate nucleus). If dopamine does not reach the caudate nucleus, a great reduction in function of the dependent dopaminergic systems occurs. Furthermore, it has recently been

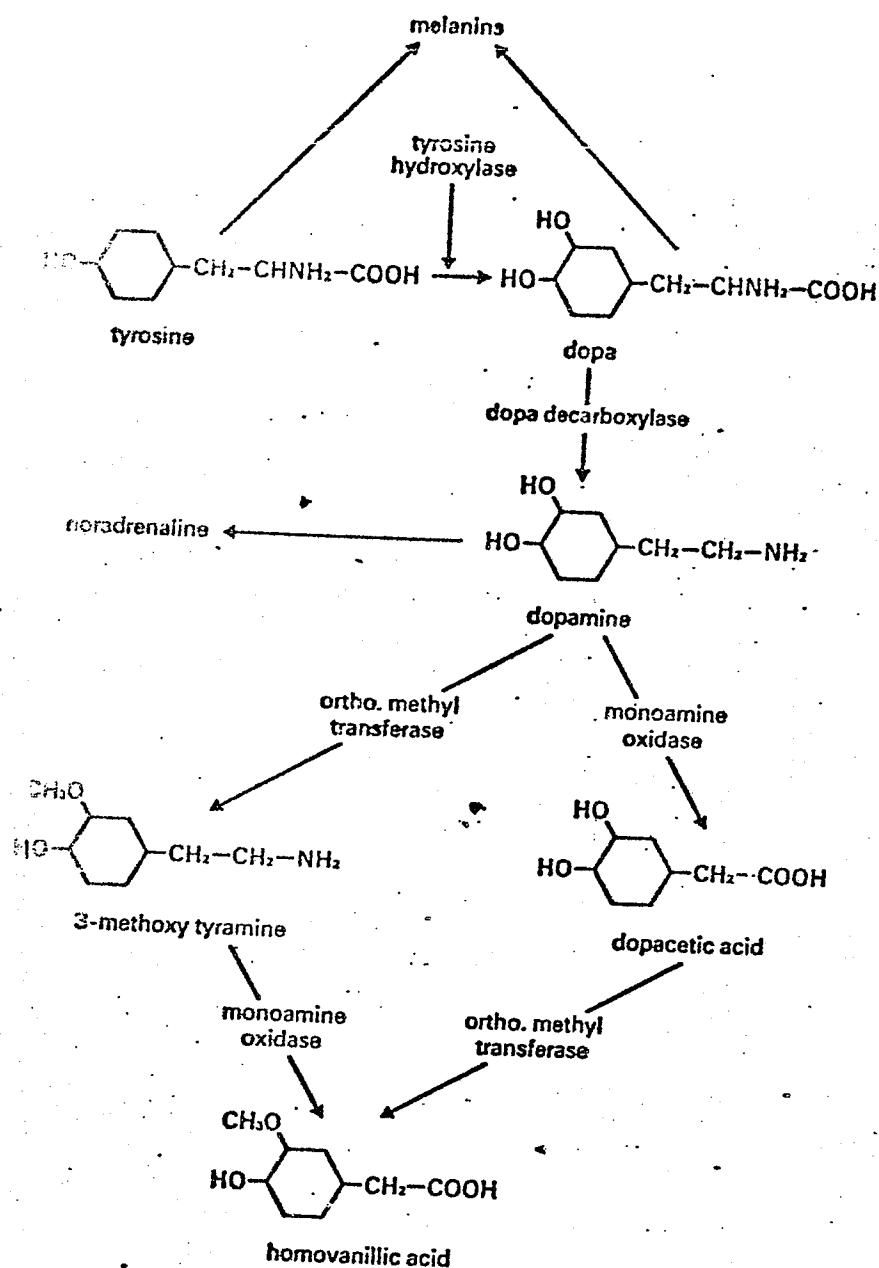


FIGURE 5. Dopamine metabolism (Roche, 1973 a).

noted (Bartholini, Stadler, & Lloyd, 1973, 1974; Vogt, 1974) that reduced dopamine activity results in heightened cholinergic activity. Few of the cholinergic cells are lost or affected in Parkinson's disease. Since dopamine appears to act as an inhibitor of cholinergic activity, if there is less dopamine, there is increased cholinergic activity. Anticholinergic medications such as Artane tend to reduce the symptoms of rigidity and tremor, but they do not reduce akinesia, which is considered to be due primarily to a dopamine deficit. The above conceptualization accounts for several observations:

1. A side-effect of neuroleptic medications such as chlorpromazine and haloperidol may be parkinsonism, particularly rigidity and tremor. Phenothiazines inhibit the uptake of dopamine in striatal synapses. This results in a depletion of dopamine, which would be followed by an unchecked increase of acetylcholine, which would result in tremor and rigidity. When phenothiazines are discontinued, the parkinson symptoms usually gradually disappear. When anticholinergic medications are given in conjunction with phenothiazines, the symptoms of rigidity and tremor seldom occur.
2. When Charcot's student prescribed belladonna, some patients improved. Belladonna's active ingredient is atropine, which is anticholinergic. Thus, symptoms of

tremor and rigidity would reduce.

3. When L-dopa (the levorotary isomer of 3, 4 di-hydroxyphenylalanine) is ingested, and if any benefit is to be derived, first akinesia reduces, then later rigidity and tremor. The reaction to L-dopa is usually much stronger than the reaction to anticholinergics alone, because the anticholinergics merely reduce the cholinergic activity without effecting the reduced dopaminergic activity. L-dopa is a double-edged sword, because as it replaces dopamine into the system, the dopamine itself tends to inhibit the cholinergic activity.

4. Ventrolateral thalamic surgery is usually followed by sudden elimination of tremor and rigidity, while akinesia is not effected. This means that the surgery essentially destroys or disrupts the unchecked cholinergic tracts.

Autopsy studies. The first parkinson autopsy was performed by Oppolzer in Vienna in 1861, but yielded little practical information (Roche, 1973b). The first autopsy study which yielded concrete results was reported by Blocq and Marinesco in 1893. They found a tuberculoma in the right side of the brain stem of a 38-year old patient with contralateral tremor and rigidity (Martinez & Utterback, 1973). In 1871, Meyert conducted an autopsy study of a person with the

parkinson symptoms, and had hypothesized that the tremor was probably caused by lesions in the basal ganglia (Greenfield, 1955). In 1919, Tretiakoff reported autopsy results which showed loss of pigmented cells in the substantia nigra. He also noted the presence of the recently discovered Lewy bodies in the substantia nigra (Selby, 1968). Tretiakoff's results were substantiated by Freeman (1925), and by Hassler's study of 32 parkinson brains in 1938 (Turner, 1968). Degeneration was most prominently noted in the zona compacta of the substantia nigra. Greenfield & Bosanquet (1953) confirmed with their autopsy studies that the pigmented cells in the substantia nigra and locus ceruleus, as well as other melanin-containing neurons in the brainstem showed consistent degenerative processes in parkinsonism. However, some studies have shown definite cellular loss in the substantia nigra without the persons having shown any signs of parkinsonism. Also, Denny-Brown (1960) described two cases of severe parkinsonism, each of which showed on autopsy to have a histologically normal substantia nigra.

In 1965, Richardson studied 119 brains of well-documented parkinson persons, and found definite examples of Alzheimer type neurofibrillar bodies in the substantia nigra of post-encephalitic persons, and Lewy bodies in

the locus ceruleus of the post-encephalitic persons was slight, while in the idiopathic persons it was moderate compared to persons with Alzheimer's disease.

Earle (1968), after reviewing 513 autopsy cases of parkinsonism, concluded that the most consistent findings were the loss of neurons in the substantia nigra, and the presence of Lewy bodies within the pigmented neurons. In 1972, Yahr, Wolf, Antunes, Miyoshi, & Duffy reported on autopsy findings of 36 parkinson persons who had all been treated with L-dopa. In all brains, the most outstanding and consistent finding was cell degeneration within the substantia nigra, with concomitant reduction in volume and increased pallor. The pallor was due to loss of neuromelanin-pigmented cells in the zona compacta of the substantia nigra. In all cases of idiopathic parkinsonism, there were Lewy bodies with an absence of Alzheimer tangles. In all cases of definite post-encephalitic parkinsonism, there was an absence of Lewy bodies, and a presence of Alzheimer tangles. The authors concluded that while most of the persons had benefitted from the L-dopa in terms of reduction of symptoms, the autopsies suggested that the progression of cell degeneration was unaffected by the medication, and continued until the person died. There also appeared to be no clear-cut relationship between the

degree of cell loss in the substantia nigra and degree of response to L-dopa.

Martinez & Utterback (1973) presented detailed information on a single case with definite left hemiparkinsonism, who showed on autopsy marked depigmentation of the substantia nigra and locus ceruleus on the contralateral side. There was extensive loss of melanin-containing and melanin-free neurons in the right substantia nigra and right locus ceruleus, with moderate gliosis. The case history of severe influenza in 1918 was substantiated with the presence of only one Lewy body and large numbers of interlacing Alzheimer tangles. The results were felt to account for why some parkinson patients do not respond to L-dopa. It is believed that at least some functioning nigro-striatal paths must be available if L-dopa is to arrive at the corpus striatum as dopamine.

Alvord, Forno, Kusske, Kauffman, Rhodes, & Goetowski (1974) reported on autopsies completed on 532 cases, of which 129 were definite parkinson persons. All had hospital records available. In addition to checking for cell changes in the substantia nigra, they also examined parts of the hippocampal formation, and parts of the frontal cortex. They found that all parkinson persons had a greater degree of cortical degeneration than age-matched non-parkinson controls.

The degree of cortical degeneration did not correlate with the degree of severity of symptoms of parkinsonism. While both groups showed fairly linear relationships between increasing age and increasing dementia, the parkinsons showed a considerably higher degree of dementia than age-matched non-parkinson controls. Measures of dementia were not reported. They found an orderly pattern of increasing severity of parkinsonism with increasing degeneration of the substantia nigra, independent of age. They found no correlation between amount of substantia nigra degeneration and cortical degeneration. While non-parkinson controls also showed increasing cortical degeneration with increasing age, they showed no dementia. The authors concluded that:

1. Cortical degeneration appears to start in all persons at about age 50-60, and increases linearly with increasing age.
2. The presence of Lewy bodies in the substantia nigra begins at about age 60, and remains constant.
3. All parkinson persons have more cortical degeneration and disproportionately more dementia than age-matched non-parkinson peers; but these differences are not proportionate to the degree or type of parkinsonism.
4. Parkinsonism appears to occur in people with diffuse degenerative changes in the brain. There are at

least two types which can be distinguished: (a) "Lewy body disease" which is relatively stereotyped, in which the degree of parkinsonism is correlated with the degree of neuronal loss in the substantia nigra and the degree of dementia is correlated with the degree of all cerebral cortical degenerations; and (b) "Alzheimer tangle disease," which includes post-encephalitic cases and others, possibly a variant of senile dementia, in which the degree of cortical degeneration correlates with degree of dementia but the degree of neuronal loss in the substantia nigra correlates only with the more severe degrees of parkinsonism.

Forno & Alvord (1974) reported electron microscopic examination of the same autopsy material mentioned above, and reported the rather critical finding that what had always appeared as deficient melanin was actually more a displacement of the neuromelanin to the periphery of the abnormal cells by Lewy bodies. There was much more convincing depigmentation in cases where Alzheimer tangles were found. Normals rarely showed such focal unpigmentation. Curiously enough, albino people who have lost melanin throughout their body still show the normal amount of melanin in the substantia nigra (Cotzias & McDowell, 1971).

Autopsy studies on parkinson persons who have

had thalamic surgery also indicate that the progressive degeneration of the cells in the substantia nigra continues independently of surgery (Cooper, 1968).

Treatment

Medication. While Parkinson's primary treatment for paralysis agitans was blood-letting from the upper part of the neck, he also prescribed antimony to induce sweating, and calomel to induce bowel movements (Parkinson, 1817). Ordenstein in 1867 was the first to prescribe belladonna alkaloids for treatment for Parkinson's disease (the name was changed from paralysis agitans by Ordenstein's mentor, Charcot) (Selby, 1968). Other early medications included intravenous iodine arsenic, mercury, parathormone, trypan blue, and X-ray irradiation of the head (Onuagulchi, 1968). The belladonna alkaloids were seen to be the only medications which reduced parkinson tremor and rigidity during the 19th century (Yahr & Duvoisin, 1968). During the epidemic of encephalitis lethargica from 1918-1927, these medications were still the only effective treatment for post-encephalitic parkinsonism, oculogyric crises, torticollis, and dystonia. It is now known that the therapeutic effect of these medications derived from atropine and scopolamine, which act as central anticholinergics. In 1946, the first synthetic anticholinergic preparations were made, and these

medications continue to be used at the present time.

The most popular anticholinergic medication used presently is Artane (benzhexal hydrochloride), a piperidyl compound. Other piperidyl compounds include Kemadrin (procyclidine), Pagitane (cycrimine), and Akineton (biperiden). Belladonna alkaloids still used include scopolamine, hyoscine, and stramonium. A related preparation is Cogentin (benztropine methanesulfonate). Approximately 20 other anticholinergic medications are now available. Three anti-histamines are also presently in use: Benadryl (diphenhydramine), Disipal (orphenadrine hydrochloride), and Phenoxene (chlorphenoxene) (Duvoisin, 1965).

The research to evaluate the effectiveness of these medications has been plagued with methodological difficulties. In addition to the lack of adequate placebo controls, crossover designs, or multiple-blind procedures, the peculiarities of the syndrome include the following:

1. At least 70% of all parkinson patients respond to almost any medication given to them if they believe it will help them.
2. The symptoms of the syndrome show irregular diurnal variations. Such persons generally feel better in the morning and early evening, and generally feel worse in the afternoon and later evening (Barbeau, 1974).

3. Nearly all symptoms drop out when the person sleeps.
4. There appears to be an inverted U-shaped functional relationship between the progression of the disease and the response to a standard dose of medication.
5. There appears to be an inverted U-shaped functional relationship between dosage of medication and response to medication.

Dopamine was first discovered in 1957. By 1959, its distribution in the brain was known. By 1961, dopa was synthesized and first tried as a therapeutic agent (Schwarz, 1970). In 1967, Cotzias, Van Woert, & Schiffer brought to an end six years of frustrating contradictory results by switching from a D-isomer to an L-isomer of dopa, and increasing the dosage. Clear therapeutic results were noted. Treatment with phenylalanine, the amino acid precursor of dopa, was ineffective (Cotzias, Papavasiliou, & Gellene, 1969). L-dopa became available for research purposes in 1968, and became commercially available in 1970. While L-dopa has limitations, it continues to be the treatment of choice for parkinsonism at this time (Bunney, 1970).

