

AMNESIA CAN FACILITATE MEMORY PERFORMANCE: EVIDENCE FROM A PATIENT WITH DISSOCIATED RETROGRADE AMNESIA

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Abstract—A case is reported of a patient who experienced numerous episodes of transient global amnesia (TGA) during which anterograde amnesia was less prominent than usual, and who developed a permanent selective retrograde amnesia. On formal testing, he performed well on traditional verbal memory tests, but showed marked retrograde amnesia for verbal material, including items on a famous voices recognition test. He was administered a paired-associate learning test where the names of famous personalities for which he was amnesic were associated with incongruous activities (e.g. John Newcombe—singing). Our patient performed better on this task than a group of five matched control subjects. Our observations indicate that in the organization of human memory retrograde amnesia may be fractionated from anterograde amnesia and that in certain situations specific types of amnesia can produce a facilitation effect compared to the performance of control subjects.

INTRODUCTION

RECENT studies of the amnesic syndrome [21, 25] have shown that, far from being a global phenomenon, amnesia can be fractionated such that certain components of memory may be intact while others may be markedly impaired. The aim of the present investigation was to provide further evidence of such fractionation and to show how a patient who is amnesic with respect to certain aspects of his memory can perform better than normal subjects on tasks that tap other components of memory.

Patients with a classical amnesic syndrome usually exhibit both anterograde and retrograde memory difficulties. Some cases have been reported of patients who suffer from anterograde memory impairment without showing a corresponding retrograde memory loss [16]. The converse type of patient (i.e. one with a significant degree of retrograde amnesia in the presence of normal anterograde memory functioning) has, until recently, been reported relatively seldom. WILLIAMS and SMITH [26] referred to patients who had suffered from TB meningitis and who showed normal performance on traditional memory tests but who had memory loss both for the period of the illness and for a number of years extending prior to the onset of the illness. In the past few years several cases have been reported of patients who similarly show relatively normal learning of new material in the presence of marked impairment for information that was learned prior to the onset of the illness [6, 14]. One of these patients had incurred a severe head injury and in the other two cases the

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condition resembled that of transient global amnesia, with a possible aetiology involving the posterior cerebral arteries. This paper concerns a patient who had a similar clinical history to the latter two reported cases. Detailed psychological testing confirmed that there was a significant dissociation between intact memory for new verbal material and his impaired memory for verbal information acquired prior to the onset of his illness. We were able to examine his performance on a number of tests of retrograde amnesia and compare his memory with that shown by a group of alcoholic Korsakoff patients. In the experimental study reported here, we were able to take advantage of this dissociation and design a memory test which required the patient to learn associations that competed with the usual past connotations to the target stimuli. We were thus able to test the hypothesis that our patient should perform better than normal subjects because the interfering effects of these new associations would not affect his learning performance but would affect that of the control subjects.

CASE REPORT

E.D., a right-handed, 70-yr-old retired company director, experienced 20 episodes of transient amnesia, lasting from 15 min to several hours, over the previous 6 yr. During attacks he showed a notable loss of memory for current events, yet he did not appear perplexed, he knew who he was and was able to give a speech or drive a car. There were no precipitating factors, aura, migrainous phenomena or accompanying neurological deficit. Following attacks he could recollect some information about events during the attack, although this was patchy. There was usually some memory loss for events prior to the attack but this was variable in duration and usually recovered. During the 6 yr when E.D. experienced these attacks, he developed an impairment in the ability to recall past information, such as significant past events, names of people, etc. that had been learned up to 20 yr previously.

Physical examination showed marked arcus senilis and tortuous retinal arterioles, but no other evidence of cardiovascular disease. Examination of cranial nerves and limbs was normal. Blood count, sedimentation rate, urea and electrolytes, liver function tests, fasting blood glucose and lipids, W.R., chest and skull X-rays and computerized tomography scan, standard and 48-hr EEG investigations were all normal.

