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The Use of Repeated Acquisition as a Technique for Establishing a Behavioral Baseline with Victims of Alzheimer's Disease

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THE USE OF REPEATED ACQUISITION AS A TECHNIQUE
FOR ESTABLISHING A BEHAVIORAL BASELINE WITH
VICTIMS OF ALZHEIMER'S DISEASE

by

Michelle D. Stone

A Thesis
Submitted to the
Faculty of The Graduate College
in partial fulfillment of the
requirements for the
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Department of Psychology

Western Michigan University
Kalamazoo, Michigan
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THE USE OF REPEATED ACQUISITION AS A TECHNIQUE FOR ESTABLISHING A BEHAVIORAL BASELINE WITH VICTIMS OF ALZHEIMER’S DISEASE

Michelle D. Stone, M.A.
Western Michigan University, 1986

Three subjects suspected as Alzheimer’s Disease (AD) victims were trained on a behavioral chain of three sets of three poker chips, each set differing in color and position, to investigate subtle changes in learning and performance behaviors over time. For each session the subjects learned a new three-response sequence with a marked poker chip serving as the correct response for each set. A within-session learning curve resulted for all three subjects with the errors in the learning trial (LT) exceeding the errors for the performance trials (PT1 and PT2). Only Subject III demonstrated a stable pattern of learning, with the number of errors reaching a steady state from session to session. Procedural rules were enforced to foster the discrimination of the correct stimulus within each set of the behavioral chain. Between-trial reinforcement "breaks" and individualized reinforcers were delivered contingent upon responding during the sessions. The results of the study indicated that the repeated acquisition technique could be used with suspected AD subjects and has demonstrated that the technique was sensitive to subtle changes in behavior over time.
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This thesis is dedicated with my deepest appreciation and affection to Mr. George Treudt and Mrs. Harriet Weaver, and to the loving memory of Mr. Carl Wilcox.

Michelle D. Stone
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ESTABLISHING A BEHAVIORAL BASELINE WITH VICTIMS OF ALZHEIMER'S
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Alzheimer's Disease (AD), an organic deterioration of the brain, has been called "the disease of the century" as it is estimated to afflict approximately 1.5 to 2.5 million persons in the United States alone (Butler & Emr, 1983; U.S. Department of Health & Human Services, 1983). Presently there is no established treatment available to cure, reverse, or halt the progression of this devastating disorder. The life-altering effects of AD have become so widely recognized that the Department of Health and Human Services assembled a Special Task Force to investigate the current state of medical knowledge of AD and provide recommendations for the needed future research "that would put the (AD) victims, their loved ones, and medical science on the pragmatic and scientific road to progress" (U.S. Department of Health and Human Services, [DHHS] 1983, p. iii).

In a special declaration to the Special Secretary's Task Force on AD in 1983, President Ronald Reagan proclaimed the month of November as National Alzheimer's Disease Month by recognizing that:

Science and clinical medicine are striving to improve our understanding of what causes Alzheimer's Disease and how to treat it successfully. Right now, research is the only hope for victims and families. (DHHS, 1983, p. ix).

The Task Force developed specific recommendations for future basic and applied research in all of the biological, clinical, behavioral, and
social science fields. These research recommendations are extensive and address nine main areas. For the purpose of this research, two specific recommendations in two main areas deserve mention. First, for research on the clinical course of this disease, the Task Force has recommended the development of longitudinal assessment instruments capable of reliably measuring behavioral and biological changes over multiple points in time. Second, in the area of treatment, the recommendation is to establish more sensitive measures of cognitive, affective, somatic, and other changes for use in drug outcome studies.

Generally, the purpose of this study is to combine these two recommendations by investigating an assessment instrument which has been demonstrated to be sensitive to subtle changes in behavior over time, and to apply this assessment instrument to AD victims. In addition, since a chronic disease such as AD affects its victims differently, this study will investigate changes in behavior for subjects in their natural setting.

In the remainder of this chapter, we will discuss Alzheimer's disease: (a) the pathological evidence, clinical symptomatology and pharmacologic interventions; (b) operant conditioning, neurological research, applied behavioral research and behavioral pharmacology; (c) the repeated acquisition technique; and (d) the rationale for the present study.

Alzheimer's Disease

Since the cause of AD is unknown, data concerning its prevalence varying, the estimates of incidence uncertain, differential diagnosis
impossible, and the knowledge concerning its clinical course limited, major gaps are left in treatment, both in efforts to assist AD victims and their families, and in outlining desired systems for care and habilitation. Therefore, a brief overview detailing what is known at present about AD is presented.

Pathology

AD is not a recent discovery; however, its chronic and debilitating nature has recently become the subject of much research. Since the initial description of AD in 1906 (Wells, 1978) as a malady which affects its victim's ability to reason, retain and recall events from his/her environment, two independent approaches have emerged to advance our understanding of the pathophysiology of this disease. The first of these approaches is neurophysiological.

Neurophysiological descriptions of AD include gross atrophy of the brain with widening of the cortical sulci and enlarged ventricles, and neuronal loss in the amygdala, hippocampus and cerebral cortex. In addition, researchers have analyzed two other major pathologies associated with AD. These include neurofibrillary tangles and neuritic plaques, both commonly found in the hippocampus and cortex regions of the brain (Bondareff, 1984; Butler & Emr, 1983; Terry & Davies, 1980; and Perry & Perry, 1985). The severity of the illness has been shown to correlate with the number of neuritic plaques and the abundance of tangles in the cerebral cortex.

The second approach to studying the pathophysiology of AD is neurochemical. From this field of research a number of studies have
demonstrated major disruptions in the cholinergic neurotransmitter system (Bartus, Dean, Beer & Lippa, 1982; Davis et al., 1983; Terry, Davies, DeTeresa & Katzman, 1981; Spar, 1984; Perry & Perry, 1985). While several reports suggest that other neurotransmitter systems may also be abnormal with AD (Bondareff, 1984; Comfort, 1984; Spar, 1984), the presynaptic cholinergic deficiency in AD is most dramatically marked by the reduction in both choline acetyltransferase (CAT) and acetylcholinesterase (ACHE). Both of these enzymes affect the synthesis and disposition of acetylcholine. Acetylcholine is the chemical needed for the transmission of nerve signals, most specifically affecting behavioral functions such as memory and learning (Bartus et al., 1982; Spar, 1984; Whitehouse et al., 1983).

Neuropathological and neurochemical investigations, while independent, combine to show that the degree of reduction in CAT activity with AD is related both to the density of neuritic plaques and the severity of clinically apparent dementia. This relationship suggests a link between the neurochemical alterations and specific qualitative neuropathological features of this disease (Spar, 1984; Whitehouse et al., 1983).

Advances in the knowledge of pathophysiology of AD have, in part, derived from advances in current medical technology (e.g., computerized tomography [CAT scan], positron tomography [PET scan], and nuclear magnetic resonance tomography [NMR scan] (Benson, 1984) as well as from autopsy of the diseased brain tissue. Diagnosis of AD in vivo is usually approached as a diagnosis of exclusion whenever a specific cause for the intellectual impairment has not otherwise been found. Most

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often the diagnosis is only suspected; an autopsy is needed to verify the existence of the pathologies outline above.

Basing a diagnosis on the pathological characteristics alone, however, often leads to the risk of over diagnosis, since all unrecognized dementing disorders are included in the AD category. Therefore, it is advisable to consider AD as a clinicopathologic entity; that is, a disease with clinical as well as pathological characteristics (Jervis, 1972). Once AD is so considered and carefully evaluated on both fronts, the diagnosis will be suitable for disease-specific treatment plans, management, and realistic prognosis (Cummings, 1984).

Clinical Symptomatology

The clinical symptoms of AD are far less consistent and definite than are the pathophysiologies just discussed. Research into the clinical aspects of AD is also less abundant.

The third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III), refers to AD as "primary degenerative dementia of the Alzheimer's type," and defines this malady as "usually insidious in onset and slowly, but relentlessly, progressive to death over a period of several years" (DSM-III, 1983, p. 111).

According to DSM-III criteria, AD can be viewed as consisting of three components: (a) memory and other cognitive impairment—the core features of dementia; (b) functional and structural impairment of the brain; and (c) behavioral manifestations that affect the patient's ability for self-care, interpersonal relationships, and adjustment in the community (DHHS, 1983). While these three components are probably
interconnected, they do not necessarily parallel each other in clinical evaluations of the AD victim. For example, in some cases memory loss may be severe, with only minimal evidence of other cerebral dysfunctions such as loss of verbal skills or self-care skills. Although the severity of cognitive deficit is usually correlated with the severity of brain changes, there remain observable discrepancies among these variables making it difficult to distinguish AD, particularly in the early course of the disease, from multi-infarct dementia, depression, or other dysfunctional states (Cummings, 1983; DHHS, 1984; LaRue, 1984; Wells, 1978).

Dementia is the most classic of the clinical symptoms representing a wide variety of central nervous system disorders, including AD. Dementia is defined as an acquired persistent compromise in intellectual function with impairments in at least three of the following spheres of mental activity: language, memory, visuospatial skills, and cognitions such as abstraction, judgement and logic (Cummings, 1984). This working definition of the dementia syndrome is based on a neuropsychiatric mental status examination and traditional psychological testing procedures (Cummings, 1984; LaRue, 1984; Wells, 1978).