A variety of medication strategies have been developed. One is to prescribe both L-dopa and anticholinergics. Another is to prescribe L-dopa with other medications which inhibit the metabolism of L-dopa into dopamine outside of the brain (Ericsson &

McMann, 1974; Pletscher, 1973). Presently, the dosage range for L-dopa varies from about 1 gram/day to about 5 grams/day, with the average initial dosage about 2-4 grams/day, and the average maintenance dosage about 3 grams/day. When decarboxylase inhibitors are used, L-dopa dosages vary from 300 mg to 1.5 grams/day. Frequent side effects from L-dopa include nausea (55% of patients report this side effect), involuntary movements such as chewing (50%), hypotension (27%), anorexia (24%), and mental changes (24%) (Roche, 1973b)

Surgery. In the 1930's, it was observed that persons with parkinsonism who had a cerebrovascular accident which resulted in hemiplegia also lost their tremors on the same side as the hemiplegia. Researchers erroneously reasoned that the tremors were thus caused by impulses from the motor cortex. In 1935, Bucy performed the first excisions of the motor cortex. In 1938, Putman incised the pyramidal tract at the level of the second cervical spinal segment. In 1940, Klemme excised parts of the premotor cortex. In 1952, Walker cut the cortico-spinal tract at the base of the cerebral peduncle. Oliver in 1953 attempted to sever the lateral column of the spinal cord. None of these procedures had any effect upon rigidity, but did eliminate tremor in the newly paralyzed part of the body.

In 1954, Cooper was attempting to section the cerebral peduncle of a man severely afflicted with

parkinsonism. During the course of the operation, the anterior choroidal artery was nicked, and subsequently tied off. On the following day, it was noted that the tremor and rigidity in the limbs contralateral to the tied-off artery had disappeared. As a result, Cooper initiated a vigorous research program into the effects of surgery on the nuclei served by the anterior choroidal artery: the substantia nigra, the ansa lenticularis, the red nucleus, the globus pallidus, and the ventrolateral part of the thalamus (Walsh, 1973).

Such surgery is performed under local anesthetic with the patient fully awake and communicating with the surgeon. Using a combination of two-plane X-ray photographs while inserting a cannula through a burr hole, the surgeon can position the cannula fairly close to the target area. Using cryosurgical procedures, the surgeon can temporarily cool an area and observe the responses in the patient. When a target area is pinpointed, the temperature is rapidly lowered to about -50°C to produce a permanent lesion. The target area is in the ventrolateral thalamus. Such a lesion results in the loss of both rigidity and tremor, with an improvement in some postural deformities, and does not cause paralysis or other neurological deficits. Such surgery also does not compromise motor strength. It will essentially have no effect on akinesia, but may

reduce bradykinesia secondary to rigidity. It will also not effect the decrease in voice volume or the monotony of the parkinson voice. Such surgery has been shown to have no harmful effects on long-term intellectual functions (Riklan, 1973).

While Cooper did about 100 such operations each year, he discontinued all parkinson surgery in 1970, when he became convinced of the effectiveness of L-dopa. Since 1971, he has resumed doing surgery, but has restricted such operations to a very few parkinson patients who have failed to benefit from L-dopa. In 1971, Cooper stated "...it's my opinion that L-dopa, not surgery, is the treatment of choice for the average parkinsonian patient (Cotzias & McDowell, 1971, p. 52)."

In summary, Parkinson's disease is a term used to describe a syndrome which is characterized by tremors, rigidity, and akinesia. Present methods of treatment are ameliorative, but not curative. The cause of the syndrome is still unknown, but appears to be related to the impaired function of the dopamine system and the pigmented cells of the substantia nigra in the brain stem. Research in 1976 continues to try to sort out the variables involved in dopamine metabolism, and is presently focusing upon finding the precursors to the dopamine system deterioration. While it may possibly be that a small peptide called MIF (melanocyte

inhibiting factor) is involved, further basic and clinical research is needed. Solution of the puzzle seems to be contingent upon finding a method to convince policy-makers of the tremendous need for research funds.

APPENDIX B
SCREENING TEST RESULTS

TABLE 9, Part 1
Summaries of One-Way Analyses of Variance
on Screening Test Data

Measure	Source of Variance	Sum of Squares	df	Mean Square	F
Age	Between	6766.44	3	2255.48	30.03***
	Within	8111.31	108	75.10	
	Total	14877.75	111		
Education	Between	24.02	3	8.01	0.87
	Within	989.97	108	9.16	
	Total	1014.99	111		
Weight	Between	298.69	3	99.56	0.69
	Within	15652.69	108	144.93	
	Total	15951.38	111		
MG/Med.	Between	132943696.00	3	44314560.00	31.25***
	Within	153170688.00	108	1418247.00	
	Total	286114304.00	111		
Ratio MG/KG	Between	28657.02	3	9552.34	33.48***
	Within	30811.41	108	285.29	
	Total	59468.43	111		
FTR	Between	9958.31	3	3319.44	20.27***
	Within	17181.00	105	163.63	
	Total	27139.31	108		
FTL	Between	9594.44	3	3198.15	23.20***
	Within	14339.81	104	137.88	
	Total	23934.25	107		

***p < .001

TABLE 9, Part 2
Summaries of One-Way Analyses of Variance
on Screening Test Data

Measure	Source of Variance	Sum of Squares	df	Mean Square	F
RTR	Between	27545.88	3	9181.96	12.16***
	Within	80043.63	106	755.13	
	Total	107589.50	109		
RTL	Between	23767.06	3	7922.35	11.91***
	Within	69844.19	105	665.18	
	Total	93611.25	108		
Chair	Between	280.63	3	93.54	6.67**
	Within	1360.14	97	14.02	
	Total	1640.77	100		
WAIS VIQ	Between	1942.00	3	647.33	3.79*
	Within	18445.00	108	170.79	
	Total	20387.00	111		
Info.	Between	53.46	3	17.82	3.04*
	Within	632.22	108	5.85	
	Total	685.68	111		
Comp.	Between	216.31	3	72.10	7.30***
	Within	1066.25	108	9.87	
	Total	1282.56	111		
Arith.	Between	133.64	3	44.55	4.51**
	Within	1066.79	108	9.88	
	Total	1200.43	111		

*p < .05

**p < .01

***p < .001

TABLE 9, Part 3
Summaries of One-Way Analyses of Variance
on Screening Test Data

	Source of Variance	Sum of Squares	df	Mean Square	F
Sim.	Between	62.39	3	20.80	1.93
	Within	1161.29	108	10.75	
	Total	1223.68	111		
Digits	Between	26.09	3	8.70	1.06
	Within	885.97	108	8.20	
	Total	912.06			
Vocab.	Between	83.32	3	27.77	3.13*
	Within	958.65	108	8.88	
	Total	1041.97	111		
MMPI D-Score	Between	5296.88	3	1765.63	12.75***
	Within	14959.25	108	138.51	
	Total	20256.13	111		
Cat. Pr. Time	Between	240443.00	3	80147.63	6.47**
	Within	1313181.00	106	12388.50	
	Total	1553624.00	109		
Errors	Between	14.24	3	4.75	1.91
	Within	268.04	106	2.48	
	Total	282.28	109		
Correct out of 4	Between	9.96	3	3.32	5.66**
	Within	63.32	106	0.59	
	Total	73.23	109		

*p < .05

**p < .01

***p < .001

TABLE 10, Part 1
Post-hoc Comparisons (Dunn Multiple t)
on Screening Data

Measure	Comparison	df	t-value
Age	PD - A	54	0.68
	PD - O	54	-7.83*
	PD - N	54	1.23
	A - O	54	-8.11*
	A - N	54	0.52
	O - N	54	8.63*
MG/Med.	PD - A	54	1.90
	PD - O	54	5.41*
	PD - N	54	6.13*
	A - O	54	7.01*
	A - N	54	7.63*
	O - N	54	1.49
Ratio MG/KG	PD - A	54	-2.02
	PD - O	54	5.42*
	PD - N	54	6.13*
	A - O	54	7.41*
	A - N	54	8.09*
	O - N	54	1.43
FTR	PD - A	51	0.72
	PD - O	51	-4.15*
	PD - N	51	-7.49*
	A - O	54	-3.28*
	A - N	54	-5.76*
	O - N	54	-4.28*
FTL	PD - A	50	-1.22
	PD - O	50	-3.60*
	PD - N	50	-7.87*
	A - O	54	-3.39*
	A - N	54	-6.36*
	O - N	54	-4.92*
RTR	PD - A	52	-0.81
	PD - O	52	-2.06
	PD - N	52	-5.21*
	A - O	54	-2.47
	A - N	54	-5.02*
	O - N	54	-3.28*

*p < .05/6, or p < .008

TABLE 10, Part 2
 Post-hoc Comparisons (Dunn Multiple t)
 on Screening Data

Measure	Comparison	df	t-value
RTL	PD - A	51	-0.93
	PD - O	51	-2.36
	PD - N	51	-5.51*
	A - O	54	-2.58
	A - N	54	-4.87*
	O - N	54	-2.81
Chair	PD - A	43	2.70
	PD - O	53	-2.50
	PD - N	53	-3.71*
	A - O	44	-0.25
	A - N	44	-1.41
	O - N	54	-1.63
WAIS VIQ	PD - A	54	-2.33
	PD - O	54	-2.42
	PD - N	54	-3.13*
	A - O	54	0.34
	A - N	54	0.57
	O - N	54	-1.06
Info.	PD - A	54	-1.95
	PD - O	54	-0.32
	PD - N	54	-2.02
	A - O	54	2.22
	A - N	54	0.44
	O - N	54	-2.24
Comp.	PD - A	54	-2.87*
	PD - O	54	0.05
	PD - N	54	-3.36*
	A - O	54	3.23*
	A - N	54	0.40
	O - N	54	-3.82*
Arith.	PD - A	54	-1.73
	PD - O	54	0.41
	PD - N	54	-2.51
	A - O	54	2.55
	A - N	54	0.84
	O - N	54	-3.46*

*p < .008

TABLE 10, Part 3
Post-hoc Comparisons (Dunn Multiple t)
on Screening Data

Measure	Comparison	df	t-value
Vocab. ^a	PD - A	54	-2.23 ^b
	PD - O	54	-0.37
	PD - N	54	-2.26
	A - O	54	2.04
	A - N	54	0.12
	O - N	54	-2.07
MMPI D-Score	PD - A	54	0.63
	PD - O _q	54	2.88*
	PD - N	54	-5.43*
	A - O	54	2.69
	A - N	54	5.90*
	O - N	54	2.33
Cat. Pr. Time	PD - A	52	3.05*
	PD - O	52	0.88
	PD - N	52	3.85*
	A - O	54	1.88
	A - N	54	1.73
	O - N	54	2.77
Errors	PD - A	52	1.55
	PD - O	52	0.57
	PD - N	52	2.37
	A - O	54	0.88
	A - N	54	0.78
	O - N	54	1.59
Correct out of 4	PD - A	52	-2.22
	PD - O	52	0.26
	PD - N	52	-2.78
	A - O	54	3.00*
	A - N	54	1.21
	O - N	54	-3.72*

^aSubtests not showing significant F on ANOVA were not computed for t-values, and do not appear in Table 8.

^bSince the alpha-level was reduced to .008, many of the above comparisons are non-significant even though the ANOVA indicated otherwise.

*p < .008

TABLE 11

Post hoc Comparisons (Dunn Multiple t) on
Screening Measures of Parkinson Persons on L-dopa
versus Parkinson Persons not on L-dopa

Measure	df	t-value
FTR	23	1.70
FTL	22	0.54
RTR	24	0.02
RTL	23	0.46
Chair	25	1.72
WAIS VIQ	26	1.70
Info.	26	1.09
Comp.	26	0.63
Arith.	26	2.33 ^a
Sim.	26	2.98
Digits	26	0.75
Vocab.	26	0.76
MMPI	26	0.22
Cat. Pr. Time	24	0.14
Errors	24	0.20
Correct/4	24	1.13

^aThe alpha-level was set at .05/16, or .003.

TABLE 12

Post hoc Comparisons (Dunn Multiple t) on
Screening Measures of Arthritis Persons on Entrophen
versus Arthritis Persons not on Entrophen

Measure	df	t-value
FTR	26	0.00
FTL	26	0.06
RTR	26	1.19
RTL	26	1.34
Chair	16	0.20
WAIS VIQ	26	2.58
Info.	26	2.17
Comp.	26	3.01 ^a
Arith.	26	1.92
Sim.	26	0.23
Digits	26	0.92
Vocab.	26	0.70
MMPI	26	0.89
Cat. Pr. Time	26	1.07
Errors	26	0.55
Correct/4	26	1.10

^aAlpha-level set at .003.

