The nature of the memory impairment in Korsakoff patients.

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THE NATURE OF THE
MEMORY IMPAIRMENT IN KORSAKOFF PATIENTS

A Thesis Presented

By

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MEMORY IMPAIRMENT IN KORSAKOFF PATIENTS

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Abstract

Korsakoff and Alcoholic patients were examined under a variety of conditions which were designed to delineate the functional nature of the amnesics' memory impairment. The Korsakoff patients were found to exhibit the classical anterograde amnesia when the information to be recalled was verbal in nature. In general, the impairment was not observed in non-verbal or verbal recognition tests. It was concluded that the behavioral deficit observed in Korsakoff patients stems from an inability to generate verbal codes for information acquired after the development of the neural lesion.
INTRODUCTION

Korsakoff patients and patients sustaining bilateral hippocampal resection exhibit a phenomenon referred to as anterograde amnesia (A.A.). Researchers have generally interpreted this memory deficit as being an impairment of transfer from short term store (STS) to long term store (LTS). This disruption of the transfer process is often described as an impairment in consolidation. The primary symptom exhibited by both classes of patients is an inability to remember events which they have just experienced; they seem incapable of learning anything new. However, they do retain motor and verbal behaviors acquired prior to the onset of the disease which enable them to respond appropriately to their environments.

The Korsakoff syndrome was first described in 1889 by the Russian psychiatrist, Sergei S. Korsakoff. He believed that chronic alcoholism was the sole cause of this degenerative psycholpolyneuritis, as did most investigators until recent research suggested that alcoholism acted only indirectly as the cause by inducing a group B avitaminosis (particularly thiamine) as a consequence of the chronic alcoholics impaired gastrointestinal absorption and a diet primarily restricted to vitamin-free alcohol. (Barbizet 1963, 1970; Brio 1969; Kolb 1968; Talland 1965). Recent evidence has suggested that low levels of endogenous magnesium may be responsible for the thiamine deficiency (Traviesa 1974). Even more recent evidence (Korsten, et al., 1975) however, has questioned the
involvement of nutritional deficiencies as the cause of the disease. It now appears that the high acetaldehyde levels in alcoholics, due to their inability to break it down as quickly as non-alcoholics, may be responsible for the degenerative polyneuritis. As a result of the biochemical lesion, Korsakoff patients suffer from diffuse neural lesions which enroach bilaterally upon the medial diencephalon. Currently, there is no definite consensus as to which structure(s) must be damaged to produce the disease. Most investigators, however, implicate the mammillary bodies as the primary site of neural degeneration in the Korsakoff amnesic syndrome (Barbizet 1963, 1970; Barbizet and Cany, 1969; Brio, 1969; Talland, 1965).

Much experimental evidence has accrued which indicates that amnesia patients apparently have deficits in short term memory (STM). Talland (1965) reports that Korsakoff patients are able to keep information in STM by rehearsal as long as their attention is not diverted. However, these patients forget as soon as their attention is shifted from the information to be remembered. Milner (1970) has observed the same finding with her hippocampal patients. She reports that her most famous patient, H.M., was able to retain the number 584 for at least 15 minutes by means of an elaborate mnemonic scheme. After a new, unrelated topic of conversation was introduced, H.M. was asked how he was able to retain the number for so long. He was unable to recall the number, the mnemonic device, or that he had been given a number to remember. Milner
states that H.M. forgets the instant his focus of attention is diverted and that the only manner in which he can keep new information accessible is through constant verbal rehearsal. Cermack, Butters, and Goodglass (1971) found deficits of STM in Korsakoff patients when they were tested in the Peterson and Peterson paradigm. Subjects were presented with consonant trigrams, single three letter words or groups of three 3-letter words, and then distracted from rehearsing by being required to count backwards from 100 by three's. This distracting task lasted either zero, three, nine or 18 seconds. At the end of this interval, the subjects were asked to recall the items which were initially presented. In the consonant trigram and the one word conditions, the Korsakoff subjects were impaired relative to normals and alcoholics at the nine and 18 second delays. In the word triad condition, the Korsakoff subjects were impaired relative to normals and alcoholics at all delays. The authors took these results to indicate that Korsakoff patients have a deficiency in STS. Corsi (1969—cited by Milner, 1970) observed deficits in the Peterson task in left hippocampal patients when consonant trigrams were used as stimuli. Prisko (1963) found a STM deficit in H.M. when non-verbal stimuli were used in a delayed-pared comparison task. The classes of stimuli were click frequency, flash frequency, tones, shades of color, and nonsense figures. The task consisted of successive presentations of two stimuli, separated by some variable time interval. The subjects were required to compare the second stimulus with the initial stimulus and
respond "same" if they matched, or "different" if they differed. H.M. was impaired on all five classes of stimuli when the delay was 60 seconds. Prisko and others have taken this to indicate that hippocampectomized patients have deficits in STS for non-verbal stimuli.

Standard clinical tests of memory indicate that Korsakoff and hippocampectomized patients exhibit deficits in consolidation (the transfer of information from STS to LTS), for both verbal and non-verbal stimuli. Barbizet (1970) has observed that Korsakoff patients are unable to consolidate memories, in varying degrees, after the onset of their disease. Cermak, Butters and Goodglass (1971) demonstrated the Korsakoff patients' deficit in consolidation by means of a paried-associate (PA) task. Six PA three letter words of high frequency (Thorndike-Lorge A or AA) were presented for a maximum of 16 trials or until the subjects correctly anticipated each of the PAs. This procedure was repeated over four successive days. The Korsakoff subjects were found to be significantly impaired, in terms of mean number of trials to learn the PA list, relative to alcoholic controls on every day except the fourth. The authors concluded that one component of the impairment appears to be a problem of transferring information from STS to LTS. Scoville (1954), Scoville and Milner (1957) and Penfield and Milner (1958) have reported that patients sustaining bilateral resection of the hippocampal zone are also impaired in their transfer of STM to LTM. Penfield and
Milner (1958) report deficits in consolidation as measured by story reproductions of the Logical Memory test. In this task, short stories are read to the subject and at variable time intervals afterwards, the subjects are asked to recall the story from memory. The hippocampal subjects' performance on immediate recall is consistently poor and on subsequent retests the patients often do not recall ever having heard the story. Thus, patients sustaining bilateral resection of the hippocampal formation suffer from "a generalized memory impairment which cuts across the distinction between verbal and nonverbal material or between one sense modality and another", (Milner and Teuber, 1968).

The results of the preceding studies might suggest that since the consolidation mechanism in amnesics is impaired, then their LTM should also be deficient. However, this in fact is not the case. In both Korsakoff and hippocampectomized patients, a retrograde amnesia (RA) is observed (Barbizet, 1970; Penfield and Milner, 1958). RA is a memory deficit for information acquired prior to the precipitating event that induced the impairment. The RA is transient, in the sense that it attenuates with time; usually however, there exists a complete amnesia for experiences immediately prior to the precipitating event. The period where RA and AA diverge in Korsakoff patients is hard to determine since all estimations of the point in time where they separate are made ex post facto. In addition to this, Korsakoff patients develop the
lesions gradually, over the period of years. As such, a precise estimate may not be possible. However, for both types of patients, memories for remote events consolidated before the RA are intact and remain quite good. Seltzer and Benson (1974) gave Korsakoff patients a questionnaire which dealt with public events that covered the past 50 years. They found that Korsakoff subjects performed very poorly on items dealing with recent events which presumably fell in the period of their RA, but performed at the same level as normals for the remote events. Hippocampal patients are also able to remember events in their distant past when asked (Scoville and Milner 1957). These findings verify that the mechanisms of LTS and retrieval are intact in amnesic patients. It will be recalled that Cermack et al., (1971) reported deficits in Korsakoff subjects' PA performance on the first three of the four days of testing. On the fourth day of PA testing, the Korsakoff subjects were found to be unimpaired as compared to control alcoholics. This would suggest that some information does get consolidated into LTM and is retained at levels comparable to those of normal subjects. Warrington and Weiskrantz (1968) reported savings over a period of three days on a perceptual task in amnesic patients (five Korsakoff patients and one temporal lobectomized patient). Subjects were presented with a graded series of fragmented words (the words were selected from the high frequency category of the Thorndike-Lorge word count) and fragmented drawings as stimuli. There were five different drawings of the same object, ranging from very incomplete to complete
representation. The words also ranged from very incomplete to complete, but there were only four representations of each word in a given series. In both sets of stimuli, the most incomplete representation was presented first and the more complete drawings followed until the subjects correctly identified the stimulus. Testing was carried out over three successive days. Their results indicated that amnesics performed more poorly than control patients sustaining peripheral neurological lesions on both sets of stimuli over trials and over days in terms of their recognition error rates. Although the amnesic patients were impaired relative to the controls, they did show savings across the three test days with higher savings demonstrated for the non-verbal stimuli than for the verbal stimuli.

In 1970, Weiskrantz and Warrington conducted a similar study to the one just described which also showed evidence of LTM. They presented fragmented words which were presented in the order of 20% complete, 50% complete and totally complete as stimuli to 6 amnesic patients and 6 neurologically diseased patients without memory deficits. On each trial, eight words were presented. All subjects were trained to a criterion which required that they recognize all the words of two successive trials at the level of 20% representation. All the subjects were tested for retention one, 24 and 72 hours after the initial criterion was met. At retention delays of one and 24 hours, the amnesic patients were found to be impaired relative to the controls. But at the 72 hour delay, the amnesic
patients were performing as well as the control patients, arguing again for some information being consolidated. Milner (1962) has gathered comparable results from H.M.'s performance on the incomplete drawing task. During the first test, H.M. performed better than the normal controls, which is consistent with his high performance on various other perceptual tests. One hour after initial testing, H.M. was retested and was found to have reduced his errors by 48%. Twenty weeks later, H.M. was retested again and his performance was virtually identical to the initial retest. However, on being confronted with the task in each retest situation, he reported never having seen the test before. H.M. also showed nearly normal learning curves across sections in three non-verbal tasks: Rotary Pursuit, Bimanual Tracking, and Tapping (Corkin, 1968). Since LTS for recent events has been demonstrated in amnesic patients, it follows that consolidation has also been demonstrated. Although the nature and extent to which these patients possess these capacities are markedly different from that of normals, in certain circumstances and situations these phenomena are capable of being demonstrated.

