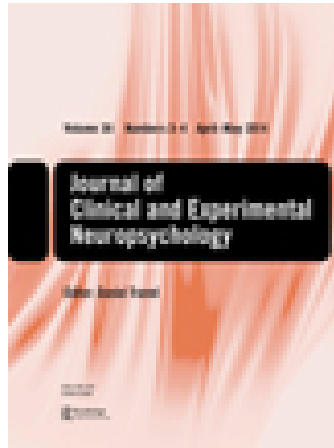


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Are Some Memory Deficits Unique to Lesions of the Mammillary Bodies?

Helmut Hildebrandt^{1,2}, Sandra Müller³, Barber Bussmann-Mork¹,
Sabine Goebel⁴, and Norbert Eilers⁴

¹ Health Research Unit, Ossietzky University Oldenburg, Germany, ² Central Hospital Bremen-Ost, Germany,

³ Department of Neuropsychology, Otto-von-Guericke-University Magdeburg, Germany, and

⁴ Rehabilitation Hospital Oldenburg, Germany

ABSTRACT

The role of the mammillary bodies in human memory is still in debate. A recent model of human amnesia proposes similar functions for the mammillary bodies and the hippocampus. But the main evidence for this model comes from animal studies using the delayed non-matching to sample paradigm. We describe a patient who developed a severe memory impairment after surgical removal of a germinoma. Postsurgical high resolution MRI revealed bilaterally shrunken mammillary bodies and an infarct of the left mammillary body. There were no other relevant lesions. Neuropsychological testing showed mildly impaired frontal lobe functions (executive functions, working memory and word fluency), almost intact learning and recognition, but severely impaired free and delayed recall. Experimental investigations revealed a reduced but preserved release of proactive interference and a pronounced impairment of recency and source judgments. We conclude that the mammillary bodies do play a prominent role in human memory, although the role differs slightly from that of the hippocampus.

INTRODUCTION

The mammillary bodies are one of the major efferent targets of the hippocampus (Nieuwenhuys, Voogd, & van Huijzen, 1991). Together with the fornix, the mamillo-thalamic tract, the anterior thalamic nuclei and the gyri cinguli form the well known Circuit of Papez, which plays a central role in episodic memory. Bilateral lesions of the hippocampus are accompanied by a dense and persistent amnesia (Cramon, Hebel, & Schuri, 1985; Kolb & Wishaw, 1993; Squire, 1992). Bilateral lesions of the thalamus often lead to severe memory disorders (Markowitsch, 1992). But it is still unknown whether bilateral lesions of the fornix (Gaffan, Gaffan, & Hodges, 1992; Hodges & Carpenter, 1991) or the mammillary bodies *per se* (Markowitsch, 1992; Cramon &

Hebel, 1989) result in an amnesic syndrome. In their recent review of studies on human amnesia and lesion studies in animals, Aggleton Brown (1999) have argued for a characteristic dissociation of impaired free recall and intact recognition after bilateral destruction of the hippocampi. According to their neuroanatomical model, a bilateral lesion of the mammillary bodies should have a similar functional consequence, whereas lesions outside the Circuit of Papez, i.e., involving the parahippocampal gyrus or the dorso-medial nuclei of the thalamus, should not lead to a dissociation between free recall and recognition.

Korsakoff's syndrome involves a lesion of the mammillary bodies, and these patients show a severe memory deficit (Kopelman & Stanhope, 1998; Kolb & Wishaw, 1993) concerning

Address correspondence to: Helmut Hildebrandt, Department of Psychology, University of Oldenburg, D-26111 Oldenburg. E-mail: hildebrandt@psychologie.uni-oldenburg.de

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recognition and recall (Kopelman, 1995). Recent imaging techniques and earlier neuroanatomical studies have shown that in these patients, not only the mammillary bodies are involved but also thalamic nuclei and the frontal lobes (Bitsch, Seipelt, Rustenbeck, Haug, & Nall, 1998; Harding, Halliday, Caine, & Kril, 2000; Kopelman, 1995; Visser et al., 1999). A clear correlation between the functional deficit and a local neuro-pathological process is therefore not possible.