TABLE 13

Summary of One-Way Analysis of Variance of
WAIS VIQ as a Function of Diagnostic Group

Source Variance	Sum of Squares	df	Mean Square	F
Between Groups	1942.00	3	647.33	3.79*
Within Groups	18445.00	108	170.79	
Total	20387.00	111		

*p < .01

TABLE 14

Post-hoc Comparisons (Dunn Multiple t)
Between Groups on WAIS VIQ

Comparison	df	t-value
PD - A	54	-2.33
PD - O	54	-2.42
PD - N	54	-3.13*
A - O	54	0.34
A - N	54	0.57
O - N	54	-1.06

*p < .008

TABLE 15

Summary of One-Way Analysis of Variance on
MMPI Depression Scale Scores

Source Variance	Sum of Squares	df	Mean Square	F
Between Groups	5296.88	3	1765.63	12.75**
Within Groups	14959.25	108	138.51	
Total	20256.13	111		

**p < .01

TABLE 16

Post-hoc Comparisons (Dunn Multiple t) Between
Groups on MMPI Depression Scale Scores

Comparison	df	t-value
PD - A	54	0.63
PD - O	54	2.88
PD - N	54	5.43*
A - O	54	2.69
A - N	54	5.90*
O - N	54	2.33

*p < .008

TABLE 17
Means and Standard Deviations of
Hours of Sleep, by Group

Group	\bar{X}	s
Parkinson	7.18	1.49
Arthritis	6.93	1.33
Older	7.14	1.11
Normal	6.64	0.99

TABLE 18
Summary of One-Way Analysis of
Variance on Hours of Sleep

Source of Variance	Sum of Squares	df	Mean Square	F
Between Groups	5.10	3	1.70	1.09
Within Groups	167.82	108	1.55	
Total	172.92	111		

TABLE 19, Part 1
Raw Data on Parkinson Persons

S#	Sex	Age	Ed.	MG. Med.	KG. Wt.	Ratio	FTR	FTL	RTR	RTL	Chair
1	M	57	12	0	83	0	33	30	85	59	10
2	F	63	6	0	65	0	25	27	52	56	10
3	F	53	10	2000	63	32	8	11	30	38	20
4	M	62	12	0	75	0	27	28	46	60	10
5	F	63	7	3000	58	52	9	14	35	34	15
6	M	40	16	2200	58	38	42	43	115	73	11
7	M	71	8	4000	58	69	20	23	25	29	35
8	M	50	9	2000	76	26	20	18	45	44	12
9	F	44	11	0	68	0	20	11	39	39	8
10	F	52	8	4000	63	64	16	16	55	49	10
11	M	60	3	0	99	0	25	26	55	55	12
12	F	48	8	3500	87	40	19	21	51	59	9
13	F	71	11	3000	54	37	14	15	45	46	20
14	M	70	12	2000	82	27	20	29	62	69	13
15	M	54	9	2000	70	29	17	28	73	68	13
16	F	64	8	0	59	0	24	26	51	52	10
17	F	63	3	0	59	0	18	21	30	25	17
18	F	62	8	1500	45	33	/	/	9	16	20
19	M	53	5	4000	75	53	/	/	/	/	11
20	M	64	10	0	53	0	45	/	71	/	10
21	M	72	8	4000	83	48	24	22	53	51	13
22	F	65	9	3000	79	38	13	13	62	65	10
23	M	64	11	3500	88	40	29	30	42	42	10
24	M	52	12	2000	72	28	20	22	45	45	14
25	F	70	16	0	65	0	/	/	/	/	/
26	F	68	13	0	57	0	19	22	37	32	12
27	M	60	5	2000	83	24	14	16	25	38	17
28	F	66	8	2000	59	34	35	32	104	97	12

TABLE 19, Part 2
Raw Data on Parkinson Persons

S#	VIQ	Info.	Comp.	Arith.	Sim.	Digit	Vocab.	MMPI	Time	Error	Correct
1	115	13	16	12	6	12	12	70	410	3	4
2	89	9	4	7	4	10	11	72	335	3	3
3	83	9	6	7	5	4	10	84	530	2	2
4	93	9	7	10	8	6	9	87	385	2	4
5	91	10	7	10	7	4	9	99	345	3	3
6	136	14	18	16	13	19	17	73	140	0	4
7	115	13	10	7	10	10	13	82	420	4	4
8	92	9	6	16	8	9	10	63	240	1	4
9	99	12	8	9	7	10	13	63	236	0	4
10	90	10	5	7	10	7	9	77	280	3	4
11	84	8	7	7	9	4	5	43	445	3	0
12	107	11	10	9	16	7	12	50	106	1	4
13	104	8	11	13	8	9	13	99	205	1	4
14	119	10	7	16	8	15	11	72	225	1	4
15	101	11	9	11	10	11	7	63	144	0	3
16	78	6	6	7	3	6	5	63	285	2	4
17	86	10	7	4	7	4	10	76	300	0	1
18	93	9	9	5	10	7	9	86	/	0	1
19	81	8	7	6	8	4	6	68	480	0	3
20	91	11	10	5	2	10	9	60	165	0	4
21	106	10	9	11	8	7	9	60	280	0	4
22	102	11	10	8	9	7	11	53	175	2	4
23	102	11	9	10	8	9	11	68	323	0	3
24	113	13	15	11	9	9	14	48	215	0	4
25	94	9	9	2	5	6	11	85	/	0	2
26	90	6	6	7	6	9	10	71	108	0	4
27	90	9	7	10	4	10	6	50	600	8	4
28	85	7	4	7	6	9	9	70	375	0	4

TABLE 20, Part 1
Raw Data on Arthritis Persons

S#	Sex	Age	Ed.	MG. Med.	KG. Wt.	Ratio	FTR	FTL	RTR	RTL	Chair
1	F	65	11	2400	75	32	/	/	/	/	/
2	F	70	6	1800	52	35	/	/	/	/	15
3	M	66	9	1200	63	19	27	27	46	46	9
4	F	51	9	0	37	0	/	/	/	/	/
5	M	59	8	3600	90	40	23	25	52	52	8
6	M	62	11	3600	61	59	56	56	91	87	12
7	M	60	11	4800	79	61	52	48	75	60	8
8	M	67	16	4800	82	59	/	/	/	/	/
9	F	35	11	0	53	0	24	24	76	72	/
10	F	62	12	3600	65	55	30	32	32	26	/
11	M	50	11	4800	79	61	2	3	52	40	10
12	F	71	11	800	64	13	26	28	49	47	10
13	M	51	8	3600	72	50	23	28	55	57	7
14	M	54	6	0	61	0	33	23	84	86	7
15	F	73	16	2400	63	38	15	14	40	40	/
16	F	63	8	2400	70	34	23	18	93	95	12
17	M	58	10	1800	77	23	63	49	106	90	9
18	M	56	10	0	63	0	39	40	70	70	9
19	M	65	7	3000	61	49	/	/	/	/	/
20	M	53	8	4800	77	62	37	35	80	84	10
21	M	50	10	2400	76	63	/	/	/	/	/
22	M	47	11	4800	77	62	34	26	70	67	10
23	F	61	6	0	62	0	/	/	52	52	15
24	F	41	12	3600	58	62	/	/	/	/	10
25	F	62	12	4800	72	67	/	/	/	/	/
26	F	50	10	0	71	0	20	20	79	75	9
27	F	59	11	4800	72	67	14	14	58	54	14
28	F	74	12	2400	47	51	/	/	/	/	/

TABLE 20, Part 2
Raw Data on Arthritis Persons

S#	VIQ	Info.	Comp.	Arith.	Sim.	Digit	Vocab.	MMPI	Time	Error	Correct
1	121	13	16	13	12	6	15	68	165	0	4
2	93	7	8	7	4	6	9	72	216	0	4
3	85	8	9	10	2	7	5	80	300	0	3
4	90	10	6	8	6	9	9	72	195	0	3
5	107	11	10	11	11	10	10	46	197	0	4
6	127	14	14	17	15	7	16	60	170	0	4
7	134	15	17	17	11	14	16	47	145	0	4
8	123	14	13	15	10	9	16	77	203	0	4
9	86	10	7	8	3	7	11	60	105	4	4
10	114	10	16	9	8	10	17	63	209	1	4
11	110	13	15	16	7	12	15	72	236	3	4
12	122	13	14	11	9	7	16	84	217	0	4
13	82	12	6	7	2	4	9	68	300	1	3
14	101	9	8	9	10	10	13	58	230	1	4
15	127	13	14	12	12	12	12	72	255	0	3
16	98	9	9	10	6	11	11	77	210	1	4
17	96	10	10	13	3	10	6	57	300	0	4
18	103	11	11	6	11	7	13	51	220	1	4
19	101	10	10	7	11	7	10	81	151	0	4
20	97	10	13	7	7	9	9	70	235	1	4
21	107	9	11	14	10	11	10	72	255	0	4
22	118	14	13	11	8	15	15	58	201	6	4
23	81	6	4	7	5	6	9	63	295	0	3
24	122	12	10	8	12	11	10	68	215	1	4
25	99	10	10	8	5	12	10	72	173	0	4
26	100	10	11	12	12	12	11	82	108	0	4
27	108	12	10	13	5	11	13	70	175	0	4
28	126	12	17	8	11	11	15	70	285	0	4

TABLE 21, Part 1
Raw Data on Older Persons

S#	Sex	Age	Ed.	MG. Med.	KG. Wt.	Ratio	FTR	FTL	RTR	RTL	Chair
1	F	79	6	0	61	0	23	22	73	74	11
2	M	84	8	0	47	0	35	26	75	77	13
3	M	76	13	0	69	0	31	31	24	32	15
4	M	81	12	0	56	0	27	26	57	56	7
5	F	87	8	0	76	0	24	23	66	73	13
6	M	72	8	1800	77	23	52	48	43	45	13
7	M	74	12	0	64	0	35	35	54	48	11
8	M	79	7	0	78	0	44	36	82	73	8
9	F	72	9	0	56	0	38	36	88	92	10
10	M	68	16	0	88	0	18	20	32	31	18
11	F	82	8	0	66	0	33	34	80	70	12
12	F	79	11	0	69	0	37	36	106	114	8
13	F	77	9	0	76	0	32	31	70	69	9
14	F	77	12	0	55	0	34	36	113	109	7
15	F	80	8	0	71	0	22	25	40	35	10
16	F	71	7	0	52	0	28	21	36	31	20
17	F	67	8	0	72	0	25	27	65	65	9
18	M	82	12	0	66	0	43	36	82	81	10
19	M	92	11	0	72	0	23	24	46	36	10
20	M	68	6	0	87	0	26	26	86	89	7
21	F	68	8	0	58	0	39	44	94	83	7
22	F	78	7	0	59	0	38	36	65	68	7
23	F	83	8	0	82	0	33	34	84	80	8
24	F	70	5	1800	62	29	23	22	57	56	11
25	M	75	7	0	66	0	35	33	56	45	8
26	M	66	8	0	91	0	31	29	41	39	10
27	M	84	7	0	82	0	26	14	24	24	11
28	M	65	9	0	80	0	45	42	78	74	9

TABLE 21, Part 2
Raw Data on Older Persons

S#	VIQ	Info.	Comp.	Arith.	Sim.	Digit	Vocab.	MMPI	Time	Error	Correct
1	93	6	6	6	4	9	6	42	280	0	1
2	98	7	7	7	6	6	9	41	460	1	3
3	103	11	5	8	2	10	11	65	408	0	2
4	105	7	5	9	8	10	10	53	227	3	3
5	112	11	9	7	10	9	11	52	335	0	2
6	105	8	7	9	6	11	11	60	192	0	3
7	110	12	10	10	8	7	11	53	402	0	3
8	125	10	9	13	12	12	13	60	175	1	4
9	108	9	10	9	7	9	12	53	135	0	4
10	111	13	11	11	9	7	14	92	482	0	4
11	117	11	16	8	8	7	11	57	156	0	4
12	118	9	9	14	10	9	11	63	140	1	3
13	104	8	8	8	7	7	10	53	165	3	3
14	108	11	9	9	6	7	10	53	150	4	3
15	109	10	7	7	8	11	10	80	168	0	4
16	92	10	6	7	4	4	9	60	320	0	3
17	100	9	9	8	9	9	10	53	260	0	4
18	114	10	12	8	11	10	11	58	135	0	4
19	102	9	9	9	2	7	10	63	144	0	4
20	93	10	10	7	2	10	8	60	204	0	4
21	96	12	10	7	10	6	15	63	130	1	4
22	110	10	10	8	8	9	9	57	215	3	3
23	112	9	7	10	0	11	10	96	720	0	4
24	83	5	5	6	3	7	4	70	500	6	1
25	115	13	10	9	9	6	12	60	139	0	4
26	87	9	7	7	7	2	9	43	280	1	4
27	95	8	7	6	7	4	7	51	312	7	2
28	120	14	10	14	10	12	14	51	247	0	4

TABLE 22, Part 1
Raw Data on Normal Persons

S#	Sex	Age	Ed.	MG. Med.	KG. Wt.	Ratio	FTR	FTL	RTR	RTL	Chair
1	M	43	17	0	85	0	58	57	69	63	10
2	F	63	9	0	77	0	18	21	59	52	20
3	F	48	10	0	65	0	31	31	87	83	7
4	F	49	11	0	57	0	43	43	106	107	8
5	M	44	10	0	75	0	58	52	135	131	7
6	M	53	11	0	84	0	55	48	62	61	8
7	F	41	10	0	54	0	31	32	97	90	8
8	F	60	10	0	61	0	45	44	95	95	9
9	M	59	11	0	89	0	52	53	74	73	7
10	F	57	11	0	77	0	59	58	124	121	7
11	F	56	12	0	64	0	55	55	91	94	10
12	F	57	11	0	62	0	42	45	112	99	8
13	F	59	8	0	56	0	30	24	82	42	12
14	M	54	7	0	84	0	46	45	63	65	7
15	F	46	10	0	63	0	49	48	102	100	7
16	F	67	11	0	74	0	49	47	151	121	8
17	F	50	16	0	75	0	50	58	69	58	9
18	M	61	8	0	89	0	28	30	48	62	9
19	M	50	15	0	86	0	57	54	105	100	12
20	F	47	10	0	59	0	44	37	83	85	8
21	M	66	9	0	62	0	47	37	83	82	12
22	F	50	16	0	50	0	42	44	93	88	8
23	M	75	7	0	67	0	39	38	80	80	8
24	M	71	4	0	70	0	55	52	97	98	11
25	M	66	5	0	70	0	23	25	55	55	9
26	M	74	1	0	94	0	45	45	73	73	8
27	M	66	10	0	75	0	44	43	48	38	10
28	M	66	4	0	84	0	25	32	71	58	9

TABLE 22, Part 2
Raw Data on Normal Persons

S#	VIQ	Info.	Comp.	Arith.	Sim.	Digit	Vocab.	MMPI	Time	Error	Correct
1	126	17	14	16	9	14	17	56	110	0	4
2	91	6	8	7	12	7	7	75	223	0	4
3	118	12	11	17	11	10	15	56	97	0	4
4	108	13	10	11	6	11	15	53	127	1	4
5	110	13	11	12	11	10	13	39	90	0	4
6	116	14	15	13	13	7	12	48	212	0	4
7	99	7	7	9	7	9	10	45	125	0	4
8	114	13	14	9	10	9	15	51	165	0	4
9	112	12	16	9	8	10	13	65	185	0	4
10	114	13	12	13	11	6	15	45	125	0	4
11	106	13	10	8	9	10	12	51	146	1	4
12	116	9	13	11	13	10	16	45	108	0	4
13	94	10	9	10	3	9	9	62	265	0	4
14	113	11	17	17	7	10	9	47	234	0	4
15	128	14	16	16	13	14	13	48	80	0	4
16	92	7	10	7	4	9	9	70	300	0	4
17	124	17	10	12	17	9	17	60	155	4	4
18	93	9	9	7	9	6	9	68	288	2	3
19	116	13	15	12	12	9	13	53	94	1	4
20	98	11	12	9	10	6	8	53	194	0	4
21	119	12	10	15	13	12	11	45	327	0	4
22	137	16	17	14	15	15	19	51	105	0	4
23	94	8	6	10	4	4	10	47	150	0	4
24	89	6	7	7	6	6	5	44	372	0	4
25	100	10	10	13	3	7	11	51	337	2	4
26	88	6	8	5	4	6	6	51	132	0	4
27	117	12	16	10	11	9	13	47	144	0	4
28	106	12	9	16	7	4	12	46	170	2	3

APPENDIX C

SCREENING TEST MATERIALS

Biographical Information

THE FIRST THING WE ARE GOING TO DO IS TO FILL IN SOME BASIC INFORMATION ABOUT YOU, WHICH WILL HELP ME SELECT OTHER PEOPLE WHO ARE ABOUT THE SAME AS YOU IN MANY WAYS. THIS INFORMATION WILL BE KEPT CONFIDENTIAL, AS WILL ALL PARTS OF THIS PROJECT, AND I WILL BE THE ONLY PERSON WHO WILL HAVE THIS INFORMATION. IF YOU WOULD LIKE ME TO, I WILL RETURN THIS INFORMATION TO YOU AFTER THE PROJECT IS OVER. LET'S START.