In 1970 Baddeley and Warrington conducted a classic study which demonstrated that STM functioning is intact in amnesic patients. In their first experiment, a free recall task was employed. A total of 20 lists of 10 nouns were used. For 10 of the lists, recall was tested immediately following presentation of the tenth word. For the other 10 lists, recall was
tested 30 seconds after presentation of the tenth word with an interpolated counting task interjected to minimize rehearsal. It has been demonstrated that one component of this task reflects STM by a high probability of recall for the last few items (the recency effect). In the immediate recall condition, amnesic patients performed as well as, if not better than, control patients with peripheral lesions on the last few items. In the delayed condition, amnesics also performed comparable to controls on the last one or two items. This finding strongly suggests that amnesic patients have unimpaired STM. In a second experiment, subjects were presented with sequences of three 3-letter words. Recall was tested zero, five, 10, 15, 30 and 60 sec. after initial presentation. At delays greater than zero seconds, subjects were required to count backwards from a given number which was presented after the test stimuli were administered. They found no differences between amnesics and controls in terms of percent recall. This finding is consistent with the hypothesis that amnesic patients have normal STM. A PA test was conducted where four pairs (stimuli were nouns, responses were adjectives) were presented on cards followed by a fifth card containing the stimulus item from one of the pairs. There were a total of 40 lists of four pairs. No significant differences were found between amnesic and control patients regardless of which item in the list of four was used as the recall stimulus. Baddeley and Warrington also conducted a digit span test, which is a
standard measure of STM. Digits were read to the subjects and there were six sequences of five, six, seven and eight digits. In terms of percent correct strings recalled, amnesics and control patients performed equally well at each string length. This again is consistent with the hypothesis that amnesic patients have a normal STM. This finding is in line with Drachman and Arbit's (1966) finding that hippocampal patients also have normal digit span when tested with a number of items that do not exceed STM capacity. Research has been cited that demonstrates the existence of STM, consolidation and its consequence, LTM. Yet, when one is confronted with these patients, after well directed questioning, it is obvious that there is some sort of a deficit that is manifested by their verbal responding. The most parsimonious account of the nature of the deficit is readily abstracted from the following studies. In 1972, Cermack and Butters read a serial list of eight words, two of which were from each of four categories: names, professions, vegetables and animals. Two of these lists were read to the subjects (Korsakoffs and alcoholic controls) with a five minute interval separating them. In the first test, subjects were simply requested to freely recall the presented words. After their recall performance, subjects were asked whether they had noticed whether the words had belonged to the categories mentioned above. Subjects were then instructed that a second list with words from the four categories would be presented and that they would be tested by
recalling words from the particular categories. The authors found no significant differences in performance between the two groups in the free recall condition, but in the cued condition the Korsakoff patients were significantly impaired relative to the alcoholic controls who had benefitted from the cuing and recalled more items than in the free recall condition. These results suggest that a failure in verbal encoding may be intrinsic to the amnesic's impairment since the patient is able to retain verbal information (words) on a rote basis for short time periods, but if forced to recall verbally encoded material, he demonstrates that his own encoding isn't precise enough to allow for retrieval. Further evidence corroborating the verbal encoding deficit hypothesis came from Cermack, Butters and Gerrein (1973). They presented 60 words to their subjects. Each word had one of the following relationships to other words on the list: 6 were unrelated or control words; 6 were synonymous (e.g., glad-happy); 6 were homophones (e.g., sea-see); 6 were high associates (e.g., table-chair); 6 were repeated within the list and the rest were filler words. Subjects were presented with list words and asked whether the item had been previously presented within the list. The subject's response was either a "yes" or a "no". Korsakoff subjects were found to make significantly more homophone errors and associate errors than did alcoholic controls. Korsakoff subjects also made more homophone and associate errors than synonym and neutral errors; however, the
number of homophone and associate errors did not differ from each other. Control subjects had no significant differences among types of errors. This demonstrated that Korsakoff subjects differ in their encoding strategies from control subjects. They rely upon associative and acoustic dimensions instead of more advanced semantic dimensions. This suggests that Korsakoff patients are either unable to spontaneously utilize semantic dimensions effectively or that they are incapable, to a degree, of semantic encoding. To further test the hypothesis that Korsakoff patients have a deficit in verbal encoding, Butters, Lewis, Cermack and Goodglass (1973) investigated Korsakoff, alcoholic and normal subjects' behavior on verbal and non-verbal tasks across three sensory modalities. The Peterson and Peterson (1959) distractor technique, with delays of zero, nine and 18 seconds and a delayed-comparison technique were used on visual, tactile and auditory tests of verbal and non-verbal memory. The visual stimuli were consonant trigrams and computer generated random figures. The auditory stimuli were consonant trigrams and a random sequence of five piano notes, both played through a headset that the subjects wore. The tactile stimuli were raised outlines of English letters and raised unfamiliar figures consisting of four serially connected lines. Across all three modalities there were no group differences at zero delays with the verbal and non-verbal stimuli. In all of the non-verbal conditions, the Korsakoff subjects did not differ from control subjects at
either nine or 18 seconds. However, in nearly all delay trials using verbal stimuli, the Korsakoff subjects made significantly more errors than control subjects. These results provide strong evidence for the hypothesis that amnesics are incapable of acquiring new information which is verbal in nature or demands verbal strategies or mediators for retention, since they were severely impaired in the retention of verbal material irrespective of the sensory modality in which the information was conveyed. This deficit in verbal encoding is also a likely candidate for the explanation of the hippocampal deficit. Hippocampal patients exhibit normal learning curves over a three day period when tested in a mirror tracing task. In this task, subjects must draw a line within the outline of a double lined five point star, observing both hand and star as reflected from a mirror (Milner, 1962). The normal performance of hippocampal patients on this task, the Incomplete Drawing, Rotary Pursuit, Bimanual Tracking and Tapping Tasks, is easily understandable since they presumably require no verbal mediation. Prisko's results would appear to contradict this interpretation except for the facts that 1) of the four hippocampal patients tested, only one showed the clear cut impairment, 2) the task instructions were given verbally, 3) H.M.'s performance on delays shorter than 60 seconds was not indicative of a complete memory loss since he was performing well above chance and 4) loss of stimulus control could not be assessed since she did not present discrimination gradients.
In 1968, Sidman, Stoddard and Mohr performed a series of tests on H.M. that demonstrated that he was capable of coming under the influence of stimulus control and that this control was maintained even after verbal distraction. H.M. was initially presented with a series of ten slides that were projected onto a panel that had key arranged in a 3 x 3 matrix. In these slides a circle was projected onto one of the nine keys while the other keys remained dark. When he pressed the key that had the circle on it, the response was reinforced with a penny. After quickly acquiring this response in the absence of verbal instructions, a new set of ten slides was introduced where one key again had the circle projected onto it and the other eight keys now had flat ellipses (vertical to horizontal ratio (vhr) = .53) projected upon them. H.M. still maintained his circle pressing behavior in this new situation. In a new set of slides, the vertical axis of the eight ellipses were lengthened from trial to trial approaching 1.0. H.M. consistently chose the circle until the ellipses reached a vhr of .93.

Next, 32 slides were shown, each having one circle and seven different shaped ellipses (vhrs of .74, .77, .80, .83, .86, .89 and .91) which changed position from trial to trial with the center key remaining dark. This test was termed "discrimination gradient series". Although H.M. did not reliably choose the circle throughout this test, his steep circle-ellipse discrimination gradients indicated that his choices were primarily determined by the stimulus dimension correlated
with reward. After completing the 32 slides, H.M. was interrupted by asking him to count his pennies and then to describe what he had done to earn them. His verbal behavior was indicative of a person who had amnesia in the sense that he talked about X's and multiplication signs (confabulation), but when he was put through the discrimination-gradient series again, he continued pressing the circle.

After several replications of the above procedure H.M. was placed on a delayed matching to sample with adjustment (titration). In this procedure, a stimulus was projected onto the center key, then concurrent with the stimulus being taken off the key, eight other stimuli were projected on the panel. The correct response was to press the outer key that had the same stimulus as the center key initially had. With each success, the delay was increased and with each failure, the delay was decreased. When consonant trigrams were used as stimuli with a four second titration step, H.M. had no problem remembering the trigrams for up to 40 seconds. When ellipses of various vhrs were used however, the titration step had to be limited to one second and the longest delay in which he could correctly respond was five seconds. Sidman et al., hypothesized that the reason for H.M.'s good performance with trigrams and poor performance with ellipses was due to the fact that the trigrams provide their own verbal code (and thus allowed him to rehearse them) whereas the ellipses don't. H.M. was observed to form the sounds of the letters with his lips, giving him access to some verbal coding system. In the case
of the ellipses however, no code is provided. Normal subjects are known to devise a verbal code such as larger than, smaller than, etc., in order to aid them in their performance. Although H.M. was not asked whether he generated such a coding system, his behavior suggested that 1) he did not devise a verbal coding system or 2) if he did generate a coding system, it was too inefficient to effectively aid him in his performance. Since his operation, H.M. has never been observed to provide himself with any sort of a verbal coding system (i.e., notes to himself as reminders).

Thus, the impairment is most often observed during verbal recall of both non-verbal and verbal material. The observations that H.M. was able to correctly recognize the trigrams at a 40 second delay, but was unable to describe the Discrimination Gradient Series stimuli in their absense or reliably recognize sample ellipses at delays longer than five seconds, suggest that the deficit may be largely specific to the self-generation of verbal codes for recently acquired information.
PURPOSE OF THE PRESENT INVESTIGATION

Since there is a paucity of data making direct comparisons between Korsakoff and hippocampal patients, on any behavioral tasks, it would seem of importance to assess the similarities or differences on identical behavioral measures in light of the fact that their disorders seem functionally equivalent. If I observe behavior that is similar to H.M.'s in Korsakoff patients, then it would seem reasonable to suggest that the functional nature of their deficits, as measured by these tasks, is similar and that their memory impairment stems from a deficit in verbal encoding. The neuroanatomical structures involved in both of these deficits are directly connected by the fornix. Sweet et al., (1959) reported that a lesion of the fornix in one patient resulted in anterograde amnesia. Although this patient has not been extensively studied to assess the nature of his deficit, the clinical report indicates that his impairment is similar to the Korsakoff and hippocampal deficit. Thus, it would appear that the hippocampal-fornix-mammillary body system may mediate a function that is similarly disrupted in Korsakoff and hippocampal patients (Adams, 1969; Barbizet, 1963, 1970; Barbizet and Cany, 1969).

