# Thalamic Amnesia Following Venous Infarction: Evidence from a Single Case Study

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The present paper describes the case of a right-handed Italian–English bilingual male patient suffering from amnesia following a bilateral thalamic lesion due to venous infarction. Within a few months from the onset, the lesion gradually shrank and was finally confined to the left thalamus only, in particular to the antero-medial portion and the pulvinar. The mammillary bodies and the cortical areas of both hemispheres were spared. After the regression of a series of generalized cognitive deficits implying slow psychic activity, confusion, and spatial and temporal disorientation, the patient presented a persistent amnesic syndrome with mild language disorders, both in Italian and in English. Major disorders of memory included a dissociation between verbal memory, which was severely impaired, and spatial memory, which was largely preserved. © 1997 Academic Press

### INTRODUCTION

Several different thalamic pathologies, such as chronic alcoholism, tumors, vascular disorders, and stereotaxic neurosurgical operations, may cause amnesia. Since lesions due to neoplastic formations or alcohol abuse are rarely confined to the thalamus only, most cases of pure thalamic amnesia

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are the consequence of vascular disorders (Schott, Mauguière, Laurent, Serclerat, & Fischer, 1980). In particular, thalamic hemorrhages are relatively frequent, accounting for 10% of all intracerebral hemorrhages (Choi, Sudarsky, Schachter, Biber, & Burke, 1983; Kawahara, Sato, Muraki, Tanaka, Kaneko, & Uemura, 1986). Since patients who have suffered from this pathology very often present with amnesia, thalamic hemorrhage represents the major cause for thalamic amnesia. On the other hand, ischemic infarctions offer the best conditions for studying thalamic amnesic syndromes, since pressure effects, such as compression of other adjacent structures, are limited (Graff-Radford, Eslinger, Damasio, & Yamada, 1984; von Cramon, Hebel, & Schuri, 1985). Thus, many cases of thalamic amnesia due to arterial infarction are reported in the literature, but, to our knowledge, no thalamic amnesia following venous infarction has been described yet. However, thalamic amnesia is still a puzzling problem. The frequent co-occurrence of cognitive deficits, other than amnesic ones, often makes it difficult to distinguish thalamic amnesia from thalamic dementia. Moreover, it is still rather unclear whether thalamic amnesia should be considered an independent form of the thalamic syndrome (Graff-Radford, Tranel, Van Hoesen, & Brandt, 1990) and whether retrograde amnesia is a constant symptom of thalamic amnesia (Butters & Stuss, 1989; Parkin, Rees, Hunkin, & Rose, 1994). Finally, the type and nature of the functional asymmetries of the thalamic nuclei still must be defined (see Parkin & Leng, 1993, for a review).

A description of a case of venous infarction provoking a bilateral thalamic lesion will follow. After a few months from the onset of the infarction, the ischemic area shrank dramatically and the patient's initial cognitive impairments cleared up, while an amnesic syndrome characterized by severe anterograde verbal amnesia and a slight, transient, retrograde amnesia became evident.

#### CASE REPORT

D.B., born in 1951, is a right-handed Italian–English bilingual engineer who lived in Australia from age 3 to 13 with his Italian family. There he went to an English school and was immersed in an English-speaking environment, whereas he spoke Italian within his family. After returning to Italy, he continued to learn and use English at least once a week. Prior to the vascular disorder that brought him to our attention, D.B. was always healthy; he had never smoked, drunk, or made use of other toxic substances. No other risk factors were present in his anamnesis. At the beginning of September 1992, his relatives noticed that his mood had substantially changed: having been gregarious and outgoing before, he became apathetic and introverted; in addition, he started to have some difficulty in remembering recent events. On September 10th, while he was on business in a town not far away from his home, he suddenly became confused and disoriented as to time and space, and he was admitted to the Neurological Department of the Hospital in Montebelluna, Treviso. During hospitalization, the patient manifested hypersomnia, disorientation as to time and space, mental slowness, and loss of memory for autobiographical events. In addition, he was hardly aware of these impairments. Spontaneous speech prosody was monotonous and with low pitch, verbal production was apparently fluent, and no confabulation was observed. The patient's neurological signs were rather mild, consisting in a deficit of the seventh right cranial nerve and a moderate hyperreflexia of the right upper and lower limbs. EEG showed generalized slowing of electrical activity, prevailingly on the left. A CT scan revealed a bilateral asymmetric lesion to the thalamus. MRI after 7 days postonset evinced an ischemic area involving the antero-medial portions of the right thalamus and the whole left thalamus, while the mammillary bodies and the cortex of both cerebral hemispheres were undamaged (Figs. 1a and 1b). An angiogram manifested the presence of an arteriovenous fistula between a choroidal branch of the carotid system and the deep venous system. An excessive blood supply in the deep venous system, thus, had provoked an ischemic infarction of the surrounding tissue. By October 6th, when D.B. was delivered to the Neurosurgical Department at the General Hospital of Verona, hypersomnia and disorientation had regressed, whereas psychomotor activity was still slow and evident anterograde and retrograde amnesic deficits persisted. The patient underwent a surgical embolization of the fistula, which was successful up to 90%, thus dramatically improving cerebral blood flow. Two weeks later he was discharged from the hospital: memory deficits persisted, while neurological disorders had disappeared. Soon after, mental slowness, confusion, and temporal and spatial disorientation also significantly regressed, allowing the patient to resume his working activity. At 3 months postonset, the patient's ability to engage in the activities of daily life (ADL) by resuming social relations ranked at level 1 of the ADL scale (see Kawahara et al., 1986). In March, 1993, D.B. underwent another surgical embolization leading to the complete occlusion of the fistula. Follow-up MRI (Figs. 2a and 2b) performed 2 months later revealed considerable restriction of the ischemic area: it had practically disappeared on the right side and only affected the left anteromedial region, in particular the anterior nucleus, the dorso-medial nucleus of the thalamus (DMNT), and the left pulvinar. Retrograde amnesia gradually disappeared and within 10 months memory for events preceding the disease was nearly normal. On the other hand, anterograde amnesia partly improved, but persists after more than 2 years from the onset of the pathology.