The number of patients with traumatic lesions of the bilateral mammillary bodies, as reported in scientific publications, is small. Several brain arteries subserve this brain structure and therefore an isolated infarct is extremely improbable. Tanaka, Miyazawa, Akaoka and Yamada (1997) reported about a patient with a resection of a craniopharyngioma. The tumor or the resection led to an infarction and a shrinking of both mammillary bodies. The patient showed impaired memory for verbal information and relatively intact memory for visuo-spatial stimuli. But the patient of Tanaka et al. also had a low verbal IQ (as measured by the revised Wechsler Adult Intelligence Scale (WAIS), Wechsler, 1981). After the resection, there was no difference anymore between verbal IQ and the revised Wechsler Memory Scale (WMS-R, Wechsler, 1987), except for delayed recall. The patient improved in memory function after the resection of the tumor, although his mammillary bodies remained shrunken. This contradicts a causal relation between mammillary bodies and memory impairment and argues for a preoperative displacement of hypothalamic or subthalamic brain tissue which caused parts of the memory impairment. And because Tanaka et al. (1997) did not differentiate between recognition and recall, nothing can be deduced from their study with regard to the model of Aggleton and Brown (1999).

Dusoir, Kapur, Byrnes, McKinstry, and Hoare (1990) described a patient with a bilateral traumatic lesion of the mammillary bodies. This patient showed impaired verbal memory and some deficits of visuo-spatial memory. Unfortunately, this patient had experienced episodes of excessive alcohol consumption which may have contributed to the memory disorders. Though not

being the focus of the report, the performance in recognition and recall tasks are given separately. In the Rivermead Behavioural Memory Test (RBMT, Wilson, Cockburn, & Baddeley, 1992) the patient successfully performed the two recognition subtests, whereas all other subtests, in which he failed, concerned free recall. The data in the Auditory Verbal Learning Test (Rey, 1964) were quite similar: the patient documented a severely impaired performance in delayed free recall but a more or less normal recognition. However, he made more intrusions during recognition than the healthy control group. The patient of Dusoir et al. (1990) may therefore be interpreted as providing some support for the model of Aggleton and Brown (1990).

In general, the role that the mammillary bodies and the thalamus play in human memory may also be discussed under the heading of "amnesia for context information". Parkin (1984) Hunkin, Parkin, & Longmore (1993); Hunkin & Parkin (1994) have shown in several studies that patients with Korsakoff's syndrome, but also patients with traumatic thalamic lesions, have problems in judging which of the two stimuli is the one they have heard last (recency judgment). And in his nowadays classic investigation, Squire (1982) showed that Korsakoff amnesics do not show a release of proactive interference (as normal controls or amnesics of different etiologies do). Following him, the major problem of this patient group to be in dating the temporal context of information.

If these deficits in the memory for context are unique to patients with Korsakoff's syndrome, and if these deficits follow from a lesion of the mammillary bodies, the functional similarity of the hippocampus and other parts of the Circuit of Papez would be minor, compared to what the model of Aggleton and Brown (1999) suggests. We recently had the opportunity to study a patient after the resection of a suprasellar germinoma over a period of 20 months. This permitted us to report about a further case of an isolated lesion of the two mammillary bodies. Like the patient of Dusoir et al. (1990) and of Tanaka et al. (1997), our patient showed a severe memory impairment, which made reintegration into a profession impossible. We investigated his memory impair-

ment in relation to other cognitive functions and we tried to analyze the internal structures of the memory disorder in order to shed some light not only on the function of the mammillary bodies in recall and recognition, but also in the memory for context.

CASE REPORT

TS is a 24-year-old right-handed man. He left school after 10 years of education. He served an apprenticeship and was then employed as an electrician until he became ill. His earlier medical history is unremarkable. In 1996, TS began to suffer from a stiff neck. Because of recurring feelings of illness and dizziness he consulted a doctor who diagnosed pathological jerks and a positive Babinsky sign on the left side. When admitted to hospital, TS complained of visual disturbances, headache, and vomiting. Except for the pathological jerks, the preoperative neurological status was regular. TS was oriented about person, time, and place. Hormonal profile studies showed no deficits.

A pterional craniotomy and tumor extirpation was carried out (tumor size: 1.5×2 cm) and a germinoma with some necrosis was found histologically. The intraoperative severance of the hypophysis stem made a postoperative total hormone substitution necessary. TS was administered hydrocortison, thyroxin, and desmopressin. Postoperatively, he showed a reduction in drive and was disoriented though he recovered within a few weeks.