If however, I observe behavior in the Korsakoff patients that is dissimilar from H.M., then it seems necessary to conclude that the functional nature of their deficits, as measured by these tasks, are not the same and that the lesioned neural structures do not mediate similar functions.
The second intent of this work is to delineate the nature and degree of possible verbal encoding deficits in Korsakoff patients. The scientific importance of such an assessment has theoretical implications for an understanding of memory function. For years, amnesia patients have been studied and treated as though they suffered specifically from a global anterograde amnesia. This investigation intends to more specifically isolate what the nature of the deficit is and consequently improve our understanding of the process(es) underlying pathological memory states. To assess this deficit, five experiments were conducted investigating perceptual performance, non-verbal recognition, verbal recognition and verbal recall.
SUBJECTS

Four patients residing at the Northampton Veterans Administration Hospital served as subjects in the following studies. The two amnesic patients were selected from a total of 26 amnesics on the basis of diagnosis, I.Q. and memory deficit, as assessed by clinical measurements. The criteria for selection were that 1) the patient was diagnosed as having Korsakoff psychosis, 2) the patient had a minimum full scale I.Q. of 89 and a minimum performance I.Q. of 84, as measured by the Wechsler Adult Intelligence Scale and 3) the Korsakoff patient's Wechsler Memory Scale Quotient was at least one standard deviation below his full scale I.Q. To assess these patients' current I.Q. and memory quotient scores, the experimenter administered the WAIS and Wechsler Memory Scale approximately one month prior to testing.

Two patients from the alcoholic ward were matched as closely as possible to the Korsakoffs for age, sex, I.Q., educational background, socio-economic history, race, and geographic background. Neither of the two alcoholic controls were observed to suffer from any apparent memory deficit. The data on the above variables for each of the patients is summarized in Table 1.
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J.P. left the hospital in the initial phase of testing however, his data from the tests he completed will be presented.
APPARATUS

The primary apparatus was originally developed in Sidman's laboratory and consisted of a panel with an array of nine translucent keys. The panel has been described in detail elsewhere (Sidman and Stoddard, 1966, 1967; Sidman, Stoddard and Mohr, 1968; Sidman, 1969). The panel used in this series of studies was slightly modified from Sidman's original apparatus. Briefly, the panel consisted of a 3 x 3 matrix of square keys. The keys were 4" squares of clear 1/8" plexiglass backed with white contact paper and were mounted with hinges behind 3" square holes in the flat black panel. The 18" square panel was mounted on a non-skidding base.

A slide projector, situated behind the panel projected stimuli onto the keys. When the stimuli were projected, the patient's task was to press one of the keys. A microswitch located behind the key signaled the patient's choice to the control panel and if the correct key was chosen, a token was automatically delivered from the dispenser into a metal hopper located next to the panel. All of the panel stimuli were photographed with H & W high contrast copy film and mounted in slide mounts. For the picture recognition task, a regular screen and two slide projectors were used. The stimuli used in various tests will be described in the procedure section.
PROCEDURE AND RESULTS

The majority of the testing was carried out in the conference room of Ward 2 lower of the Northampton Veterans Administration Hospital. Testing was carried out in 15 sessions per subject, with an individual session lasting from 10 minutes to three hours. The testing conditions and procedure were kept as uniform as possible for all subjects. The reinforcers were tokens which could be exchanged for money or cigarettes.

Shaping

The intent of this test was simply to determine whether subjects were capable of acquiring a circle-pressing response with a minimum of verbal instruction.

Procedure

All subjects were initially given a series of 10 slides, each of which presented a white outline of a circle with a grey background on one of the keys; the remaining eight keys of the matrix were grey. The only verbal instructions that the subjects received was "Press the key that you think will give you a token". The intertrial interval (ITI) was approximately one second.

Results

Figures 1K1, 1K2, 1A1, and 1A2 summarize the results of the four subjects' initial circle-pressing response. An upward step indicates a correct response. None of the subjects
encountered any difficulty acquiring this response, as indicated by the continually rising step graphs.

Circle-ellipse Discrimination

The purpose of this test was to determine whether Korsakoff patients were able to acquire a discrimination between circles and ellipses at a level similar to the controls and the reported data on H.M. in the absence of verbal instructions.

Procedure

Subjects were given a series of 10 slides in which each of the formerly grey keys now had a white ellipse on a grey background. The ellipses and the circle had the same horizontal axis length but the vertical to horizontal ratio (vhr) of the ellipses was .50. The circle and ellipses changed positions from trial to trial with the center key remaining grey. Subjects were reinforced for pressing the circle with a token; the ITI was approximately one second and no verbal instructions were given.

Results

The performance of subjects on this task appears in figures 2K1, 2K2, 2A1 and 2A2. A horizontal line with an open triangle pointed down denotes an error; a dark triangle pointed up or a step upward signifies a correct response. As can be seen in Figure 2K1, Kl's responses were not controlled by the stimulus correlated with reward since he only made two correct responses occurring on the second and fourth trials.
The shift from shaping to circle-ellipse discrimination probably was not gradual enough for him to respond appropriately to the stimulus. Since there were no intermediate fading slides, it was decided to run K1 through the shaping series for a second time. Figure 3K1.1 indicates that K1 had not forgotten the task, scoring perfectly as he did the first time through. He was then placed back on the circle-ellipse discrimination task and made no errors, as is shown by the steadily increasing plot in Figure 3.K.1.2. Both K2 and A1 made errors in the initial trials (the first four and three trials respectively), however, once they were reinforced for their first correct response, neither of them made another error (Figures 2K2 and 2A1). A2 (Figure 2A2) understood the requirements of the task from the beginning since he made no errors.

Determination of Circle-ellipse Threshold

This experiment was conducted so that a determination of the subjects' circle-ellipse difference threshold could be made. The procedure used was a modification of the measurement of difference thresholds by the method of limits, as described by Underwood (1966).

Procedure

A total of 21 slides were presented with each slide having one circle and seven ellipses (the center key had no figure on it). The vertical axes of the ellipses were
lengthened over the series, approaching a vhr of 1.0. The vhrs were .500 on two slides, .574 on two slides, .643 on three slides, .707 on three slides, .776 on three slides, .819 on four slides and .906 on four slides. Presentation of the ellipses began with .50 and would sequentially increase to a vhr of .906, given perfect performance. The position of the circle and ellipses changed from slide to slide. If the subject made a mistake, picking an ellipse instead of the circle, the last slide in which the subject correctly chose the circle was readministered. On some occasions, this back-up procedure returned the subject to an easier discrimination, while on other occasions the discrimination required was the same except that the circle was in a different position (Sidman and Stoddard, 1966; Sidman, Stoddard, and Mohr, 1968). This task allowed the experimenter to detect gross perceptual deficits, if any existed in the subjects. The ITI was approximately one second.

Results

K1 (Figure 4K1) was able to successfully discriminate between the ellipses and the circle until he reached a vhr of .906. Thus, his circle-ellipse threshold was .819 since he was unable to correctly distinguish the ellipses having a vhr of .906 from the circle.

K2 (Figure 4K2) encountered some difficulty in procedure on the first, fifth and eighth trials, making errors at a vhrs of .50 and .643. However, after his third error, he
correctly distinguished the ellipses from the circle up to a vhr of .906. Although he made an error in distinguishing between the circle and ellipses having vhrs of .819, this occurred only once in 11 trials at the .819 vhr value. Thus, it is reasonable to conclude that his circle-ellipse threshold is .819.

Both control subjects, Al and A2, were able to distinguish the circle from the ellipses having vhrs of .906 without any difficulty (Figures 5A1 and 5A2, respectively). Their performance on this task was perfect, except for one error made by Al on Trial 17, in distinguishing the circle from ellipses having vhrs of .819. This, however, only amounted to one error in six trials at the .819 vhr level. Therefore, both controls were considered to have circle ellipse thresholds of at least .906.

Discrimination Gradient Series (DGS)

The purpose of this experiment was to yield a quantitative analysis of retention and to utilize a more sensitive measure of the degree of stimulus control in both types of patients. H.M.'s behavior has been observed to remain under strict stimulus control in this task after having been distracted, although his verbal responses indicated an apparent memory loss for the task (i.e., he confabulated). This seemingly paradoxical finding warranted investigation in Korsakoff patients to determine any similarities that may exist
between both types of impaired patients on this behavioral task.

Procedure

Thirty-two slides, each of which projected a circle and seven different ellipses (vhrs of .500, .574, .643, .707, .776, .819, and .906) onto the matrix with the center key always remaining grey. The circle and ellipses changed position randomly from one slide to the next. When the subject was presented with an ellipse vhr which he could not distinguish from a circle, he would oscillate between slides, one in which he could make the discrimination and the other in which he could not tell the ellipses from the circle. The subjects' threshold was the ellipse vhr beyond which he no longer consistently made correct choices. In the graphs of this experiment, an upward step or a dark triangle pointed up indicates a correct response whereas a step downward or an open triangle pointed downward denotes an error. The key pressing response was reinforced with a token when the subjects pressed the key that had the circle on it. After the subjects had completed the first DGS series, they were asked to count their tokens and then to describe the experiment. After completing the second and fourth runs of the DGS, subjects were again asked to count their tokens and to describe the experiment. The subjects' verbal behavior was recorded on a cassette tape recorder and the ITI was approximately two seconds.
Results

The results of Kl's performance are shown in Figure 6. In all five of the tests, Kl distributed his responses between the circle and the .906 ellipse. Since his circle-ellipse threshold was .819, he was probably unable to distinguish the .906 ellipse from the circle. Because all of his responses, except one (Figure 6K1) were distributed between the .906 ellipse and the circle and he was incapable of discriminating between those figures, it may be inferred that his behavior was primarily controlled by the stimulus dimension correlated with reward. After completing the first, second, and fourth sets of 32 slides, he was asked to count the number of reinforcers that he received and then asked to describe the tasks. Below are the transcripts of the recorded conversations that took place between Kl (S) and the experimenter (E).

Following the first DGS test:

E Tell me what you did to earn the tokens.
S Over there, you mean?
E Yeah.
S I got to get the circle.
E You got to get the circle?
S Yeah.
E What do you mean, you got to get the circle?
S I had to get the circle to get a token.
E Right. So in other words you would press the circle.
S And get a token.
E And get the token. And what else was there on the board?
S Well all different shaped circles.
E All different shaped circles. Could you describe them?
S Well, there was some oval shaped and some wider than they were long and all different shapes.
E And to get the tokens you would have to do what?
S Get the perfect one.
E The perfect one. Which was the perfect one?
S The perfect circle.
E The perfect circle?
S Yeah.

Following the second DGS test:

E All right A., have you seen me before?
S I've seen you around here before.
E Where, here?
S Yeah.
E On this ward? (Nod from Kl) I see. Well, what's my name?
S I don't know your name.
E Have I ever told you?
S Not that I know of. (E had told S his name many times)
E Yeah. So we have seen each other before?
S Oh sure.
E You recognize my face?
S Yes.
E You do? OK A., what did you do to earn your tokens?
S Press the right button.
E Which one was the right button?
S The perfect circle.
E The perfect circle. And what was on the others?
S Different shaped circles.
E Different shaped circles. I see. So when you'd press that one you'd get a token.
S A token.
E And would you always press the perfect circle?
S Not always, I'd miss once in a while.