## PSYCHOMETRIC ASSESSMENT

Psychometric assessments were performed at the Institute of Human Physiology at the University of Verona. The patient was assessed four times: once during the acute phase immediately after the first neurosurgical operation

### THALAMIC AMNESIA

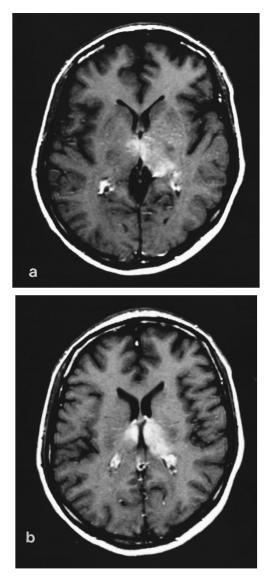


FIG. 1. (a, b). Select magnetic resonance images, T2 weighted, showing D.B.'s acute lesions. Damaged areas appear white. The left hemisphere is represented on the right. The exam was performed at the Montebelluna Hospital, on September 17, 1992.

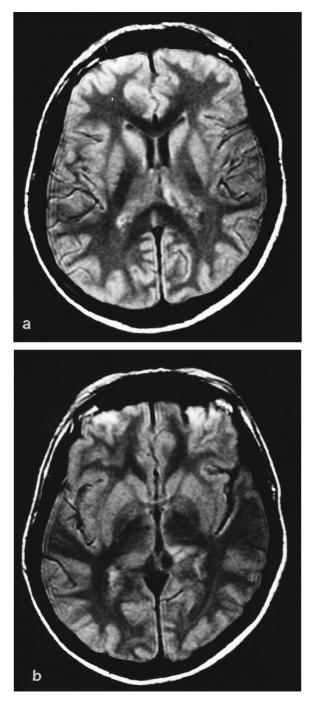


FIG. 2. (a, b). Select magnetic resonance images, protonic density, showing D.B.'s chronic lesions. Damaged areas appear white. The left hemisphere is represented on the right. The exam was performed at the Ospedale Maggiore, Verona, on May 26, 1993.

#### THALAMIC AMNESIA

		Assessmen			
				Mean	
	Ι	II	II	Score	SD
Intelligence					
WAIS					
VIQ	88	115	np		
PIQ	105	116	np		
IQ	95	116	np		
Raven PM 48	35	44	np	36.18	± 7.43
Arithmetic abilities					
Judgment test					
Calculation (max $= 5$ )	5	5	np		
Numbering $(max = 5)$	4	4	np		
Full score	9	9	np	9.88	± 0.39
Frontal lobe functions					
Verbal fluency	8	16.25	15.75	19.94	$\pm$ 5.75

## TABLE 1 Neuropsychological Examination

Note. np, not performed.

(October 1992) and three times during the chronic phase after 5, 9, and 24 months postonset (February 1993; June 1993; and December, 1994, respectively). If not otherwise specified, we refer to Spinnler and Tognoni (1987) for testing procedures and scoring. Scores below 1 *SD* of the control average are considered pathological indexes.

# NEUROPSYCHOLOGICAL EVALUATION

Even in the acute phase the patient's temporal and spatial orientation were not impaired. Attention, perception, and praxias were well preserved. Spontaneous speech in Italian was fluent, but dysprosodic and with low pitch. At times D.B. produced semantic paraphasias (e.g., *Examiner:* "How many weeks has a year?" *Patient:* "Twelve and thirteen a leap year") and was also slightly anomic. In this phase he showed the following major cognitive symptoms (see Table 1):

Intelligence D.B.'s verbal IQ according to the WAIS-R (Wechsler, 1981) scale was low, mainly because of his poor arithmetic performance (see below). On the *Raven Progressive Matrices* (1938) he scored within 1 *SD* from comparison mean; however, this score must be considered below normal given the patient's high level of education.

*Frontal lobe functions.* On the *Verbal Fluency Test*, a reliable measure of frontal lobe efficiency (Speedie & Heilman, 1983), D.B. scored very poorly and displayed also a tendency to verbal perseverations.