A spontaneous postoperative oculomotor paresis on the right side was described then, which soon regressed as well as a slight hemiparesis on the left side. One month later TS became apathic, however, this was reversible after a reduction of hydrocortison. Later, radiation therapy was started (24 Gy to the whole neuraxis and an additional 16 Gy to the sella region). During this therapy TS suffered from dysphagia and visual sensations. The ophthalmological assessment showed a bilateral hemianopsia which remitted later. Two months after this radiation therapy, TS came to our hospital. The first examination showed an alert and cooperative man, oriented

about person, place, and partially about time, but being unsure about month and day. His behavior was socially acceptable; but considering his age, it would sometimes have been more appropriate to a younger person. He exhibited no abnormalities such as episodic rage, sexual alterations, or emotional lability, except for being moody. He showed lack of drive but reported no hypersomnia. Alopecia after radiation and striae because of cortison therapy were obvious. He had a slight ptosis of the right eye, but no other signs of oculomotor paresis. Muscle stretch reflexes were slightly stronger on the left side; there was a latent hemiparesis on the left side. Other cranial nerves, sensory and cerebellar functions were all normal. Medication was: Hydrocortison 20-mg-5-mg-5 mg, L-Thyroxin 100 g-0-0, Testosteron dep. every four weeks 250 mg i.m., Desmopressin once a day. During the following weeks his weight increased about 20 kg, and TS reported increasing appetite.

Neuro-imaging was performed 7 months after the operation with a 1.5 Tesla MR scanner with standard quadrature head coil. Conventional structural high resolution images (42 coronal, 40 sagittal and 40 transversal, 2 mm thickness) were followed by T2*-weighted gradient echo sequence (3 mm slices, flip = 30°). The MRI slices showed small, atrophic mammillary bodies. The left mammillary body revealed a lower signal intensity with T2- and T2*-weighted MR images, which may be due to a hemosiderin deposit. The right mammillary body was shrunken, compared with those of normal subjects. Moreover, we found a substance loss at the anterior pole of the temporal lobe and hemosiderin deposits along the operative route to the germinoma (see Figs. 1 and 2), but no other pathological deformation or re-growth of the tumor.

NEUROPSYCHOLOGICAL INVESTIGATION

At the time of the neuropsychological examination, i.e., 4 months after resection of the germinoma, the patient's reading, writing, calculation, and praxia were normal. His general IQ was 96, his verbal IQ was 101, his performance IQ was 86 (see Table 1). The reduction of the performance

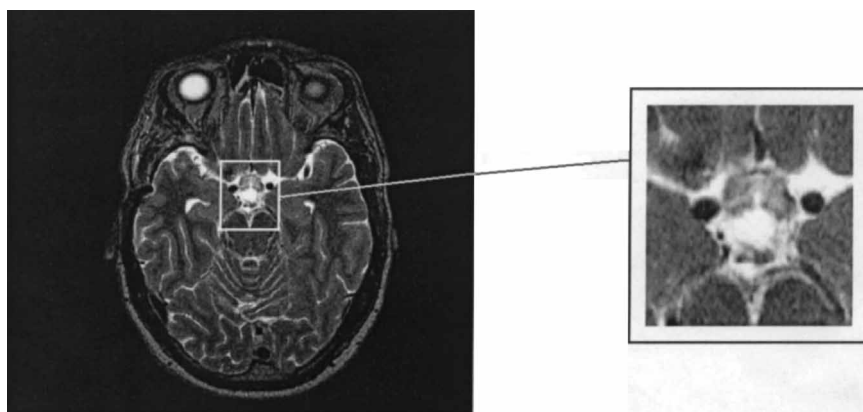


Fig. 1. T2-weighted sagittal NMR slice of the mammillary bodies of TS (see arrow). The slice shows an infarct of the left mammillary body. The right mammillary body is shrunken and smaller, compared to healthy subjects. No further substance defects are visible.

IQ can partly be explained by a general psychomotor slowing, because only the Digit-Symbol Test, which relies highly on psychomotor speed, indicated an existing impairment. We investigated TS again 16 months later and found a performance IQ of 108 and a verbal IQ of 95.