Neuropsychological investigations

On the Wechsler Adult Intelligence Scale (WAIS) [24] E.D. had a pro-rated Verbal IQ of 106, a Performance IQ of 98 and a Full-Scale IQ of 103. There was relatively little variability between subtest scores. [Age-scaled scores were: Arithmetic (9), Similarities (13), Digit Span (12), Vocabulary (13), Digit Symbol (13), Block Design (10), Picture Arrangement (9)]. On a test of pre-morbid intellectual level, the National Adult Reading Test (NART) [11], E.D.'s score was equivalent to an estimated IQ of 114 and his overall WAIS IQ scores therefore suggest a minor degree of generalized cognitive dysfunction. On the Wechsler Memory Scale [23] E.D. had a memory quotient of 120. His scores on the Wechsler Memory Subtest are indicated in Table 1. His memory for one of the test stories was assessed after an interval of 40 min, and was the same (11 out of 23) as his immediate recall. Alcoholic Korsakoff patients, in contrast, usually cannot recall any of the story even after a 5-min interval. In addition, his excellent performance on the paired-associate learning subtest contrasts with the striking difficulties found in amnesic patients [27]. On some occasions E.D.'s memory for information acquired several months earlier appeared on clinical observation to be inaccurate, but it was not possible to be certain whether this was due to subtle anterograde difficulties or to the effects of an intervening transient amnesic episode.

E.D.'s score on the memory-for-designs subtest of the Wechsler Memory Scale suggested a mild impairment, and this was further confirmed by his rather low score (three out of 10 items correct) on the Benton Visual Retention Test [1]. There was also evidence of a mild impairment on a faces-matching task [3]. On a test of retrieval from semantic memory [2], where he had to name words beginning with the letters F, A and S, his score was above average (above the 95th percentile). For the purposes of the present investigation, two tests of retrograde amnesia were administered, both of which have been used in the study of alcoholic Korsakoff patients. Details of test design and

Table 1. E.D.'s scores on subtests of the Wechsler Memory Scale

Information	6	Memory for passages	11.5
Orientation	5	Digits total	11
Mental control	3	Visual reproduction	7
		Associate learning	16.5

procedure and described elsewhere [9, 15]. The two tests of retrograde amnesia related to memory for public events and memory for voices of famous personalities. For both tests E.D. was given a recall and a recognition version of the task. E.D.'s performance on the public events questionnaire for the various decades sampled by the test (1920s–1970s) is indicated in Table 2. The performance of control patients and alcoholic Korsakoff patients, which has been presented elsewhere [9, 15], is also shown for purposes of comparison. As can be seen, E.D. showed evidence of significant retrograde amnesia, especially in the recall version of the task, for events in the 1960s and 1970s. Although Korsakoff and control patients were several years younger than E.D. and were tested approx. 5 yr before E.D., the overall effect of this is probably marginal and would not explain the specific difficulties which he had for events in the 1960s and 1970s nor would it explain his superior performance to control subjects for very remote items. The performance of E.D., alcoholic Korsakoff and control subjects on the memory for voices test is indicated in Table 3. E.D. performed poorly, especially on the recall version of the task, over the various decades which were sampled. On recognition items, he again performed worse than control patients and his performance was also slightly lower than Korsakoff patients for most of the decades sampled.

In summary, E.D. showed evidence of significant impairment on verbal tests of retrograde amnesia and on some items his amnesia was even more marked than that found in Korsakoff patients. For general events, his memory deficit is restricted to items in the 1960s and 1970s, but for identification of famous voices, his impairment covers all of the decades sampled. (Pilot studies were carried out using a nonverbal test of retrograde amnesia requiring identification of faces. Although E.D. also had difficulty on the test, this observation was not pursued in view of his impairment on anterograde nonverbal memory tests.)

EXPERIMENTAL INVESTIGATION

A further test of retrograde amnesia, The Famous Personalities Test [18], was administered in which the patient is presented with a list of 160 names, 100 of which are of personalities who were famous in the past 50 yr, and 60 of which are 'fictitious' names. The patient has to indicate for each name whether he recognizes the name, whether he does not recognize the name, or whether he is uncertain. In the original normative study [18], target test items were distinguished according to whether the items were correctly recognized by particular age groups. Some names (e.g. Adolf Hitler) were correctly recognized by subjects of all ages and were categorized as "Very Famous Names". Other names were

Table 2. Performance (% items correct) of E.D., Korsakoff and control patients on the public events test of retrograde amnesia