*Arithmetic abilities.* D.B. had no difficulties in performing the very easy mental calculation subtest of the *Arithmetic Judgement Test*, but he made 9 errors out of 14 trials on the *WAIS-R* arithmetic subtest, mainly because he needed too much time to answer the items. However, he was not able to make a progressive subtraction of 3 digits starting from 20. This test is considered an index of the correct functioning of the frontal lobes, rather than a simple arithmetic task.

After 5 months from the onset of the illness, D.B. showed a dramatic improvement of his intellectual abilities, which was verified by means of the *WAIS-R* (2 errors out of 14 trials on the arithmetic subtest) and the *Raven Progressive Matrices*. On the *Verbal Fluency Test* he performed within 1 *SD* interval from comparison mean, but, again, this was rather poor given the patient's high educational level. In this phase D.B. still presented with semantic paraphasias (e.g., *Examiner:* "What is a synagogue?" *Patient:* It is the church of the Arabic''). He was administered the *Bilingual Aphasia Test (BAT)* according to Paradis (1987), which showed fluent spontaneous speech both in Italian and in English with some phonemic and semantic paraphasias and mild disorders of comprehension, writing, and reading in both languages. The most severe disorders mainly affected lexical access of both languages (see also Fabbro, Peru, & Skrap, 1995).

In the course of the third assessment (9 months postonset), a thorough analysis of the patient's arithmetic disorders was done by means of the *Acalculia Test* (Miceli & Capasso, 1987). The knowledge of arithmetic rules and the numbering system turned out to be well preserved. D.B. performed flawlessly when reading or repeating numbers with less than 6 digits; in contrast, when presented with numbers of 6 digits, D.B. made 3 errors out of 5 trials in both the repetition (e.g., 527506 instead of 527106) and the reading tasks (e.g., the number 328762 was split as 328 and 362). D.B. made 5 errors out of 60 trials (8.33%) in the written calculation subtest; he also made 12 errors out of 55 trials (21.8%) in the mental calculation subtest requiring a greater involvement of memory.

## MEMORY ASSESSMENT

Table 2 shows D.B.'s performance in memory tasks.

## Immediate Memory

Verbal and spatial memory span were within normal scores in both the acute and chronic phase.

# Short-Term Memory

For the assessment of verbal and spatial short-term memory, the *Recency Effect of the Serial Position Curve* and the immediate reproduction of the

## THALAMIC AMNESIA

		Asses				
	I	II	Ш	IV	Mean score	SD
Immediate memory						
Verbal memory						
Digit forward test	4	6	6	np		
Bisyllabic word repetition test	4	4	4	np	4.75	0.86
Spatial memory						
Corsi's Block Tapping Test	5	5	5	np	5.11	1.01
Short-term memory						
Visuo-spatial memory						
Rey-Osterrieth complex figure	20	np	np	np	22	4.9
Verbal memory						
Serial position curve						
Recency effect	19	18	np	14	15.63	3.47
Long-term memory						
Implicit memory						
Maze learning test	14	np	np		13.41	
Visuo-spatial memory						
Rey-Osterrieth complex figure 15 min	16	26	np			
Picture						
Recall /30	np	np	15		20.4	2.07
Recognition /30	np	np	29		28	1.58
Corsi's block tapping supra span	4.53	18.04	18.9	28.28	23.18	5.67
Rey memory test						
Subtest 2	np	np	14	np	14.7	1.1
Subtest 4	np	np	19	np	16.4	1.8
Verbal memory						
Story recall						
Immediate recall	3	3.6	6.7	np		
Delayed recall	5.5	6.9	7.3	np		
Total recall	8.5	10.5	14	np	13.32	2.65
Serial position curve						
Primacy effect	1	1	np	4	5.05	2.11
Selective reminding test	22	np	np	15	135.3	25.9
Word list			-		12.2	
Immediate recall/30	np	np	7	14	13.3	2.12
Delayed recognition/30	np	np	13	15	26.6	3.07
Rey memory test			10		15.7	1.0
Subtest 6	np	np	12	np	15.7	1.8
Remote memory						
MLT '88						
Verbal	24	22	np	26	27	1
Pictorial	19	24	np	27	27	1
Famous personalities of the present	3	np	7	7	8.14	1.14
Semantic knowledge						
Animals	9	np	np	10	10	
Common objects	10	np	np	np	10	
Verbs	10	10	np	10	10	
Famous personalities of the past	1	7	np	8	8.08	0.8

Note. np, not performed.

*Rey–Osterrieth Complex Figure* (Osterrieth, 1944) have been used, respectively. In both tests D.B. scored normally.

# Long-Term Memory

# (a) Procedural Memory

The *Maze Learning Test*, following De Renzi, Faglioni, and Villa (1977), was used. The number of trials made by D.B. to reach the goal was within the normal range.

# (b) Spatial Memory

The *Corsi's Block Tapping Test*, which implies the learning of a supraspan sequence to be reproduced twice in a row without making mistakes, was used. In the acute phase D.B. was unable to meet requirements, whereas in the remaining three assessments he always scored normal.