The visual fields of the patient were investigated using the Neglect Test of the Attention Testing Battery (ATB) (Zimmerman & Fimm, 1992), and at the time of our testing no visual field defect at 20° to the horizontal and 15° to the vertical line was apparent, but there was still a relative slowing in the right visual field. Visual conjunction search (Treisman & Gelade, 1980), tested by the Visual Scanning Test of the ATB, was slowed down but the number of misses was within normal range (see Table 1).

Visual reaction times studied by the subtest "Alertness" of the ATB were slowed with and also without a warning signal. The capacity to divide attention between visual and acoustical stimuli was slowed down, but misses and false positives were within normal range. A slowing down of the reaction time without an increased error rate was obvious, also for selective visual attention. Working memory function, studied by the subtest "Working memory" of the ATB, a variant of a Two-Back paradigm (Braver et al., 1997; Kramer et al., 1997), was normal with regard to errors, but reaction time was slowed down (see Table 1). The same held for a subtest of the ATB with alternating the response between

two criteria for changing stimuli pairs. We retested TS four months later. His reaction time was still slowed down, but only during complex tasks.

TS achieved only three categories in the Modified Card Sorting Test (MCST, Nelson, 1976), but he made only four perseverative errors. He was able to solve planning problems, like finding an optimal temporal solution for five different appointments at one afternoon plus consideration of the time needed for the distance between the appointments if the ordering changed. But compared to a control group he was impaired in two fluency tasks, i.e. in first letter fluency and semantic fluency (see Table 3).

MEMORY

Clinical Observation

At the beginning of his stay at the hospital, TS showed severe memory impairments in everyday life, p.e. asking the same question several times without remembering that he had already posed them before. Only after three weeks, he learned the whereabouts of the neuropsychological office. He was not able to name the participants of the memory training group 10 minutes after the session and never managed to give a full account of where exactly each of the patients had been seated, although they only seldom changed their seats.