1. HOW OLD ARE YOU? _____ WHEN IS YOUR BIRTHDATE? _____
2. HOW FAR DID YOU GO IN SCHOOL? _____
3. WHAT IS YOUR MAIN OCCUPATION? _____
4. DO YOU HAVE ANY DIAGNOSED ILLNESS AT THIS TIME? _____
 - A) IF SO, WHAT IS IT? _____
 - B) HOW DOES IT EFFECT YOU IN YOUR DAILY LIFE? _____
5. WHAT MEDICATIONS ARE YOU PRESENTLY TAKING? _____

- HOW MUCH OF EACH DO YOU TAKE PER DAY? _____
6. HOW DO YOU SLEEP AT NIGHT? _____ DETAILS? _____
7. WHAT PART OF THE DAY ARE YOU LIKELY TO FEEL THE BEST?

8. WHAT PART OF THE DAY ARE YOU LIKELY TO FEEL THE WORST?
9. WHEN WOULD BE THE BEST TIME OF THE DAY FOR YOU TO
TAKE THESE TESTS? _____
10. ARE YOU RIGHT-HANDED, OR LEFT-HANDED? _____
11. WEIGHT? _____

TIME		DATE: _____ ACTIVITY	DATE: _____ ACTIVITY	DATE: _____ ACTIVITY
NOON	12			
	1			
	2			
	3			
	4			
PM	5			
	6			
	7			
	8			
	9			
	10			
	11			
MIDNIGHT	12			
	1			
	2			
	3			
	4			
AM	5			
	6			
	7			
	8			
	9			
	10			
	11			

Total Hours Sleep=___ Total Hours Sleep=___ Total Hours Sleep=___

MOTOR TESTS

NEXT I'D LIKE TO SEE HOW WELL YOU CAN MOVE AROUND. I'M GOING TO ASK YOU TO MAKE THREE DIFFERENT KINDS OF MOVEMENTS, AND I WILL BE TIMING YOU WITH A STOP-WATCH. THE PURPOSE OF THE STOP-WATCH IS TO MAKE SURE THAT I GIVE EVERYONE THE SAME AMOUNT OF TIME TO TRY EACH ACTIVITY.

1. FINGER TAP.

FIRST, I WOULD LIKE YOU TO PLACE YOUR HAND IN A COMFORTABLE POSITION ON THIS BOARD. I AM GOING TO SEE HOW MANY TIMES YOU CAN TAP YOUR POINTING FINGER ON THE BOARD, LIKE THIS (DEMONSTRATE), OKAY? NOW WHEN I TELL YOU TO START TAPPING, YOU TAP JUST AS FAST AS YOU CAN UNTIL I TELL YOU TO STOP. READY? OKAY, START TAPPING. (START TIMER ON FIRST TAP, COUNT TAPS, RECORD. DO FIRST WITH DOMINANT HAND, THEN WITH NONDOMINANT HAND. THREE TRIALS EACH HAND, 10 SECONDS EACH.)

DOMINANT: 1. _____

2. _____

3. _____

NON-DOMINANT: 1. _____

2. _____

3. _____

2. RULER TAP.

NOW, I'M GOING TO PLACE THIS RULER ON THE TABLE HERE IN FRONT OF YOU, AND I'M GOING TO ASK YOU TO TAP EACH END OF THE RULER, LIKE THIS: (DEMONSTRATE). I WANT TO SEE HOW MANY TIMES YOU CAN TAP EACH END OF THE RULER. DO THIS AS FAST AS YOU CAN, AND I WILL STEADY THE RULER FOR YOU. READY? OKAY, START TAPPING. (START TIMER WITH FIRST TAP. COUNT TAPS. DO FIRST WITH DOMINANT HAND, THEN NON-DOMINANT HAND. THREE TRIALS EACH HAND, 30 SECONDS EACH.)

DOMINANT: 1. _____
 2. _____
 3. _____

NON-DOMINANT: 1. _____
 2. _____
 3. _____

3. CHAIR: STAND-WALK-TURN-SIT.

TO COMPLETE THIS PART, I WOULD LIKE TO HAVE YOU SIT IN A STRAIGHT-BACKED CHAIR. WHEN I TELL YOU TO BEGIN, I WOULD LIKE YOU TO STAND UP, WALK OVER TO THIS MARK, TURN AROUND, AND WALK BACK TO THE CHAIR AND SIT DOWN. PLEASE WALK AS YOU NORMALLY DO, AND MOVE AS YOU NATURALLY DO. BEGIN.

TIME: _____ sec. OBSERVATIONS: _____

CONSENT FORM

I, _____, agree to take part in the research project conducted by Mr. Daniel L. McIvor, from the Department of Psychology at the University of Manitoba. I understand that I will be tested for about an hour, and will be paid \$5.00 for this work. I may be tested a second time, also for about an hour, and will be paid \$5.00 for this work also.

I give permission to Mr. McIvor to review my medical record to obtain medication information necessary for his project. I understand that any information obtained from the medical record or from the test results will be strictly confidential, and will be used only for the purposes of this research project.

I give permission for Mr. McIvor to come to my home and administer the necessary tests for his research project. I also give permission for another trained examiner, identified by name by Mr. McIvor, to come into my home and administer the second group of tests if I am selected.

I understand what this project is for, and will receive a letter describing the results of the project when it is completed.

Signature

Witness

Date

WAIS VERBAL SUBTESTS

NOW I AM GOING TO ASK YOU SOME QUESTIONS. SOME OF THEM ARE EASY, AND SOME OF THEM ARE VERY DIFFICULT. THE IDEA IS TO DO AS WELL AS YOU CAN. YOU'RE WELCOME TO GUESS. IF YOU DON'T KNOW, JUST SAY I DON'T KNOW, AND WE WILL MOVE ON TO THE NEXT QUESTION. IF I ASK YOU A QUESTION AND YOU CANNOT THINK OF THE ANSWER RIGHT THEN, BUT THINK OF IT LATER, PLEASE TELL ME, AND I WILL GO BACK AND GIVE YOU CREDIT FOR IT. ARE YOU READY? OKAY, WHAT ARE THE COLOURS OF THE CANADIAN FLAG? _____

PREVIOUSLY COPYRIGHTED MATERIALS,
LEAVES 140, 141 and 142, APPENDIX C,
NOT MICROFILMED.

"Wechsler Adult Intelligence Scale Record Form", The
Psychological Corporation, 304 East 45th Street, New
York, N.Y., U.S.A. 10017

WAIS RECORD FORM

Wechsler Adult Intelligence Scale


Name _____ 140 
 Birth Date _____ Age _____ Sex _____ Marital: S M D W
MO DAY YR.
 Nat. _____ Color _____ Tested by _____
 Place of Examination _____ Date _____
 Occupation _____ Education _____

TABLE OF SCALED SCORE EQUIVALENTS*

Scaled Score	RAW SCORE										Scaled Score	
	Information	Comprehension	Arithmetic	Similarities	Digit Span	Vocabulary	Digit Symbol	Picture Completion	Block Design	Picture Arrangement		Object Assembly
19	29	27-28		26	17	78-80	87-90					19
18	28	26		25		76-77	83-86	21		36	44	18
17	27	25	18	24		74-75	79-82		48	35	43	17
16	26	24	17	23	16	71-73	76-78	20	47	34	42	16
15	25	23	16	22	15	67-70	72-75		46	33	41	15
14	23-24	22	15	21	14	63-66	69-71	19	44-45	32	40	14
13	21-22	21	14	19-20		59-62	66-68	18	42-43	30-31	38-39	13
12	19-20	20	13	17-18	13	54-58	62-65	17	39-41	28-29	36-37	12
11	17-18	19	12	15-16	12	47-53	58-61	15-16	35-38	26-27	34-35	11
10	15-16	17-18	11	13-14	11	40-46	52-57	14	31-34	23-25	31-33	10
9	13-14	15-16	10	11-12	10	32-39	47-51	12-13	28-30	20-22	28-30	9
8	11-12	14	9	9-10		26-31	41-46	10-11	25-27	18-19	25-27	8
7	9-10	12-13	7-8	7-8	9	22-25	35-40	8-9	21-24	15-17	22-24	7
6	7-8	10-11	6	5-6	8	18-21	29-34	6-7	17-20	12-14	19-21	6
5	5-6	8-9	5	4		14-17	23-28	5	13-16	9-11	15-18	5
4	4	6-7	4	3	7	11-13	18-22	4	10-12	8	11-14	4
3	3	5	3	2		10	15-17	3	6-9	7	8-10	3
2	2	4	2	1	6	9	13-14	2	3-5	6	5-7	2
1	1	3	1		4-5	8	12	1	2	5	3-4	1
0	0	0-2	0	0	0-3	0-7	0-11	0	0-1	0-4	0-2	0

SUMMARY

TEST	Raw Score	Scaled Score
Information		
Comprehension		
Arithmetic		
Similarities		
Digit Span		
Vocabulary		
Verbal Score		
Digit Symbol		
Picture Completion		
Block Design		
Picture Arrangement		
Object Assembly		
Performance Score		
Total Score		
VERBAL SCORE _____ IQ _____		
PERFORMANCE SCORE _____ IQ _____		
FULL SCALE SCORE _____ IQ _____		

*Clinicians who wish to draw a "psychograph" on the above table may do so by connecting the subject's raw scores. The interpretation of any such profile, however, should take into account the reliabilities of the subtests and the lower reliabilities of differences between subtest scores.

I. INFORMATION		SCORE 1 or 0			SCORE 1 or 0			SCORE 1 or 0
1. Flag			11. Height			21. Senators		
2. Ball			12. Italy			22. Genesis		
3. Months			13. Clothes			23. Temperature		
4. Thermometer			14. Washington			24. Iliad		
5. Rubber			15. Hamlet			25. Blood vessels		
6. Presidents			16. Vatican			26. Koran		
7. Longfellow			17. Paris			27. Faust		
8. Weeks			18. Egypt			28. Ethnology		
9. Panama			19. Yeast			29. Apocrypha		
10. Brazil			20. Population					

OBSERVATIONS:

2. COMPREHENSION

SCORE
2, 1 or 0

1. Clothes	
2. Engine	
3. Envelope	
4. Bad company	
5. Movies	
6. Taxes	
7. Iron	
8. Child labor	
9. Forest	
10. Deaf	
11. City land	
12. Marriage	
13. Brooks	
14. Swallow	

4. SIMILARITIES

SCORE
2, 1 or 0

1. Orange—Banana	
2. Coat—Dress	
3. Axe—Saw	
4. Dog—Lion	
5. North—West	
6. Eye—Ear	
7. Air—Water	
8. Table—Chair	
9. Egg—Seed	
10. Poem—Statue	
11. Wood—Alcohol	
12. Praise—Punishment	
13. Fly—Tree	

3. ARITHMETIC

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	R or W	Time	SCORE
1. 15"			0 1
2. 15"			0 1
3. 15"			0 1
4. 15"			0 1
5. 30"			0 1
6. 30"			0 1
7. 30"			0 1
8. 30"			0 1
9. 30"			0 1
10. 30"			0 1
11. 60"			0 1 ¹⁻¹⁰ ₂
12. 60"			0 1 ¹⁻¹⁰ ₂
13. 60"			0 1 ¹⁻¹⁵ ₂
14. 120"			0 1 ¹⁻²⁰ ₂

5. DIGIT SPAN

SCORE

Digits Forward	Circle
5-8-2	3
6-9-4	3
6-4-3-9	4
7-2-8-6	4
4-2-7-3-1	5
7-5-8-3-6	5
6-1-9-4-7-3	6
3-9-2-4-8-7	6
5-9-1-7-4-2-8	7
4-1-7-9-3-8-6	7
5-8-1-9-2-6-4-7	8
3-8-2-9-5-1-7-4	8
2-7-5-8-6-2-5-8-4	9
7-1-3-9-4-2-5-6-8	9
Digits Backward	Circle
2-4	2
5-8	2
6-2-9	3
4-1-5	3
3-2-7-9	4
4-9-6-8	4
1-5-2-8-6	5
6-1-8-4-3	5
5-3-9-4-1-8	6
7-2-4-8-5-6	6
8-1-2-9-3-6-5	7
4-7-3-9-1-2-8	7
9-4-3-7-6-2-5-8	8
7-2-8-1-9-6-5-3	8

F + B =
Highest numbers circled

	SCORE 2, 1 or 0	6. VOCABULARY
1. Bed		
2. Ship		
3. Penny		
4. Winter		
5. Repair		
6. Breakfast		
7. Fabric		
8. Slice		
9. Assemble		
10. Conceal		
11. Enormous		
12. Hasten		
13. Sentence		
14. Regulate		
15. Commence		
16. Ponder		
17. Cavern		
18. Designate		
19. Domestic		
20. Consume		
21. Terminate		
22. Obstruct		
23. Remorse		
24. Sanctuary		
25. Matchless		
26. Reluctant		
27. Calamity		
28. Fortitude		
29. Tranquil		
30. Edifice		
31. Compassion		
32. Tangible		
33. Perimeter		
34. Audacious		
35. Ominous		
36. Tirade		
37. Encumber		
38. Plagiarize		
39. Impale		
40. Travesty		

MMPI DEPRESSION SCALE

NOW I AM GOING TO READ SOME SENTENCES TO YOU, AND
I'D LIKE YOU TO ANSWER EITHER TRUE OR FALSE AS THE
SENTENCE APPLIES TO YOU AT THIS TIME IN YOUR LIFE. IF
IT'S HARD TO DECIDE, THEN SAY THE BEST ANSWER THAT'S
EITHER MOSTLY TRUE, OR MOSTLY FALSE AS IT APPLIES TO YOU.