Memory loss and chronic confusion remain the first and most outstanding clinical symptom of AD (Burnside, 1979; Jervis, 1972; Wolanin & Phillips, 1981). Much of the traditional psychological research has emphasized the accompanying memory dysfunction associated with AD. In this context, memory "refers to the retention and recall of specific events which occurred at a given time in a given place" (Craik, 1977, p. 285). Various models of memory have emerged from the literature in
support of this popular definition. Distinctions are made in the literature between (a) episodic memory (recall of more general knowledge) and semantic memory (recall of specific events); (b) echoic memory (auditory stimulus events held in an auditory sensory site) and iconic memory (visual stimulus events held in a visual sensory store); and (c) long-term memory (a permanent storage site) and short-term memory (a limited-capacity storage) (Craik, 1977; Bartus et al., 1982). However, within the clinical literature on AD, the memory model employed in clinical research seems to relate more to the preference of the researcher than to the absolute clinical nature of AD. It therefore remains unclear which, if any, memory model is most relevant to investigating the cognitive dysfunctions observed in AD subjects.

Other prominent symptomatologies identify disturbances in behaviors. Rosin's study investigated, among other things, the extent of behavior problems associated with AD (Rosin, 1977). The results indicated that behavioral disturbances such as shouting, wandering, incontinence, and degeneration of self-care were found in a high proportion of the AD subjects studied. In addition, communication disorders, perceptual impairment, distractability, disorientation to time and place, aggression, tremors, stimuli intergration problems and depression are commonly reported (Bartal, 1979; Blass, 1984; Burnside, 1979; Sim, 1965; Wolanin & Phillips, 1981). These behavioral manifestations, while consistent in the literature with general AD clinical symptomatology, are not characteristic of all AD victims.

Since the mental and behavioral disorientations with AD are variable (Sim, 1965) the course of AD has been conventionally divided into
three, sometimes four, progressive stages: (a) gradual loss of memory, depression, loss of efficiency, and the development of a variety of defenses designed to conceal the deficits; (b) disorientation, confusion, impaired comprehension and speech, nocturnal restlessness, and alexia; and finally, (c) paraphasia, parkinsonism, eventual incontinence, inability to cooperate effectively or follow directions, and epileptic-like seizures (Jervis, 1972; Sim, 1965). These stages, along with the clinical descriptors within, vary from author to author; nonetheless, they outline the progressive nature of AD. Correct evaluation of the clinical symptomatology of AD is an absolute necessity. As with the pathophysiology of this disease, research to determine reliable and valid behavior patterns continues.

**Pharmacologic Intervention for AD**

In recent years, the numbers of clinical psychopharmacologic trials designed to test the efficacy of drug treatments have increased greatly. Generally, the research findings in this area have concluded that conventional psychopharmacotherapy is useful in managing the clinical symptomatology even though the underlying pathology cannot be repaired or arrested by currently available means (Fisk, 1983; Terry & Katzman, 1983; Spar, 1984).

The most common use of drugs in the clinical management of AD victims relates to the evidence that AD is associated with the impaired function of neurons that utilize the chemical messenger acetylcholine in the cerebral cortex (DHHS, 1983; Terry & Katzman, 1983; Spar, 1984). "Scientists have demonstrated that drugs that interfere with the action..."
of acetylcholine in the brain can produce cognitive impairments in the young that resemble those seen in AD" (DHHS, 1983, p. 34).

Double-blind crossover studies administering acetylcholine activity enhancers, such as lecithin or anticholinesterase, appear to produce no improvements (Terry & Katzman, 1983; Spar, 1984) or only minimal improvements (DHHS, 1983) in performance on a variety of memory and/or cognitive tests. In addition, similar studies which administer physostigmine either orally or intravenously have shown encouraging improvements in memory for some AD victims while not for others (Terry & Katzman, 1983; Spar, 1984).

In a recent pharmacologic investigation conducted by Spar (1984) the opiate antagonist, naloxone, was intravenously administered each morning for three consecutive days. Improvements in the psychiatric complications of AD for any subject on any psychometric testing measure were not demonstrated (Spar, 1984).

Other pharmacologic investigations include evaluations of the effects of: (a) neuropeptides such as vasopression, (b) vasodilators such as nyliddain, (c) cerebral metabolic enhancers such as dihydroergotosine, (d) chelating agents, (e) anti-depressants, and (f) psychostimulants. However, so far, clinical trials of these compounds with AD subjects have been disappointing. "While positive findings have been reported in some isolated patients, the magnitude of any drug effect on learning and memory in Alzheimer's Disease has been, at best, quite modest" (DHHS, 1983, p. 35; Spar, 1984).

Most of the pharmacologic investigations have relied primarily on neuropsychological examinations, especially norm-referenced testing.
procedures, as the dependent variable. These testing procedures tradi-
tionally focus on measuring cognitive impairment, as mediated by memory 
loss, and on group findings. To date, the norm-referenced testing 
procedures have not proved useful as either accurate indicators of 
diagnosis or as useful evaluator of treatment efficacy (DHHS, 1983; 
LaRue, 1984; Mohs, 1983).

Many of these pharmacologic agents are currently being prescribed 
by physicians to help the families and caretakers manage the clinical 
symptoms of AD. Despite the basic lack of pharmacologic research on 
"the relationship between blood levels and clinical outcomes" (DHHS, 
1983, p. 36) anti-depressants and psychostimulants are frequently used 
with AD victims even though major side effects, such as tardive 
dyskinesia, are commonly reported (DHHS, 1983; Spar, 1984). Medications 
are most frequently prescribed to manage behavioral problems such as 
wandering, sleep disorders, withdrawal, depression and/or mood dis-
orders, and agitation because "these symptoms are often seen to exacer-
bate the severity of illness, contributing to excessive disability for 
the patient, excess burden on the resources of the family, and higher 
rates of institutionalization" (DHHS, 1983, p. 37).

Pharmacologic agents which can retard or reverse the cellular 
degeneration and associated cognitive dysfunction of AD are not yet 
known. Although investigations to correlate the basic physiological or 
biochemical process which mediate drug effects continue (Terry & 
Katzman, 1983).
Summary

This very brief review of the pathologic, clinical symptomatology and pharmacology literature attempts to provide a basic understanding of AD. Since all of the subjects in this study are suspected Alzheimer's victims, the following clinicopathologic characteristics, most especially affecting behavioral functions such as learning, are relevant: (a) cerebral atrophy, neurofibrillary tangles and neuritic plaques are probably centralized in the hippocampus; (b) dramatic loss of acetylcholine and its relationship to the transmission of nerve signals; (c) dementia and associated memory impairment; and (d) the prevalent use of medications to manage behavior problems.

The conclusion drawn from this overview of AD is that the disease and its effects are as individual as the individual who has it. The lack of consistency and of specificity reported in the current literature supports that AD is an individualized phenomenon (Powell & Courtice, 1983). This study, therefore, seeks to investigate the behavioral consequences of AD on learning for each subject separately.

The methodology most appropriate for this type of investigation is the single-case design or the intrasubject-replication design because the capacity to conduct experimental investigation with the individual subject is a unique feature of this design. "The ... methodology focuses on performance of the same person over time" (Kazdin, 1983, p. 3).
The development of the single-case design can be traced back to the initial work of B. F. Skinner (1953). Skinner's method of experimentation and data evaluation, known as the experimental analysis of behavior, has several distinct characteristics which are consistent with single-case experimentation. These characteristics include the study of the frequency of performance, the use of one subject across the experimental conditions, and the systematic changes in performance observed over time as a consequence of manipulating experimental conditions. As a result, the experimental analysis of behavior and the single case design has become synonymously identified with operant conditioning research (Kazdin, 1983).

Since the methodology desired in studying the behavioral consequences of AD is the learning performance with a single-subject, over time, across a variety of experimental conditions, a review of the operant conditioning literature seems warranted. This review is organized as the review of the AD literature with the relevant nonhuman, neurological studies presented first, followed by applied behavioral reports and then, a review of the behavioral pharmacology research.

**Neurological Research**

Two studies of operant learning with nonhuman subjects seem particularly relevant to this investigation because they directly relate to some of the pathological changes occurring with AD. The first was conducted by Kimble (1969) who investigated the behavior of rats with
bilateral hippocampal damage. By removing the cortex overlying the hippocampal formation in 11 Long-Evans strain rats and 10 other rats receiving bilateral damage to the hippocampus, he compared these two experimental groups to a 12-rat matched control group. The most striking finding in a preoperation-postoperation, open-field maze learning procedure related to the extremely repetitive pattern of behavior for animals with hippocampal damage. These subjects ran rapidly along the perimeter of the open field, rarely stopping. The behavior of the other two groups (cortex experimental subjects and the control subjects) differed dramatically. Their behavior consisted of "bursts" and "stops" occurring randomly. Kimble hypothesized that:

If the behavioral deficit seen in animals with hippocampal damage indeed stems from their inability to perform adequately when all of the relevant stimuli are not immediately present, the deficit should appear selectively on the successive discrimination problem. (p. 18)

To test this hypothesis, another experiment, with the same subjects, was designed to investigate discrimination learning using a Y maze with removable walls and floors in the arms serving as the stimulus cues. In the simultaneous discrimination procedure, one of the arms was always white, while the other arm was always black. For the successive discrimination procedure, both of the maze arms were either black or white on any given trial. The results indicated that the hippocampal subjects took significantly more trials than did the other two groups to reach criterion on the successive discrimination, while no difference among the three groups appeared in the simultaneous discrimination.

