Basal Forebrain Amnesia
A Case Study

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Objective: To provide new evidence of the existence of basal forebrain amnesia, as a different entity from hippocampal or diencephalic amnesia.

Background: Some authors consider that the characteristics of amnesia do not depend on lesion site, although others claim there are neuropsychologic differences between amnesias due to hippocampal, diencephalic, and basal forebrain lesions. As to the latter, literature is scarce and controversial. The opportunity to thoroughly study J.S., a man with a high IQ and amnesia, enabled us to reinforce the second hypothesis.

Methods: J.S. is a 47-year-old man who underwent surgery for a pituitary adenoma, the resulting lesion involving only the basal forebrain. We gave him a complete neuropsychologic battery for amnesia and executive functions.

Results: J.S. showed severe amnesia with a flat learning curve, a rapid forgetting rate and good recognition, a temporal gradient of several years for remote memory, preserved semantic and procedural memory. Most of the tests for executive functions were normal, although he did have a significant personality change after surgery.

Conclusions: This patient is different from patients with hippocampal or diencephalic lesions, and is similar to other patients reported with basal forebrain lesions. The main difference is the relation between his flat learning curve and preserved recognition, both for visual and verbal material.

Key Words: basal forebrain, amnesia, pituitary adenoma

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Some authors[1–3] hold that amnesia is a unitary syndrome resulting from a lesion in any part of the memory circuit. Individual qualitative differences among patients are ascribed to "neighborhood damage," differences in methodology or premorbid personality.

The alternative, nonunitarian concept contends that amnesic syndromes can be attributed to specific neuroanatomic lesions, for example, in the diencephalon or the hippocampus.[4–6] There are reports describing a distinct amnesic syndrome caused by basal forebrain damage,[7–9] in particular with lesions of the septal region[10] and ruptured ACoA aneurysms.[11,12] However, literature on this form of amnesia is still scarce and controversial and the neuropsychologic findings are heterogeneous.

Meticulously detailed studies of patients with basal forebrain amnesia may help to clarify some of these points. We report the neuropsychologic findings of a patient with basal forebrain amnesia. He presents special characteristics which make his study interesting: he has a high level of intelligence and he has belonged to a close community for many years, so that we have been able to gather information about his premorbid personality and his present behavior in the community.

CASE REPORT

J.S. is a 47-year old, right-handed man, who was born in South Africa. As a child he spoke Russian and Yiddish at home, and English and South African in school. He always had difficulty in subjects requiring mastery of language. At age 20 he immigrated to Israel and joined the Kibbutz (communal settlement) movement. He was known as a very creative, extroverted, and sociable man, with a very "strong character."

At age 46, he was discovered to have an invasive pituitary adenoma which extended both anteriorly to involve part of the orbitofrontal area and posteriorly toward the hypothalamus. He underwent cranial surgery by a pteronian approach (the Dolen technique). This approach reaches the tumor from the base of the skull. The tumor was almost totally removed. The postoperative period was followed by confusion which lasted for 2 months. When the confusion cleared, it became apparent that he was amnesic and that his personality had changed markedly.

He was referred to us 6 months later, when his condition had stabilized. After surgery J.S. has become a very mild, placid person with no traces of the previous strong character. Nothing embarrasses or irritates him, and he accepts everything with a smile or a joke. He is still an active person, mainly socially, but he has lost his creativity, he has stopped painting or doing anything new. When asked to draw a picture he willingly and quickly grabs a pencil and draws, but his drawings are always exactly like the ones he had drawn before his disease. He has

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difficulty in making decisions such as deciding what to wear, or what to buy in the grocery store. But the most disabling symptom is his severe memory loss.

On motor sensory examination the only positive finding was a mild hyperreflexia of the right limbs. The rest of the examination was unrevealing. On mental status examination J.S. was pleasant and cooperative, well aware of his deficits, but without signs of concern about them. He tended to joke about his memory loss. His jocularity was not inappropriate. He showed no sign of disinhibition. He behaved very adequately during the repeated examinations to which he was subject.

NEUROIMAGING

Figure 1 shows the most relevant axial sections of the magnetic resonance imaging performed 10 months after surgery. The basal forebrain is markedly involved, including an area of gliosis on the anterior cingulum. Other structures known to be related to memory functions seem to be spared: in particular the mamillary bodies, the thalamus, and the hippocampus.

GENERAL NEUROPSYCHOLOGIC ASSESSMENT

Intelligence

J.S.'s performance on the Wechsler Adult Intelligence Scale-Revised[13] was high (FIQ: 128) (Table 1). Possible interpretations of the PIQ-VIQ gap may be: (1) his premorbid cognitive style which pointed to a better performance on tasks associated with the right hemisphere function; (2) an innate dyslexia as reflected by his spelling errors in previous writings and in his school records; (3) J.S. grew up in South Africa and some of the verbal subtests are culture-biased and they may not fully reflect J.S.'s verbal ability.