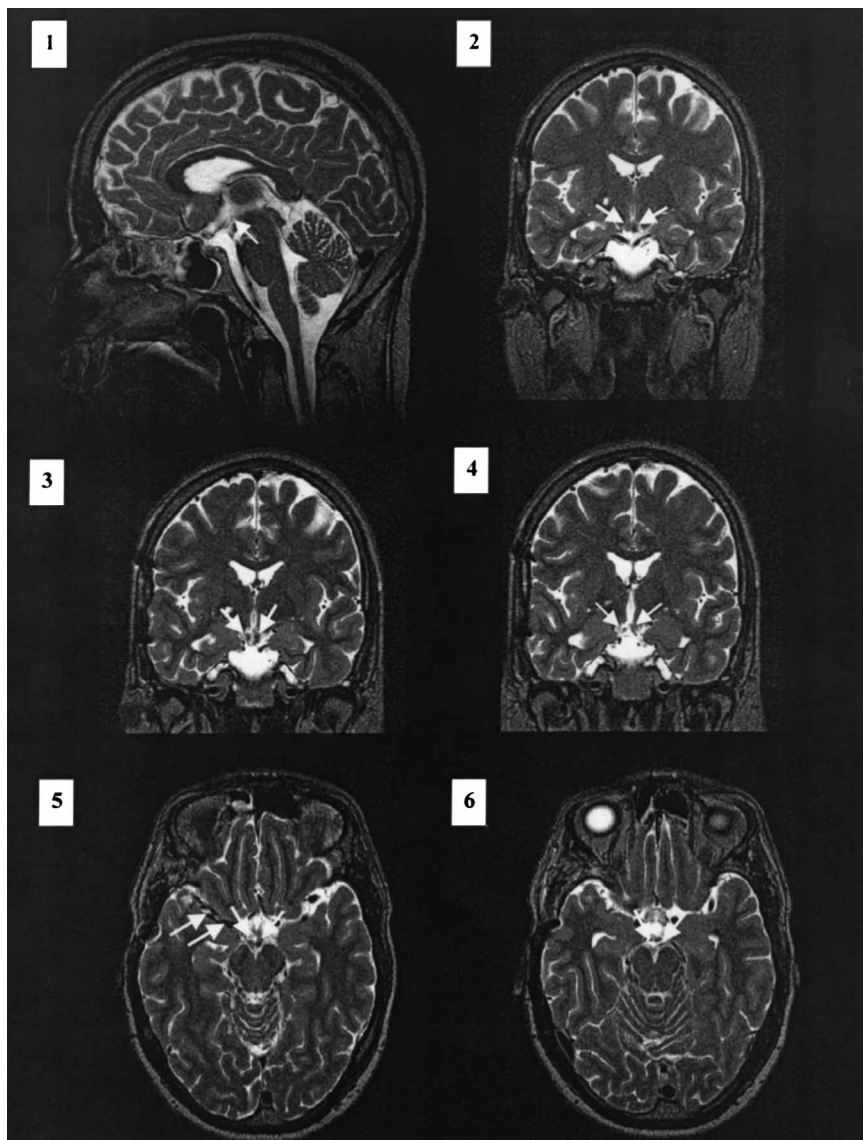


Fig. 2. T2*-weighted sagittal (1), coronal (2–4), and axial (5,6) NMR slices of the mammillary bodies of TS (see the arrows). The slices show an infarct of the left mammillary body. A T2* scan (not shown) revealed hemosiderin deposits inside this mammillary body. The right mammillary body is shrunken and smaller compared to healthy subjects. The two arrows on the left side in (5) show the operative access to the germinoma resulting in hemosiderin deposits along the anterior pole of the right temporal lobe. No other substance defects are visible.

Autobiographical memory was investigated with a structured interview covering past personal events. The items selected for this interview were based on an interview with his parents. Moreover, we tested his professional knowledge by using standard textbooks about electronics. No retrograde amnesia was apparent. But information

about the last half year before the operation seemed somewhat less complete than other biographical information.

Testing

We analyzed TS' memory with the WMS-R, the Recurring Figure Test (RFT, Rixecker, 1978), the

Table 1. Neuropsychological Results.

General Intelligence (WAIS)		
Verbal intelligence	61 Value Points	IQ = 101
Performance intelligence	44	IQ = 86
General intelligence	105	IQ = 96
Vision		
Left visual field: Reaction time	784 msec	T < 25
Right visual field: Reaction time	629 msec	T < 25
Conjunction search: Response time to feature absent trials	13772 msec	T < 25
Conjunctions search: Omissions	2	PR < 27
Attention		
Visual reaction time not warned	335 msec	T: 25
Visual reaction time warned	275 msec	T: 34
Divided attention: Reaction time	762 msec	T: 33
Divided attention: Omissions	3	T < 39
Executive Funktion		
Working memory: Reaction time	1155 msec	T: 31
Working memory: Omissions	1	T > 44
Alternate response: Reaction time	1635 msec	T: 29
Alternate response: Errors	7	T: 40
Modified Card Sorting Test (MCST) categories	3	
Total error (TE)	15	-0.6
Perseveration error (PE)	4	+1.3
PE/TE × 100	26.6	+0.26

recurring words test (RWT, Stollmann, 1990), the Rey-Osterrieth Figure (Rey, 1941), the RBMT (see Table 2), and the German version of the California Verbal Learning Test (CVLT, Ilmberger, 1988) (see Table 2).

The WMS-R showed a normal attention/concentration index of 93, a somewhat reduced verbal memory performance of 85, a severely impaired visual memory of 67, and for delayed recall an index of 75. The RBMT also yielded a severe memory disorder. Only the recognition of pictures and faces was within normal range. Immediate recall of the Rey-Osterrieth Figure was unimpaired. The RFT was used twice with an interval of one and a half year: at the first testing TS scored at the 3rd percentile rank for his recognition performance in the RFT, but he made only few false alarms (56th percentile rank). One and a half year later he scored at the 2nd percentile rank for recognition and at the 62nd percentile for the number of false positives. For the RWT he scored below the first percentile for recognition hits and at the 54th percentile for the number of false alarms.

The CVLT revealed a somewhat impaired learning rate, a nearly normal sum of learned items during the five presentations of the learning list and a pronounced forgetting ratio after interference. TS profited, moreover, from semantic cues and showed a normal recognition performance. The number of intrusions during the recognition testing was definitely higher than that of healthy controls (Hildebrandt, Gießelmann, Sachsenheimer, & Schwendemann, 2001).