	Recall			Recognition		
	E.D.	Korsakoffs	Controls	E.D.	Korsakoffs	Controls
1970s	25%	13%	64%	65%	50%	83%
1960s	25%	21%	56%	63%	60%	80%
1950s	60%	26%	38%	85%	63%	67%
1940s	40%	21%	46%	75%	61%	66%
1930s	55%	18%	34%	60%	50%	61%
1920s	20%	3%	6%	20%	36%	23%

Table 3. Performance (% correct responses) of E.D., Korsakoff and control patients on the voices recognition test of retrograde amnesia

	Recall			Recognition		
	E.D.	Korsakoffs	Controls	E.D.	Korsakoffs	Controls
1970s	6%	13%	64%	35%	58%	92%
1960s	11%	18%	58%	72%	56%	88%
1950s	8%	14%	38%	31%	51%	78%
1940s	8%	26%	49%	67%	73%	88%
1930s	5%	13%	29%	35%	45%	48%

Table 4. Number of errors on items in the Famous Personalities Test

	Fictitious names	Items recognized by 20-70-yr-olds	Items recognized by 30-70-yr-olds	Items recognized by 40-70-yr-olds	Items recognized by 50-70-yr-olds	"Very Famous Names"
E.D.	0	7	7	3	9	0
Control mean	0.4	0.6	2.2	3.4	4.8	0.5

recognized most frequently by certain age groups (e.g. names from the distant past tended to be correctly recognized best by 50-70-yr-old subjects) and the resultant classification is indicated in Table 4, which also shows the performance of E.D. and five control subjects matched for age and estimated IQ level (mean age = 66.8 yr, mean IQ rating based on NART = 111.6). As can be seen E.D. failed to recognize as familiar a number of names of famous personalities and his performance on this test was lower than that of five control subjects. Some famous personalities whom E.D. failed to recognize were used as the basis for an experimental investigation into his verbal memory performance. We carried out a study of E.D.'s verbal memory by assessing his performance on a paired-associate learning test which was designed such that associations to the target items would, in the case of normal subjects, interfere with memory performance. As was pointed out earlier, E.D. showed unimpaired paired-associate learning ability but failed to recognize a number of names of famous personalities on the test of retrograde amnesia. We selected six names that E.D. had failed to recognize and constructed a paired-associate learning test where the subject had to learn an activity associated with the target name, but where this activity was different from that for which the target name was famous. The paired-associate items used in the present study were as follows:

Garry Glitter-tennis
 Tony Jacklin-music
 Donny Osmond-golf
 John Newcombe-singing
 Telly Savalas-athletics
 Mary Peters-films

The following procedure was followed to administer this paired-associate learning test. The paired associate items were listed on a card and the card was exposed to the patient for 30 sec. He was asked to remember the activity indicated with each stimulus name, and was told that his memory for the associated activity would be tested afterwards. The 10 sec following presentation of the stimuli were filled with instructions for recall. The patient was then told one of the original six names and asked to state the activity which was associated with that name. He was given 10 sec to offer a response and if no response was made he was told the correct answer. He was provided with feedback after each response, i.e. if he was correct he was told so and if he was incorrect he was provided with the correct response. After the six items had been tested in this way a further 10 sec intervened before the six paired-associate items were again presented as before. Items were presented in the same order but the order of items on test trials was randomized. Five presentation and test trials were administered in this way with a total possible score of 30 correct responses. Identical items and procedure were used in the test administered to control subjects. Figure 1 shows E.D.'s performance and that of five matched control subjects (mean age = 67 yr, mean estimated pre-morbid IQ

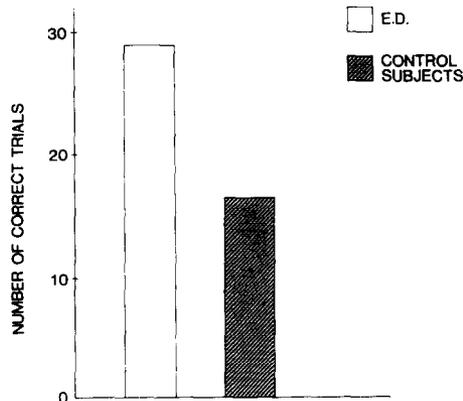


FIG. 1. The performance of E.D. and control subjects on the paired-associate learning test comprising famous names.

on the basis of the NART = 113.8). As can be seen E.D.'s performance was much better than that of control subjects, with none of the control subjects scoring as well as E.D. on this task. After the experiment, most control subjects reported that the past associations related to each of the stimulus names interfered with their memorizing of the paired-associate items, but this was not reported by E.D. As a check on the possibility that E.D. was significantly better than controls at performing paired-associate learning tests in general, a further set of control subjects (mean age = 67.8 yr, mean IQ based on NART = 112.4) were given the Wechsler Memory Scale paired-associate learning test and their mean score of 14.4 was not significantly different from E.D.'s score of 16.5.