Spared Functions

J.S. performed very well on different tests of attention, a frequent finding in basal forebrain amnesia.[14–16] There were no indications of any visuospatial deficit in the Hooper Visual Organization Test,[17] or his copy of the Rey-Osterriecht Complex Figure.[18] Spontaneous language was fluid, with no signs of dysphasia. On the Boston Naming Test[19] his score was 59/60.

Executive Functions

There was only subtle impairment in some of the tests, despite the patient's very impaired executive functions in daily life. On the Wisconsin Card Sorting Test[20] he achieved 6 categories in 72 trials. He also showed a very good ability to estimate length, ages, temperature, distances, and weights. Abstraction was preserved. His problem-solving ability was preserved as reflected by the base-line performance on the Tower of Hanoi puzzle, which was within normal range. This test is viewed as sensitive to frontal lobe functioning.[21]

Performance was impaired on Porteus Mazes,[22] showing difficulties in planning. On Word List Generation[23] there was a suggestive gap between the generation of words according to initial sound (FAS = 9) and according to category 16. Temporal order judgment was significantly below normal on the temporal order list of the Rey Auditory Verbal Learning Test (AVTL).[24]

In sum, formal testing of executive functions only showed subtle impairment in some of the tasks, not reflecting the difficulties J.S. experienced in daily life. These findings are similar to previous studies reporting extensively discussed low intercorrelations among tasks designed to test executive functions and between these tasks and behavior.[25,26]

MEMORY INVESTIGATION

J.S.'s MQ, as measured by the Wechsler Memory Scale-Revised (WMS-R),[27] was 86 (see Table 1). This impairment is even more pronounced when compared with J.S.'s IQ (FIQ 128-MQ 86 = 42), indicating a severe amnesia.

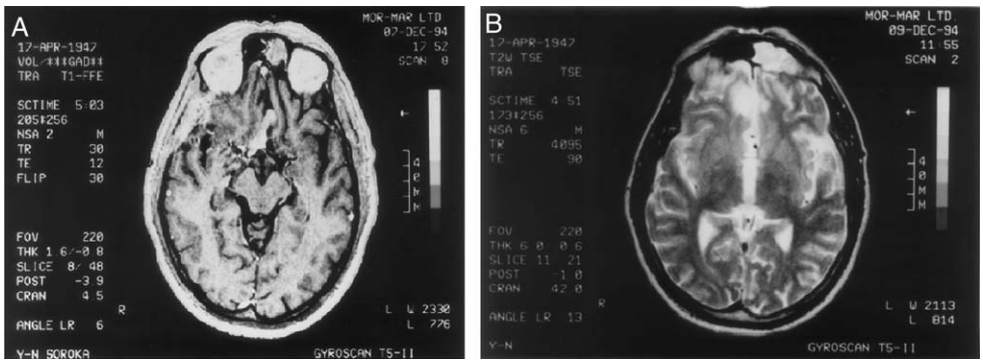

FIGURE 1. A, An axial T1-weighted image at the level of the midbrain after IV injection of gadolinium reveals evidence of a right temporal craniotomy. The residual lesion at the anterior forebrain includes the septum, most of the gyrus rectus to the right, anterior column of the fornix and anterior hypothalamus. On the left there is mild lateral displacement of the gyrus rectus. Adjacent to the craniotomy area there is evidence of postoperative changes extending to the frontal cortex. B, An axial T2-weighted image at the thalamic level reveals an area of elongated relaxation time that includes the anterior cingulum on both sides, although most prominent to the right, representing postsurgical gliosis.

TABLE 1. JS' Results on the Wechsler Adult Intelligence Score-Revised (WAIS-R) and on the Wechsler Memory Scale—Revised (WMS-R)

Subtests	Scores	Subtests	Scores
WAIS-R			
Information	12	Arithmetic	11
Picture completion	14	Object assembly	18
Digit span	10	Comprehension	16
Picture arrangement	13	Digit symbol	11
Vocabulary	11	Similarities	11
Bock design	13		
FSIQ = 128			
VIQ = 114			
PIQ = 128			
WMS-R			
Mental cont.	6	Verbal paired association I	16
Figural memory	7	Verbal paired association II	7
Logical memory I	16	Logical memory II	2
Visual paired association I	11	Visual reproduction I	33
Visual paired association II	2	Visual reproduction II	15
Visual memory span	18	Digit span	17
MQ = 86			
Verbal MQ = 82			
Visual MQ = 98			

Working Memory

J.S.'s performance on the Digit Span subtest, and in Serial 7's suggested an intact working memory. For further assessment he was tested with the Brown and Peterson task with interference (BP)[28] in the following way: consonant trigrams were printed in black on 15×10 cm white cards. Each card was presented for 2 seconds and he read it aloud. When the card was withdrawn he was given a number from which to count backwards. Recall was tested after intervals of 6, 9, 15, 24, and 30 seconds. There were a total of 30 trigrams, that is, 6 times for each interval. J.S. had no error in any of the intervals. These results indicate that J.S. has no impairment of working memory or in the very early stages of memory processing.