Experimental Investigations

In order to specify TS' memory disorder, we executed several experimental investigations and compared his performance with that of a control group matched for age and education.

One of the major results of memory testing was a discrepancy between impaired delayed recall and almost intact learning. In order to investigate the ability to retain information over time, we presented a short story of about 50 separate items to TS and asked him to reproduce this story immediately and after a delay of 15 minutes (see Table 3). Compared to a healthy

Table 2. Memory Test Results.

Wechsler Memory Scale	Raw score	z-score
Mental Control	6	+0,7
Figural Memory	5	-1,2
Logical Memory I	19	-0,9
Visual Paired Associates I	6	-3,1
Verbal Paired Associates I	20	-1,0
Visual Reproduction I	29	-0,8
Digit Span	16	-0,05
Visual Memory Span	19	+0,3
Logical Memory II	11	-1,4
Visual Paired Associates II	6	+0,4
Verbal Paired Associates II	7	-1,1
Visual Reproduction II	18	-2,1
Recurring Figures Test RFT		
Ri +	22	-2,8
Fa +	8	+0,4
Diff.	14	-1,7
Rivermead Behavioural Memory Test	9	-2,7
		CON (<i>n</i> = 20)
CVLT, Trials 1-5*	7, 8, 10, 10, 9	8.1, 11.2, 13.4, 14.6, 14.8
Immediate postinterference free recall	5	12.95 (<i>SD</i> : 2.48)
Recognition	16 hits out of 16, and 3 intrusions	Hits: 15.5 (<i>SD</i> : 0.95) Intrusions: 0.1 (<i>SD</i> : 0.31)
AVLT, Trials 1-5*	6, 8, 8, 10, 11	8.7, 11.2, 13.0, 13.8, 13.9
Immediate postinterference free recall	6	12.75 (<i>SD</i> : 1.65)
Recognition	15 hits out of 15, and 1 intrusion	Hits: 14.5 (<i>SD</i> : 0.89) Intrusions: 0.32 (<i>SD</i> : 0.48)

*There are no standardized German versions of the CVLT and the AVLT available. We investigated therefore 20 control subjects of the same age and a similar education for the control figures.

control group, TS scored below two standard deviations already in the immediate reproduction. But he was even more impaired after a retention interval of 10 minutes. He lost 11 items during this period (about 50%), whereas the control group lost only about 1.3 items (about 3%).

In a second step we investigated his recognition performance and his ability to judge the relative position of items in a sequence (recency judgment). We presented a series of 200 words. After every 10 words, we presented word pairs and asked either if one of these had been presented earlier or which of the two words had been presented more recently. One word of the pairs or in the case of recency judgment both had been part of the list of 10 words which the patient had just seen. TS recognized all items in the recognition condition. His performance was better than that of a healthy control group (see

Table 3). In the recency condition he made 40% errors. This is near chance level (50%) and more than one standard deviation below the performance of the control group.

The next experiment examined the patient's source memory function. We presented the patient with 10 categories for which he then had to name and write down one item each. Subsequently, the two experimenters chose two items, different from those the patient had named for each of the 10 categories. The three lists were presented in the following order: Patient's list read by the patient himself, list 2 presented by Experimenter 1, and list 3 presented by Experimenter 2. Each list was presented twice and after the presentation of the three lists the same procedure was started again. Ten minutes after the last learning trial of list 3, the three words of each category (the one of the patient and the two

Table 3. Experimental investigations.

Tasks	TS Mean	Mean (<i>SD</i>) CON (<i>n</i> = 18)
Fluency Tasks		
First Letter Fluency	24	29.9 (6.9)
Semantic Fluency	24	31.1 (4.8)
Text reproduction		
Immediate recall	23	34.91 (5.18)
Delayed recall (10 minutes)	12	33.68 (5.20)
		CON (<i>n</i> = 10)
Proactive interference		
1. List	5.5	6.00 (0.64)
2. List	5.0	5.00 (1.08)
3. List	3.5	4.67 (0.85)
4. List	4.5	4.94 (0.86)
5. List	5.0	4.83 (0.82)
Release List	6.0	6.78 (0.58)
Source judgment (three lists)		
Permutation errors – experimenters' lists	9.00	3.9 (2.30)
Permutation errors – own list with experimenters' 1 list	3.00	0.25 (0.46)
Permutation errors – own list with experimenters' 2 list	3.00	0.25 (0.71)
		CON (<i>n</i> = 18)
Source judgment (two lists): Errors	6.00	0.32 (0.95)
Recognition: Errors	0.00	1.39 (1.24)
Recency: Errors	4.00	1.72 (1.41)