DISCUSSION

A case was presented in this paper of a patient who experienced transient amnesic attacks and who showed retrograde amnesia for verbal material without any corresponding anterograde verbal memory deficit. On an events questionnaire, our patient's retrograde amnesia was restricted to items relating to events in the 1960s and 1970s and was more marked on recall than recognition tests. In this respect, E.D.'s performance paralleled those of alcoholic Korsakoff patients. On voices' identification, his impairment was even more marked than that shown by Korsakoff patients, and this deficit was also attenuated on recognition testing. We were also able to show that a focal retrograde amnesia for verbal material in the presence of normal performance on traditional verbal memory tests can be manipulated such that the effect of prior associations on learning can be diminished. We have further been able to show that in these circumstances a patient with such a condition can perform better on a memory task than normal control subjects.

Our present findings provide additional support for the idea [21] that human amnesia is not necessarily a global deficit but can be fractionated such that one aspect of memory may be impaired while another aspect can be intact. A number of studies of amnesic patients have shown the material-specific [10] and process-specific [12] nature of their memory deficits. The findings of our case study lend support to the view that such fractionation of memory can be extended to different components of a single aspect of memory, in this case anterograde and retrograde memory for verbal material.

Although there were no precise indicators as to the neuroanatomical locus of our patient's memory difficulties, we would speculate, in the light of clinical/pathological studies [13] of patients with retrograde amnesia and studies of positron emission tomography in patients with transient global amnesia [19], that our patient's retrograde amnesia was due to a focal vascular lesion in the region of the medial temporal/hippocampus region. It would appear from the present case study that this region itself can be fractionated such that certain areas may be damaged while neighbouring structures are spared.

The short period of permanent pre-ictal retrograde amnesia found in patients with TGA implies obliteration of previously stored memories and hints at the possibility that even more distant memories may be susceptible to at least minor erasure. Occasional cases have shown more extensive permanent retrograde amnesia following repeated [17] or prolonged [14] episodes of TGA. The evolution of a marked permanent retrograde amnesia in our patient may have been caused by the cumulative effect of minor memory erasure with each attack. It is therefore possible that in previous cases of TGA isolated retrograde amnesia may have been latent or clinically undetected, and that a more systematic clinical and neuropsychological investigation of such cases may yield a higher incidence of isolated retrograde amnesia that has hitherto been considered.

There were similarities and differences between the retrograde amnesia shown by our patient and that which has been demonstrated in alcoholic Korsakoff patients. While our patient did show marked memory loss for events in the 1960s and 1970s, in common with Korsakoff patients, on the events questionnaire his performance improved considerably for earlier decades and the temporal gradient of his absolute scores was not 'flat', as is the case with Korsakoff patients. On the voices' identification test, our patient did show a 'flat' gradient and in this respect his retrograde amnesia paralleled that found in alcoholic Korsakoff patients.

Our findings also have implications for the type of methodology used in clinical neuropsychology. Neuropsychological investigations have traditionally been 'deficit based', i.e. a statement about brain function has often been dependent upon particular patients showing impaired performance on certain tasks relative to control subjects. Some recent studies [4, 5, 7, 8] have shown that in certain conditions brain-damaged patients can perform better than normal subjects. Since impaired performance in brain-damaged patients would tend to be expected by chance factors alone and may be compounded by factors such as nonspecific effects of brain damage, we would suggest that in some cases such factors can be circumvented by the use of 'functional facilitation paradigms', i.e. an experimental design which incorporates the possibility of brain-damaged patients performing better than normal subjects. It is possible that there are a number of neurological conditions where sufficiently focal patterns of cerebral dysfunction can provide an opportunity for investigation using a 'functional facilitation paradigm', and that amnesic patients in particular may prove to be a useful population with which to explore the scope of such facilitation effects.

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