The performance of organic amnesics on BP seems to be related, at least in part, to the locus of the lesion. Extensively studied diencephalic amnesic patients, such as BY[29] or NA[30] were impaired at short intervals on this task. By contrast, patients with amnesia owing to lesions of the hippocampus show a normal decay over short periods of time.[31–33]

Learning Ability

J.S. performed poorly on the visual and verbal learning subtests of WMS-R. For further assessment, more specific tests were used. On Rey AVLT[21,34,35] he showed a flat curve, (5,8,9,8,8) and delayed recall was 1.

For the following tests 10 volunteers matched for age and education served as controls.

Verbal Paired Associate Learning: we used Winnocur's method,[31] with repeated learning trials and immediate recall until the list was learned or until 9 trials were completed. The pairs were presented at a rate of 4 seconds in printed 15×10 cm. white cards. Firstly, we used a list of 12 pairs of words of high associative strength to learn, and then 12 pairs of low associative strength. Like most amnesics, J.S. was able to learn the first list quite easily, although with more difficulty than controls, but he was unable to learn the second list.

In a third experiment, 12 pairs of low associative strength were presented in a similar fashion to the latter test but this time he was instructed that for each pair he should try to imagine a picture connecting both words. J.S.'s performance was much improved, although still below normal performance. Thus, when encoding was reinforced through visual imagery, J.S. was able to retain material better, confirming the usefulness of dual encoding of information.[36]

For assessment of visual learning ability we used Biber's Visual Learning Test (VLT).[37] He showed a flat learning curve, retrieving between 4 and 6 pictures. An analysis of performance on these lists shows that his learning ability is limited to a reduced amount of material after which he cannot acquire any additional information. In the third or fourth trial, the number of stimuli that was recalled remained more or less stable and sometimes even decreased; the stimuli recalled did not always remain consistent. The impression is that J.S. relies mainly on his working memory, actually considering each trial as new material to be learned, with very limited benefit from previous exposures. On the basis of criterion of learning ability J.S. resembles Luria's description of the flat learning profile of frontal patients.[38]

These findings have also been reported in several patients with basal forebrain damage.[39] Information is more readily acquired when encoding is enhanced through visual imagery strategies. Improvement of learning through visual imagery techniques is seldom mentioned in case studies of amnesia, but when it was assessed it was similar to J.S.'s performance.[10]

Forgetting Rate

The distinction between deficits in initial acquisition and deficits in retention of information has been claimed to characterize different forms of amnesia.[40,41] The former has been described as the hallmark of diencephalic amnesia; the latter as a typical feature of mid-temporal lesions. The combination of good working memory, poor delayed recall, and good recognition has been reported to be the amnesic profile of basal forebrain amnesia.[39,42] J.S. had a very poor delayed recall on the Delayed Recall subtests of the WMS-R, Rey AVLT, and the Biber VLT. To further assess this issue, a new 15-word list was aurally presented in 10 consecutive trials and delayed recall was retested at different intervals (30 min, 1 h, 2 h, 2 days and 1 wk). The same test was given to the controls, but the learning trials were discontinued when the subjects reached J.S.'s maximum level (9 words), which they obtained in an average of 4.1 trials. As seen in Figure 2, J.S.'s rate of forgetting was dramatically steeper than that of controls.

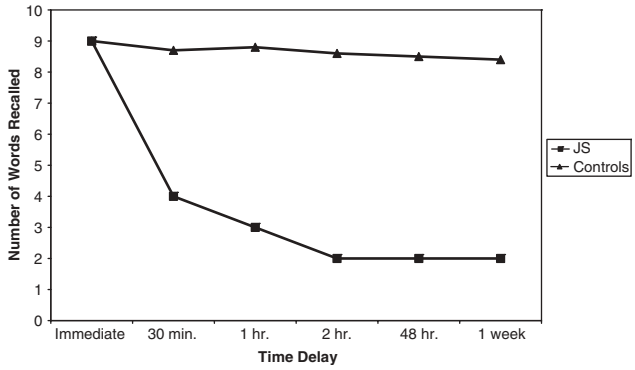

FIGURE 2. The forgetting rate of a list of words by J.S. and a control group.