For an explanation of tasks see the text.

of the experimenters) were presented on the computer screen and the patient had to decide which word had been taken from his own list, which word from the list of Experimenter 1 and which word from Experimenter 2. Each word was presented three times and the position on the screen was interchanged semi-randomly. Analysis of errors showed that TS scored near chance (30% false decisions) for discriminating between the lists of the two experimenters, but above chance for his own list (10% false decisions). Compared to the performance of the control group TS was severely impaired in all respects, and the better performance with his own list may be caused by the order of presentations.

We focused on source amnesia in a second experiment where TS had only to discriminate between items which he had read himself and items he had heard by one of the experimenters. This was done to examine if the number of lists might play a role. This time TS made 20% errors in source discrimination, i.e., was definitely better

than chance (which would have meant about 15 errors). Again he scored clearly below the control group (see Table 3).

The release of proactive interference was analyzed twice. The patient was asked to memorize 40 items (first trial: animals; second trial: professions), separated each into five lists of eight items. After these five lists, the release list was presented. In the case of animals, the release list entailed eight furniture items, in the case of professions the second list was made up of eight different articles of clothing. We averaged the results of both trials and compared them with scores of a healthy control group (see Table 3). During the presentation of the first two lists TS remembered as many items as the control group. At the third list, his performance dropped below that of controls, but recovered at the fourth and fifth list. At the release list, TS performance was impaired again (one standard deviation below the control group) but he reproduced more items than with the lists 2 to 5. Thus he showed effects of proactive

interference similar to the control group, but a quantitatively reduced release effect. In addition, we analyzed the perseverations and intrusions of TS at the proactive interference and at the release lists. No difference was found in this respect.

DISCUSSION

Although neuroanatomical models predict a major role of the mammillary bodies for memory, empirical findings about their function are limited. Earlier theories stem from investigations of patients with Korsakoff's syndrome. But most of these cases show a loss of substance, not only of the mammillary bodies but also of the anterior and mediodorsal thalamic nuclei and the frontal lobes. We were able to investigate a patient with a bilateral shrinking of the mammillary bodies and an infarct of the left mammillary body. His severe memory impairment prevented a regular employment. Seven months after the resection of a germinoma and three months after the end of the radiation, the MRI scans did not show brain atrophy and only one additional abnormal region in front of the anterior pole of the right temporal lobe, which was due to the operative access and which does not explain the memory deficit.

The memory disorder of our patient resembled the one shown by patient BJ, as reported by Dusoir et al. (1990) and that of SS (Tanaka et al., 1997), i.e., a further two patients with isolated lesions of the mammillary bodies. In the case of Dusoir et al., his patient's periods of heavy drinking make an interpretation of the memory impairment difficult; Tanaka et al.'s patient recovered partly from his memory problems though his mammillary bodies remained shrunken.

Is the memory disorder of our patient specific or could it be the result of an additional impairment? As a first step it can be argued that what we have found is a selective memory impairment without a general intelligence reduction. Our patient showed an average verbal IQ and, at least at a second investigation, a normal performance IQ. The memory quotient of the WMS-R was significantly reduced and only the attention/concentration index of the WMS-R reached a normal score. Thus the memory

disorder cannot be traced back to a general reduction of the IQ.

On the other hand, the memory impairment could be the result of a general cognitive slowing, because of the increase in the reaction time in attention tasks. There are at least two aspects which contradict such an interpretation. First, the memory impairment was not an impairment of the learning process (encoding) but of free recall. One would expect that an attentional slowing should also impair the encoding process, but this was not the case. Even more to the point is a second argument: the patient was able to reduce his reaction times during the time of the investigation but this recovery process was not paralleled by an increase in his memory performance.

TS had some problems with word fluency tasks, such as first-letter fluency or categorial fluency, in the MCST categories but did not show perseverations. Additional testing did not reveal impairments of working memory, of planning or in alternate response, leaving aside his moderate but general slowing which might also explain the results of the verbal fluency tasks. We would therefore argue that both neuroanatomical aspects and additional neuropsychological investigation point to a specific memory disorder due to a lesion of the mammillary bodies.