One possible explanation for the results of this stimulus discrimination study is that the hippocampal Ss are impaired on complex tasks,
but relatively unimpaired on simpler discriminations. Therefore, an experiment was designed to test this complexity of task dimension. With the same subjects, Kimble (1969) used two mazes, one very simple (M1), and one relatively complex (M6). The results showed that the hippocampal Ss made significantly more errors than either of the other two groups on both maze problems. In addition, when placed in the start box, the hippocampal Ss:

typically entered one of the two side alleys, progressed to the end and then turned and entered the other alley. These Ss, in sharp contrast to the other two groups, then re-entered the originally chosen alley, rather than moving into the center of the maze. This repetitive tracing of the two side alleys continued 50-75 times. (p. 22)

This repetitive behavior suggests that Ss with hippocampal damage may be unable to inhibit their motor responses and perhaps even display a tendency for hyperactivity (Kimble, 1969).

The second study of operant learning with nonhuman subjects worthy of mention was conducted by Banks and Russell (1967). The experiment was designed to study the effects upon serial problem solving behavior with subjects with acetylcholine (ACHE) levels 40% below normal. Banks and Russell were interested in whether the chronic ACHE levels would result in extinction on problem solving tasks which were reinforced by food contingent upon the correct response. The results indicated that the greater the reduction in ACHE activity, the greater the difficulty in shifting from problem to problem and hence the Ss had larger total error scores. In addition, Banks and Russell have concluded that there were no gross signs of peripheral sensory involvement, nor any significant difference between the experimental and control groups in
speed of locomotion or in consummatory responses on the peripheral motor side.

Both of these studies demonstrate the behavioral effects of removal or damage to the hippocampus, and the critical and chronic loss of acetylcholine in experimental laboratory animals. Similar findings are also reported in the clinical literature with human subjects (Carlson, 1977).

**Applied Clinical Research**

Perhaps the most famous single-account of memory impairment associated with hippocampal damage in the behavioral literature is reviewed by Milner (1970). The case of H. M. demonstrates the effects of bilateral, removal of the medial temporal lobes, which included removal of the amygdala, the uncus and the hippocampus. H. M.'s intellectual ability and short-term memory appeared unchanged following surgery, however, with few exceptions, he demonstrated an inability to learn anything new. "He cannot identify people he met since the operation, nor can he find his way back home if he leaves his house" (Carlson, 1977, p. 562). If left undistracted, H. M. could perform simple discriminations especially when presented with verbal stimuli. He did this by continuous, uninterrupted rehearsal for short durations. If the interval between the stimulus and the response was too long, or if he became distracted, he "forgot" the appropriate response. Milner (1970) concludes that the hippocampus plays a vital role in the consolidation process of memory. The removal of the hippocampus dramatically affected H. M.'s ability to retain and recall events from his environment.
Sidman, Stoddard and Mohr (1968) tested H. M. on a delayed matching-to-sample problem with verbal and nonverbal material. H. M.'s performance on problems for nonverbal stimuli were better when there was no delay, with his performance quickly deteriorating as the delay interval gradually increased. On the other hand, he was able to select the matching stimulus to a three-letter nonsense word after a 40 second delay. Sidman and others concluded that H. M. can easily rehearse verbal information but cannot construct verbal codes for nonverbal stimuli (Carlson 1977).

Skinner refers to the construction of verbal codes for nonverbal stimuli as "rule-governed behavior." People learn to be affected by a description of a contingency somewhat as they would be affected by the contingency itself" (Peterson, 1980, p. 6). The findings presented by Milner (1970), Sidman et al. (1968), and others suggests that the loss or damage to the hippocampus will result in a deficit of this rule-governed ability.

Clearly, discrepancies which exist between the effects of hippocampal lesions in human and nonhuman studies relate to the ability to engage in rule-governed behavior. Since humans have a large verbal repertoire, the ability to verbally reproduce stimulus contingencies greatly enhances our interaction with the environment; while its loss can only be a hinderance.

**Behavioral Pharmacology**

Behavioral pharmacology has grown out of the integration of experimental psychology and pharmacology to investigate the behavioral
actions of drugs (Thompson & Boren, 1977). The goal of behavioral pharmacology is to describe the behavioral mechanisms by which drugs alter behavior. Drugs used in a clinical setting are of particular interest to the experimental researcher. The researcher may seek to understand the behavioral mechanisms responsible for a particular clinical effect or to determine if laboratory procedures are relevant to a clinical problem. In either case, drug effects are more readily manipulated with nonhuman subjects.

Early nonhuman experiments have clarified that operant baselines are among the most sensitive measures of behavioral action with drug use (Thompson & Boren, 1977). These investigations have focused on the analysis of how incoming sensory stimulation can serve to change the state of the central nervous system in such a way "that the modification can alter subsequent information processing and behavior of the organism" (Stein & Rosen, 1974, p. 1). These changes, if repeated over time, are referred to as "learning."

Investigations into the factors central behavior change, the operant behavioral pharmacology approach most often involves the intensive study of a single individual subject. The single-case design emphasizes a close observation of a single subject for maximum experimental control:

If the experiment is successful, a subject will behave predictably from session to session and even from minute to minute. Thus, when an effective drug is administered in the middle of a session, a change from the dependable baseline behavior should be readily apparent in an individual subject. Furthermore, on different sessions, a range of drug dosages can be studied in the same subject with a sound basis for comparison (Thompson & Boren, 1977, p. 541).
The repeated acquisition procedure, by Boren and Devine (1968) (described in detail below) is just such a single subject design and has been used to investigate drug effects in the behavioral pharmacology literature.

Moerschbaecher and Thompson (1980) have reported that the technique of repeated acquisition has been found to be a sensitive method by which a drug's effects on learning may be studied. Their experiments using a wide variety of drugs have shown that responding under an acquisition baseline is disrupted (i.e., errors increase) at doses lower than those that disrupt a comparable performance baseline, where the discrimination is the same each session (Moerschbaecher, Boren, Schat & Fontes, 1979; Thompson, 1970, 1971, 1974, 1975; Moerschbaecher & Thompson, 1980). For example, Moerschbaecher et al. (1979) used a multiple schedule of repeated acquisition and performance of a conditional discrimination to study the effects of cocaine and D-amphetamine in pigeons. They found that on both an acute and chronic basis, responding in the acquisition component was disrupted at doses that had little or no effect on responding in the performance component.

One possible reason that an acquisition baseline is more sensitive to disruptive drug effects than a comparable performance baseline may be related to different conditions of stimulus control. "Behavior under strong control by external stimuli are generally less affected by drugs than behavior under weak control by external stimuli" (Moerschbaecher & Thompson, 1980, p. 370). Variations in stimulus control, therefore, may function to modulate drug action.
Another possible rationale for the sensitivity of repeated acquisition baseline to drug effects may involve the complexity of the task itself. A belief commonly held is that difficult tasks are more susceptible to drug effects than are simple tasks (Polidora, 1963; Thompson, 1975, 1974, Kimble, 1969).

Summary

Whereas, single-subject design studies are abundant in the operant conditioning literature, this brief review of some findings relevant to AD perhaps demonstrates the utility of the operant approach in investigating human performance and the benefits of applying findings of animal laboratory research to human subjects. For the most part the operant literature presented reflects the behavioral mechanisms affected by organic changes in the brain. It has also been useful in locating a longitudinal assessment instrument necessary for the present study.

The repeated acquisition technique described in the behavioral pharmacology literature is an assessment technique demonstrated to be sensitive to subtle changes in learning. It has, therefore, been selected as the assessment technique, capable of measuring behavioral changes over multiple points in time.

Repeated Acquisition

The repeated acquisition technique was developed by Boren and Devine (1968) to study acquisition with an individual subject by studying behavioral chains. Each subject acquires different but similar behavioral chains (patterns of responding), a large number of times, so
that a pattern of learning and the number of errors reach a "steady state" (leveling off of errors) from session to session (Boren & Devine, 1968).

This technique is a "learning to learn" process. The transfer of learning from problem to problem, which Harlow (1949) calls the formation of a learning set, is a highly predictable orderly process which can be demonstrated as long as controls are maintained over the subject's experience and the difficulty of the problem. Thompson (1971), in a report describing the transition to a steady state, developed an index of the rate of learning based on the errors made during a trial. He concluded that the transitional data obtained from his investigation illustrated two types of learnings: (1) learning within each session, as shown by the systematic decrease in errors across trials, and (2) "learning to learn" or "learning set formation," as shown by the gradual decrease in total errors across sessions (Thompson, 1971).

Basically, the procedure requires the subject to emit a specific chain or serial sequence of responses on different manipulanda to obtain reinforcement. The correct behavior chain is then altered from session to session, thereby requiring the subject repeatedly to acquire different chains of responses. After preliminary training, subjects typically demonstrate a stable state, in terms of a stable within-session error reduction, and rapid acquisition, in terms of between-session error rates, each time a new chain is required (Boren & Devine, 1968; Hursh, 1977; Thompson, 1975).