Following the fourth DGS:

E Why don't you tell me what you did to earn your tokens.
S Press the right button.
E You'd press the right button? Uh...
S The circle, got to get the circle.
E Got to get the circle. OK.

(A few minutes later)

E A., did you get any tokens?
S When?
E A little while ago.
S I didn't do anything with any tokens.
E You didn't do anything? Did you do anything just a few minutes ago? What were you doing a few minutes ago?
S Press the right button to get the right circle.
E Were you getting tokens?
S Well, if you got the right circle.
E You did very well; you got a lot of money. What else was there? Was there anything besides the right circle?
S There was all shaped circles?
E All different shapes. And what did you have to do to get the tokens?
S Hit the right one.
E Which one was the right one?
S The perfect circle.
E You did well at it.
S It wasn't hard. I'm just waiting to go home...

The fact that Kl evidenced no apparent memory deficit was not a surprise to the experimenter since it was known that Kl could tolerate retroactive interference over short periods of time, as assessed by questioning when I first met the patient. It was then decided to run Kl through the task once more, 24 hours after completing test D. The following
is a transcript of the dialogue that took place between the
E and S immediately prior to the fifth DGS test.

24 hours later in the same room:

E Have you ever been in this room before?
S No.

E A., then you've never been in this room before?
S Of course not.

E But you've seen me before.
S Oh yes.

E Have I ever told you my name?
S No.

E Well I'm Fred. A., did we see each other yesterday?
S Gosh, I don't remember if we did or not.

E Well, we did.
S We did.

E You see that panel over there?
S Yeah.

E Have you seen that before? Put on your glasses please.
S I don't need my glasses, I can see it all right.

You mean the wall there?

E No, no the thing thats got...
S Oh, this here?
E Yeah.

S Oh, I don't remember seeing that before.
E Are you sure?
S Yeah. It might have been here yesterday, but I....
E Look at it; look at it closely.
S Well I've seen it, one like it or that one before.
E Do you remember what you did with it to get tokens?
S No, no.

The following passage is the conversation that took place after KL completed the fifth DGS test.

E Tell me what you did to earn your tokens.
S Well, I had to press the right lever or right button or whatever you call it.
E And what was on the right button?
S Perfect circle, wasn't it?
E Yeah. And what was on the rest of them?
S Odd shaped circles.
E Had you ever done it before?
S No.
E This is the first time you saw it?
S Yeah.
E I see.

E So the one you were supposed to pick...
S Was the perfect circle.
E OK.
Figure 14Kl.1 demonstrated that Kl was still responding along the dimension correlated with reward even after 24 hours, even though his verbal behavior would have suggested otherwise. A possible explanation for this discrepancy could be that Kl actually had forgotten the task but had relearned it rapidly. This interpretation is rather unlikely since his choices were quite accurate, even on the first trial of the fifth DGS test (Figure 14Kl.2).

K2's responses were also under good stimulus control in this task as shown by the relatively steep gradients (Figures 6-9K2). If there had been no relation between the stimuli correlated with reward and his selections, the gradients would have been flat. K2 was asked what he had to do to earn his tokens after the first, second, and fourth DGS tests. The conversations are presented below.

Following the first DGS test:

E  Now tell me, what did you do to earn those coins?
S  What did I do to earn 'em?
E  Yeah.
S  Well I did that number on the...
E  Numbers on the?
S  On the board there. Whatever they are, first read on 'em. Had 'ta listen 'ta the click of yours then.
E  So there were numbers on the board.
S  Well, uh, such as they are. I call 'em numbers.
They were. What type of numbers?

Well, whatever you press there. There was no number on there only light flickering.

There was light flickering? Well, what kind of light?

Well it goes, changes colors on me, get tired. Sure, yeah, colors change on me and it goes out the others stay on.

Right, I see. Um, so then the color would go out and then they would come back on again.

That's right.

I see, well what colors were they? What kind of colors?

Well they just, well they all were the same dark color.

They were?

Yeah, all the same color one goes off the others stay lit.

So that's what you do to earn your money?

That's what I had to do there.

I see, OK.

Following the second DGS test:

Tell me what you did to earn those tokens.

Well I had to pay attention to the light there.

The light?

And listen.
And listen?
To the sound.
But what did you do to earn the tokens?
Well I had to make sure I watched the lights and kind of obeyed like you'd say, had my mind on my work; whatever I'm doing.
And what was your work?
Be sure to watch them lights.
The lights?
Yes, hit them right so I'd earn some money.
And how do you get the lights right?
Why by watching it, make sure I was watching them lights there and listen to the click.
What did the lights look like?
Well, different colors, one's dark and the other's light.
I see.
What's the difference on them buttons there is that one would be light and the other would be dark. That's what I followed by.
You followed the one that was light?
Yeah.
And the other one was dark?
Right.
I see.
We had two differences in the lights.
So you're saying there were two, one was light and one was dark.

Yeah.

You picked the light one?

Well I had to be talking to which ever one that was snapped on there. The light one or, the way it lit up. Which was one went darker and pick on that one if you're gonna light it or one that flickered different one and pick on that one.

I see.

Opposite from the rest of them there.

Opposite from the rest of them?

Yeah, they had to be different from what the rest were.

How?

Why, by one bein' light and the rest be dark.

I see.

Or else that one would be dark and the rest of 'em would be light.

Uh huh, I see, OK.

Following the fourth DGS test:

Tell me what you did to get these tokens.

Well, I had to obey the, according to the lights there.

What lights? What were the lights like?

Why, certain ones there'd light up, I had to be quick to make sure I got the right light on, I'd have to press the right button for the right light.
What did the light look like?
Well, one was light and the other would be dark.
So there were two then?
Yeah, there were two of them. You had to make sure to get the right light.
So which one was the good one?
Which one, the one that was on from the rest of them.
I see.
Had to make sure I hit that one.
So the rest of them were off?
Yeah.
And the one that was on?
That's the one I had to hit every time.
I see. And, uh, what did the light look like, or was it just a light.
Well, one was light and the other one was....
Was dark.
Was dark, that's all.
I see. So it was just a light.
Like yeah,...and one was dark and the other was a just like a bulb goin' on and off.
So there was no figure?
No, blank like.
I see.

Clearly, K2's verbal responding was characteristic of amnesic patients; upon questioning, he confabulated. However,
inspection of Figures 6-9K2 and 10-13K2 indicates that his choice, like Kl's were related to the dimension correlated with reward.

Both of the control subjects' responses were largely controlled by the circle. Al made a few errors (Figures 6-9A1 and 10-13A1) but these were made when he chose the ellipse which most closely resembled the circle. A2's responses (Figures 6-9A2 and 10-13A2) were under complete stimulus control since he did not make one error (i.e., he always chose the circle). These two subjects' verbal accounts of the task demonstrated that they understood the requirements of the task.

Below are sample descriptions of the task from each patient.

Al: "Push the circles instead of the oval ellipse."
   "Look for the perfect circle; the others were a little thinner."

A2: "Pick out the round circle."
   "Concentrate to pick out the circle from the others."

Delayed Matching to Sample (DMS) - Trigrams and Ellipses

These two tests are replications of the work conducted on H.M. by Sidman et al., (1968). The DMS task permits a more precise index of STM than those previously described. Sidman et al., found that H.M. was able to perform the DMS when the stimuli were trigrams but not when they were ellipses. This would seem to suggest that H.M.'s impairment is a verbal encoding deficit since the trigrams provide a verbal code whereas
the ellipses do not. In the ellipse condition, the subjects must rely on their own verbal coding strategies (i.e., larger than, smaller than, etc.). This inability to self-generate verbal codes appears to be the nature of the deficit observed in Korsakoff patients as postulated in the introduction.

Procedure

The sample stimulus was first projected on the center key. The sample stimulus terminated when the subject pressed it and the matching stimuli appeared on the matrix $4 \times n$ ($n=0-10$) seconds later. A titrating delay method was used. This procedure begins with a zero second delay. As soon as the sample stimulus disappears, the choices appear. When a subject made a correct response, the delay on the subsequent trial was increased. If a subject made an incorrect response, the delay was shortened on the next trial. The subjects were reinforced with a token when they pressed the outer key that matched the sample stimulus. In the trigram DMS task, the incorrect choices on each trial were all permutations of the three letters that served as the sample. The matching stimuli were projected upon six outer keys of the matrix since three letters can make six possible combinations. In the ellipse DMS task, the stimuli consisted of eight different ellipses (vhrs of .259, .342, .423, .500, .643, .776, .906, and 1.0). Each of the ellipses served as choices on every trial; however, the largest and smallest ellipses were not used as sample stimuli. The procedure was the same as the one for the trigram DMS task. Initially the delay was titrated at $4 \times n$ ($n=0-10$)
seconds; however, in all cases the titrated delay was changed to $2 \times n$ and finally to $n$ since all subjects found this task to be difficult. Each task consisted of 20 trials and the ITIs were approximately two seconds.

Results

In this test of STM, H.M. (the hippocampal amnesic patient) was found to have no difficulty remembering the trigrams for up to forty seconds (Sidman et al., 1968). Kl gave the same results (Figure 15K1); he did not make any errors. Although Kl was able to remember the trigrams for as long as forty seconds, (Kl was heard rehearsing the letters during the delay), he was not able to remember the sample ellipse for more than two seconds, and this only occurred once in two trials (Figure 17K1). Out of a total of 20 trials, Kl correctly chose the sample on only seven trials (six of them were at a zero second delay). H.M. was also impaired on this task. Out of three test sequences, the longest delay at which he correctly chose the original sample, was five seconds, and that occurred only once in four trials at the five second delay.

K2's performance during the trigram condition was relatively good (Figure 15K2). He was able to remember the trigrams for as long as 38 seconds. He made a total of five errors; however, the first two, on trials one and three, were in all probability due to a misunderstanding of the requirements of the task, or otherwise (i.e., if it were a memory
problem) he never would have been able to remember the trigrams for as long as 38 seconds. K2's ellipse-DMS performance was impaired (Figure 17K2) as was K1's. The longest delay at which K2 correctly recognized the sample, was eight seconds, and this only occurred once in two trials at the eight second delay. By the 20th trial, K2 had titrated back to a zero second delay. Out of 20 trials, he correctly recognized the sample only eight times.

Except for A2's two errors on the first two trials, both control patients did not have any problem remembering the trigrams for as long as forty seconds (Figures 16A1 and 16A2). In the ellipse DMS test, the longest delays at which A1 and A2 correctly recognized the sample were seven and six seconds, respectively (Figures 18A1 and 18A2). Out of the 20 trials, A1 had 10 correct and A2 had 11 correct.

In summary, it was found that all subjects were able to remember the trigrams at delays of at least 38 seconds. However, the longest delay at which any subject could remember the ellipses was eight seconds. Since the subjects were impaired when the stimuli were ellipses, they were given massed trials in an ellipse DMS test (48 trials at each of five delays) so that discrimination gradients could be analyzed.