MMPI "D" SCALE

- ___ 1. I am easily awakened by noise.
- ___ 2. My judgment is better than it ever was.
- ___ 3. I usually feel that life is worthwhile.
- ___ 4. I go to church almost every week.
- ___ 5. I believe in the second coming of Christ.
- ___ 6. I do not worry about catching diseases.
- ___ 7. I have never had a fit or convulsion.
- ___ 8. I enjoy many different kinds of play and recreation.
- ___ 9. I believe I am no more nervous than most others.
- ___ 10. When I leave home I do not worry about whether the door is locked and the window closed.
- ___ 11. Everything is turning out just like the prophets of the Bible said it would.
- ___ 12. At times I feel like picking a fist fight with someone.
- ___ 13. Sometimes, when embarrassed, I break out in a sweat, which annoys me greatly.
- ___ 14. Once in a while I laugh at a dirty joke.
- ___ 15. At times I am full of energy.
- ___ 16. At times I feel like swearing.
- ___ 17. I am troubled by attacks of nausea and vomiting.
- ___ 18. At times I feel like smashing things.
- ___ 19. I have never felt better in my life than I do now.
- ___ 20. It takes a lot of argument to convince most people of the truth.
- ___ 21. I have periods in which I feel unusually cheerful without any special reason.
- ___ 22. I am very seldom troubled by constipation.
- ___ 23. My sleep is fitful and disturbed.
- ___ 24. I have a good appetite.
- ___ 25. I am about as able to work as I ever was.
- ___ 26. I am in just as good physical health as most of my friends.
- ___ 27. During the past few years I have been well most of the time.
- ___ 28. I feel weak all over much of the time.
- ___ 29. I am neither gaining or losing weight.
- ___ 30. I have never vomited blood nor coughed up blood.
- ___ 31. I am happy most of the time.
- ___ 32. My daily life is full of things that keep me interested.
- ___ 33. I find it hard to keep my mind on a task or job.
- ___ 34. Sometimes without any reason or even when things are going wrong I feel excitedly happy, "on top of the world".
- ___ 35. I wish I could be as happy as others seem to be.
- ___ 36. I sometimes tease animals.
- ___ 37. I cry easily.
- ___ 38. I am certainly lacking in self-confidence.

MMPI "D" SCALE continued

- ___ 39. I certainly feel useless at times.
- ___ 40. I seldom worry about my health.
- ___ 41. I seem to be about as capable and smart as most others around me.
- ___ 42. Most nights I go to sleep without ideas or thoughts bothering me.
- ___ 43. I have had periods of days, weeks, or months when I couldn't take care of things because I couldn't "get going".
- ___ 44. I cannot understand what I read as well as I used to.
- ___ 45. I am afraid of losing my mind.
- ___ 46. My memory seems to be all right.
- ___ 47. I prefer to pass by school friends, or people I know but have not seen for a long time, unless they speak to me first.
- ___ 48. I don't seem to care what happens to me.
- ___ 49. I have difficulty in starting to do things.
- ___ 50. I work under a great deal of tension.
- ___ 51. I brood a great deal.
- ___ 52. Criticism or scolding hurts me terribly.
- ___ 53. I am a good mixer.
- ___ 54. I like to flirt.
- ___ 55. I dream frequently about things that are best kept to myself.
- ___ 56. I sometimes keep on at a thing until others lose patience with me.
- ___ 57. I have at times stood in the way of people who were trying to do something, not because it amounted to much, but because of the principle of the thing.
- ___ 58. I sweat very easily even on a cold day.
- ___ 59. I do not blame a person for taking advantage of someone who lays himself open to it.
- ___ 60. I do not have spells of hay fever or asthma.

Category Practice List

a Biblical name

Jesus
Mary
Jacob

a part of a boat

sail
oar
cabin
anchor

a sport played with a ball

baseball
volleyball
golf

a drug

heroin
morphine
methedrine
opium

a composer

Bach
Chopin

a philosopher

Plato

a cleaning instrument

vacuum
dustpan
brush
mop

a month of the year

March

an item of sports equipment

tennis racket

a piece of jewelry

necklace
earring
brooch

a planet

Jupiter
Mercury

a milk product

butter
yogurt

CATEGORIZING PRACTICE TASK

THE LAST THING WE ARE GOING TO DO TODAY IS TO PRACTICE SORTING SOME WORDS INTO CATEGORIES. I WILL GIVE YOU SOME CATEGORY WORDS, AND PLACE THEM HERE. THEN I WILL GIVE YOU THESE OTHER WORDS, WHICH BELONG WITH ONE OF THE CATEGORY WORDS. WHEN I TELL YOU TO BEGIN, GO AHEAD AND SORT THE WORDS INTO THE CATEGORIES YOU THINK THEY BELONG IN. TAKE AS MUCH TIME AS YOU NEED TO DO ALL OF THEM.

TIME: _____ ERRORS: _____

MEMORY PRACTICE

OK, NOW I'D LIKE TO SEE IF YOU CAN DO THE SAME IF
I DO IT WITHOUT THE CARDS. FOR EXAMPLE, IF I SAY THE
CATEGORY WORDS ARE PART OF A BOAT, THEN SAY THE WORDS
ARE SAIL, OAR, CABIN, AND ANCHOR, CAN YOU THEN TELL ME
THE WORDS WHICH ARE PART OF A BOAT?

_____, _____, _____, _____.

APPENDIX D

MEMORY TEST MATERIALS

FIRST IPC TASK: ADMINISTRATION

FIRST, WE ARE GOING TO DO SOMETHING SIMILAR TO WHAT YOU PRACTICED WITH MR. MCIVOR WHEN YOU WORKED WITH HIM. REMEMBER THOSE WORDS THAT YOU SORTED INTO CATEGORIES? WELL, I'M GOING TO READ YOU A LIST OF GROUPS OF FOUR WORDS. REPEAT EACH WORD AFTER ME AS I SAY IT. EACH GROUP OF FOUR WORDS WILL BE PRECEDED BY ANOTHER WORD OR PHRASE WHICH DESCRIBES THE WORDS TO BE REMEMBERED, BUT WHICH ITSELF DOESN'T NEED TO BE REMEMBERED. AFTER I READ THROUGH THE LIST OF 24 WORDS, 6 CATEGORIES, WITH 4 WORDS IN EACH CATEGORY, I WOULD LIKE TO SEE HOW MANY WORDS YOU CAN REMEMBER. LISTEN CAREFULLY, BECAUSE I CANNOT REPEAT THE WORDS. OKAY, ARE YOU READY? THE FIRST CATEGORY IS _____. THE FOUR WORDS ARE ____, ____, ____, AND _____. NEXT, _____: ____, ____, ____, ____.

List 1. IPC=4

a precious stone

pearl
topaz
amethyst
gem

a unit of time

century
millisecond
era
millenium

a relative

aunt
father
nephew
son

a type of reading material

textbook
comic book
journal
encyclopedia

a four-footed animal

cat
horse
cow
elephant

a kind of cloth

silk
rayon
satin
orlon

FIRST IPC TASK: NON-CUED RECALL

OK, NOW YOU TELL ME AS MANY WORDS AS YOU CAN
REMEMBER. YOU HAVE THREE MINUTES.

- | | |
|-----------|-----------|
| 1. _____ | 13. _____ |
| 2. _____ | 14. _____ |
| 3. _____ | 15. _____ |
| 4. _____ | 16. _____ |
| 5. _____ | 17. _____ |
| 6. _____ | 18. _____ |
| 7. _____ | 19. _____ |
| 8. _____ | 20. _____ |
| 9. _____ | 21. _____ |
| 10. _____ | 22. _____ |
| 11. _____ | 23. _____ |
| 12. _____ | 24. _____ |

FIRST IPC TASK: CUED RECALL

OKAY, NOW I WILL READ OFF THE CATEGORIES, AND YOU
TELL ME AS MANY WORDS AS YOU CAN REMEMBER. THE FIRST
CATEGORY IS _____. (WE HAVE THREE MINUTES AGAIN.)

A PRECIOUS STONE

A TYPE OF READING MATERIAL

A UNIT OF TIME

A FOUR-FOOTED ANIMAL

A RELATIVE

A KIND OF CLOTH

WECHSLER MEMORY SCALE

This test WILL TAKE US ABOUT 20 MINUTES TO COMPLETE.

I'M GOING TO ASK YOU SOME QUESTIONS AND ASK YOU TO DO
SOME THINGS. THE IDEA IS TO DO AS WELL AS YOU CAN.

FIRST I'LL ASK YOU SOME QUESTIONS LIKE THIS: HOW OLD
ARE YOU?

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10017



WECHSLER MEMORY SCALE FORM I

David Wechsler
Bellevue Hospital, New York

154

Score

I. Information _____
II. Orientation _____
III. Mental Control _____
IV. Memory Passages _____
V. Digits Total _____
VI. Vis. Reprod. _____
VII. Associate Lng. _____
Total Raw Score _____
Age Correction _____
Corrected Score _____
MQ (Table 3) _____

NAME _____ AGE _____ SEX _____

REFERRED FOR _____ DATE _____ EXAMINER _____

I. PERSONAL AND CURRENT INFO.	Score	II. ORIENTATION	Score
1. Age	_____	1. Year	_____
2. When born	_____	2. Month	_____
3. President of U.S.	_____	3. Day	_____
4. Before him	_____	4. Where now	_____
5. Governor	_____	5. City in	_____
6. Mayor	_____	Total	_____
Total	_____		

III. MENTAL CONTROL (Circle omits; cross out errors.)

1. (30") 20 19 18 17 16 15 14 13 12 11 10 9 8 7 6 5 4 3 2 1
2. (30") A B C D E F G H I J K L M N O P Q R S T U V W X Y Z
3. (45") 1 4 7 10 13 16 19 22 25 28 31 34 37 40

Time	Errors	Score	Total Score
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____

IV. LOGICAL MEMORY

(A) Anna Thompson/ of South/ Boston/
employed/ as a scrub woman/
in an office building/ reported/
at the City Hall/ Station/
that she had been held up/
on State Street/ the night before/
and robbed/ of fifteen dollars/.
She had four/ little children/ the rent/
was due/ and they had not eaten/
for two days/. The officers/
touched by the woman's story/
made up a purse/ for her/.

(B) The American/ liner/ New York/
struck a mine/ near Liverpool/
Monday/evening/. In spite of a blinding/
snowstorm/ and darkness/ the sixty/
passengers including 18/ women/
were all rescued/ though the boats/
were tossed about/ like corks/
in the heavy sea/. They were brought
into port/ the next day/ by a British/
steamer/.

(A) Number of Memories _____ (B) Number of Memories _____ Average Score = $\frac{(A+B)}{2} = \frac{\quad}{2} = \quad$

V. (A) DIGITS FORWARD	Score	(B) DIGITS BACKWARD	Score	
6-4-3-9	4	Draw a line	2-8-3	3
7-2-8-6	4	through any	4-1-5	3
		series failed.		
4-2-7-3-1	5	Circle score	3-2-7-9	4
7-5-8-3-6	5	for maximum	4-9-6-8	4
		number repeated		
6-1-9-4-7-3	6	correctly.	1-5-2-8-6	5
3-9-2-4-8-7	6		6-1-8-4-3	5
5-9-1-7-4-2-3	7		5-3-9-4-1-8	6
4-1-7-9-3-8-6	7		7-2-4-8-5-6	6
5-8-1-9-2-6-4-7	8		8-1-2-9-3-6-5	7
3-8-2-9-5-1-7-4	8		4-7-3-9-1-2-8	7

Forward Score _____ Backward Score _____ Digits Total _____

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C-1. C-2

old Part VII under on broken line before giving paper to subject for drawing in Part VI.

VI. VISUAL REPRODUCTION A _____ B _____ C-1 _____ C-2 _____ Total _____

VII. ASSOCIATE
LEARNING

First Presentation

Metal - Iron
Baby - Cries
Crush - Dark
North - South
School - Grocery
Rose - Flower
Up - Down
Obey - Inch
Fruit - Apple
Cabbage - Pen

First Recall Easy Hard

North _____
Fruit _____
Obey _____
Rose _____
Baby _____
Up _____
Cabbage _____
Metal _____
School _____
Crush _____
TOTAL _____

Second Presentation

Rose - Flower
Obey - Inch
North - South
Cabbage - Pen
Up - Down
Fruit - Apple
School - Grocery
Metal - Iron
Crush - Dark
Baby - Cries

Second Recall Easy Hard

Cabbage _____
Baby _____
Metal _____
School _____
Up _____
Rose _____
Obey _____
Fruit _____
Crush _____
North _____
TOTAL _____

Third Presentation

Baby - Cries
Obey - Inch
North - South
School - Grocery
Rose - Flower
Cabbage - Pen
Up - Down
Fruit - Apple
Crush - Dark
Metal - Iron

Third Recall Easy Hard

Obey _____
Fruit _____
Baby _____
Metal _____
Crush _____
School _____
Rose _____
North _____
Cabbage _____
Up _____
TOTAL _____

Easy 1) _____
2) _____
3) _____
(A) Total _____
A ÷ 2 _____
Hard 1) _____
2) _____
3) _____
(B) Total _____
SCORE
A
2 + B = _____

SECOND IPC TASK: ADMINISTRATION

NOW WE ARE GOING TO TRY ANOTHER LIST LIKE WE DID A LITTLE WHILE AGO. I'LL READ YOU A LIST OF GROUPS OF FOUR WORDS. YOU REPEAT EACH WORD AFTER ME. EACH GROUP OF WORDS WILL BE PRECEDED BY A DESCRIPTIVE WORD OR PHRASE WHICH NEED NOT BE REMEMBERED. AGAIN, THERE WILL BE 24 WORDS, 6 CATEGORIES, AND 4 WORDS IN EACH CATEGORY. ARE YOU READY? OKAY, THE FIRST CATEGORY IS _____:

_____, _____, _____, _____. _____: _____, _____, _____, _____.

List 2. IPC=4

a type of foot gear
shoe
boots
slipper
sandal

a building for religious services
church
temple
chapel
shrine

a fruit
apple
banana
lemon
cherry

a type of fuel
oil
coal
gas
wood

a type of vehicle
train
boat
car
airplane

a vegetable
carrot
corn
lettuce
bean

SECOND IPC TASK: CUED RECALL

OKAY, NOW I WILL READ OFF THE CATEGORIES, AND YOU TELL ME AS MANY WORDS AS YOU CAN REMEMBER. THE FIRST CATEGORY IS _____. (WE HAVE THREE MINUTES AGAIN.)