The serial position sequence has several useful methodological features for studying acquisition behavior. First, the experimenter
introduces quantifiable levels of difficulty by lengthening the behav-
ioral sequence. Second, once the subject has learned a particular
sequence, a new randomly assigned serial position is introduced. Third,
the stimulus position, itself, serves as either a reinforcer or a
discriminative stimulus (Sidman & Rosenberger, 1967). "Each successive
member of the sequence acts as a reinforcer for the previous response"
(Sidman & Rosenberger, 1967, p. 478). Or "acts as discriminative
stimuli if they alter the probability of subsequent new responses"
(Hursh, 1977, p. 315). In other words, if the selection of the correct
stimulus in any given sequence increases the probability that the sub-
ject will make the same selection in the next sequence, then the serial
position of the stimulus has served as a reinforcer. On the other hand,
if the selection of the correct stimulus in one set increases the
correct stimulus selection in the next set, then the subject has
discriminated between the two sets and, hence, the correct stimulus has
served as a discriminative stimulus in the behavioral chain. The
subject can, therefore, make use of the stimulus position as a relevant
dimension for responding and consequently reduce error rates until a
"steady state" is observed.

Besides the stimuli in the behavioral chain serving as a
reinforcers and/or discriminative stimuli, Thompson (1970) and
Moerschbaecher, Boren & Schrot, (1978) have shown that the subject’s own
behavior can serve similarly as a reinforcer and/or discriminative
stimulus.

Thompson (1970), has shown that serial position or a subject’s own
behavior may control responding in a tandem schedule of repeated
acquisition of response sequences. Pigeons were able to acquire a new response sequence on a session-to-session basis with their own behavior functioning as the discriminative stimuli.

Likewise, Moerschbaecher et al. (1978), investigated the extent to which color, rather than the subject's own behavior controlled responding in the repeated acquisition procedure. Total percentage of errors increased in the tandem condition where colors marking the sequences were absent. From this procedure, therefore, color or the subject's own behavior (body position) could function as a discriminative stimulus for chain position.

A steady state may be generally defined as "a behavioral state in which the behavior of interest exhibits relatively little variation in its measured dimensional qualities over a period of time" (Johnston & Pennypacker, 1979, p. 455). A stable steady state over a number of sessions is then used as a behavior baseline for studying the effects of a variety of independent variables that may influence learning (Thompson, 1970). Furthermore, disruptions in this steady state of errors in the rate of acquisition between sessions can be influenced by stimulus events occurring during the session both external (i.e., conditioned reinforcement, time-out, etc.) and internal to the subject (i.e., drugs, toxic agents, etc.) (Hursh, 1977; Moerschbaecher & Thompson, 1980; Thompson, 1975, 1971, 1970).

Much of the literature on repeated acquisition reports its effects on learning. Experiments concerned with such variables as differential pretraining (Behar & LeBedda, 1974), conditioned reinforcement (Hursh, 1977) and conditional discrimination (Moerschbaecher et al., 1978)
investigate their effect on the repeated acquisition of response sequences.

Moerschbaecher et al. (1978) investigated the effects of variables previously demonstrated to affect the repeated acquisition of response sequences such as time-out duration, extended training and tandem scheduling. The results from the experiment investigating time-out showed that time-out duration had no effect on either percent errors or response rate (Moerschbaecher et al., 1978). Similarly, the results from the effects of extended training experiment showed errors generally decreased as a function of sessions of extended training on a single sequence of responses.

The repeated acquisition procedure was developed as an individual subject design. This procedure has several important advantages over the more conventional "independent group" design, including elimination of intergroup variability, direct behavioral measures of the individual's responses (versus statistical derivations), and the direct applicability of the findings to the behavior of the individual (Boren & Devine, 1968).

The experimental design requires that: (a) each subject serves under all experimental conditions, (b) before any variables are manipulated the subjects are trained until the behavior under study reaches a steady state, and (c) the effects of an independent variable are seen as a change in the steady state (Boren & Devine, 1968).

With regard to AD, changes in both the trend and range of the steady state would be expected to be both gradual and progressive, in keeping with the commonly held nature of AD. That is to say, the
between-session error rate would gradually increase over time (trend) in keeping with the progressive and insidious decline which has been reported as a characteristic of AD; and the total number of errors within-session or between trials would increase (range) in keeping with the reported loss of the consolidation process reported by Milner (1970). Collecting data for a significant length of time would provide the needed data to determine both the trend and the range for the AD subject, and would address the issue of behavioral degeneration or decline.

The common assumption is that AD progresses without remissions (Butler & Emr, 1978; Hayter, 1974). Although Sim's (1965) study yielded a fairly recognizable clinical picture, the lack of consistency in the descriptions of AD patients and the relatively small numbers of subjects in the AD research (Schoenberg, 1978; LaRue, 1984), suggest that the progressively degenerative progress of AD is specific to the individual afflicted. Since this disease is variable both in duration and rate of progress (Sim, 1965), it is of interest to study how the disease progresses for individuals and how this progression affects learning. This can be accomplished only by comparing the individual's behavior on one occasion to his/her behavior on another occasion.

Rationale for this Study

The purpose of this study is to determine if repeated acquisitions is a viable technique for studying subtle behavior change, over time, with persons suffering from Alzheimer's Disease.
The review of the AD literature has provided a basic understanding of the pathology and clinical symptomatology of the AD victim. Advances in both medical technology and psychometric testing procedures have contributed to a general agreement that AD is a progressive deterioration in physical and mental health, resulting in behavioral deficits such as learning and retention. However, good behavioral assessments of the subtle changes over time are not available with human subjects suffering from AD. In addition, behavioral assessments of treatment efficacy are also absent with AD subjects.

Behavioral psychologists in the past two decades, having concerned themselves with treatment efficacy guidelines, have investigated the use of learning and its sensitivity to environmental changes. Results of experiments with laboratory animals using the repeated acquisition technique have demonstrated that learning behaviors are sensitive to drug effects (Moerschbaecher & Devine, 1978).

Since clinical investigations into the nature of AD indicate the extreme loss of memory with the advance of the disease and subsequent loss in the AD victim's ability to learn, learning itself has not heretofore been considered as a viable research variable.

The dependent variable for the present study is the repeated acquisition technique reported in the behavioral pharmacology literature; more specifically, the total number of within-session and between-session errors of the behavioral chain. Herein, learning is defined as the acquisition of a steady state from session to session, and from trial to trial within a session. Direct obtrusive observations for this exploratory study are made in the setting in which the subject normally
functions (e.g., the home). Restrictions are placed on the family caregivers to minimize distractibility for the subjects. Therefore, the actual circumstances of the assessment are somewhat contrived since the absence of the primary caregiver departs from the ordinary living conditions during the testing procedure.

Since the purpose of this study is to investigate the utility of the repeated acquisition technique as an assessment tool, sensitive to subtle behavior changes over time, with persons suffering from Alzheimer's disease, only the baseline phase of the method was conducted.

The baseline phase is the initial period of observation prior to any intervention. Data collection during this baseline phase should yield a description of the existing level of learning performance as well as provide a basis for predicting the level of learning performance for the immediate future without an intervention.

By combining an understanding of the nature of AD with an investigation of learning behaviors, this study will contribute to the body of literature on AD in two ways: first, by studying learning as a single variable with AD subjects, and second, by utilizing the repeated acquisition technique with AD subjects to investigate the potential feasibility of this technique as a possible tool for further studies in either the behavioral consequences of this progressive illness or of possible treatment efficacy studies with AD victims.
CHAPTER II

METHOD

Subjects

Three subjects, two males and one female, between 62-68 years of age participated in the study. All subjects were diagnosed by a neurologist three to four years prior to the start of this study as suspected Alzheimer’s patients. All three subjects’ family members participated in the Kalamazoo Alzheimer’s Family Support Group where volunteers for this study were originally solicited. Two of the subjects received neuropsychological testing to confirm this initial diagnosis. It is likely that all subjects were in stage 2, using a three stage disease process, which is characterized by evidence of disorientation, confusion, impaired comprehension and speech, nocturnal restlessness and alexia. At the start of the study all subjects were taking the nutritional supplement lecithin in addition to maintenance doses of medications prescribed by their physicians for either the AD itself or other chronic conditions. Therefore, none of the subjects were totally drug free.

The study was conducted in the subjects’ apartments with their family members (primary care givers), for the most part, absent. Subjects II and III were living with their spouses while Subject I lived alone. All subjects and all caregivers signed informed consent prior to the study.
Apparatus

The testing apparatus involved a 24" x 8" green felt cloth, nine poker chips (three sets of three: red, blue & white) with a happy face sticker affixed to one chip in each set. The sets of poker chips were placed horizontally on the felt cloth background. The sets were positioned approximately three inches apart, with a within-set chip distance approximately 1/2". The three sets were arranged in exactly the same order for every session in the study, with the red set on the left, the blue set in the middle, and the white set on the right.

Within each set, one chip position was the correct choice and was identified by the sticker, while the other two chips positions were incorrect (See Figure 1). Notice that one chip in each set is marked and the other two are blank.) When the poker chips were turned down all chips were flush against the cloth. The scheduling of events was accomplished manually by the experimenter. A recording sheet was designed to allow the researcher to quickly record every response. A stop watch was used to measure the duration of each trial as well as the total session length.