## (c) Visuo-Spatial Memory

D.B. had no problems in reproducing the *Rey–Osterrieth Complex Figure* after a delay. In the *Picture Display Test* D.B. behaved as follows: For 5 min we displayed, on a table in front of him, 30 pictures taken from children playing bingo; after the pictures were removed, he was asked to recall as many pictures as possible. He only recalled 15 of 30. After 15 min the same 30 pictures were presented mingled with another 30 new pictures: D.B.'s task was to sort out the first 30 pictures he had been shown. Although he had performed very poorly in free recall, he turned out to be perfectly able to recognize the pictures and sort the old from the new ones (29 of 30). Similar results have been observed also in the *Rey Memory Test*, which is quite reliable for the purpose of discriminating between pretenders and patients with genuine memory disorders (Rey, 1964). In this test D.B. clearly showed a dissociation between visual memory, which was still well preserved, and verbal memory, which turned out to be rather impaired.

# (d) Verbal Memory

*Recall of stories.* The examiner read a short story and required the patient to recall it immediately (IR, immediate recall). The story was then repeated and, after a 10-min break, an unexpected delayed recall was required (DR, delayed recall). Correctly recalled story items in IR and DR were counted separately. D.B.'s performance in the acute phase was very poor, whereas he showed a slight improvement 5 months postonset and improved up to a normal level 9 months postonset. In all assessments, however, IR was worse than DR.

Serial position curve. This test consists in the immediate free recall of 10 lists with 12 words each read out loud by the examiner at a pace of 1 word every 2 sec. In normals, the chance to recall a given word depends on its position within the list: words in the initial and final position have far better chances to be recalled than the others (primacy and recency effect, PE, and RE, respectively). Thus, RE and PE scores are considered to be a reliable index of recall abilities from short-term and long-term memory, respectively (Glanzer & Cunitz, 1966). D.B. has always shown a high RE, whereas PE was below normal standards in the first three evaluation sessions. Only during the fourth evaluation session (24 months postonset) did his PE reach a normal level, but his RE had worsened, though it was still within normal values.

Selective reminding test. The procedure suggested by Buschke and Fuld (1974) was slightly revised in this test. The patient did not meet requirements in 18 trials and in all trials recall from long-term memory was very poor.

*Word list.* For 5 min the patient was allowed to read through a list of 30 words which he then was asked to recall. After a 15-min break, the patient was asked to detect these 30 words introduced at random within a longer list of 60 words. In the third evaluation session D.B.'s recall was below standard, whereas it improved up to a normal level in the fourth session. Word recognition was below normal standards in both sessions.

# **Remote Memory**

A slightly revised version of the *MLT'88* test according to Andreani, Amoretti, and Baldi (1988) was used for the purpose of investigating D.B.'s memory for events having occurred from his birth year to 1988. Two versions of this test were administered: one with verbal stimuli and the other with pictures. Each version included 22 questions, 2 for each lustrum. Each completely correct answer scored 2 points; each partially correct answer scored 1 point. Five other subjects matching in age and education level acted as controls. In the acute phase, D.B. scored significantly lower than controls in both versions; then his retrograde memory deficit gradually improved to reach nearly normal level in the following evaluation sessions.

# Famous Personalities of the Present

Further data on D.B's memory for events of the recent past have been obtained by means of this test, which includes questions on 10 famous people from the past 20 years. In this test the patient also had severe memory problems in the acute phase, but he gradually returned to normal in the chronic phase.

#### Semantic Knowledge

In the *WAIS* subtests referring to information, analogies, sentences, and vocabulary, D.B. showed severe deficits in the acute phase, which had substantially improved as early as in the second evaluation session. The patient was also administered three other questionnaires with 10 items each concerning animals' habits, the use of objects, and sentence completion. In the acute phase, D.B made a single mistake when naming the elephant as the king of the jungle, instead of the lion. In subsequent sessions he made no mistakes at all.

A test of *Famous Personalities of the Past* with 10 questions on famous people from Italian history was also administered. D.B. performed very poorly in the acute phase and nearly reached normal standards in the chronic phase. As in the *MLT*'88 test, he made some semantic paraphasias by confusing Leonardo da Vinci with Michelangelo and Amerigo Vespucci with Christopher Columbus.