Delayed Matching to Sample (Ellipse Gradient)

Procedure

Since many errors at longer delays in the ellipse DMS may indicate a loss of accuracy rather than a loss of stimulus
control (e.g., subject chooses .423 ellipse when the sample was .500), subjects were given 48 trials of zero, eight, 16, 24, and 32 second delays in the ellipse-DMS test. The stimuli and procedure, except for delay, were the same as described for the ellipse DMS test. Correct responses were rewarded with a token and the ITI was approximately 2 seconds.

Since the analysis of the ellipse DMS series did not investigate the degree to which the subjects were responding along the sample stimulus dimension, all subjects went through an extended series of 48 trials of zero, eight, 16, 24, and 32 second delays. The curves of Figures 19K1, 19K2 and 19A1 show the subjects' gradients of control by sample stimuli at a zero second delay. The manner in which the scale values of these figures were determined was to rank of each of the possible choices (.259, .342, .423, .500, .643, .776, .906, and 1.0) on each trial with respect to its deviation from the sample. If the sample was the .500 vhr ellipse, values of +1, +2, +3, and +4 were assigned to the .643, .776, .906, and 1.0 ellipses respectively; values of -1, -2, and -3 were assigned to the .423, .342, and .259 ellipses respectively. The correct choice, the sample, was assigned a value of 0. The two extreme choices of .259 and 1.0 were never used as samples; they were, however, used as choices. Since the number of opportunities for a subject to deviate a given value from the sample stimulus was directly related to the sample size, the number of times the subject chose each deviation was divided by the number of opportunities he had to make each choice.
The ratio at the 0 absissa value designated the percentage of opportunities in which the subject correctly chose the sample stimulus. The ratio at +1 corresponds to the proportion of opportunities in which the subject chose the ellipse which was one size larger than the sample. The remaining ratios were determined by the procedure described above.

Results

At the zero, eight and 16 second delays (Figures 19, 23 and 27) all subjects tended to respond along the dimension correlated with reward. The sample stimuli controlled their selection, as indicated by the sharply peaked curves at the abscissa value of 0 and by the fact that the errors tended to decrease in an orderly manner as the selections deviated more from the sample stimulus. At 24 and 32 second delays, the behavior of the Korsakoff patients deteriorated (Figures 31 and 35). At the 24 second delay (Figure 31K1), K1 chose ellipses which were one value larger than the sample as often as the samples. At the 32 second delay (Figure 35K1), he selected ellipses which were one value larger than the samples more often than the samples and predominantly overestimated the samples; he had very few underestimations. Although K1's data points do not exactly coincide with H.M.'s, (graphs labeled A of Figures 41-43) the trend toward overestimations are seen at longer delays. K2 on the other hand, showed a general trend towards clustering his responses around the -1, 0, +1 and +2 deviations equally often at the 24 and 32 second delays (Figures 31K2 and 35K2). This diminution of stimulus control
which was found in the Korsakoff subjects' behavior at the 24 and 32 second delays, was not observed in the control subjects' behavior (Figures 31A1 and 35A1). The control subject's gradients remained sharp and were very narrow at both of these delays.

Because the control exerted on the behavior of the Korsakoff subjects by the sample (sample control) lessened in longer delays, it might have been possible that the attenuation was a result of a change in the stimulus dimension controlling the behavior. For example, both K1 and K2 might have selected one ellipse more frequently than the others, regardless of sample size, or they might have chosen a particular key more often than the others, again independent of sample size. Therefore, the subjects' choices were grouped as 1) absolute ellipse size and 2) key position. Both of these measures are indications of choice control. The ellipse sizes were denoted as relative frequencies (number of choices per number of trials) and the key selections were expressed as a proportion of the number of times the subjects chose each key relative to the number of times each key contained the sample within any given series of 48 trials. The curves denoting both of these measures are depicted in Figures 20, 21, 24, 25, 28, 29, 32, 33, 36 and 37. The dotted lines in these graphs indicate maximal performance (i.e., if subjects only chose the samples).

In zero delay matching, all subjects distributed their
choices relatively evenly among the six ellipses used as samples. This came as no surprise, since the subjects were generally accurate at this delay. There was a slight key position preference in both K2 and A1 (Figures 21K2 and 21A1) however, this factor did not have much influence on their performances, as evidenced by their steep sample control gradients. At the eight second delay, the Korsakoff subjects' proportion of correct choices (0 deviation) fell approximately 15% from the 0 delay. Inspection of Kl's key preferences (Figure 25K1) suggests that his reduction in correct choices was partially due to a shift from sample to key position control, since he tended to over-select key #6 by 14% and under-select key #1 by 13%. K2's poorer performance was in part due to the fact that he chose the .64 ellipse 25% more than he should have for optimal performance (Figure 24K2). At the 24 second delay, the behavior of both Korsakoffs shifted from sample control towards choice control; their sample gradients (Figures 31K1 and 31K2) no longer peaked at 0 deviation and there was a slight shift in each case toward positive deviations (overestimations). The choice control gradients of Kl and K2 (Figures 32K1 and 32K2) clearly show the tendencies toward choosing the fourth, fifth and sixth largest ellipses. Both curves sharply peak at the .643 ellipse (both subjects chose the .643 ellipse 25% more than they should have for optimal performance) and the gradients fall from that point in an orderly fashion. In the 32
second delay condition, the behavior of the Korsakoffs as even less under sample control and more under choice control than it is in the 24 second condition (Figures 35K1, 36K1, 35K2 and 36K2). Both Korsakoff patients perserverated on the .643 ellipse, as they did at the 24 sec. delay and K2 chose that ellipse about 44% more often than he should have. K1 chose the .643 ellipse 25% more and there was a slight shift towards selecting the larger ellipses. In contrast, Al's sample gradients (Figures 19A1, 23A1, 27A1, 31A1 and 35A1) remained sharply peaked through all of the delays, his choices (Al graphs of the Figures 20, 24, 28, 32 and 36) were fairly evenly distributed among the six ellipses and his key selections (Al graphs of Figures 21, 25, 29, 33 and 37) coincided fairly well with the expected frequencies for optimal performance.

Having demonstrated that the controlling stimuli tended to shift from the sample towards the choices, it was necessary to determine the nature of this combined control. In order to assess the combined control of the sample sizes and ellipse-choice displays, the mean vhrs of stimuli chosen for each sample were calculated. Figures 22, 26, 30, 34 and 38 illustrate the mean choice vhr as a function of sample size at the five delays. If the subjects' choices were strictly dependent upon the sample sizes, then the solid curve would fall exactly on the 45° dotted line. If subjects responded independently of the sample, being entirely a function of the
choice display, the curve would be parallel to the abscissa. Points below the 45° line represent underestimations whereas points above the diagonal represent overestimations.

At the 16 second delays (Figure 30K1) K1's curve between the .423 and .776 ellipses was nearly horizontal, suggesting a loss of differential sample control by these ellipses and the development of control by the choice display. For example, the .423 ellipse had little differential control on the .500, .643, and .776 ellipses but exerted substantial control when measured against the .906 sample and to a lesser extent, the .342 ellipse. Therefore, the sample or choice control that varies with the size of the sample indicates an interaction and results in a non-linear, non-diagonal curve. At the 32 second delays (Figure 38K1), K1 shows only minor evidence of differential sample control. In general, all samples were overestimated; he was unable to distinguish between the .423, .500, and .643 ellipses and between the .776 and .906 ellipses. He was, however, able to differentiate between the smallest ellipse, the three next larger ellipses, and the two largest ellipses.

For the most part, K2 was unable to distinguish among the second, third, fourth and fifth largest samples at the 32 second delay (Figure 38K2); their points lie on or close to a horizontal line. He was, however, able to correctly discriminate between the smallest, largest and four intermediate sized samples.
Al, on the other hand, was differentially controlled by the samples at all delays (Graphs labeled Al of Figures 22, 26, 30, 34, and 38). The only deviations he made from strict sample control were general tendencies to overestimate the smallest samples and to underestimate the largest samples. When he completed the entire series, he was asked how he was able to remember the sample stimuli. He reported that he developed a verbal mediating behavior that allowed him to place verbal tags onto the stimuli. He categorized the stimuli into two groups; one group contained the four largest stimuli and the other group included the four remaining smaller ellipses. He assigned numbers to the stimuli in each group (i.e., the four largest were given codes of 1-4 and the four smallest were also coded 1-4). The Korsakoff patients were not asked how they were able to remember the stimuli since their performance was very poor at the end of the series. It may be assumed, however, that they either generated a coding system that was inefficient or that they did not devise such a system.

In summary, both Korsakoff subjects exhibited substantial degrees of choice control at every delay. Choice control was primarily evident within the range of the intermediate sized ellipses and was most pronounced at delays of 16, 24, and 32 seconds. Al's behavior was almost completely controlled by the sample. K1 and K2's performances were controlled by the sample to a certain degree, however, the extent of sample control in the Korsakoff's behavior was clearly
more independent of the samples. The data reported on K1 and K2 are quite comparable with data gathered from another male Korsakoff patient (Sidman, 1969). Thus, it appears that these findings are consistent across males diagnosed as having Korsakoff's syndrome.

Delayed Matching to Sample (DMS) —

Homophones and Non-Homophones

This task specifically addresses itself to the deficit in verbal encoding hypothesis. If Korsakoff patients are unable to employ verbal or semantic encoding strategies, then their performance on a DMS task that utilizes homophones should be impaired. Cermack et al., (1973) found that the performance of Korsakoff patients on a recognition task employing homophones was poor. This test was designed to determine whether the same deficit is found in Korsakoff patients when tested in a different paradigm.

Procedure

The procedure was the same as for the ellipse-DMS except that the stimuli were words and there were only two matching stimuli (located on the outer keys of the second row of the matrix) presented per trial. The stimuli were 30 pairs of homophones and 30 pairs of unrelated words which were matched with the homophones for frequency of occurrence in the English language, part of speech, and number of letters (+1). There were a total of four testing sessions. The first was
as described; however, in the second and third sessions the subjects were required to count backwards from 100 by threes during the delay. The fourth session was like the first in that there was no interpolated distraction task. Al and K1 received stimuli which were blocked into sequences of 15 non-homophones followed by 15 homophones per session. K2 was presented with sequences of 15 homophones followed by 15 non-homophones per session.