Procedures

General Procedures

A three step preliminary training procedure was necessary prior to the baseline phase. This procedure was exactly the same for both the preliminary training and the baseline phase.
Figure 1. Apparatus.

The repeated acquisition apparatus used for this study. Circles represent poker chips, and the dots the markers or stickers used. The felt cloth was 24" X 8" and each set was placed 1/2" apart.

*These color cues were removed for Subject III.
The subjects were seated at the kitchen table in their own homes, with their family caregiver absent. The felt cloth background was placed in front of the subjects with the poker chips placed in the horizontal fashion described above. Within each set the marked chip was placed in a different position. For example, in the left position for the red set, in the middle position for the blue set, and in the right position for the white set. The marked chip remained in the same position for the entire session and was changed prior to the beginning of the next session. Only one session was conducted each day.

To begin the session, all poker chips were turned up, so that the marked chips’ positions were displayed, and the subject was asked to point to the marked chip in each set. Then all the chips were turned over in a sequential fashion, starting with the chip in the left position in the red set, followed by the chip in the middle position in the red set, then the chip in the right position in the red set, and so on, until all of the chips were turned down. Chips were turned down in this way (R-B-W) for the entire study.

Once the chips were turned over, the subject was asked to point to, or turn over the marked chip in the red set, followed by a request to point to or turn over the marked chip in the blue set, and finally, the white set. The subject or the experimenter would turn the chip selected face up. A correct response was defined by correctly identifying the marked chip in the appropriate set. Praise for the correct response was delivered on a continuous reinforcement schedule (CRF). Once a marked chip was turned over in a set, it remained face-up until the entire behavior chain was completed. At the end of the behavior chain, all of
the marked chips were displayed for five to fifteen seconds depending on the number of sets in the behavior chain (i.e., five seconds for one set, ten seconds for two sets and fifteen seconds for three sets), which served as a time-out (TO) duration after which the experimenter would turn down the marked chips in the correct serial order (R-B-W) to begin the next response sequence.

An incorrect response, or an error, was defined as the subject pointing to or turning over any chip other than the marked chip for the appropriate set of the three component behavior chain. An error was followed by the experimenter saying: "No! That's not the chip with the happy face sticker," turning over the chip selected, and recording the response. After a three second delay interval, the subject was prompted to turn over the marked chip for the appropriate set.

The response sequences continued until the subject correctly completed the behavior chain with no errors for three consecutive sequences, or until ten minutes had elapsed. These response sequences or ten minute intervals are referred to as a trial. Three trials were conducted during a session with two ten minute between trial rest intervals (Lindsley, 1966). Individualized reinforcers (i.e., walks, exercise, coffee, etc.) were used during these rest intervals or "breaks," and were delivered on a fixed ratio reinforcement schedule (FR3). If the subject did not meet criterion during the trial, the individualized reinforcer was not delivered, and the subject and the experimenter would leave the testing area and go to another area of the home for the ten minute "break".
Preliminary Training

For the preliminary training procedure, all sets were displayed exactly as they would appear throughout the study and involved the following three steps:

Step I: Only the red poker chip set was used. The subjects were shown the red poker chips and asked to point to the marked chip. The three chips were then turned over, with the marked chip remaining in the same position. The subjects were then asked to turn over the red poker chip marked with the happy face. All responses were recorded. Three consecutively correct responses (FR3) or a lapse of 10 minutes completed a trial. Praise was delivered on a CRF schedule within the trial, 10 minute "break" intervals were contingent upon completing a trial, and individualized reinforcers were delivered contingent on meeting criterion within the trial. For example, after the trial was completed, the subject received a 10 minute "break" interval, however if the subject completed the FR3 behavior chain, an individualized reinforcer was provided (e.g., go for coffee). The subject was required to complete three consecutively correct responses (FR3) for three consecutive trials to proceed to step II.

Step II: The red and blue sets of chips were used. The subjects were shown the red set and asked to point to the marked chip and then shown the blue set with the same request. The red set of chips were then turned over, followed by the blue set. The subjects were asked to turn over the marked chip in the red set, and, once correct, were immediately asked to turn over the marked chip in the blue set. Again, all
responses were recorded. A correct response sequence required the subjects to turn over the marked chip in the red set, followed immediately by turning over the marked chip in the blue set, with no errors within the behavior chain. Three consecutively correct response sequences of the two-component behavior chain completed the trial. Reinforcement was delivered as described in step I. Correct sequential responding of the session was necessary to proceed to step III.

**Step III:** All three sets of poker chips were used. The subjects were shown the red set and asked to point to the marked chip, then the blue set, and finally the white set with the same request. The red set was then turned over, followed by the blue set and then the white set. The subjects were asked to turn over the marked chip in the red set in the same manner as in steps I and II. A correct response was followed by a request to turn over the marked chip in the blue set. A correct response with the blue set was followed by a final request to turn over the marked chip in the white set. All responses were recorded and a correct response sequence required the subject to turn over the marked chip for the specific color set, with no errors within the three component behavior chain. Criteria for trial and session completion and the contingencies of reinforcement were the same as for Steps I and II. Achievement of the criterion of the behavioral chain for the session was necessary to proceed to the baseline phase.

**Baseline Phase**

The baseline phase began in the session immediately following the successful completion of the third step of the preliminary training and
was the same as Step III with the following additions: rule 1, the marked chip's serial position in each set differed; and rule 2, the marked chip was changed for every session and was never the same for consecutive sessions. The subjects were told the following at the beginning of each session: "The chip with the happy face sticker will be in a different position in each set." For example: if the marked chip was in the first position (reading from left to right) in the red set it would not be in this same position for either the blue set or the white set. In fact, if this were true for the red set, and blue marked chip was in the second position, the marked chip would, therefore, be in the third position for the white set.

All responses were recorded. Both within-session (between-trial) and between-session error rates were calculated. Learning was defined by the decrease in errors between trials within a session. Stable levels of overall accuracy, as measured by total error per session, characterized the steady state of responding desired. Nineteen sessions were completed with Subjects I and III, and thirteen sessions were completed with Subject II in this study.

Alternate Baseline Procedure

As a result of the unexpectedly rapid rate and ease of acquisition of the preliminary training procedure for Subject III, an alternate baseline procedure was designed. Basically the characteristics of the original baseline procedure were the same for Subject III with the following two exceptions: (1) the color discrimination was removed; therefore, all of the chips were white (Moerschbaecher et al., 1978).
The remaining discriminative stimuli were the position of the marked chip with respect to the unmarked chips in each set, the serial position of the sets on the felt background, and the subject's own body position with respect to the apparatus and (2) the sessions began with all chips turned down, hence each session began with the subject locating the marked chip by trial and error for the first set and by making use of the procedural rule (1) on serial position for the subsequent sets.
CHAPTER III

RESULTS

The results of this study were treated in terms of the number of times the subjects selected incorrect chips within the three component behavior chain. Each incorrect selection is counted as an error. As can be seen in Table 1, the mean errors for all three subjects are presented. All subjects performed better (made fewer errors) on the performance trials (PT1 and PT2) than for the learning trial (LT). In addition, the mean errors for subjects I and II were slightly greater for PT1 than for PT2. The mean errors for Subject III were the same for the two performance trials. Since the overall learning trial errors are greater than the overall errors for both performance trials, all subjects demonstrated a capacity to learn the three component response chain to some degree. Furthermore, since the errors for PT1 were slightly greater or equal to the errors for PT2, all subjects demonstrated a capacity to retain the initial learned responses.

This finding was somewhat unexpected. As was mentioned earlier, the total number of within-session errors (range) was expected to increase in keeping with the reported loss of the consolidation process reported in the literature. This, however, was not found to be the case in the present study. All subjects successfully transferred behavior acquired in the initial learning trial to the performance trials. Thus, with regard to within-session learning, all subjects demonstrated the capacity to learn to some extent. This finding is consistent with the
repeated acquisition literature for nonhuman subjects (Moerschbaecher et al., 1978; Boren & Devine, 1968; Thompson, 1971, 1970).

TABLE I

<table>
<thead>
<tr>
<th>SUBJECT I</th>
<th>LT mean</th>
<th>PT1 mean</th>
<th>PT2 mean</th>
<th>Total mean</th>
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<td>35</td>
<td>28</td>
<td>27</td>
<td>30</td>
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<tr>
<td>•</td>
<td>BV</td>
<td>AV</td>
<td>BV</td>
<td>AV</td>
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<tr>
<td>•</td>
<td>31</td>
<td>40</td>
<td>23</td>
<td>32</td>
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<tr>
<td>SUBJECT II</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>•</td>
<td>BV</td>
<td>AV</td>
<td>BV</td>
<td>AV</td>
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<tr>
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<td>8</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>•</td>
<td>10/10</td>
<td>10/30</td>
<td>10/10</td>
<td>10/30</td>
</tr>
<tr>
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<td></td>
<td>3.9</td>
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</tbody>
</table>

* BV = Before Vacation
  AV = After Vacation

** 10/10 = Session 1-6: 10 minute "break" periods LT to PT1 and PT1 to PT2
  10/30 = Session 7 - 19: 10 minutes and 30 minute "break" periods LT to PT1 and PT1 to PT2, respectively

All subjects were given identical instructions prior to each session. As can be seen by the extreme number of learning trial errors, no subject made use of the procedural rule (2) that no marked chip would be in the same position in the sequence on two consecutive days. Hence, this procedural rule was not a sufficient discriminative stimulus across sessions.