## DISCUSSION

Immediately postonset, D.B. presented with disorders of awareness that were not accompanied by impairments of ocular motility, a rather frequent symptom observed in diencephalic lesions (Castaigne, Lhermitte, Buge, Escourolle, Hauw, & Lyon-Caen, 1981; Antonini, Rasura, Paolini, Mercieri, Pantano, Gragnani, & Argentino, 1994). This shows that the lesion only affected the nuclei of the median thalamic line (Guberman & Stuss, 1983; Gentilini, De Renzi, & Crisi, 1987), thus sparing the thalamo-encephalic structures with oculomotor innervations. As soon as the patient's disorders of awareness improved, he showed a severe amnesia associated with a state of confusion, disorientation as to time and space, and mild language disorders. These additional cognitive impairments are a consistent feature of thalamic amnesia. In some cases, however, they are so severe that memory loss represents only one symptom of thalamic dementia (Guberman & Stuss, 1983; Graff-Radford et al., 1984; Muller, Destée, Steinling, Pruvo, & Warot, 1989). Two main criteria may be used to disentangle amnesia from dementia: (1) Amnesic patients present permanent memory deficits even after the recovery of other cognitive disorders. (2) Patients with pure amnesic syndrome usually perform within normal range on a digit span, while a bad performance on this task is frequently reported in patients with generalized mental deterioration (Parkin & Leng, 1993). D.B. surely fulfilled these two criteria. Recovery of generalized cognitive disorders occurred relatively early, as early as in the second evaluation (5 months postonset), and the patient's IQ ranked normal. However, disorders of long-term memory still persisted, while his performance on immediate memory tasks was always within normal range. Finally, D.B.'s picture is also consistent with the typical features of thalamic amnesia: a total recovery of anterograde deficits very seldom occurs, but the persistence of a psychometric impairment does not prevent patients from returning to their previous activities (Signoret & Goldenberg, 1986). D.B. can therefore be viewed as a case of pure thalamic amnesia.

# Thalamic Amnesia Provoked by Venous Infarction

To our knowledge, this is the first description of a case of pure thalamic amnesia following a venous infarction. Angiographic investigations of our patient revealed that the venous infarction had been provoked by an arteriovenous fistula. This is a rare finding, since arteriovenous malformations generally produce arterial hemorrhages. On the other hand, venous infarctions, which are mainly due to coagulation disorders or complications of heart diseases, are less frequent than arterial infarctions. The remarkable recovery of cognitive and mnestic functions in D.B. suggests that venous infarctions may have a better prognosis than arterial infarctions.

# Additional Cognitive Impairments

In the chronic phase, the patient showed, in addition to mnestic disorders, two other major cognitive deficits affecting language and arithmetic calculation, respectively. Language disorders have been relatively frequently observed in thalamic amnesia (Crosson, 1992). Speech abnormalities have been described in connection with lesions of the left thalamus affecting both the paramedian territory (Meissner, Sapir, Kokmen, & Stein, 1987; Stuss, Guberman, Nelson, & La Rochelle, 1988; Ghidoni, Pattacini, Galimberti, & Aguzzoli, 1989) and the tuberothalamic territory (Bogousslavsky, Regli, & Uske, 1988), and also the following thalamic nuclei: the antero-lateral thalamus (Goldenberg, Wimmer, & Maly, 1983), the DMNT and the MMT (Mori, Yamadori, & Mitani, 1986), and the pulvinar (Castaigne et al., 1981; Meissner et al., 1987; Fensore, Lazzarino, Nappo, & Nicolai, 1988). D.B.'s language disorders were consistent with those described by Fensore et al. (1988) in "thalamic aphasia": fluent speech with phonemic and semantic paraphasias with unimpaired comprehension and repetition. D.B. turned out to be also mildly anomic, as also observed by Meissner et al. (1987) in their clinical data. As opposed to the relatively high frequency of language impairments, disorders of arithmetic calculation have been rarely observed in thalamic amnesia (McEntee, Biber, Perl, & Benson, 1976; Guberman & Stuss, 1983; Fensore et al., 1988). In the case of our patient, a thorough analysis of these symptoms revealed that both enumeration abilities and awareness of arithmetic calculation rules were undamaged. Thus, D.B. did not show a genuine acalculia, but failed in all the trials requiring the involvement of verbal memory beyond his actual possibilities.

# Functional Thalamic Asymmetry

In the initial phase, the ischemic lesion affected both the left and the right thalamus; after a few months, however, it was confined to the left thalamus

only. D.B.'s mnestic impairments selectively regarded verbal material. During the acute phase, the patient was unable to learn a spatial supraspan sequence, and this was probably due to an initial deterioration of frontal functions. This transient deficit of spatial memory cleared up, whereas disorders of verbal memory persisted chronically. Although some authors deny the existence of a functional specialization of the two thalami (von Cramon et al., 1985; Rousseaux, Cabaret, Lesoin, Dubois, & Petiti, 1986), data obtained from D.B. actually corroborate the hypothesis of a functional asymmetry of the left and right thalamus for verbal material (Squire & Moore, 1979; Michel, Laurent, Foyater, Blanc, & Portafaix, 1982; Ojemann, 1982; Goldenberg et al., 1983; Graff-Radford et al., 1984; Mori et al., 1986; Parkin et al., 1994) and nonverbal material, respectively (Guberman & Stuss, 1983; Speedie & Heilman, 1983; Graff-Radford et al., 1984; Crosson, 1992).