Results

Figure 44.A shows the recognition scores, in terms of percent correct, for subjects across homophone and non-homophone conditions as well as the effect of interference vs. non-interference. Figures 44.B and C show the percentage of correctly recognized non-homophones and homophones respectively, for all subjects. An analysis of variance, conducted on the first two sessions revealed that Korsakoff patients made more recognition errors than the controls (p < .025). Furthermore, there was a significant effect for homophone vs. non-homophone errors (p < .025), and a significant groups x part of speech interaction (p < .05). Subjects were found to be adversely affected by interference (p < .05) and Korsakoffs were more adversely affected by the interference than were the controls (p < .05). These findings are entirely consistant with the observations reported by Cermack et al., (1973).
Trigram Recognition Test

Recent evidence (Martin and Melton, 1970) has shown that normal subjects are facilitated in their performance on a recognition task when the material is of high meaningfulness. If Korsakoff patients suffer only from an inability to generate verbal codes, then the more meaningful the stimuli are, the more one would expect to observe a corresponding facilitation of recognition. Since the verbal codes are supplied in this task, the performance of the Korsakoff subjects should improve with practice (i.e., they show evidence of memory). This study is directed at assessing this hypothesis.

Procedure

The stimuli consisted of 30 trigrams on slides. Ten of the trigrams were of a low meaningfulness level (4%), ten of medium (43-58%) and ten of high (three letter words) as determined by the Underwood and Schultz meaningfulness tables (1960). Each trigram was presented for 15 seconds. The subject was instructed to spell the trigram out loud when it was projected on the center key. The meaningfulness levels for the trigrams were not blocked; the trigrams were presented in random order. After completing the first series of 30 slides, there was a three to four minute break in which the subject smoked and engaged in casual conversation after which the 30 slides were presented for a second time, following the same
procedures as in the first presentation. The following day, 30 pairs of trigrams were presented on the matrix (each pair was located on the outer two keys of the second row of the matrix). Each item of the pair was from the same meaningfulness level, but only one item of the pair had been presented on the previous day. This paradigm was run twice for each subject in order to determine whether a practice effect would be observed in the Korsakoff subjects. If Korsakoff patients suffer from an inability to generate verbal codes then a facilitation would be expected to occur, and the meaningfulness level should affect their recognition. Subjects were reinforced with a token when they pressed the key that had the trigram which had been previously presented. The ITI was approximately 1 second.

Results

Figure 45 illustrates the performance of subjects in the two trigram recognition tasks expressed in percentage of correct responses. An analysis of variance, conducted on the first test, suggested that Korsakoffs tended to perform more poorly than the controls (p < .10). It was found that the level of meaningfulness affected performance (p < .005) and that there was a groups X meaningfulness level interaction (p < .025); Figure 45 indicates that Korsakoff subjects were, in general, affected more by the meaningfulness levels than were the controls.
If the Korsakoff patient's deficit is specific to generating verbal codes, then when he is placed in a situation where the opportunity for reliance on his own verbal codes is minimized, and the opportunity to employ rich non-verbal cues is maximized, or the verbal code is supplied, his performance should demonstrate substantial retention. If however, the subject must supply his own verbal coding as a necessary strategy of the task, then the Korsakoff patient's performance should show the classical deficit.

Procedure

Day 1. Twenty-nine slides of common objects were projected on a screen at the rate of one every 15 seconds. Each object was named by the experimenter (example: "This is a picture of a river"). The series of 29 slides were presented twice with a three to four minute interval between presentations.

Day 2. Picture recognition: Twenty-nine pairs of pictures, each of the same type of object (e.g., two different houses, two different doors, etc.) were presented to the subjects; however, only one of the pictures was shown on Day 1. The experimenter asked the subjects, "Which picture did you see yesterday?" during the first few trials. If the subject didn't remember, he was asked to guess. The experimenter manually recorded the subjects' responses and the
subjects were reinforced with a token after each correct response.

Day 3. Verbal recognition: The experimenter asked the subject which of two objects he had seen before. Only one of the two objects was presented on Day 2 (e.g., "Did you see a house or a school?"). All stimuli presented on Day 1 were represented in the 29 questions. Each pair of stimuli, on a given trial, were matched for frequency of occurrence in the English language, part of speech and number of letters (+ 1). The experimenter manually recorded each subject's response and tokens were given for each correctly recognized. Subjects were again presented with the sample pictures and took the picture recognition test three and four days, respectively, after completing the verbal recognition test. After taking the second picture recognition test, subjects were instructed to recall the objects.

Verbal recall: Twenty-four hours after completing the second picture recognition task, subjects were required to freely recall as many of the 29 objects as they could. Each subject was given three promptings (e.g., "Are you sure you can't remember any more items?"). If the subject asked, the experimenter would indicate how many items were left that had not been recalled. Other than this, the promptings and the delivery of tokens after each item correctly recalled, the experimenter did not interact with the subject. One week after the verbal recall test, a second verbal recall test was
administered following the same procedures. Ten days after the second verbal recall test, a third verbal recall test was given. Immediately after the third verbal recall test, the subjects were given a third picture recognition task. The instructions differed slightly from the first and second picture recognition tests. Subjects were instructed to choose the picture which they saw when the items were initially presented (e.g., "Choose the picture that you saw when the pictures were presented one at a time").

Results

Figure 46 summarizes the performance of subjects, in terms of percent correct, across conditions. Figure 46.A shows that all subjects were able to recognize, well above the chance level, the stimuli presented to them on the previous day. On the day after the first picture recognition test, all subjects were able to correctly recognize the items they had seen previously when two choices were read to them (Figure 46.B). Each set of choices contained the correct item and another item matched for frequency, part of speech, and number of letters. The subjects were given the picture presentations and the picture recognition task for a second time on the third and fourth days, respectively, after completing the verbal recognition task. The results of the second picture recognition task are presented in Figure 46.C. Although there was a slight decrease in the number of correct responses, as compared to the first test, for both K1 and K2
(23 and 28 correct responses in test A vs. 22 and 24 correct responses in test C, respectively), their performances were still well above chance. Graphs A, B, and C of Figure 46 demonstrate that Al had no difficulty whatsoever with the picture and verbal recognition tasks. Twenty-four hours after the second picture recognition test was administered, a verbal recall test was given. The results of this test (Figure 46.D) clearly indicate that the Korsakoff subjects were severely impaired at this task. K2 was only able to recall 10 items and K1 two, in direct contrast with Al who was able to correctly recall all 29 items. This test was then readministered one week later. The results (Figure 46.E) again clearly demonstrate the Korsakoff subjects were not able to adequately perform the task, relative to the control subject. K1 could not recall any of the items, K2 recalled seven, three less than he recalled on the first recall test, whereas Al again was able to recall all 29 items. Ten days after the second recall test was given, a third recall test was administered (Figure 46.F). K1 did not remember seeing any pictures, K2 only recalled six items and Al was able to recall nearly all of the items (26 out of 29). Al reported that he used a method to recall the items. He categorized the items into various classes (e.g., animals, water related items, etc.). Since K1 did not remember seeing any pictures and K2 performed so poorly, the experimenter did not ask them how they remembered the items. Immediately after completing
the third recall test, the subjects were given a third picture recognition task; they had not seen the sample stimuli for 18 days. All subjects were able to correctly identify the correct pictures well above the chance level (Figure 46.G). Three Chi Square analyses comparing the performances of K1 and A1 are included in Appendix A.

The sequence of testing was mixed yet relatively constant across subjects. The reasons for this were that 1) the design was modified as testing proceeded and thus did not follow the procedure outlined in the prospectus, 2) the alcoholic control patient that completed all tests was only available four days out of each week and 3) variations in test order would avoid boredom or adverse conditions which were generated by many of the tests. The order in which the tests were taken by each subject is summarized in Table 2.
<table>
<thead>
<tr>
<th>A.R. (K1)</th>
<th>W.G. (K2)</th>
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<tbody>
<tr>
<td>1    shape</td>
<td>shape</td>
</tr>
<tr>
<td>2    circle-ellipse discrimination</td>
<td>circle-ellipse discrimination</td>
</tr>
<tr>
<td>3    shape</td>
<td>circle-ellipse threshold</td>
</tr>
<tr>
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<td>DGS - 4 tests</td>
</tr>
<tr>
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<td>DGS - 1 test</td>
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<tr>
<td>6    DGS - 4 tests</td>
<td>DMS-trigrams</td>
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<td>DMS-Homoph/non-homoph w/o interference</td>
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<td>9    DMS ellipses</td>
<td>trigram presentation</td>
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<tr>
<td>10   DMS-homoph/non-homoph w/o interference</td>
<td>trigram recognition</td>
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<td>DMS homoph/non-homoph w/ interference</td>
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<td>ellipse-dMS gradient</td>
</tr>
<tr>
<td>13   DMS-homoph/non-homoph w/ interference</td>
<td>picture presentation</td>
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<td>2nd DMS-homoph/non-homoph w/ interference</td>
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<td>19   2nd trigram recognition</td>
<td>2nd picture presentation</td>
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<td>2nd picture recognition</td>
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<td>verbal recall</td>
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<td>22   2nd picture recognition</td>
<td>2nd verbal recall</td>
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<tr>
<td>23   verbal recall</td>
<td>2nd DMS-homoph/non-homoph w/o interference</td>
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<td>24   2nd verbal recall</td>
<td>3rd verbal recall</td>
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<tr>
<td>25   2nd DMS homoph/non-homoph w/o interference</td>
<td>3rd picture recognition</td>
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<tr>
<td>26   3rd verbal recall</td>
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<tr>
<td>27   3rd picture recognition</td>
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The lines separate the sessions
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<th>J.P. (A2)</th>
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<tbody>
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<td>1  shape</td>
<td>shape</td>
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<tr>
<td>2  circle-ellipse discrimination</td>
<td>circle-ellipse discrimination</td>
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<tr>
<td>3  circle-ellipse threshold</td>
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<td>16 2nd trigram recognition</td>
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<td>23 3rd verbal recall</td>
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<tr>
<td>24 3rd verbal recognition</td>
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DISCUSSION

The results of this investigation suggest that the behavioral impairment suffered by Korsakoff patients is not due to loss of memory per se, but rather, the deficit appears to stem from an inability to generate verbal codes for information acquired, presumably, after the development of the neural lesion. This interpretation receives corroborative support from animal studies in which mammillary body lesions did not produce a memory loss, as measured by simple nonverbal tasks (Kim, Chang and Chu, 1967; Thompson and Hawkins, 1961; Woody and Ervin, 1966). Since infrahuman animals do not exhibit verbal ability, the impairment seen in humans after mammillary lesions would not be expected to be seen in animals sustaining such lesions, assuming the deficit is a result of a failure to generate verbal codes. Throughout this study, Korsakoff patients exhibited nearly normal memory functioning except in situations where they had to produce their own verbal codes (e.g., recall).