Another objective of this study was to determine the extent it was possible to achieve a steady state of relearning from session to session; to see if a stable baseline could be obtained with subjects.
suspected of having AD. As Boren and Devine (1968) have indicated, "the features of the procedure important in establishing a stable state of relearning are: (1) a set of equivalent problems to be learned, and (2) a sufficient number of training sessions to produce stability." (p. 652) Because of the variability among subjects, each will be reported individually.

Subject I

As can be seen in Figure 2 (total errors per trial and session), Subject I did not achieve a steady state for the 19 sessions. However, Subject I did meet criterion in 39 of the 57 trials (or 68% of the time) even though the number of errors far exceeded what would be expected. In addition, note the incidence of PT1 errors in the first seven sessions. The increase in errors for PT1 seem to indicate, at least initially, that learning did not transfer. However, errors were lower for the second performance than for either of the first two trials. The subject's overall performance, however, eventually demonstrated positive transference of learned behavior.

Session 19 again shows a greater number of errors for PT1 than for LT and suggests the lack of transfer. However, it should be noted that the subject was found wandering in the early morning hours outside the apartment on the day of this session. The literature on AD suggests that confused wandering is an indication of an exacerbation of the resulting dementia. Had the study been continued it would have been interesting to see if this lack of transference between LT and PT1 would
Figure 2. The total number of errors per trial for Subject I (a suspected victim of Alzheimer's disease) are located on the ordinate and trials/sessions along the abcissa. Three trials were conducted for every session, hence trials are presented first along the abcissa with the corresponding sessions underneath. The solid dots reflect trial data in which criterion was reached (3 consecutively correct responses on the 3 component behavior chain.) Open circles reflect trial data in which criterion was not reached, but ten minutes had elapsed during the trial. The first trial (1) in all 19 sessions refers to the learning trial (LT) and the second and third trials (2, 3) in all 19 sessions refers to the performance trials, with the second trial referred to as the first performance trial (PT1) and the third trial referred to as the second performance trial (PT2).
Figure 2. The Total Number of Errors Per Trial and Session for Subject I.
continue. This potentially dangerous situation, however, resulted in the subject being relocated to an institutional setting to ensure the subject's safety; therefore, the study was terminated.

During the three months of the study the subject's overall errors seem quite high. It should be noted, however, that it was common for the subject to repeat the same systematic errors in the response chain. In other words, if the subject had selected the middle chip in the first set in the previous sequence, the subject was likely to select the middle chip in the first set again during the next sequence, even though the response was incorrect. This suggests that the position of the correct chip in a set was not a sufficient discriminative stimulus for correct responding until a relatively high number of sequences had been attempted.

In addition, Subject I did not seem to benefit from the procedural rule (1) that no correct chip was in the same position at any one time in the three sets, even when the rule was provided as a prompt prior to any response for each set. Responding seemed to be trial and error until a high number of behavior chains had been attempted.

Subject II

Subject II also did not achieve a steady state of responding over time. Figure 3 indicates the overall high level of errors. Unlike Subject I, however, Subject II did not have a higher number of errors in either of the performance trials than for the learning trial. Only in session 3 did performance trial errors exceed learning trial errors; and
Figure 3. The total number of errors per trial for Subject II (a suspected victim of Alzheimer's disease) are located on the ordinate and trials/sessions along the abcissa. Three trials were conducted for every session, hence the trials are presented first along the abcissa, with the corresponding session underneath. The solid dots reflect trial data in which criterion was reached (3 consecutively correct responses on the 3 component behavior chain.) Open circles reflect trial data in which criterion was not reached, but ten minutes had elapsed during the trial. The first trial (1) in all 13 sessions refers to the learning trial (LT) and the second and third trials (2, 3) in all 13 sessions refer to the performance trials, with the second trial referred to as the first performance trial (PT1) and the third trial referred to as the second performance trial (PT2). The data plotted to the left of the dotted line were collected prior to a 3 week hiatus, with the data plotted to the right collected after the subject's return.
Figure 3. The Total Number of Errors Per Trial and Session for Subject II Both Before and After Vacation

SUBJECT II

Key

• = Trial Criterion Reached
○ = Trial Criterion Not Reached
then only minimally. This suggests for Subject II positive transferance from the learning trial to the two performance trials. Subject II met criterion in only 9 of the total 39 trials (or 29% of the time). More often than not time would run out before the subject could master the response chain three consecutive times. For further study with Subject II it might have been beneficial to drop criterion from three to one correct behavior chain and, hence, simplify the learning task.

After session 7, the subject traveled to Hawaii for a three week vacation. As can be seen, the overall total errors have an upward trend following this hiatus. Prior to this delay, no trend is apparent in these data. Table 1 shows the mean errors for each trial prior to and after the vacation. On the average, Subject II made nine more errors across all trials after the three week interruption. Again, the AD literature suggests that travel to foreign territory disorients the AD victim and the lack of routine further exacerbates the dementia. As can be seen, these data tend to support this conclusion.

Interestingly, prior to session 11, Subject II was observed being physically abusive to the caregiver and it was necessary to restrain the subject for some time. Session 11 was postponed for a full day as a result, to allow the subject to calm down. The high number of errors in the remaining three sessions corresponds to the subject's continued abusiveness and, as with Subject I, it became necessary to institutionalize Subject II for safety. The study was, therefore, terminated.

Subject II made the same systematic errors throughout this study. Once the subject found the correct chip in the first set, the chip in the same position was selected in the next set. The subject was
reinforced by the correctness and the spatial position of the chip in the previous set and generalized this reinforcement contingency to the next set. This behavior pattern, however, was counter to the procedural rule (1), that no correct chip would be in the exact same position for the three sets. Rather than responding to the rules, the subject was reinforced by correct selection. In addition, it was also noted that Subject II would make the same error in any given set more than once: the subject would turn over the same incorrect chip two or three times in a row. This systematic error related to Subject II's overall high error rate throughout the study.

Subject III

The only subject consistently to meet criterion (100% of the time) and achieve a steady state of relearning from session to session was Subject III. This subject established a stable relearning performance after six sessions. Mean errors for these six sessions for LT and for PT1 and PT2 were 10, 2 and .5 respectively (Table 1). These data are consistent with data achieved from other baseline studies of repeated acquisition (Peterson, 1980; Thompson, 1971). For Subject III, as for Thompson's pigeons, the color cue of the sets were removed, so that only serial position and the subject's kinesthetic feedback system were cues for correct responding (Thompson, 1971). The sharp decrease in errors from LT to PT1 and between the two performance trials may indicate learning of the three component behavior chain on the basis of these two remaining cues.
Figure 4 shows the number of errors made for each trial across all sessions. In the first six sessions, the number of errors for LT were relatively few, considering that the subject was not shown the position of the marked chip in each set prior to the beginning of the trial. Subject III, in effect, had to "find" the marked chip in each set by trial and error with the help of the procedural rule (2). The subject did not seem to benefit from this procedural rule, relying almost solely on a trial and error behavior pattern. However, the object did seem to benefit from rule 1, as most of these learning trial errors were made in the first set of the behavior chain and as few as two total errors in the third set for all of the trials. In addition, the serial position of the marked chip in each set seemed to be under strong stimulus control, as the subject would often attempt to "abort" a selection response without necessarily checking for the sticker. Subject III would begin to turn over an unmarked chip, say "no" and put the chip back without looking at the underside for confirmation. All selection responses were recorded whether the subject actually turned over the chip or not. Interestingly, these aborted responses decreased considerably after the first seven sessions and their absence may indicate the presence of covert rule-governed behavior generated by the subject since the learning trial errors also decreased somewhat after session 7.

Subject III was the only subject to meet criterion in every trial for all sessions, and all trials were completed in under five minutes. In fact, the longest trial time was 4:38, occurring in LT for session 2, in which the subject made eleven errors for that trial.
Figure 4. The total number of errors per trial for Subject III (a suspected victim of Alzheimer's disease) are located on the ordinate and trials/sessions along the abscissa. Three trials were conducted for every session, hence the trials are presented first along the abscissa, with the corresponding sessions underneath. The solid dots reflect trial data in which criterion was reached (3 consecutively correct responses on the 3 component behavior chain.) The first trial (1) in all 19 sessions refers to the learning trial (LT) and the second and third trials (2, 3) in all 19 sessions refers to the performance trials, with the second trial referred to as the first performance trial (PT1) and the third trial referred to as the second performance trial (PT2). The data plotted to the left of the dotted line were collected with 10 minute between-trial "break" periods. The data plotted to the right were collected with a 10 minute between-trial "break" between LT and PT1 and a 30 minute between-trial "break" between PT1 and PT2.
Figure 4. The Total Number of Errors Per Trial and Session for Subject III During 10 Minute "Breaks" and 10 and 30 Minute "Breaks".
Since a steady state of relearning was established by Subject III in only six sessions, the between-trial reinforcement durations were altered within the session to test the effect on the contingencies of reinforcement and retention. Ten and thirty minute between-trial reinforcement periods were implemented for sessions 7 through 19. For this condition, the ten minute duration between LT and PT1 remained, and a thirty minute between-trial reinforcement period was imposed for the performance trials.