A TYPE OF FOOT GEAR

A TYPE OF FUEL

A RELIGIOUS BUILDING

A TYPE OF VEHICLE

A FRUIT

A VEGETABLE

SECOND IPC TASK: CUED RECALL

NOW I WILL AGAIN READ YOU THE CATEGORIES, AND YOU
TELL ME AS MANY WORDS AS YOU CAN REMEMBER. WE HAVE
THREE MINUTES AGAIN. THE FIRST CATEGORY IS_____.

A TYPE OF FOOT GEAR

A TYPE OF FUEL

A RELIGIOUS BUILDING

A TYPE OF VEHICLE

A FRUIT

A VEGETABLE

TRANSITION

OKAY, THAT FINISHES THIS PART OF TODAY'S WORK.
WE'RE HALF DONE. NOW LET'S MOVE INTO THE OTHER POSITION
WE ARE TO WORK IN TODAY. ARE YOU COMFORTABLE? WE'LL
START WITH ONE LIKE WE FIRST DID IN THE OTHER POSITION.

THIRD IPC TASK: ADMINISTRATION

ONCE AGAIN, I'LL PRESENT YOU WITH A LIST OF 6
GROUPS OF 4 WORDS EACH. YOU REPEAT EACH WORD AFTER ME.
AGAIN, EACH GROUP OF WORDS WILL BE PRECEDED BY A
DESCRIPTIVE PHRASE WHICH NEED NOT BE REMEMBERED. AS
BEFORE, THERE WILL BE 24 WORDS ALTOGETHER. ARE YOU
READY? OKAY, THE FIRST CATEGORY IS ____:____,____,____,____.

List 3. IPC=4

a unit of distance

kilometer
foot
decimeter
mile

a military title

lieutenant
major
commander
corporal

a part of the human body

foot
mouth
heart
nose

an alcoholic beverage

beer
champagne
wine
scotch

a country

Spain
Brazil
Russia
Germany

a sport

tennis
hockey
badminton
baseball

THIRD RECALL TASK: CUED RECALL

AGAIN, I WILL READ OFF THE CATEGORIES, AND YOU TELL
ME AS MANY WORDS AS YOU CAN REMEMBER. WE HAVE THREE
MINUTES AGAIN. THE FIRST CATEGORY IS _____.

A UNIT OF DISTANCE

AN ALCOHOLIC BEVERAGE

A MILITARY TITLE

A COUNTRY

A PART OF THE HUMAN BODY

A SPORT

THIRD IPC TASK: CUED RECALL

AGAIN, I WILL READ OFF THE CATEGORIES, AND YOU TELL
ME AS MANY WORDS AS YOU CAN REMEMBER. WE HAVE THREE
MINUTES AGAIN. THE FIRST CATEGORY IS _____.

A UNIT OF DISTANCE

AN ALCOHOLIC BEVERAGE

A MILITARY TITLE

A COUNTRY

A PART OF THE HUMAN BODY

A SPORT

WECHSLER MEMORY SCALE: SECOND TIME

NOW WE ARE GOING TO GO THROUGH SOME OF THE SAME QUESTIONS WE DID A LITTLE WHILE AGO, BUT SOME OF THEM ARE DIFFERENT. AGAIN, THE IDEA IS TO DO AS WELL AS YOU CAN. FIRST I'LL ASK YOU YOUR NAME AGAIN.

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WECHSLER MEMORY SCALE FORM II

166

Calvin P. Stone
Stanford University, California

David Wechsler
Bellevue Hospital, New York

Score

NAME _____ AGE _____ SEX _____

REFERRED FOR _____ DATE _____ EXAMINER _____

I. PERSONAL AND CURRENT INFO. Score

1. Age
2. When born
3. President of U.S.
4. Before him
5. Governor
6. Mayor

Total

II. ORIENTATION Score

1. Year
2. Month
3. Day
4. Where now
5. City in

Total

III. MENTAL CONTROL (Circle omits; cross out errors.)

1. (30") A B C D E F G H I J K L M N O P Q R S T U V W X Y Z
2. (30") 20 19 18 17 16 15 14 13 12 11 10 9 8 7 6 5 4 3 2 1
3. (45") 1 5 9 13 17 21 25 29 33 37 41 45 49 53

Time	Errors	Score	Total Score
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____

IV. LOGICAL MEMORY

(A) Dogs/ are trained/ to find/
the wounded/ in war time/. Police dogs/
are also trained/ to rescue/
drowning people/. Instead of running/
down to the water/ and striking out/
they are taught/ to make/ a flying leap/
by which they save/ many swimming strokes/
and valuable/ seconds of time/.
The European sheep dog/ makes the best/
police/ dog/.

(B) Many/ school/ children/ in northern/
France/ were killed/ or fatally hurt/
and others/ seriously injured/
when a shell/ wrecked/ the schoolhouse/
in their village/. The children/
were thrown/ down a hillside/
and across/ a ravine/ a long distance/
from the schoolhouse/. Only two/
children/ escaped uninjured/.

(A) Number of Memories _____ (B) Number of Memories _____ Average Score = $\frac{(A+B)}{2}$ = $\frac{\quad}{2}$ = _____

V. (A) DIGITS FORWARD Score

- | | | |
|-----------------|---|--|
| 2-8-6-1 | 4 | Draw a line
through any
series failed.
Circle score
for maximum
number repeated
correctly. |
| 5-3-9-4 | 4 | |
| 7-4-2-9-6 | 5 | |
| 8-5-1-6-4 | 5 | |
| 8-4-2-7-5-1 | 6 | |
| 7-2-9-5-3-6 | 6 | |
| 7-4-8-2-5-9-1 | 7 | |
| 8-3-9-6-1-5-2 | 7 | |
| 2-6-9-5-8-3-7-1 | 8 | |
| 3-7-2-9-4-1-5-8 | 8 | |

*5-9-4-8-2-7-3-1-6
*4-2-9-3-8-6-1-7-5

*5-2-7-1-8-4-9-3-6-2
*4-9-7-3-6-1-5-8-4-7

(B) DIGITS BACKWARD Score

- | | |
|---------------|---|
| 7-5-1 | 3 |
| 2-9-6 | 3 |
| 3-5-8-2 | 4 |
| 9-6-1-7 | 4 |
| 4-7-1-8-6 | 5 |
| 3-9-2-6-1 | 5 |
| 6-3-9-1-5-8 | 6 |
| 4-8-1-6-3-7 | 6 |
| 5-4-9-2-7-3-6 | 7 |
| 2-5-1-9-4-7-3 | 7 |

*2-7-1-5-3-9-6-4
*3-8-5-9-4-7-1-6

*9-1-6-4-8-3-7-5-2
*5-2-7-1-8-4-9-3-6

*NOT COUNTED IN SCORE IF USED.

Forward Score _____ Backward Score _____ Digits Total _____

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VI. 2

VI. 3-L; 3-R

 Fold Part VII under on broken line before giving paper to subject for drawing in Part VI.

VI. VISUAL REPRODUCTION 1 _____ 2 _____ 3-L _____ 3-R _____ Total _____

VII. ASSOCIATE LEARNING

First Presentation

Come - Go
 Lead - Pencil
 In - Although
 Country - France
 Dig - Guilty
 Lock - Door
 Jury - Eagle
 Murder - Crime
 Knife - Sharp
 Necktie - Cracker

Second Presentation

Knife - Sharp
 Jury - Eagle
 Country - France
 Lead - Pencil
 Necktie - Cracker
 Murder - Crime
 Lock - Door
 Come - Go
 Dig - Guilty
 In - Although

Third Presentation

Country - France
 Necktie - Cracker
 Murder - Crime
 Dig - Guilty
 Come - Go
 In - Although
 Lock - Door
 Jury - Eagle
 Lead - Pencil
 Knife - Sharp

<u>First Recall</u>	<u>Easy</u>	<u>Hard</u>	<u>Second Recall</u>	<u>Easy</u>	<u>Hard</u>	<u>Third Recall</u>	<u>Easy</u>	<u>Hard</u>	<u>Easy</u>	<u>1)</u>
Knife	_____	_____	Lock	_____	_____	Lead	_____	_____		2)
Lead	_____	_____	Dig	_____	_____	Lock	_____	_____		3)
Jury	_____	_____	Come	_____	_____	Necktie	_____	_____	(A) Total	_____
Country	_____	_____	Jury	_____	_____	Come	_____	_____	A ÷ 2	_____
In	_____	_____	Knife	_____	_____	Dig	_____	_____	Hard	1)
Murder	_____	_____	Country	_____	_____	Country	_____	_____		2)
Necktie	_____	_____	In	_____	_____	Jury	_____	_____		3)
Lock	_____	_____	Murder	_____	_____	Knife	_____	_____	(B) Total	_____
Come	_____	_____	Necktie	_____	_____	In	_____	_____		
Dig	_____	_____	Lead	_____	_____	Murder	_____	_____		
									<u>SCORE</u>	
TOTAL	_____	_____	TOTAL	_____	_____	TOTAL	_____	_____	$\frac{A}{2} + B =$	_____

LAST IPC TASK: ADMINISTRATION

WE ARE ALMOST DONE. THIS IS OUR LAST THING TO DO TODAY. ONCE AGAIN, I WILL PRESENT YOU WITH A LIST OF GROUPS OF WORDS. AGAIN, THERE ARE 24 WORDS, 6 CATEGORIES, AND 4 WORDS IN EACH CATEGORY. PLEASE REPEAT EACH WORD AFTER ME WHEN I SAY IT. YOU NEED NOT REMEMBER THE DESCRIPTIVE PHRASE WHICH PRECEDES EACH GROUP OF FOUR WORDS. ARE YOU READY? OKAY, THE FIRST CATEGORY IS

_____:_____,_____,_____,_____.

List 4. IPC=4

a color

- yellow
- pink
- grey
- gold

a kitchen utensil

- pan
- spatula
- stove
- mixer

a part of speech

- noun
- verb
- participle
- words

an article of furniture

- chair
- bed
- bureau
- bookcase

an elective office

- vice-president
- chairman
- alderman
- councilman

a type of human dwelling

- house
- hut
- mansion
- igloo

IPC TASK: NON-CUED RECALL

OK, NOW YOU TELL ME AS MANY WORDS AS YOU CAN
REMEMBER. YOU HAVE THREE MINUTES.

- | | |
|-----------|-----------|
| 1. _____ | 13. _____ |
| 2. _____ | 14. _____ |
| 3. _____ | 15. _____ |
| 4. _____ | 16. _____ |
| 5. _____ | 17. _____ |
| 6. _____ | 18. _____ |
| 7. _____ | 19. _____ |
| 8. _____ | 20. _____ |
| 9. _____ | 21. _____ |
| 10. _____ | 22. _____ |
| 11. _____ | 23. _____ |
| 12. _____ | 24. _____ |

LAST IPC TASK: CUED RECALL

AGAIN, I WILL READ OFF THE CATEGORIES, AND YOU TELL
ME AS MANY WORDS AS YOU CAN REMEMBER. WE HAVE THREE
MINUTES AGAIN. THE FIRST CATEGORY IS _____.

A COLOR

AN ARTICLE OF FURNITURE

A KITCHEN UTENSIL

AN ELECTIVE OFFICE

A PART OF SPEECH

A TYPE OF HUMAN DWELLING

APPENDIX E

CORRESPONDENCE MATERIALS

University of Manitoba
Faculty of Medicine
Winnipeg, Manitoba

Dear

A research project is being conducted through the University of Manitoba, Department of Psychology, and Faculty of Medicine, to investigate how different conditions effect the memory functions of persons with Parkinson's disease and persons with arthritis. Such research may provide useful answers to how memory works, and what might be done to help those persons who are having difficulties with their memories.

The project will be conducted by Mr. Daniel L. McIvor, a Ph.D. Candidate at the University of Manitoba. I will be providing some support for the project as it relates to arthritis, and Michael Newman, M.D. will be providing support as the project relates to parkinsonism.

The persons who volunteer to take part in this project will be interviewed by Mr. McIvor, and will take some brief question-and-answer tests. This will take about an hour, will be done at your convenience at your home, and you will be paid \$5.00 for your time. After the first testing, several persons will be tested a second time, which will also take about an hour, and for which they will also be paid \$5.00

I have given your name to Mr. McIvor as a possible participant in his project. He will be calling you soon to ask you if you would be willing to participate. While this would give you an opportunity to contribute to research in parkinsonism and arthritis, you should also feel free not to participate, and this choice would not in any way effect my continued care for you.

Sincerely yours,

Fletcher Baragar, M.D.

University of Manitoba
Rheumatic Disease Unit

Alvin E. Miller, Pastor,
Our Saviour's Lutheran Church
Winnipeg, Manitoba

Dear Church Member,

A research project is being conducted through the University of Manitoba, Faculty of Medicine, and Department of Psychology. The purpose of the project is to learn how different conditions effect the memory functions of persons with Parkinson's disease and persons with arthritis. Such research may provide useful answers to how memory works, and what might be done to help people who are having trouble with their memory.

This project will be conducted by Mr. Daniel L. McIvor, a Ph.D. Candidate at the University of Manitoba. Our church may be able to assist with this research project, and will also be able to earn some funds to be used by the church. Mr. McIvor needs about 40 persons who have neither arthritis nor parkinsonism, to serve as normal control subjects for his study. Persons between the ages of 40 - 70 are needed, preferably with 3 - 11 years of education in English. Some persons with more than 11 years of education may also be needed. Such persons should be healthy, able to walk unaided, able to see, hear, and use hands and fingers. Both men and women are needed.

The persons who volunteer to take part in this study will be interviewed by Mr. McIvor in their own homes at their own convenience, and will take some brief question - and - answer tests which take about an hour and do not require any detailed personal information. Each person who takes part in this project will be paid \$5.00 for his time, or \$5.00 will be credited to the church. After the first testing, several will be tested a second time, again in your own home at your own convenience, again for an hour, and again you will be paid \$5.00 for your time, or \$5.00 will be credited to the church.

If you are interested in helping with this project, please fill out the form at the bottom and return to me by April 13. I will pass the forms on to Mr. McIvor, who will then call you to make an appointment for the first testing. While such participation is voluntary, it is an opportunity to both contribute to the church, and to also contribute to ongoing medical-psychological research in Manitoba.

Sincerely yours,

Alvin E. Miller, Pastor

Please return this portion by April 13.

Name _____ Age _____

Address _____ Education _____

Phone number _____

FIRST PHONE CALL

HELLO, MR. _____,

THIS IS DAN MCIVOR SPEAKING. I AM THE PH.D. STUDENT AT THE UNIVERSITY OF MANITOBA WHO IS DOING THE RESEARCH PROJECT ON MEMORY FUNCTIONS AND PARKINSON'S DISEASE. DID YOU GET THE LETTER FROM DR. BARAGAR OR DR. NEWMAN LAST WEEK?