Recognition

J.S.'s performance on the recognition part of Rey AVLT and the Biber VLT was very good. On the former, after a 20 minute delay, he was able to recognize 11 words out of the original 15, with 3 false positive answers. On the Biber test, after a 1 hour delay, he recognized all the figures without false positive answers. This disproportional advantage of recognition over free recall is usually attributed to retrieval deficits and is characteristic of basal forebrain amnesia.[11,43]

Retrograde Amnesia

Standardized tests for retrograde public memory could not be used owing to J.S.'s life history. In our interviews it was clear that he had had retrograde amnesia for the last 10 or 15 years. To obtain more objective information we used the Crovitz Test,[44] a structured questionnaire for semantic and autobiographic memory, prepared with the assistance of people from the Kibbutz, and a structured questionnaire on public events for the last 20 years. The structured interview for autobiographic memory contained 30 questions dating from the last 20 years. The questionnaire on public events contained 60 questions covering the last 20 years. It was also given to 10 normal controls matched for age and education, recruited from the same Kibbutz.

On the Crovitz Test, J.S. was given 10 words and asked to recall specific events of his life related to those words. For each event recalled he was asked about details. He was only able to recall with details 3 incidents, dated 1957, 1959, and 1967. A fourth word was "accident"; he recalled being in an accident in the Kibbutz, but he did not remember details. He was unable to recall anything related to the other 6 words.

On the autobiographic questionnaire, J.S. was able to answer most of the questions referring to 1970 to 1980, only 50% of the questions about 1980 to 1985, and 20% of the questions about the 1985 to 1991 period. The questionnaire on public events showed a similar gradient.

These results suggest that J.S. has a retrograde amnesia for autobiographic and public events covering a period of about 10 years before his disease, with temporal gradient. This profile has been described for patients with basal forebrain amnesia and Alcoholic Korsakoff syndrome.[45]

Semantic memory was investigated using similar methods and it was normal.

Procedural Memory

One of the most classical tests of procedural memory is the Tower of Hanoi Puzzle. Amnesic patients show a preserved ability to learn this task.[46] J.S. was tested on a computerized version of the task showing a normal ability to learn the procedural task within 3 sessions and to retain his learning between sessions.

GENERAL DISCUSSION

J.S. has an above-average IQ, preserved language functions, and visuospatial ability. His attention and concentration are preserved. Executive functions are quite impaired as judged by his everyday behavior, although this is not systematically documented by formal testing. He shows normal performance in Wisconsin Card Sorting Test and preserved working memory and abstraction ability but impaired temporal order judgment. The latter has been reported to be impaired in basal forebrain amnesics.[47]

J.S. meets the criteria for amnesia: loss of memory with preserved intellectual functions, a greater than 20-point discrepancy between FIQ and MQ, and demonstrable episodic memory deficits, with intact semantic and procedural memory.

An analysis of results lends support to the concept that lesions in different loci may be expressed as different forms of amnesia. J.S.'s amnesia is quite similar to that reported in patients with basal forebrain damage. He shows a decreased immediate free recall, a flat learning curve, and decreased delayed recall, both for verbal and visual material, with preserved recognition. A review on literature confirms that these are practically constant findings in basal forebrain damage, constituting a distinct form of amnesia. Other accompanying features are more variable. Attention and working memory may or may not be impaired, as well as results on tasks of PI. Considering the complexity of the frontal lobes, these variations probably reflect minimal differences in the extension of the lesion. Postoperative magnetic resonance imaging shows that the classical areas associated with memory, that is, the diencephalon and the hippocampus, are intact. Damage of the basal forebrain is quite extensive.

The theory that relates specific anatomic targets to specific stages of memory processes considers the diencephalon as an important locus for acquisition—the initial mnemonic processes—and the hippocampus as an anatomic substrate for consolidation. The basal forebrain would have an important role in retrieval.[48] A pure retrieval deficit, although clear in J.S., could not explain all of the results of the memory tests.

A different view of the nature of amnesia considers memory processing as the result of a complex network, in which certain anatomic loci would have a more prominent role in some stages of the process, but the final result would depend on the intactness of the net and the fiber bundles connecting the different loci.[49]

In the past decades, the contribution of the septal region to memory processing has been stressed.[10] The role of the septum is suggested by its direct and reciprocal connections with hippocampal formation. Positron emission tomography studies of amnesia after rupture and repair of aneurysm of the anterior communicating artery[50] have shown a secondary remote effect on the mid-temporal area suggesting that this amnesia may be owing to an indirect effect on the hippocampus. Whether the amnesia is the result of direct damage to septal nuclei or to the disruption of their connections to other memory centers remains an unanswered question and requires further investigation. Yet, the interest of this work is not to distinguish, among forebrain structures, the specific loci of amnesia, but to confirm previous works on the specific profile of forebrain amnesia and to provide detailed neuropsychologic testing.

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