The Korsakoff patients' impairment was first observed during the verbal descriptions of what they did to receive tokens in the discrimination gradient series. It will be recalled that K2 spoke of numbers, lights flickering, colors changing and lights going on and off. Kl, 24 hours after completing the fourth DGS test, did not remember taking the test, let alone what he did to earn his tokens. Yet the
circles and ellipses which they were unable to describe were subsequently found to control their key pressing behavior quite precisely. Sidman et al., (1968) reported virtually identical findings from H.M. Both H.M. and Kl showed normal learning curves on the DGS series as evidenced by the fact that they began each session at the same levels of performance which they had attained on the previous day. Furthermore, H.M. claimed that he had never taken a mirror drawing test before, even though he had been given the test the day before (Milner, 1962). Kl also expressed the belief that he had never taken the DGS test although he had taken it four times, 24 hours before. These data strongly indicate a dissociation between the amnesic patients' verbal behavior and their accurate perceptual-motor behavior. This dissociation between the amnesic patients' inappropriate verbal behavior and accurate non-verbal behavior readily explains the observation that mammillary body and hippocampal lesions in animals do not produce memory deficits. When amnesic patients are tested on tasks that do not require the presence of verbal mediating behaviors, they show near normal learning and retention curves, as do animals sustaining similar lesions. However, when amnesic patients must generate verbal behaviors to perform a task, they show the classical deficits. Lesioned animals, on the other hand, are never required to generate such behaviors and thus do not show the gross behavioral impairments seen in humans. Accordingly, this dissociation
suggests that the factor which is responsible for these patients' behavioral deficit is the impairment of an appropriate system for verbal mediating behaviors.

The results of the trigram-DMS test further support the hypothesis that a failure in verbal encoding is the cause of the amnesic's behavioral impairment. The performances by Kl and H.M., on this task, were identical in every observable respect. Neither of the amnesic patients encountered any difficulty, both of them titrating up to forty seconds without making a single error. These observations, unlike the performances with the ellipses, are understandable when one considers the nature of the stimuli. Trigrams consist of letters and thus possess a verbal code. It may be surmised that both patients were able to make use of the readily available verbal tags since H.M. was seen shaping the sounds of the letters with his lips, as was Kl. Kl was also heard rehearsing the letters to himself. In this situation, the amnesic and the normals did not have to devise or rely upon a code of their own.

The results from the ellipse delayed-matching-to-sample gradient task corroborates and further delineates the nature of the verbal encoding deficit observed in amnesic patients. In the ellipse-DMS gradient task, Korsakoff subjects were observed to respond inappropriately at delays of 24 seconds and longer. On the other hand, the control subject responded along the stimulus dimension correlated with reward at
each delay. After completing the task, the control subject reported that he had developed a verbal mediating strategy that enabled him to verbally code the stimuli. This allowed him to "tag" the stimuli so that he could determine which stimulus was associated with reward. Because the Korsakoff subjects were performing so poorly at the end of the task, they were not asked how they remembered the stimuli. Their results, however, indicate that they either did not develop such a coding system or that they did generate a coding system, but it was ineffective. Kl and K2, and H.M. (Sidman, et al., 1968) have never been known to spontaneously develop self-stemmed coding systems to help them overcome their impairments. Kl, like H.M., has never, since his hospitalization, written notes to himself to assist as reminders.

The homophone and non-homophone-DMS test revealed an additional facet of the verbal encoding impairment sustained by Korsakoff patients. Amnesics were found to be adversely affected by interference, and this susceptibility to retroactive inhibition was found to be most pronounced in the homophone condition. The most parsimonious explanation for this finding is that Korsakoff patients encode information on an acoustic rather than a semantic basis and that this strategy is more subject to disruption than is semantic encoding. This interpretation is supported by the fact that Korsakoff subjects were able to remember the non-homophones more reliably than the homophones, in both interference and
non-interference conditions. These findings indicate that amnesic patients are either impaired in their ability to employ semantic encoding strategies or that they are unable to spontaneously employ their own semantic dimensions effectively.

The results of the picture recognition, verbal recognition and verbal recall tasks specifically show that the behavioral impairment suffered by Korsakoff patients is an inability to effectively produce verbal codes for information recently acquired. Amnesic patients' memories were shown to be relatively intact as long as the mediating verbal stimuli were externally generated or that the demands placed upon verbal coding was either minimized or absent. During the picture recognition tests, two different pictures were simultaneously presented, each of which was of the same type of object. Since each pair of pictures was of the same type of object, the verbal coding in this task was minimized. The subjects had to rely on stimuli in the pictures other than the primary verbal codes, which were the same in each case (i.e., two doors, two houses, etc.). In the picture recognition test, all of the subjects were able to recognize the correct stimuli at levels well above chance. When the subjects were administered a verbal recognition questionnaire 24 hours after completing the first picture recognition task, again all subjects were able to recognize the correct stimuli at levels well above chance. In this situation, subjects
were given verbal codes and did not have to rely on generating their own. The first verbal recall test, administered the day after the second picture recognition test, gave quite different results, however. Both Korsakoff subjects were severely impaired relative to the control subject. In this condition, when the Korsakoff patients had to rely on endogenously generated verbal codes, they were unable to devise a strategy which would enable them efficiently to recall the stimuli. The control subject, however, developed a technique in which he could organize the stimuli and thus allowed him to recall every item. He grouped the items into headings such as water-related items, animals and so forth. These observations are paralleled by the results gathered from the trigram DMS and ellipse DMS gradient tasks.

The alcoholic subject devised a code for himself in the DMS-ellipse task whereas the performance of the Korsakoff subjects at long delays indicated that they devised no such strategy or that the strategy which they did generate was ineffective. When the verbal code was supplied in the trigram DMS test, the Korsakoff subjects were found to be virtually unimpaired, compared to control subjects. The second recall test, administered one week after the first, showed a decrement in the performance in the Korsakoff group, in contrast to the perfect performance exhibited by the control subject. The third recall test, given 10 days after the second, showed a further reduction in the performance of one of the
Korsakoffs (K1 could not recall any stimuli in either the second or third tests), whereas the control subject maintained a nearly perfect level of recall. Immediately after the third recall tests, all subjects took the third picture recognition test. Both Korsakoff subjects were able to recognize the sample stimuli at levels comparable to those they reached 18 days earlier (they had not seen the sample stimuli since the second picture recognition test). The Korsakoff group's performance on the third picture recognition test is in direct contrast to their recall test performances. This disparity is readily reconciled when one considers the factors involved in each test. In the third recall task, the subjects had to generate their own verbal stimuli which gave them a handle, so to speak, on their memories. In all three of the recall tests, they were incapable of efficiently developing such a strategy. In the picture recognition task, however, they were not required to gain access to their memories verbally and therefore were able to effectively gain access to the necessary stored information. Another possible explanation for their good performance in the picture recognition task is that, since they were responding to rich sets of stimuli, they may have been able to take advantage of the extra cues. This interpretation has yet to be tested in a verbal task.

Although the evidence presented thus far suggests that the memory deficits observed in Korsakoff patients stem from
an impairment in their ability to generate verbal codes, they do show memory loss in daily situations that are presumably mediated non-verbally. Both KI and H.M. will get lost in the hospital if they are not constantly supervised. These findings seemingly contradict the hypothesis that the nature of amnesic patients' deficit is one of verbal encoding. This disparity is reconciled by the results of experimentally produced lesions in animals. Many studies have shown that animals sustaining bilateral destruction of the hippocampal formation are severely impaired in the acquisition of complex mazes (Gross, Chorover and Cohen, 1965; Jackson and Strong, 1969; Kaada, Rasmussen and Kvein, 1961; Kveim, Setekleiv and Kaada, 1964; Kimble, 1963; Thomas, 1971).

Humans sustaining bilateral hippocampal destruction are likewise impaired in their acquisition of complex mazes (Corkin, 1965; Milner, 1962, 1969; Milner, Corkin and Teuber, 1968).

If it is assumed that maze performance consists of responses to sequential spatial stimuli and that maneuvering about a hospital ward also involves responses to sequential spatial stimuli, then the inability of hippocampal patients to find their way about their environment is thus explained.

The above observations have certain implications for the function of the hippocampus. It may be that in human evolutionary development, the hippocampus assumed an additional function of being a verbal coding system. This may account for the fact that very few impairments, comparable to those
found in humans, are observed in non-verbal organisms. If one assumes that the anatomical connections of the hippocampus-fornix-mammillary body system comprises an anatomical pathway which mediates common functions (Barbizet, 1963; Papez, 1937; Penfield and Milner, 1958; Sweet et al., 1959), then the fact that Korsakoff patients have trouble finding their way about the hospital wards is readily explained. Korsakoff patients sustaining bilateral mammillary body lesions presumably are also impaired in their ability to respond effectively to sequential spatial stimuli. Unfortunately, however, Korsakoff patients have never been tested to assess their performance on complex mazes, nor have there been any experiments on complex maze performance using animals that have had bilateral mammillary body lesions.

In summary, the functional nature of Korsakoff and hippocampectomized patients' impairments, as measured by the DGS and DMS tasks, are quite equivalent. Their impairment is not one of global amnesia, but rather the deficit appears to be quite specific to an inability to endogenously produce verbal stimuli (codes) for information in memory. The information is accessible, but only by means of exogenously-produced verbal stimuli, which can then gain access to the information, as was demonstrated in the verbal recognition and trigram DMS tests, or by means of an accessory system which does not require verbal mediation, as was demonstrated in the visual recognition task. This deficit is not an all-or-none
phenomenon. Mammillary body, hippocampal and fornical patients show various degrees of the impairment. There are cases in which no memory impairments have been observed in patients sustaining these lesions (Adams, 1969; Garcia Bengocheta, de la Torre, Esquivel, Vieta, and Fernandez, 1954; Prisko, 1963). Since the major afferent input to the mammillary bodies is the hippocampal formation via the fornix, it is reasonable to conclude that the components of this hippocampal-fornix-mammillary body system mediate similar functions in humans. In most instances, when there are lesions of any of these structures, gross behavioral deficits are observed in the patients (i.e., AA). Thus, the interpretation that Korsakoff and hippocampal patients suffer from equivalent impairments is supported by the fact that the neuroanatomical substrates affected in these two classes of patients are intimately related.

The tentative conclusions which can be drawn at this point are for the most part quite general and do not isolate the exact nature of the deficit observed in these patients. The amnesic patients were able to recognize pictures (images) after 18 days and the verbal codes for them after 24 hours. In each case however, the stimuli were not self-generated; they were generated externally by the experimenter. This study does not indicate whether Korsakoff patients are able to generate images from memory, although the results strongly suggest that they can recognize images and verbal stimuli
which are presented to them. If they are impaired in their ability to generate images, this could explain the poor performance in the recall tests relative to the recognition tests (assuming that image generation is not necessary for recognition). However, since the contingencies (i.e. instructions) of the recall tests did not specifically require that the patients "mentally" picture the stimuli, it may be that they were able to recall images but that the contingencies did not lend themselves to such a response or strategy. This would imply that amnesic patients might improve their recall performances by means of the recall of visual images and subsequent verbal coding of the images (Patton, 1972). Before this possibility can be investigated, certain questions have to be answered in order for a more comprehensive theoretical analysis to be accomplished.