The function of the mammillary bodies plays a prominent role in two different models of human memory. Aggleton and Brown (1999) have published a model which postulates a Circuit of Papez proper, involving hippocampus, fornix, mammillary bodies and the anterior nucleus of the thalamus. According to their theory, lesions of this circuit produce an amnesia of free recall but leave recognition performance more or less intact. Recognition performance stems from two different sources: conscious recollection and a feeling of familiarity. While conscious recollection is made possible by the Circuit of Papez, the feeling of familiarity may depend on the parahippocampus, the inferior thalamic pedunculus, and the dorso-medial nucleus of the thalamus. Because these latter structures remain preserved, patients with isolated lesion in the Circuit of Papez do not show impaired recognition performance.

Characterizing the memory impairment of our patient, who had a bilateral lesion of the Circuit of

Papez at the level of the mammillary bodies, our main focus concerned free recall, especially delayed free recall; in contrast to this, the encoding process was more or less normal. TS reached an almost normal learning rate and an almost normal total recall during the five learning trials of the CVLT. Recognition performance was also less impaired than delayed free recall. In the RBMT the items concerning recognition of faces and of pictures were in the normal range. TS also showed normal recognition in the CVLT, but similar to the patient of Tanaka et al. (1997) he made more false alarms.

Most of the neuropsychological tests on recognition involve a judgment of items taken from target and irrelevant interfering lists. This kind of recognition testing implies a confounding with context memory (irrelevant but learned information may intrude into critical items). We were able to demonstrate experimentally that TS's recognition performance was normal, if the testing did not involve those items already heard in different contexts. TS actually performed better in this situation than the control group, a result which speaks clearly in favor of a dissociation between recognition and free recall, as proposed by the model of Aggleton and Brown (1999).

On the other hand, RWT and RFT also use only new items as distractors. Despite that, TS documented an impaired recognition performance in both tests. One possible explanation for these contradictory results might be that the deficit in recognition by awareness cannot be compensated fully by recognition by familiarity. In the case of RFT and RWT, the load on recognition may be too high to be compensated for, whereas for RBMT, CVLT and in our experimental investigation, the load remains beneath a threshold and thus a compensation is made possible.

Such a view would endorse the second finding of an additional impairment of source and context memory in our patient. The experimental investigation showed a severe impairment in recency judgment despite an intact recognition of the items. We also found an increase of source errors in judging the items of different lists. And a reduced memory for context may also explain the increased rate of false alarms in the CVLT despite the low rate of false alarms in the RWT and RFT.

These tests differ in either using distractors which have been presented before or not. Deficits in source memory and recency judgment have been found after frontal lobe lesions (Nelson, 1976) or thalamic lesions (Parkin, Rees, Hunkin, & Rose, 1994) including also Korsakoff's syndrome (Hunkin & Parkin, 1993; Parkin, Leng, & Hunkin, 1990; Mayes et al., 1989) but have not been studied in patients with isolated lesions of the mammillary bodies. Our results therefore provide additional evidence for the assumption that diencephalic structures play a role in contextual memory. Concerning the lack of release of proactive interference which has been shown in patients with Korsakoff's syndrome but not for temporal lobe patients (Squire, 1982), the situation is less clear because our patient showed a small release effect and did not produce more intrusions than patients with Korsakoff's syndrome (Warrington & Weiskrantz, 1978).

Although this is not discussed by Aggleton and Brown (1999) as a consequence of their theory, one may argue that bilateral lesions of the mammillary bodies should not lead to an impairment of the memory for context, because earlier studies (Parkin et al., 1990) on patients with lesions in the medio-temporal lobe did not reveal deficits in this respect. But, one should keep in mind that these patients had extended medio-temporal lesions involving also the parahippocampus, and therefore, had the capacity of recognition by familiarity. Theoretically, an impairment of context memory may be a direct consequence of a recognition process based – due to an isolated lesion of the hippocampi – only on familiarity which should lead to an increased number of intrusions if concurrent and irrelevant information has been presented recently. But, as far as we know, studies dealing with this question have not yet been carried out.

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