## Retrograde Deficits in Thalamic Amnesia

It is still unclear whether retrograde amnesia is a constant symptom of thalamic amnesia (Butters & Stuss, 1989), but as far as retrograde thalamic amnesia is concerned, this is always associated with a bilateral thalamic lesion (McEntee et al., 1976; Barbizet, Degos, Louarn, Nguyen, & Mas, 1981; Goldenberg et al., 1983; Signoret & Goldenberg, 1986; Rousseaux et al., 1986; Meissner et al., 1987; Muller et al., 1989; Graff-Radford et al., 1990; Antonini et al., 1994), the only exception being a patient described by von Cramon et al. (1985), and it tends to regress in the course of time (Winocur, Oxbury, Roberts, Agnetti, & Davis, 1984; Nichelli, Bahmanian-Behbahani, Gentilini, & Vecchi, 1988). Unilateral lesions, however, do not provoke retrograde amnesia (Squire & Moore, 1979; Speedie & Heilman, 1983; patient no. 4 in von Cramon et al., 1985; Mori et al., 1986; Fensore et al., 1988); only Graff-Radford and colleagues (1984) found persistent retrograde amnesic deficits with unilateral thalamic lesions, though it should be pointed out that all their patients presented multiple lesion accompanied by generalized cognitive impairments.

Our patient, D.B., manifested transient deficits of remote memory, but after 10 months postonset he had already recovered memory for events that occurred before the ischemic lesion. Our data thus support the hypothesis according to which preserved unilateral thalamic functions may compensate for the retrograde deficit caused by contralateral lesions (Parkin et al., 1994).

# Anterograde Deficits: How Many Types of Thalamic Amnesia?

Several cases of amnesia following focalized lesions in specific thalamic structures have been described in the literature (see Markowitsch, 1988, for a review), and in particular lesions to the nucleus dorso-medialis (Schott et al., 1980; Guberman & Stuss, 1983; Speedie & Heilman, 1982; Kawahara

et al., 1986), to the mammillo-thalamic tract (von Cramon et al., 1985; Mori et al., 1986; Gentilini et al., 1987), to the anterior nucleus (Graff-Radford et al., 1990; Parkin et al., 1994), to the intralaminar nuclei (Goldenberg et al., 1983), and to the lamina medullaris interna (von Cramon et al., 1985; Calabrese, Haupts, Markowitsch, & Gehlen, 1993). On the other hand, some authors argue that thalamic lesions alone are not sufficient to induce amnesia and that a combination of damage is required to produce severe memory impairments (Mishkin, 1982; Aggleton & Mishkin, 1983; Kritchevsky, Graff-Radford, & Damasio, 1987). Thalamic amnesia may thus manifest itself following lesions to various thalamic structures; however, it is generally considered a unitary syndrome, irrespective of the site of the lesion. Some authors believe that thalamic amnesia resembles diencephalic amnesia (McEntee et al., 1976; Winocur et al., 1984; Swanson & Schmidley, 1985; Parkin et al., 1994), others assimilate it to hippocampal amnesia (Schott et al., 1980; Goldenberg et al., 1983), and still others maintain that it should be viewed as an autonomous syndrome, differing from both diencephalic and hippocampal amnesia (Barbizet et al., 1981). A few authors suggested that specific focalized lesions may determine specific patterns of amnesia (Lhermitte & Signoret, 1972; Signoret & Goldenberg, 1986), but the limited number of cases with localized lesions to definite thalamic structures described in the literature so far does not allow a specification of the role of each structure in amnesic syndromes (Aggleton, 1986).

D.B. had a lesion to the left thalamus involving three nuclei: the DMNT, the anterior nucleus, and the pulvinar. Since the DMNT is interconnected with the frontal lobes, the amygdala, and the hippocampus, a lesion to this structure may explain the frontal deficits observed in several other patients (Lhermitte & Signoret, 1972; Speedie & Heilman, 1983). In our patient, frontal deficits were quite evident in the acute phase, whereas in the chronic phase the only evidence of frontal deficits was the poor performance in the *Verbal Fluency Test*. The anterior nucleus is apparently involved in the process of consolidation of memory traces (Mishkin, 1978) and in working memory, and this would explain D.B.'s deficits of recognition in the *Word List Test*, in the *Selective Reminding Test*, and in the recall of stories: in all these tests delayed recall was always better than immediate recall, thus suggesting possible impairments of the phonological loop within the working memory system (Baddeley, 1990).

Lesions to the anterior nucleus and the pulvinar may account for mild aphasic disorders (e.g., anomias, semantic paraphasias, syntactic mistakes) that have been observed in D.B.'s two languages (see also Crosson, 1992; Fabbro & Paradis, 1995). The neuropsychological symptoms of this patient thus depend on a lesion to specific thalamic nuclei. This observation, however, is in contrast with the hypothesis that thalamic amnesia is a unitary syndrome. In our opinion, neuropsychological deficits occurring after a thalamic lesion may differ from patient to patient and may also depend on the nature, the site, and the extention of the lesion (Rousseaux et al., 1986; Ghi-doni et al., 1989).