The first and most basic question to be answered is whether there is a significant impairment in the patients' ability to assign verbal codes to common images, presumably experienced prior to the onset of their retrograde amnesia. In order to answer this question, common pictures and figures will be presented to the patients, and they will be asked to verbally identify them. This is important since the Korsakoff patients were unable to describe the circles and ellipses in their absence. Thus, it would appear necessary to determine whether they can generate codes for images presented to them. If they are unable to do this, then it may be concluded that
they are impaired in their ability to name objects and, as such, suffer from aphasia. The second question to be answered is whether amnesic patients can generate images from both verbal and non-verbal stimuli presented by the experimenter. In the verbal stimulus condition, the experimenter will ask the patient to draw a picture of a common item. In the non-verbal stimulus condition, the experimenter will present a picture of a common object (or the object itself) and after removing the picture (or the object) will ask the patient to draw it from memory. If the patients are unable to perform adequately in these two conditions, it may be that their memories are bad or that they are impaired in their abilities to express or generate images. The third question to be answered is whether it is possible for Korsakoff patients to recall images from memory and subsequently assign verbal codes to them. This question will be investigated by attempting to train the patients to use mnemonics. If they are unable to use mnemonics it may be that they are unable to recall the images, or that they are unable to assign codes to images recalled from long term store. If the patients are able to use mnemonics, then the fourth question to be investigated is whether the patients can link several images together, thus enabling them to recall a number of items from the same number of recalled images. If they are unable to link images it may be that they are not able to make image associations. These are the major questions which require investigation in
future research in order to more specifically isolate the exact nature of the coding deficit observed in these patients.

The following quotation portrays a different approach to the problem of amnesia, since it deals with non-verbal animals, but it nevertheless emphasizes that coding is fundamental to memory disturbances:

"...the impairment is not so much a removal of localized engrams as an interference with the mechanisms that code neural events so as to allow facile storage and retrieval. Thus, the evidence shows that anatomically the memory trace is distributed within a neural system by means of an encoding system process, while as a function of decoding the engram is reassembled, that is, remembered. Thus, what and whether something is remembered is in larger part dependent on how it is, and that it is, adequately coded."

-- Pribram, 1969
FIG. 2

Circle-Ellipse Discrimination

Circle-Ellipse Discrimination

Circle-Ellipse Discrimination

Circle-Ellipse Discrimination
FIG. 3

K1.1

Correct Response

Trial 1 2 3 4 5 6 7 8 9 10

Shaping

K1.2

Correct Response

Trial 1 2 3 4 5 6 7 8 9 10

Circle-Ellipse Discrimination
FIG. 5

Determination of Circle-Ellipse Threshold

Determination of Circle-Ellipse Threshold
Ellipses Choice Distributions

**K1**

- **Axis Measurement:** 1.0 to 0.71
- **Selection Count:** 30 to 10

**K2**

- **Axis Measurement:** 1.0 to 0.71
- **Selection Count:** 30 to 10

**A1**

- **Axis Measurement:** 1.0 to 0.71
- **Selection Count:** 30 to 10

**A2**

- **Axis Measurement:** 1.0 to 0.71
- **Selection Count:** 30 to 10
Ellipses Choice Distributions

FIG. 7

Number of Selections in DGS

Minor / Major Axis
Ellipse Choice Distributions

Number of Selections in DGS

Minor/Major Axis
Ellipse Choice Distributions
Fig 10
Trial by Trial Record of Choice

K1

K2

A1

A2
Fig. 11

Trial by Trial Record of Choice

K1

K2

A1

A2

Trial

40  50  60
Fig. 12
Trial by Trial Record of Choice

K1

K2

A1

A2
Fig. 13

Trial by Trial Record of Choice

K1

K2

A1

A2
Fig. 14

Trial by Trial Record of Choice
Fig. 15

Trigram - DMS Performance

Ki

K2
Fig. 16

Trigram- DHS Performance

A1

Delay (sec)

Trial

A2

Delay (sec)

Trial
Fig. 17
Ellips - DHS Performance
Fig 18

Ellipt - DNS Performance

A1

Delay (sec)

Trial

A2

Delay (sec)

Trial
FIG 19

Sample Control Gradient

K1

K2

Ai

ORDINAL DEVIATION FROM SAMPLE
Choice Gradient - (Ellipse Size)

Fig. 20

Delay 0 sec.

K1

K2

A1

Choice (inches)

Percent choice

Choice (inches)
FIG. 21

Choice Gradient (Key Position)

Delay 0 sec.

K1

K2

A1

Percent choice vs. Key#
FIG. 22

Analysis of Interaction Between Sample and Choice

( Ellipse ) Control

Delay 0 sec.
FIG. 23

Sample Control Gradient

K1

PERCENT CHOICE

ORDINAL DEVIATION FROM SAMPLE

K2

PERCENT CHOICE

ORDINAL DEVIATION FROM SAMPLE

A1

ORDINAL DEVIATION FROM SAMPLE

Delay 8 sec.
Fig. 24
Choice Gradient (Ellipse Sign)

Delay 8 sec.
Fig. 25
Choice Gradient (Key Position)

Delay 8 sec.

K1

K2

A1
Fig. 24

Analysis of Interaction Between Sample and Choice (Elliptic) Control

Delay 8 sec.
FIG. 27

SAMPLE CONTROL GRADIENT

Delay 16 sec.

K1

K2

A1

ORDINAL DEVIATION FROM SAMPLE

ORDINAL DEVIATION FROM SAMPLE

ORDINAL DEVIATION FROM SAMPLE
FIG 28

Choice Gradient (Ellipse Size)

Delay 16 sec.

K1

K2

A1
Fig. 29

Choice Gradient (Key Position)

Delay 16 sec.

K1

K2

A1

Percent choice

Key

Percent choice

Key

Percent choice

Key
Analysis of Interaction Between Sample and Choice (Ellipse) Control

FIG 30

Delay 16 sec.
Fig. 31. Sample Control Gradient

Delay 24 sec.

K1

Percent Choice

Ordinal Deviation from Sample

K2

Percent Choice

Ordinal Deviation from Sample

A1

Percent Choice

Ordinal Deviation from Sample
Fig. 32

Choice Gradient (Elliptical Size)

K1

K2

A1

Delay 24 sec.
Fig. 33

Choice Gradient (Key Position)

Delay 24 sec

<table>
<thead>
<tr>
<th>Percent Choice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Key# 1 2 3 4 5 6 7 8 9</td>
</tr>
<tr>
<td>K1</td>
</tr>
<tr>
<td>K2</td>
</tr>
<tr>
<td>A1</td>
</tr>
</tbody>
</table>

Key#: 1 2 3 4 5 6 7 8 9
Fig. 34

Analysis of Interaction Between Sample and Choice (Ellipses) Control

Delay 24 sec.

K1

Average Size of Choice (inches)

Sample (inches)

K2

Average Size of Choice (inches)

Sample (inches)

A1

Average Size of Choice (inches)

Sample (inches)
Fig 35
Sample Control Gradient

Delay 32 sec.

Percent Choice

K1

Percent Choice

K2

Percent Choice

A1

Ordinal Deviation from Sample

Ordinal Deviation from Sample

Ordinal Deviation from Sample
Fig. 26
Choice Gradient (Ellipse Size)

Delay 32 sec.

K1

K2

A1
Fig. 37
Choice Gradient (Key Position)

Delay 32 sec.

K1
Percent Choice

K2
Percent Choice

A1
Percent Choice

Key: 1 2 3 4 5 6 7 8 9
Analysis of Interaction Between Sample and Choice (Ellipse) Control
Analysis of Interaction Between Sample and Choice (Ellipse) Control
A

Percent Choice

-5-4-3-2-1 0 1 2 3 4 5 6

Ordinal Deviation from Sample

Sample Control Gradient

B

Choice Gradient (Ellipses Size)

C

Analysis of Interaction Between Sample and Choice (Ellipses) Control
FIG 41

A

B

Sample Control Gradient

Choice Gradient (Ellipse Size)

C

Analysis of Interaction Between Sample and Choice (Ellipse) Control
FIG. 42

A

Sample Control Gradient

B

Choice Gradient (Ellipse Size)

C

Analysis of Interaction Between Sample and Choice (Ellipse) Control

Delay 24 sec

115
FIG 43

A

B

C

Delay 32 sec.

Analysis of Interaction Between Sample and Choice (Elliptic) Control
FIG. 44

Combined Hom. and Nonhom. Performance

A

Percent Correct

B

C

Subjects

First Session
(No Interference)

Second Session
(With Interference)

Third Session
(With Interference)

Fourth Session
(No Interference)

Hom. Performance

Nonhom. Performance
FIG. 46

Performance on Picture Recognition, Verbal Recognition and Verbal Recall
REFERENCES


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Thomas, G.J. Maze retention by rats with hippocampal lesions and with fornicotomies. *Journal of Comparative and Physiological Psychology.* 1971, 75, #1, 41-49.


The Pearson $X^2$ test was used to make a statistical comparison between the matched Korsakoff (Kl) and alcoholic (Al) patients on the picture recognition task. The most conservative test, comparing Kl's worst performance on picture recognition (the second picture recognition test) with Al's best performance (he recognized at the 100% level on all three tests) found that the patients differed ($X^2 = 7.96, p < .01$). All comparisons on this task were significant. However, it would be more salient to the theory under consideration to show that verbal recall and picture recognition performances differed for the Korsakoff patient but did not differ for the alcoholic patient. The most conservative test, a comparison of Kl's best verbal recall performance (first verbal recall test) to his worst picture recognition performance (second), still yielded significant differences ($X^2 = 28.2, p < .001$). A comparison of Al's worst recall performance (third recall test) and his best picture recognition performance was found not to be significant ($X^2 = 3.17, p$ not significant).

Since Kl was found to be impaired, relative to Al on the picture recognition tasks, it was decided to test whether Kl performed better than chance. Since there were two alternatives presented, $p$(correct) = .5, the binominal distribution is appropriate to describe the outcome, if a chance process
was at work. The most rigorous method of testing whether or not a chance performance did generate the data is to find the confidence interval for the probability parameter of the binomial distribution. Once again, doing the most conservative test, on Kl's second picture recognition performance, the 95% confidence interval was found to be \( p \leq .46 \). Since .5 is not in this interval, we may reject the hypothesis that Kl's performance was a result of chance.