In addition, a reinforcement activity was imposed during the thirty minute between-trial reinforcement period in which the subject and the experimenter left the testing environment for the duration of the "break". As the data in Table 1 indicate, PT2 errors were slightly greater than the PT1 error rate. The mean error for PT1 was 1.5, while the mean error for PT2 was 3.2.

As Figure 4 indicates, on three occasions the PT2 errors were slightly greater than, slightly less than or equal to the initial LT errors, perhaps indicating a trial and error performance pattern rather than a positive transference of stimulus discrimination from learning to performance trials. On the other hand, on five occasions PT2 errors were equal to or only slightly greater than PT1 errors, indicating positive transference. Overall, PT2 error rates are considerably lower than for the learning trial error rates. This suggests that the total fifty minute delay between the end of the learning trial and the beginning of the 2nd performance trial only minimally affected the acquisition behavior studied.
Inter-Observer Reliability

Inter-observer reliability checks were conducted on two separate occasions (two different sessions) for all subjects or eleven percent of the total number of sessions. The family caregivers responsible for the subjects were trained on the data collection instrument and were used as reliability observers due to availability and accessibility, and to minimize observer influence or "reactivity" (Bailey, 1977). The overall percent agreements were 95, 100 and 89 for Subjects I, II and III respectively. The largest inter-observer reliability disagreement (82 & 91 percent) occurred during the last trial in both sessions for Subject III. It was noted that the reliability observer was not recording this subject's "abortive" response attempts and only recorded responses which resulted in the subject completely turning over the poker chips.

Summary

The data indicate that all three subjects were able to successfully transfer behavior acquired in the initial learning trial to the performance trials, as seen by the overall fewer total errors for the performance trials. Further, only Subject III achieved a steady state of relearning from session to session, and did so consistently throughout the study. Subject II, on the other hand, was the only subject to demonstrate an increasing trend in errors over time, but then, only after a three week hiatus which interrupted the testing procedure. None of the subjects utilized rule 2, seemingly all responding in a trial and
error fashion each session. However, Subject III did make use of rule 1 as demonstrated by the "abortive" response attempts and the overall few performance trial errors.
CHAPTER IV

DISCUSSION

This study demonstrates the use of the repeated acquisition technique with individual subjects suspected of having Alzheimer's Disease (AD) serving as their own control to test the possibility of achieving a stable pattern of learning and relearning. Each subject had a preliminary training procedure with increasing problem complexity until criterion on a three component behavior chain was successfully achieved. Following this initial training, a baseline condition was implemented and errors on the three component behavior chain were recorded. The repeated acquisition technique required each subject to respond repeatedly on different but equivalent behavior chains until a stable and predictable baseline rate of performance was obtained. Color cues, serial position, procedural rules or instructions, and reinforcing break periods were used to study the acquisition behavior of AD subjects over a three month period.

Typically, subjects demonstrate a stable state of learning (within-session error reduction) and a rapid acquisition (between-session error reduction) with this behavioral procedure (Thompson, 1970, 1971). While, overall, the subjects in the present study demonstrated a within-session error reduction, only Subject III successfully achieved a steady state. Nonetheless, all subjects' error rates were, for the most part, greater during the initial learning trial than for either of the
performance trials. Generally, the greatest number of errors occurred for all subjects during the learning trials.

The overall error rates were higher for Subjects I and II than were expected, suggesting that the within-session error reduction was likely due to continued trial and error responding or to accidental reinforcement of errors by the experimenter.

The color cues and serial position of the poker chips were sufficient discriminative stimuli for the correct component of the behavior chain for Subject I but were insufficient for Subject II. Subject II required numerous verbal prompts to move between the components of the behavior chain during a session.

Even though Subjects I and II began every session with the marked chips face up in each set of the three component behavior chain, they could not complete the sequence without numerous errors. In fact, Subject II never met criterion without error, while Subject I could only do so on three separate performance trials in three different sessions, and then only after numerous errors in the previous trial.

With the color cues in the behavior chain removed for Subject III, the serial position seemed to serve as an adequate discriminative stimulus to evoke correct responses, independent of color. Subject III did not rely on the color discrimination for chain position requiring this subject to be sensitive to the consequences of each response (Moerschbaecher et al. 1978).

The procedural rules were found to have no positive consequence for either Subject I or II. For two of the subjects, the rules were ineffective in evoking the correct response. This lack of control could
be due to sensorimotor and perceptual impairments associated with AD (Bartal, 1979; Burnside, 1979; Wolanin & Phillips, 1981). Subject III, on the other hand, did benefit from procedural rule 1 (the marked chip's serial position in each set differed) suggesting that these impairments may be individualized.

Although the achievement of a steady state of baseline responses was of major interest in this research, there are other behavioral characteristics observed that should be mentioned.

The individualized reinforcers seemed to have no positive consequence for either Subject I or II but were effective for Subject III. In fact, it should be mentioned that for both Subjects I and II there was no reinforcement carry over from one session to another. An example occurred with Subject I. During session 5, the subject met criterion (three sequentially correct responses for the three component behavior chain without error) on all three trials. The contingent reinforcement was a handful of unsalted cashews. The next session, session 6, when the subject met criterion during PT1, cashews were, again, given. The subject brushed the cashews on to the floor stating, "There's dirt on the table." It may be possible that reinforcement for AD victims depends less on their past history of reinforcement and more on the physical properties of the stimulus they perceive at any given moment. While the reinforcing stimulus remained consistent during any given session for Subjects I and II, it differed on any number of occasions between sessions, resulting in a decreased probability of correct responding. This, however, was not observed with Subject III. The
individualized reinforcer remained consistent for this subject throughout the duration of the research.

During preliminary training with Subject II, only two poker chips were presented in front of the subject on the felt cloth board. One chip was marked with the happy face and the other was blank. The procedure started with both chips face up. After the subject pointed to the marked chip, both chips were turned over remaining in the same position. On one occasion the subject turned over the unmarked poker chip 45 times in a row and finally reported to the experimenter: "You are going to run out of chips." The subject's response did not demonstrate the perception of a relationship between the two poker chips. Similar observations were reported by Kimble (1969) with the hippocampal rats running to either side of the maze 50 to 75 times, and by Banks and Russell (1967) with the difficulty of the experimental rats with at least a 40 percent reduction in ACHE in shifting from problem to problem.

One final observation relates to the subjects' motor responses. Kimble (1969) observed that the hippocampal rats may be unable to inhibit their motor responses and perhaps may even display a tendency for hyperactivity. Similar effects were noted for all three subjects in this study on different occasions. All of the subjects responded very quickly, turning over the chips in rapid succession. Except for the first four sessions, Subject III completed each trial in all subsequent sessions in an average time of 1:43. In addition, all of the subjects paced rapidly during the between-trial breaks. While out for coffee, during the thirty minute between-trial reinforcement condition, Subject
III would rapidly drink his cup of coffee no matter how many times it was refilled, get in and out of his seat two or three times, and walk briskly to and from the restaurant. Subject I, on the other hand, would respond in bursts and stops similar to Kimble's findings with the hippocampal rats. These rapid motor responses relate directly to the large number of errors for Subject I. Conversely, Subject II responded more slowly to the testing apparatus over time even though he would move rather quickly during the between-trial reinforcement intervals.

For the most part, this study investigated the baseline phase of the repeated acquisition technique. However, an intervention was imposed for Subject III to study the effects of between-trial reinforcement "breaks" on positive transference. During this intervention (sessions 7 to 19), the subject made slightly more errors in PT2 and in PT1. While the subject did demonstrate a stable baseline performance, these increased errors suggest that the repeated acquisition technique is sensitive to subtle changes in behavior, over time.

Similarly, a delay of three weeks during the baseline phase for Subject II resulted in an increased error rate in all three trials for sessions 7 to 13. Even though the subject did not demonstrate a stable baseline performance prior to the trip, the error rate increased by seven following the hiatus. Again, this suggests that the repeated acquisition technique is sensitive to subtle changes in behavior with a semi-stable baseline performance.

Finally, Thompson and Boren (1977) have advised that in order "to understand the way in which drugs alter behavior, it is first necessary to understand the factors which control behavior" (p. 540). By way of
understanding possible external factors which seemed to control the behavior of Subject I, (see Figure 2, session 13), this subject made more errors during this session than in any other session in the study, with the highest recorded error rate within every trial. While direct causation is difficult to determine, a few concurrent circumstances should be mentioned.

There was a five day delay between session 12 and 13 which spanned a weekend. During this time, the subject awaited the return of the primary caregiver from vacation. Because of the anticipated return of the primary caregiver, a surrogate caregiver neglected to look in on the subject. While alone during this weekend, the subject did not eat properly, bathe, change clothing, or admit anyone into the apartment. After gaining entrance, I found the subject nervously pacing the floor, expressing fears of an angry male stranger locked in the bathroom. The bathroom door was closed. In attempting to problem-solve with the subject, I happened to notice a scene on the television which dramatized a man pounding on a door in anger, with a frightened woman on the other side. Immediately after turning off the television, the subject stopped pacing. I reassured the subject that no one was in the bathroom and we delayed the session for 2 hours which allowed the subject to bathe, change clothing and eat a balanced meal.