## REFERENCES

- Aggleton, J. P., & Mishkin, M. 1983. Memory impairments following restricted medial thalamic lesions in monkeys. *Experimental Brain Research*, 52, 199–209.
- Aggleton, J. P. 1986. Memory impairments caused by experimental thalamic lesions in monkeys. *Revue Neurologique*, **142**, 418–424.
- Andreani, O., Amoretti, G., & Baldi, P. 1988. MLT '88 Test di memoria di eventi storici. Firenze: Organizzazioni Speciali.
- Antonini, G., Rasura, M., Paolini, S., Mercieri, M., Pantano, P., Gragnani, F., & Argentino, C. 1994. Paramedian diencephalic syndrome. *The Italian Journal of Neurological Sciences*, **15**, 285–290.
- Baddeley, A. D. 1990. Human memory: Theory and practice. Howe/London: Erlbaum.
- Barbizet, J., Degos, J. D., Louarn, F., Nguyen, J. P., & Mas, J. L. 1981. Amnésie par lésion ischémique bi-thalamique. *Revue Neurologique*, 137, 415–424.
- Bogousslavsky, J., Regli, F., & Uske, A. 1988. Thalamic infarcts: Clinical syndromes, etiology, and prognosis. *Neurology*, 38, 837–848.
- Buschke, H. & Fuld, P. A. 1974. Evaluating storage retention and retrieval in disordered memory and learning. *Neurology*, **11**, 1019–1025.
- Butters, N., & Stuss, D. T. 1989. Diencephalic amnesia. In F. Boller & J. Grafman (Eds.), *Handbook of neuropsychology*. Amsterdam: Elsevier.
- Calabrese, P., Haupts, M., Markowitsch, H. J., & Gehlen, W. 1993. Case Report: The cognitive-mnestic performance profile of a patient with bilateral asymmetrical thalamic infarction. *International Journal of Neuroscience*, **71**, 101–106.
- Castaigne, P., Lhermitte, F., Buge, A., Escourolle, R., Hauw, J. J., & Lyon-Caen, O. 1981. Paramedian thalamic and midbrain infarcts: Clinical and neuropathological study. *Annals of Neurology*, **10**, 127–148.
- Choi, D., Sudarsky, L., Schachter, S., Biber, M., & Burke, P. 1983. Medial thalamic hemorrhage with amnesia. Archives of Neurology, 40, 611–613.
- von Cramon, D. Y., Hebel, N., & Schuri, U. 1985. A contribution to the anatomical basis of thalamic amnesia. *Brain*, **108**, 993–1008.
- Crosson, B. 1992. Subcortical functions in language and memory. New York: Guilford Press.
- De Renzi, E., Faglioni, P., & Villa, P. 1977. Topographical amnesia. Journal of Neurology, Neurosurgery and Psychiatry, 40, 498–505.
- Fabbro, F., & Paradis, M. 1995. Differential impairments in four multilingual patients with subcortical lesions. In M. Paradis (Ed.), *Aspects of bilingual aphasia*. London: Pergamon Press.
- Fabbro, F., Peru, A., & Skrap, M. 1995. *Bilingual aphasia following thalamic lesions*. Paper accepted for the International Symposium on Communication Disorders in Bilingual Populations, Haifa, Israel, August 1–4, 1995.
- Fensore, C. Lazzarino, L. G., Nappo, A., & Nicolai, A. 1988. Language and memory disturbances from mesencephalothalamic infarcts. *European Neurology*, 28, 51–56.
- Gentilini, M., De Renzi, E., & Crisi, G. 1987. Bilateral paramedian thalamic artery infarcts: Report of eight cases. *Journal of Neurology, Neurosurgery and Psychiatry*, **50**, 900–909.
- Ghidoni, E., Pattacini, F., Galimberti, D., & Aguzzoli, L. 1989. Lacunar thalamic infarcts and amnesia. *European Neurology*, **29**, 13–15 (Suppl. 2).
- Glanzer, M., & Cunitz, A. R. 1966. Two storage mechanisms in free recall. *Journal of Verbal Learning and Verbal Behavior.* **5**, 351–360.