THE REASON I CALLED TODAY WAS TO ASK YOU IF YOU UNDERSTOOD WHAT THE PROJECT WAS ABOUT, AND IF YOU WOULD LIKE TO TAKE PART IN IT. HOW DO YOU FEEL ABOUT IT AT THIS POINT?

IF YES: GOOD, I'M GLAD. I THINK YOU WILL FIND IT AN INTERESTING EXPERIENCE. WHEN WOULD BE A CONVENIENT TIME FOR US TO WORK TOGETHER? OKAY, I WILL SEE YOU THEN. THANKS FOR YOUR HELP.

IF NO: OK, WELL THAT'S CERTAINLY OK. I HOPE I HAVEN'T TROUBLED YOU TOO MUCH SO FAR. THANK YOU JUST THE SAME.

POST-SCREENING INFORMATION SHEET

Thank you for taking part in this research project. Today, you have completed the first half of the study, which is called the "screening" part. This means that you and several other people will be given these same tests, and those who perform within a certain range of similarity will be "screened" to be included in the actual memory tests.

Thus, you may, or may not take any additional tests after today. It depends upon how other people do. It also depends on how many years of school they have had, and what their language skill is.

If you are selected for further testing, you will receive a call from my assistant, Mr. Steve Dunsiger. Steve is an experienced graduate student on the Ph.D. level in the Department of Psychology at the University of Manitoba. The reason I needed to hire Steve to do the memory tests was to keep me from influencing the results of the tests because of my own beliefs or theories, and because of my personal involvement with this project. Steve will call you and make an appointment to come and give you the memory tests.

If you are not selected, your screening test data will still be very useful to this research project. I will call you, and let you know that there will be no further calls or tests to take. When this project is completed in the spring, I will send you a letter describing the total project and the results we found.

If you are selected, Steve will come to your home at the appointed time and administer the memory tests. You will probably find these tests easier than the ones you took today. These tests will be divided into two parts. Half of the tests will be given to you while you are sitting up, in the same way as we worked today. The other half of the tests will be given to you while you are lying down, either on a couch or on your bed, whichever you feel most comfortable with. These tests will take about an hour altogether.

Steve will not know anything about you except your name and address. He will not know whether you have arthritis, parkinsonism, or neither. Please do not discuss your illness with him, because it is important that he be "blind" to this aspect of the research. When he has completed his testing, he will pay you \$5.00, and will have you sign a receipt. That will be all.

Again, in the spring, I will send you a letter describing the total project, and will tell you what we learned.

Thank you again for your very special help with this project. Without your assistance, we would not be able to advance in these field of research.

Daniel L. McIvor, Ph.D. Candidate

REJECT LETTER

Department of Psychology
University of Manitoba
Winnipeg, Manitoba

Dear

It was such a pleasure to work with you, and I really appreciate your helpfulness.

I feel that enough data is in at this point so that I am able to begin giving the second set of tests to some of the persons who took the first tests.

As I mentioned to you before, the purpose of the first tests was to help to select a group of people who had about the same level of education, same age, and could move with about the same ability. It was not a matter of passing or failing the tests as it was a matter of selecting people who were about the same.

As it turns out, you function somewhat above the level of most of the persons seen so far, so I will not require further testing help from you for this project. Thus, you need not take the memory tests I had mentioned before. The information you provided during our time together will be combined with that of others, and will be used to help find what some of the general relationships are between different conditions of health, age, education, and performance on the tests you took.

When the entire project is completed sometime next spring, I will be sending you a letter describing what the whole project was about, and what we learned from the study. I would like to thank you again for your help to contribute to the knowledge of how Parkinson's disease and arthritis effect how we think and remember.

Sincerely yours,

Daniel L. McIvor
Ph.D. Candidate

INTRODUCTORY CALL FROM ASSISTANT

HELLO, MR. _____.

THIS IS STEVE DUNSIGER SPEAKING. I AM A GRADUATE STUDENT AT THE UNIVERSITY OF MANITOBA, AND HAVE BEEN HIRED BY MR. MCIVOR TO DO SOME TESTING FOR HIS RESEARCH PROJECT. I CALLED YOU TODAY, BECAUSE I WILL BE THE PERSON WHO WILL WORK WITH YOU ON THE SECOND TESTING. I WILL BE COMING OUT TO YOUR HOME SOON AND WILL WORK WITH YOU FOR ABOUT AN HOUR, SORT OF LIKE YOU DID WITH MR. MCIVOR.

I THOUGHT TODAY WE WOULD MAKE AN APPOINTMENT FOR US TO GET TOGETHER AT YOUR HOME SOON. WHEN WOULD BE A GOOD TIME FOR YOU? OK, HOW ABOUT NEXT TUESDAY, NOVEMBER_____, AT____A.M., AT YOUR PLACE. IS THAT OK? GOOD. I'LL BE LOOKING FORWARD TO MEETING YOU THEN. BYE.

THIRD FOLLOW-UP LETTER

Dear _____,

This letter is to verify our conversation on the telephone on _____, when we decided to meet at your home on _____ at _____ a.m. to complete the second tests for Mr. McIvor's research project. As I said on the phone, it will take us about an hour to complete the tests. Half of the tests will be given as you sit at a table, while half of the tests will be given while you are lying down on a couch or bed. When we are finished, I will pay you \$5.00, just as Mr. McIvor did when he tested you the last time.

I am looking forward to seeing you then, on _____,
at _____ A.M.

Sincerely yours,

Graduate Student

WHAT THIS RESEARCH PROJECT WAS ALL ABOUT:

Thank you for taking part in this research project. With the completion of the memory tests today, you have finished your contribution to this research.

Your test results will now be given to another person who will score them. This person will not know anything about who took the tests, nor will she know whether the tests were taken sitting up or lying down. After this person scores the tests, the data will be run through a computer, and we will then be able to see what effect sleep, medications, age, illness, and position in space have on the ability to remember words.

The reason you were tested the first time was to help select a group of people with about similar abilities. The reason you were tested both sitting and lying today was to find out if this would have an effect on the ability to remember words. Sometimes we can do better if we are given a hint, but sometimes not. Since you were tested both sitting and lying, both with hints and without hints, this will help us learn if these different ways are important. Also, we tested some persons with Parkinson's disease, some with arthritis, some older persons, and some younger persons. This will help us learn what effects these different conditions might have on the ability to remember words.

When this project is completed, probably in the spring or summer, I will send you a letter describing the total project, and will tell you what we learned. This way, you will see how your work contributed to the whole project and how the results might be used to help people who are having difficulty with their memory.

Thank you so much for letting us come into your home and work with you. Your willingness to participate in this project is greatly appreciated.

If you have any questions about the project, feel free to call me at home or at the University of Manitoba.

Sincerely yours,

Daniel L. McIvor
Ph.D. Candidate

Department of Psychology
University of Manitoba
Winnipeg, Manitoba

Dear

Thank you so much for participating so willingly in this research project. Much important research into human problems could not be done without such support from the general public.

When all results are in, I will send you a brief summary of the project, along with a summary of the results and what the results might mean. I would imagine this last letter should come to you sometime this spring or summer.

Once again, thank you so much for your help with this project. It could not have been done without your time and efforts.

Best wishes to you and your family.

Sincerely yours,

Daniel L. McIvor
Ph.D. Candidate

APPENDIX F
MEMORY TEST RESULTS

TABLE 23

Means and Standard Deviations of WMS
Subtest Scores, by Position and Group

	Group							
	PD		A		O		N	
Variable	\bar{X}	s	\bar{X}	s	\bar{X}	s	\bar{X}	s
<u>Information:</u>								
Sit:	5.29	0.90	5.61	0.57	5.25	0.65	5.61	0.57
Recline:	5.43	0.74	5.57	0.57	5.36	0.62	5.68	0.55
<u>Orientation:</u>								
Sit:	4.64	0.68	4.50	0.57	4.64	0.49	4.82	0.39
Recline:	4.64	0.56	4.57	0.50	4.89	0.31	4.75	0.44
<u>Mental Control:</u>								
Sit:	5.54	2.38	6.71	2.31	6.29	1.92	6.79	2.10
Recline:	5.21	2.33	6.79	2.36	5.93	1.82	6.93	1.92
<u>Logical Memory:</u>								
Sit:	6.21	2.81	8.18	2.78	6.64	2.85	8.75	4.08
Recline:	6.71	2.36	7.61	2.73	6.21	2.95	9.29	4.27
<u>Digit Span:</u>								
Sit:	10.04	2.28	11.25	2.32	10.14	2.10	10.79	2.02
Recline:	9.68	2.36	11.21	2.23	10.00	1.59	9.29	4.27
<u>Associative Learning:</u>								
Sit:	10.00	2.88	12.82	3.17	10.54	3.64	13.89	4.25
Recline:	11.36	3.00	12.75	3.48	9.96	3.00	13.39	4.37

TABLE 24

Summary of Analysis of Variance on MQ data
by Group and Position

Source of Variance	Sum of Squares	df	Mean Square	F
Groups	7616.91	3	2538.97	6.39*
Error	42881.61	108	397.05	
Position	6.11	1	6.11	0.10
Groups x Position	118.97	3	39.66	0.67
Error	6440.33	108	59.63	

*p < .001

TABLE 25

Summary of One-Way Analysis of Variance of MQ Scores

Source of Variance	Sum of Squares	df	Mean Square	F
Between Groups	3808.00	3	1269.33	6.39*
Within Groups	21441.00	108	198.53	
Total	25249.00	111		

*p < .01

TABLE 26

Summaries of One-Way Analyses of Variance on
WMS Subtests, Summed Across Positions in Space
and Showing Effect of Diagnostic Group

Subtest	Source of Variance	Sum of Squares	df	Mean Square	F
<u>Information:</u>	Between Groups	2.36	3	0.79	2.13
	Within Groups	40.05	108	0.37	
	Total	42.42	111		
<u>Orientation:</u>	Between Groups	1.15	3	0.38	1.81
	Within Groups	22.85	108	0.21	
	Total	24.00	111		
<u>Mental Control:</u>	Between Groups	39.27	3	13.09	3.56*
	Within Groups	397.67	108	3.68	
	Total	436.95	111		
<u>Logical Memory:</u>	Between Groups	130.74	3	43.58	5.36**
	Within Groups	878.74	108	8.14	
	Total	436.95	111		
<u>Digit-Span:</u>	Between Groups	33.69	3	11.23	2.84
	Within Groups	426.77	108	3.95	
	Total	460.46	111		
<u>Associative Learning:</u>	Between Groups	224.61	3	74.87	7.77***
	Within Groups	1041.00	108	9.64	
	Total	1265.61	111		

*p < .05

**p < .01

***p < .001

TABLE 27

Post-hoc Comparisons (Dunn Multiple t)
Between Groups on Significant WMS Subtests,
Summed Across Positions in Space

Subtest	Comparison	df	t-value
<u>Mental Control:</u>	PD - A	108	-2.68*
	PD - O	108	-1.42
	PD - N	108	-2.89*
	A - O	108	1.25
	A - N	108	-0.21
	O - N	108	-1.46
<u>Logical Memory:</u>	PD - A	108	-1.87
	PD - O	108	0.05
	PD - N	108	-3.35*
	A - O	108	1.92
	A - N	108	-1.48
	O - N	108	-3.40*
<u>Associative Learning:</u>	PD - A	108	-2.54
	PD - O	108	1.52
	PD - N	108	-3.57*
	A - O	108	3.01*
	A - N	108	-1.03
	O - N	108	-4.04*

*p < .008

TABLE 28, Part 1

Raw Data on Parkinson Persons on WMS
in Sitting Position

S#	Info.	Orient.	M. Con.	Logical	Digit	Assoc.	MQ
1	6	5	8	6	13	9	105
2	5	5	7	4	12	9	99
3	5	4	4	3	10	11	84
4	5	5	4	8	9	9	96
5	5	5	6	8	8	10	99
6	6	5	9	8	15	13	112
7	6	4	3	6	11	10	96
8	6	4	2	11	11	16	106
9	5	5	9	10	10	16	110
10	6	5	6	7	10	8	93
11	6	4	3	4	9	7	81
12	6	5	4	12	11	15	110
13	5	5	8	6	12	12	132
14	5	5	9	7	14	10	114
15	4	5	7	4	12	11	94
16	6	5	6	7	10	8	97
17	6	5	5	2	9	7	87
18	6	4	5	4	9	8	89
19	6	5	2	5	7	9	80
20	6	5	7	7	9	15	112
21	3	4	5	4	10	7	84
22	6	5	7	7	8	9	99
23	5	5	4	5	11	8	92
24	6	5	7	6	10	10	99
25	4	2	0	0	3	5	59
26	5	5	5	6	8	7	89
27	5	5	9	12	10	12	122
28	3	4	4	5	10	9	87

TABLE 28, Part 2

Raw Data on Parkinson Persons on WMS
in Reclining Position

S#	Info.	Orient.	M. Con.	Logical	Digit	Assoc.	MQ
1	6	5	8	8	13	12	116
2	5	5	7	3	11	13	100
3	5	4	5	4	9	13	90
4	5	5	3	11	10	13	108
5	5	5	3	13	7	10	100
6	6	5	7	6	15	11	100
7	6	4	5	5	10	16	106
8	6	4	3	6	11	13	94
9	5	5	9	9	13	12	106
10	6	5	7	11	9	12	106
11	6	4	5	4	8	6	81
12	6	5	4	11	11	16	110
13	5	5	4	7	10	13	100
14	5	5	9	8	12	16	126
15	5	4	5	7	10	9	90
16	5	5	3	4	9	9	87
17	6	5	6	6	4	9	89
18	6	4	2	3	9	10	86
19	6	5	2	2	8	8	74
20	6	5	5	6	7	13	99
21	3	4	3	4	8	8	79
22	6	5	7	7	10	15	114
23	6	5	7	7	11	12	110
24	6	5	6	12	11	12	112
25	5	3	0	4	5	3	63
26	5	5	5	6	8	11	96
27	6	5	9	8	11	13	120
28	4	4	7	6	11	10	99