The primary caregiver returned prior to session 14. Notice the reduction in errors for this session (see Fig. 2). At this time the primary caregiver was again caring for the subject. It is possible from these data to conclude that the repeated acquisition technique
demonstrated sensitivity to the measurement of environmental factors which affected Subject I's responses to the behavioral chain.

These findings are consistent with the operant literature on repeated acquisition which suggests that errors in the rate of acquisition between sessions can be influenced by external as well as internal stimulus events occurring during the session (Hursh, 1977; Moerschbaecher & Thompson, 1980; Thompson, 1975, 1971, 1970). The repeated acquisition technique in this study has demonstrated consistent sensitivity to behavior changes with and without the establishment of a steady state during baseline. This evidence, while not conclusive, does suggest that future research specifically designed to investigate a wide variety of stimuli, however, requires careful consideration of the following limitations found with the present exploratory study.

Limitations and Recommendations

Generally, single-case research designs, such as repeated acquisition, are useful in applied behavior analysis to evaluate the effectiveness of a wide variety of interventions (Kazdin, 1983). Because this study was designed to investigate the nature of a specific problem (AD) and the nature of learning performance during a baseline condition, limitations concerning interventions will not be addressed. Rather, the following discussion will address the limitations of the methodology and its application for future research. Methodological issues, such as generalization, stimulus control, the experimental setting, and the system for data collection are presented.
First, comparisons between the subjects are difficult to make with regard to the variation in performance in this study, due to the variety of variables which may control responding for the subjects. Generalization to persons suspected as AD victims not included in this study are impossible with this design. However, since the focus of this investigation was an attempt to discover the laws of individual performance with the repeated acquisition technique, and since the effects of AD are probably individualized, the inability to generalize to the AD population may be of minor importance. What may be generalized, however, is the sensitivity of the technique to subtle changes in behavior over time, as demonstrated by all three subjects. The key to evaluating the generality of this sensitivity with other AD subjects is through replication.

Second, in regard to stimulus control, strong control is maintained through contingencies of reinforcement. The procedures enforced in the present study inadvertently limited the establishment of effective discriminative stimuli, and/or the management of appropriate schedules of reinforcement for the AD subjects.

The discriminative stimuli were the color cues (for Subject I & II), the position of the marked chip within the sets, the serial position of the sets, the subject's own body position with respect to the apparatus, and the procedural rules. All of these stimuli were established to alter the probability of the subjects' responses. If these discriminations had been effectively established, then altering the probability of the correct response could have been accomplished by presenting or removing any of the discriminative stimuli (Skinner,
This kind of stimulus control was established with Subject III. The other two subjects' responses were under weaker stimuli control since fewer correct responses were reinforced, as evidenced by the large number of errors and the failure to meet criterion. Correct responding in the presence of a discriminative stimulus occurs only when the response is likely to be reinforced (Skinner, 1953). It is likely, for these two subjects, that the reinforcement contingencies for correct responding were ineffective in strengthening the stimulus control. Other immediate reinforcers may have been needed to increase the probability of correct responses.

The fixed ratio reinforcement schedule (FR3) imposed for this study (i.e., three consecutive correct responses for the behavioral chain) required a series of events to occur prior to the subject receiving reinforcement. This type of intermittent reinforcement is consistent with most social situations and with the operant literature on repeated acquisition (Boren & Devine, 1968; Moerschbaecher et al. 1978; Thompson, 1970, 1971, 1975). Nonetheless, Lindsley (1966) suggests that this type of reinforcement contingency may be inappropriate for the geriatric population.

deficits in responding (with intermittent reinforcement) are probably attributable to deficits in recent memory and in formation of conditioned reinforcement...It is very possible that many geriatric patients will...prove unable to maintain high rates of responding on intermittent schedules and will have to be kept on regular reinforcement contingencies in which every response is immediately followed with a reinforcing episode. (p. 164)

It is, therefore, probable that the FR3 schedule of reinforcement was ineffective, if not inappropriate, for these AD subjects, with Subject III being the exception, rather than the rule. Interval and continuous
reinforcement schedules need further investigation with the AD population.

Third, since environment-behavior relationships are determined with tight experimental control, the setting must guarantee this requirement so as not to preclude definitive research findings (Bailey, 1977). The present study was conducted in the subject's home to maximize accessibility, and minimize environmental effects, as may occur with a novel setting. The instability in the naturalistic setting limited the between-trial reinforcement "breaks," and, at times, limited the scheduling of the experimental sessions and the subjects' performance. In addition, the natural home setting was somewhat contrived, since restrictions were placed on the primary caregiver's attendance during the testing procedure. It has been documented in the AD literature that the victim's dependency on the primary caregiver is extremely high, especially for AD victims in the latter stage(s) of the disease (Mace & Rabins, 1981; Powell & Courtice, 1983; Wolanin & Phillips, 1981). Requiring the caregiver to be out of the room during testing altered the ordinary living conditions for the subjects and may have compromised experimental control.

Fourth, experimental control may have been further compromised by the manual recording of the data and the measurement of the duration of the trials by the researcher. These manual measurement techniques, while easily transported, may actually distract subjects from the learning task. Distractibility was noted by Milner (1970) to affect performance with a hippocampal damaged subject. Therefore, in research with suspected AD subjects, an automated data recording procedure may
have minimized reactivity on the part of the subjects (Bailey, 1977). Systematic investigations which foster possibilities for tighter experimental control need further consideration.

Finally, the length of a study with AD subjects also should be considered further. The most fundamental design requirement of single-case experimentation is the reliance on repeated observations of performance over time. Exactly how much time varies from study to study. The present study examined baseline performance with suspected AD victims for three months or nineteen sessions. Since AD is an insidious and progressive disease, longitudinal research which spans at least one stage transition is needed. Since the stage model of AD is not exact (Sim, 1965), the duration required to accomplish this goal is not clear. The present study was ended, for the most part, as a result of subject drop-off, an expected consequence in the study of chronically ill, elderly, subjects.

Summary

Historically, relatively few systematic applications of operant methods have been conducted with the geriatric population, and no applied behavior analysis has been applied to victims of AD. This study is an initial attempt to fill this void by exploring the utility of the repeated acquisition technique with AD subjects. The data presented and this discussion of the limitations suggest a wide variety of operant research possibilities.
Conclusions

In conclusion, the repeated acquisition technique was found to be a sensitive measure of the chronic effects of an internal variable affecting the subjects, such as AD. In addition, the between-session acquisition behavior was influenced by external events, such as vacation (Subject II) and disorientation to time and place (Subject I). While internal events, such as chronic AD, and effects of the subjects' medications and lecithin, may have affected the within-session error rates, all subjects demonstrated a learning curve.

There were no positive consequences observed by the between-session procedural rules, with only one subject (Subject III) demonstrating a positive consequence for the within-session rule (1). Therefore, this study found similar effects from verbal instruction as was found by others (Milner, 1970; Sidman et al., 1968). Similarly, the reinforcement contingencies had no demonstratable positive consequence (i.e., error reduction) for two of the subjects, with a positive effect observed for one subject. It seems possible to assert, therefore, that the effects of AD are as individual as the individual afflicted. If this is the case, then the single-case design characteristic of repeated acquisition seems appropriate for further investigation into the clinical nature of AD and the consequent behavior change for its victims.

The achievement of a stable baseline, or a steady state, by only one subject was disappointing since the behavioral pharmacology literature reports a relationship between the degree of stability and
the magnitude of effect (Thompson & Boren, 1977). In other words, the greater the stability, the smaller the effect which can be reliably studied. Hence, with regard to drug efficacy, the more stable the baseline, the easier it is to evaluate small drug dosage or subtle changes due to drug effects.

The original purpose of this study was to see to what extent it was possible for AD subjects to respond to changing behavioral chains with stability during a baseline condition. Since only one subject accomplished this goal, it may seem unreasonable to recommend the repeated acquisition technique as a testing procedure for drug outcome studies with AD. However, Thompson and Boren (1977) have suggested that absolute baseline stability may not be a necessary antecedent to the evaluation of drug effects:

Baseline stability, while it makes drug work more convenient and exact, need not be a critical consideration. Semi-stable procedures may permit useful observations which are not possible with the more conventional techniques (p. 543).

Kazdin (1983) further supports this argument by suggesting that an initial trend during baseline need not interfere with drawing inferences about interventions when various design options, such as reversal designs, multiple-baseline designs, and changing-criterion designs are employed. In addition, he suggests that a variety of data evaluation techniques which utilize statistical applications, such as time-series analysis, can reduce ambiguity about intervention effects. However, both Kazdin (1983) and Thompson and Boren (1977) caution that with relatively large variability in baseline data, stronger interventions are needed to infer a systematic change in behavior. Therefore, further
research is necessary to investigate the complexities of experimental control with this technique prior to applying repeated acquisition to drug outcome studies with suspected AD subject.

The present study has, however, demonstrated that the repeated acquisition technique is sensitive to subtle behavior change with suspected Alzheimer's subjects. And, further, has attempted to provide insight into the eventual emergence of operant research into the study of the behavioral consequences of Alzheimer's disease.


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