292

- Goldenberg, G., Wimmer, A., & Maly, J. 1983. Amnesic syndrome with a unilateral thalamic lesion: A case report. *Journal of Neurology*, 229, 79–86.
- Graff-Radford, N. R., Eslinger, P. J., Damasio, A. R., & Yamada, T. 1984. Nonhemorrhagic infarction of the thalamus: Behavioral, anatomic and physiological correlates. *Neurology*, 34, 14–23.
- Graff-Radford, N. R., Tranel, D., Van Hoesen, G. W., & Brandt, J. P. 1990. Diencephalic amnesia. *Brain*, 113, 1–25.
- Guberman, A., & Stuss, D. T. 1983. The syndrome of bilateral paramedian thalamic infarction. *Neurology*, 33, 540–546.
- Kawahara, N., Sato, K., Muraki, M., Tanaka, K., Kaneko, M., & Uemura, K. 1986. CT classification of small thalamic hemorrhages and their clinical implications. *Neurology*, 36, 165–172.
- Kritchevsky, M., Graff-Radford, N. R., & Damasio, A. 1987. Normal memory after damage to medial thalamus. Archives of Neurology, 44, 959–962.
- Lhermitte, F., & Signoret, J. L. 1972. Analyse neuropsichologique et différenciation des syndromes amnésiques. *Revue Neurologique*, **126**, 161–178.
- Markowitsch, H. J. 1988. Diencephalic amnesia: A reorientation towards tracts? Brain Research Review, 13, 351–370.
- McEntee, W. J., Biber, M. P., Perl, D. P., & Benson, F. D. 1976. Diencephalic amnesia: A reappraisal. *Journal of Neurology, Neurosurgery and Psychiatry*, **39**, 436–441.
- Meissner, I., Sapir, S., Kokmen, E., & Stein, S. D. 1987. The paramedian diencephalic syndrome: A dynamic phenomenon. *Stroke*, 18, 380–385.
- Miceli, G., & Capasso, R. 1987. Batteria di test per la valutazione dei disturbi del sistema dei numeri e del calcolo. Milan: Masson.
- Michel, D., Laurent, B., Foyater, N., Blanc, A., & Portafaix, M. 1982. Infarctus thalamique paramédian gauche: Étude de la mémoire et du langage. *Revue Neurologique*, **138**, 533– 550.
- Mishkin, M. 1978. Memory in monkeys severely impaired by combined but not by separate removals of amygdala and hippocampus. *Nature*, **273**, 297–298.
- Mishkin, M. 1982. A memory system in the monkey. *Philosophical Transactions Royal Society B*, **298**, 85–95.
- Mori, E., Yamadori, A., & Mitani, Y. 1986. Left thalamic infarction and disturbance of verbal memory: A clinicoanatomical study with a new method of computed tomographic stereotaxic localization. *Annals of Neurology*, **20**, 671–676.
- Muller, J. P., Destée, A., Steinling, M., Pruvo, J. P., & Warot, P. 1989. Infarctus bithalamique paramédian: 1 cas avec IRM et: tomographie d'émission gamma. *Revue Neurologique*, 145, 732–734.
- Nichelli, P., Bahmanian-Behbahani, G., Gentilini, M., & Vecchi, A. 1988. Preserved memory abilities in thalamic amnesia. *Brain*, **111**, 1337–1353.
- Ojemann, G. A. 1982. Interrelationships in the localization of language, memory, and motor mechanisms in human cortex and thalamus. In R. A. Thompson & J. R. Green (Eds.), *New perspectives in cerebral localization.* New York: Raven Press.
- Osterrieth, P. A. 1944. Le test de copie d' une figure complèxe. Archives de Psychologie, **30**, 206–356.
- Paradis, M. 1987. The assessment of bilingual aphasia. Hillsdale, NY: Erlbaum.
- Parkin, A. J., Rees, J. E., Hunkin, N. M., & Rose, P. E. 1994. Impairment of memory following discrete thalamic infarction. *Neuropsychologia*, 32, 39–51.
- Parkin, A. J. & Leng, R. C. 1993. Neuropsychology of the Amnesic Syndrome. Hove/London: Erlbaum.
- Raven, J. C. 1938. Standard progressive matrices: Sets A, B, C, D and E. London: HK Lewis.
- Rey, A. 1964. L' examen clinique en psychologie. Paris: Presses Universitaires de France.
- Rousseaux, M. Cabaret, M., Lesoin, F., Dubois, F., & Petiti, H. 1986. L' amnésie des infarctus thalamiques. *Encephale*, XII, 19–26.

- Schott, B., Mauguière, F., Laurent, B., Serclerat, O., & Fischer C. 1980. L' Amnésie Thalamique. *Revue Neurologique*, 136, 117–130.
- Signoret, J. L., & Goldenberg, G. 1986. Trouble de mémoire lors des lésions du thalamus chez l'homme. *Revue Neurologique*, **142**, 45-448.
- Speedie, L. J., & Heilman, K. M. 1982. Amnesic disturbances following infarction of the left dorsomedial nucleus of the thalamus. *Neuropsychologia*, **20**, 597–604.
- Speedie, L. J., & Heilman, K. M. 1983. Anterograde memory deficits for visuospatial material after infarction of the right thalamus. *Archives of Neurology*, **40**, 183–186.
- Spinnler, H., & Tognoni, G. (Eds.), 1987. Standardizzazione e Taratura Italiana di Test Neuropsicologici. *The Italian Journal of Neurological Sciences* (Suppl. 6).
- Squire, L. R., & Moore, R. Y. 1979. Dorsal thalamic lesions in a noted case of human memory dysfunction. *Annals of Neurology*, **6**, 603–606.
- Stuss, D. T., Guberman, A., Nelson, R., & La Rochelle, S. 1988. The neuropsychology of paramedian thalamic infarction. *Brain and Cognition*, **7**, 1–31.
- Swanson, R. A., & Schmidley, J. W. 1985. Amnesic syndrome and vertical gaze palsy: Early detection of bilateral thalamic infarction by CT and NMR. *Stroke*, 16, 823–827.
- Wechsler, D. A. 1981. WAIS-Revised. New York: Psychological Corp.
- Winocur, G., Oxbury, S., Roberts, R., Agnetti, V., & Davis, C. 1984. Amnesia in a patient with bilateral lesions to the thalamus. *Neuropsychologia*, 22, 123–143.