TABLE 29, Part 1

Raw Data on Arthritis Persons on WMS
in Sitting Position

S#	Info.	Orient.	M. Con.	Logical	Digit	Assoc.	MQ
1	6	4	9	6	8	15	110
2	6	5	2	6	9	12	96
3	6	5	5	5	10	13	101
4	5	4	8	11	10	10	103
5	6	5	7	8	9	12	105
6	4	5	9	11	11	11	114
7	6	5	9	9	15	14	131
8	6	5	9	10	10	8	110
9	6	5	9	11	9	19	116
10	6	5	6	5	14	5	97
11	6	5	9	10	12	18	132
12	6	4	7	7	8	16	110
13	6	4	4	8	8	16	100
14	6	4	8	16	13	11	116
15	5	3	6	10	12	13	112
16	5	4	8	7	10	16	106
17	6	4	6	4	10	16	103
18	5	5	5	8	11	10	99
19	5	4	4	7	10	13	100
20	6	4	2	6	12	10	90
21	5	4	7	6	12	11	99
22	6	5	9	12	15	13	126
23	5	4	2	3	8	13	87
24	6	4	8	8	14	12	105
25	5	5	6	8	12	17	118
26	5	5	6	11	14	15	120
27	6	5	9	9	14	11	120
28	6	5	9	7	15	9	118

TABLE 29 , Part 2

Raw Data on Arthritis Persons on WMS
in Reclining Position

S#	Info.	Orient.	M. Con.	Logical	Digit	Assoc.	MQ
1	6	4	9	10	10	16	126
2	6	5	2	5	9	13	96
3	6	4	5	4	11	19	112
4	5	4	5	8	9	14	84
5	6	5	6	8	8	8	94
6	4	5	9	11	12	13	124
7	6	5	9	9	14	16	140
8	6	5	9	12	11	16	140
9	6	5	8	13	9	10	100
10	6	5	7	6	12	11	108
11	5	5	9	14	13	12	126
12	6	4	5	8	8	10	97
13	6	4	9	8	7	10	96
14	6	4	9	8	13	10	106
15	6	4	8	3	13	14	110
16	5	5	8	7	11	20	129
17	6	4	2	9	10	11	96
18	5	5	3	5	12	10	93
19	5	4	8	5	11	13	106
20	6	5	3	5	11	7	84
21	5	5	9	7	13	6	99
22	6	5	9	7	15	11	106
23	5	4	4	6	7	17	100
24	6	4	6	8	15	14	103
25	5	5	8	6	13	11	110
26	5	5	6	4	13	17	100
27	6	5	9	7	13	16	124
28	5	4	6	10	11	12	110

TABLE 30, Part 1

Raw Data on Older Persons on WMS
in Sitting Position

S#	Info.	Orient.	M. Con.	Logical	Digit	Assoc.	MQ
1	5	4	6	4	7	6	81
2	6	5	8	7	6	4	89
3	6	4	7	4	11	10	99
4	6	5	7	7	9	10	101
5	6	4	5	9	10	14	110
6	6	5	9	4	13	8	106
7	6	4	7	9	11	8	106
8	6	4	9	9	13	14	126
9	5	5	3	6	12	14	106
10	5	5	7	7	9	6	94
11	5	5	7	10	12	7	106
12	6	4	7	8	10	13	110
13	4	4	8	6	11	16	112
14	5	5	8	6	9	11	101
15	5	5	5	2	8	9	86
16	5	5	7	7	7	19	114
17	5	5	7	10	10	17	124
18	4	4	5	14	14	11	120
19	5	5	7	4	9	9	94
20	5	5	6	4	11	10	97
21	5	4	5	12	9	12	108
22	5	5	9	4	13	9	106
23	5	5	6	4	13	11	101
24	5	5	2	3	9	13	90
25	6	5	5	10	8	10	101
26	5	5	4	8	8	7	90
27	4	4	2	4	10	5	77
28	6	5	8	8	12	12	118

TABLE 30, Part 2

Raw Data on Older Persons on WMS
in Reclining Position

S#	Info.	Orient.	M. Con.	Logical	Digit	Assoc.	MQ
1	6	5	5	5	10	8	94
2	5	5	5	7	8	7	90
3	6	5	7	6	11	10	106
4	6	5	8	3	9	9	96
5	6	4	8	4	10	9	97
6	5	4	9	7	12	9	106
7	6	5	6	7	10	9	100
8	6	5	6	9	12	14	120
9	6	5	7	9	8	9	101
10	5	5	8	7	11	8	101
11	6	5	5	10	10	8	101
12	6	5	8	6	9	13	108
13	6	5	5	3	10	9	92
14	4	5	7	5	11	11	100
15	5	5	7	4	11	6	92
16	5	5	5	3	8	14	96
17	5	5	6	10	10	16	120
18	5	4	6	8	11	12	106
19	5	5	5	2	9	12	92
20	5	5	5	5	9	9	92
21	6	5	5	15	11	13	126
22	5	5	5	6	8	8	90
23	5	5	6	7	14	9	106
24	5	5	2	2	10	5	77
25	5	5	5	9	9	13	106
26	5	5	5	7	8	8	92
27	4	5	1	2	8	5	70
28	6	5	9	6	13	16	126

TABLE 31, Part 1

Raw Data on Normal Persons on WMS
in Sitting Position

S#	Info.	Orient.	M. Con.	Logical	Digit	Assoc.	MQ
1	6	5	7	12	14	21	137
2	4	5	4	5	11	6	87
3	6	5	9	8	14	17	124
4	6	5	9	15	14	20	143
5	6	5	7	13	11	17	120
6	6	5	9	14	8	15	124
7	6	5	9	5	11	16	105
8	6	5	5	8	8	17	112
9	5	5	6	4	12	13	101
10	5	4	9	13	11	15	124
11	5	5	8	6	12	13	108
12	6	5	8	7	10	16	116
13	6	5	9	10	11	19	137
14	6	5	9	5	9	11	99
15	5	4	7	7	12	19	112
16	6	5	4	8	11	14	110
17	6	5	5	10	11	17	116
18	6	4	9	4	10	6	94
19	6	5	4	15	11	13	116
20	5	5	8	7	10	11	97
21	6	5	9	10	13	16	140
22	5	4	6	18	15	16	143
23	5	4	3	5	8	9	86
24	6	5	6	4	8	10	94
25	6	5	6	2	8	10	90
26	6	5	2	8	8	7	89
27	5	5	7	9	11	8	105
28	5	5	6	13	10	17	129

TABLE 31, Part 2

Raw Data on Normal Persons on WMS
in Reclining Position

S#	Info.	Orient.	M. Con.	Logical	Digit	Assoc.	MQ
1	6	4	9	15	15	21	143
2	4	5	5	4	8	12	92
3	6	5	9	16	14	18	143
4	6	5	7	14	13	19	140
5	6	5	9	18	11	20	143
6	6	5	9	10	10	14	116
7	6	5	8	6	12	13	100
8	6	5	5	10	12	17	126
9	6	5	8	5	10	12	103
10	5	4	9	11	9	11	105
11	5	5	5	9	13	12	108
12	6	5	7	8	12	12	110
13	6	5	6	13	11	19	137
14	6	5	9	8	7	13	103
15	6	5	9	11	14	18	137
16	5	4	7	6	10	10	99
17	6	5	7	12	8	13	110
18	6	4	5	2	8	7	81
19	6	5	8	14	11	15	129
20	6	5	8	7	10	7	92
21	6	5	7	11	11	17	135
22	5	4	9	13	15	19	143
23	5	4	7	3	9	9	90
24	6	5	6	5	9	8	92
25	6	5	3	7	9	8	92
26	6	4	2	2	8	9	80
27	5	5	6	11	11	7	105
28	5	5	5	9	11	15	114

TABLE 32

Summary of Analysis of Variance for #C by Group,
Position, and Recall Condition

Source of Variance	Sum of Squares	df	Mean Square	F
Groups (G)	1696.02	3	565.34	8.21*
Error	7437.84	108	68.87	
Position (P)	0.40	1	0.40	0.03
G x P	47.13	3	15.71	1.15
Error	1475.08	108	13.66	
Recall Condition (C)	6159.03	3	2053.01	276.63*
G x C	100.10	9	11.12	1.50
Error	2404.54	324	7.42	
P x C	37.07	3	12.36	1.55
G x P x C	85.35	9	9.48	1.19
Error	2581.44	324	7.97	

*p < .001

TABLE 33

Summary of One-Way Analysis of Variance on #C in
Non-cued Recall Condition,
Summed Across Positions in Space

Source of Variance	Sum of Squares	df	Mean Square	F
Between Groups	198.17	3	66.06	3.57*
Within Groups	1996.25	108	18.48	
Total	2194.42	111		

*p < .05

TABLE 34

Post-hoc Comparisons (Dunn Multiple t)
Between Groups on #C in Non-cued Condition,
Summed Across Positions in Space

Comparison	df	t-value
PD - A	108	-1.83
PD - O	108	0.09
PD - N	108	-2.58
A - O	108	1.93
A - N	108	-0.75
O - N	108	-2.67*

*Alpha level set at $.05/6 = .008$; obtained
p = .009.

TABLE 35

Summary of One-Way Analysis of Variance on #C in
Cued Recall Condition, Summed
Across Positions in Space

Source of Variance	Sum of Squares	df	Mean Square	F
Between Groups	252.85	3	84.28	5.52*
Within Groups	1650.01	108	15.28	
Total	1902.86	111		

*p < .01

TABLE 36

Post-hoc Comparisons (Dunn Multiple t)
Between Groups on #C in Cued Recall Condition
Summed Across Positions in Space

Comparison	df	t-value
PD - A	108	-2.26
PD - O	108	-0.27
PD - N	108	-3.49*
A - O	108	1.98
A - N	108	-1.23
O - N	108	-3.21*

*p < .008

TABLE 37, Part 1

Raw Data on Parkinson Persons on
Memory Tasks in Sitting Position

Recall Condition ^a		
Non-cued Recall		Cued Recall
S#	#C	#C
1	7	15
2	5	15
3	10	13
4	7	7
5	7	11
6	14	16
7	4	3
8	3	10
9	4	10
10	11	15
11	10	11
12	12	21
13	8	13
14	6	14
15	2	15
16	15	8
17	8	11
18	10	20
19	11	13
20	7	15
21	3	7
22	7	13
23	4	12
24	11	22
25	0	3
26	2	17
27	3	13
28	6	11

^aOnly the first recall trial
results are presented.

TABLE 37, Part 2

Raw Data on Parkinson Persons on
Memory Tasks in Reclining Position

Recall Condition ^a		
Non-Cued Recall		Cued Recall
S#	#C	#C
1	0	11
2	5	14
3	8	14
4	5	7
5	8	14
6	13	13
7	8	17
8	15	16
9	9	17
10	8	15
11	7	16
12	9	20
13	12	14
14	12	15
15	0	10
16	10	11
17	1	14
18	0	10
19	6	12
20	14	12
21	0	11
22	3	13
23	4	15
24	7	17
25	0	5
26	4	11
27	5	14
28	2	14

^aOnly the first recall trial
results are presented.

TABLE 38, Part 1

Raw Data on Arthritis Persons on
Memory Tasks in Sitting Position

Recall Condition ^a		
Non-cued Recall		Cued Recall
S#	#C	#C
1	12	16
2	5	11
3	2	15
4	9	20
5	13	18
6	9	21
7	15	18
8	11	5
9	13	18
10	10	16
11	12	18
12	9	18
13	11	15
14	7	16
15	5	15
16	10	14
17	10	7
18	6	13
19	7	11
20	1	13
21	6	11
22	11	18
23	3	13
24	11	18
25	16	16
26	14	19
27	7	11
28	11	16

^aOnly the first recall trial
results are presented.

TABLE 38, Part 2

Raw Data on Arthritis Persons on
Memory Tasks in Reclining Position

Recall Condition ^a		
Non-cued Recall		Cued Recall
S#	#C	#C
1	15	16
2	4	14
3	13	12
4	16	21
5	9	19
6	16	15
7	10	24
8	5	14
9	13	19
10	12	11
11	14	19
12	15	22
13	7	14
14	5	18
15	9	11
16	14	16
17	5	16
18	12	10
19	7	14
20	11	10
21	7	9
22	9	16
23	7	16
24	15	16
25	7	13
26	15	19
27	6	15
28	6	16

^aOnly the first recall trial
results are presented.

TABLE 39 , Part 1

Raw Data on Older Persons on
Memory Tasks in Sitting Position

Recall Condition ^a		
Non-cued Recall		Cued Recall
S#	#C	#C
1	6	13
2	10	11
3	7	13
4	12	13
5	10	14
6	0	15
7	13	16
8	13	16
9	14	16
10	14	11
11	6	12
12	10	16
13	9	14
14	9	16
15	8	10
16	6	9
17	9	15
18	6	10
19	4	11
20	1	12
21	2	14
22	0	11
23	8	18
24	5	7
25	5	15
26	1	10
27	0	6
28	6	18

^aOnly the first recall trial
results are presented.

TABLE 39, Part 2

Raw Data on Older Persons on
Memory Tasks in Reclining Position

Recall Condition ^a		
Non-cued Recall		Cued Recall
S#	#C	#C
1	11	3
2	4	16
3	6	13
4	5	14
5	4	16
6	7	10
7	6	18
8	7	13
9	13	19
10	5	14
11	6	18
12	7	17
13	3	13
14	4	14
15	5	14
16	3	12
17	9	14
18	2	13
19	0	17
20	3	15
21	6	16
22	7	19
23	2	16
24	5	12
25	5	16
26	0	9
27	0	11
28	7	15

^aOnly the first recall trial
results are presented.

TABLE 40, Part 1

Raw Data on Normal Persons on
Memory Tasks in Sitting Position

Recall Condition ^a		
Non-cued Recall		Cued Recall
S#	#C	#C
1	11	21
2	3	9
3	21	22
4	15	22
5	13	23
6	7	17
7	18	19
8	9	19
9	7	14
10	17	14
11	14	18
12	11	15
13	13	21
14	10	18
15	18	14
16	8	12
17	6	18
18	3	13
19	13	17
20	11	16
21	7	14
22	10	20
23	8	15
24	4	11
25	5	12
26	4	19
27	3	7
28	13	16

^aOnly the first recall trial
results are presented.

TABLE 40, Part 2

Raw Data on Normal Persons on
Memory Tasks in Reclining Position

Recall Condition ^a		
Non-cued Recall		Cued Recall
S#	#C	#C
1	14	22
2	3	12
3	9	20
4	15	21
5	23	23
6	13	15
7	9	17
8	9	17
9	12	17
10	11	19
11	12	13
12	16	20
13	18	19
14	13	16
15	14	20
16	8	18
17	4	9
18	4	12
19	17	13
20	5	9
21	8	17
22	13	12
23	3	17
24	2	20
25	3	16
26	3	15
27	12	15
28	7	14

^aOnly the first recall trial
results are presented.