

## The Neuropsychology of Paramedian Thalamic Infarction

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A longitudinal study of three patients with CT-scan documented paramedian thalamic infarctions (bilateral, primarily right, unilateral left) is reported and the neuropsychology of human paramedian thalamic infarction is reviewed. The neuropsychological deficits following these selected lesions, the nature of the clinical memory disorder, and the neuroanatomy of memory are discussed. The significance of cortical/subcortical relationship in explaining observed behavioral changes is emphasized. Brain damage with maximum involvement in the dorsomedial nuclei and mamillothalamic tracts appears to cause primarily a memory disorder and frontal-limbic behavioral changes, the severity and profile of deficits depending on lesion extent and location. Both anterograde and remote memory loss may be present. Asymmetry in memory at the level of the thalamus was observed, following the left-verbal, right-nonverbal dichotomy. © 1988 Academic Press, Inc.

Two distinct forms of human amnesia have been distinguished in the last 15 years (Huppert & Piercy, 1978, 1979; Lhermitte & Signoret, 1972; Mattis, Kovner, & Goldmeier, 1978; Signoret, 1983; Squire, 1980, 1981, 1982; Squire & Cohen, 1982; Squire & Zola-Morgan, 1983): diencephalic and bitemporal amnesia. Diencephalic amnesia is typified by Korsakoff patients (Victor, Adams, & Collins, 1971) and by cases of trauma or infarction such as NA (Squire & Moore, 1979; Teuber, Milner, & Vaughan, 1968). Bitemporal amnesias are exemplified by surgical cases such as H.M. (Milner, 1966, 1972; Scoville & Milner, 1957), and by patients who

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have received electroconvulsive therapy (Squire, 1981, 1982; Squire & Zola-Morgan, 1983).

Anatomical investigations have probed the role of damage to various brain areas in the two clinical types of amnesia. In bitemporal amnesia, lesions in both the amygdala and hippocampus appear necessary to produce an obvious memory deficit (Andersen, 1978; Andy, Jurko, & Hughes, 1975; Duyckaerts et al., 1985; Halgren, 1981; Milner, 1966; Mishkin, 1978; Sarter & Markowitsch, 1985; Saunders, Murray, & Mishkin, 1984; Scoville & Milner, 1957; Squire & Zola-Morgan, 1983; Woods, Schoene, & Kneisley, 1982; Zola-Morgan, Squire, & Mishkin, 1982). Controversy surrounds the role of various diencephalic regions in memory, in particular the dorsomedial (DM) nucleus and the mamillary bodies (MB). Many publications emphasize the role of the MB or mamillothalamic tract (bundle of *vicq d'Azyr* which runs from the MB to the anterior thalamic nuclei), particularly in patients with Korsakoff's disease (e.g., Brierly, 1977; Brion & Mikol, 1978; Brion, Mikol, & Plas, 1983; Castaigne et al., 1966, 1981; Delay & Brion, 1954; Delay, Brion, & Elissalde, 1958, a,b; Gruner, 1956; Grunthal, 1939; Jarho, 1973; Mair, Warrington, & Weiskrantz, 1979; Pierrot-Deseilligny, Chain, & Lhermitte, 1982; Swanson & Schmidley, 1985). The anterior thalamic nuclei themselves have also been implicated in memory functioning (Barbizet, Degos, Louarn, Nguyen, & Mas, 1981; Hassler, 1962; Mark, Barry, McLardy, & Ervin, 1970).

The DM nucleus, however, has been suggested most frequently as the diencephalic area most related to memory (Ignelzi & Squire, 1976; McEntee, Biber, Perl, & Benson, 1976; Mills & Swanson, 1978; Schott, Maugiere, Laurent, Serclerat, & Fischer, 1980; Smyth & Stern, 1938; Squire, 1980; Squire & Moore, 1979; Victor et al., 1971; Williams & Pennybacker, 1954). It has been proposed that lesions of the MB and DM nucleus may individually cause some amnesia but that damage to both are necessary for more profound deficits, the severity correlating with the degree of involvement (Aggleton & Mishkin, 1983; Riggs & Boles, 1944; Schott et al., 1980; Speedie & Heilman, 1982, 1983; Squire, 1980, 1982).

In addition to further characterizing diencephalic memory problems, other issues concerning neuropsychological changes after pathology in this region are relevant. If the lesion is unilateral, will asymmetrical memory deficits be confirmed (verbal-nonverbal)? Are other functions disturbed: language, visual-spatial abilities, attention, etc.? Do lesions in the diencephalic area result in a "dementia" and, if so, can this more general disorder be characterized according to common typologies such as cortical or subcortical dementia? Because of the intimate interconnections of the dorsomedial nucleus with the frontal lobes, are there parallels with frontal lobe disturbance?

There remains a need for assessment of patients with well-defined focal diencephalic lesions to elucidate further the specific role of lesions

in this region in producing specific behavioral abnormalities (Aggleton & Mishkin, 1983; Markowitsch, 1982, 1984; Zola-Morgan & Squire, 1985). Computed tomographic scanning (CT) has allowed more precise localization of focal brain pathology and has led to progress in the delineation of brain-behavior relations. We have studied three patients with isolated vascular infarcts in the paramedian thalamic areas: one with bilateral lesions, one with maximal right-sided involvement and one with only left-sided damage. These cases provided an opportunity to investigate the effects of specific subcortical lesions and to assess possible clinical differences arising from lesions of left versus right thalamic areas. We also studied specific characteristics of the memory process, including memory encoding and retrieval, and remote memory. Finally, repeated assessments yielded information on the prognosis of acute paramedian thalamic infarction.

## METHOD

### *Subjects*

Table 1 outlines the demographic characteristics of the three patients. Details of cases 1 and 2 were originally presented previously (Guberman & Stuss, 1983) and are only summarized here. In the chronic state, all three patients were fully alert and cooperative for neuropsychological assessment.

*Case 1.* This 54-year-old man (R.C.) was in excellent health except for long-standing mild untreated hypertension until one morning he was found unarousable in bed. On admission, he was deeply stuporous but arousable with vigorous stimulation. The following day, the level of consciousness fluctuated. Vertical (especially saccadic) gaze was impaired. For 2 to 3 weeks he was disoriented, hypokinetic, apathetic, and hypersomnolent. CT (see Fig. 1) at that time showed small ill-defined hypodense areas in both medial thalami. He was eventually discharged home, where he remains apathetic and unconcerned, under the care of his wife.

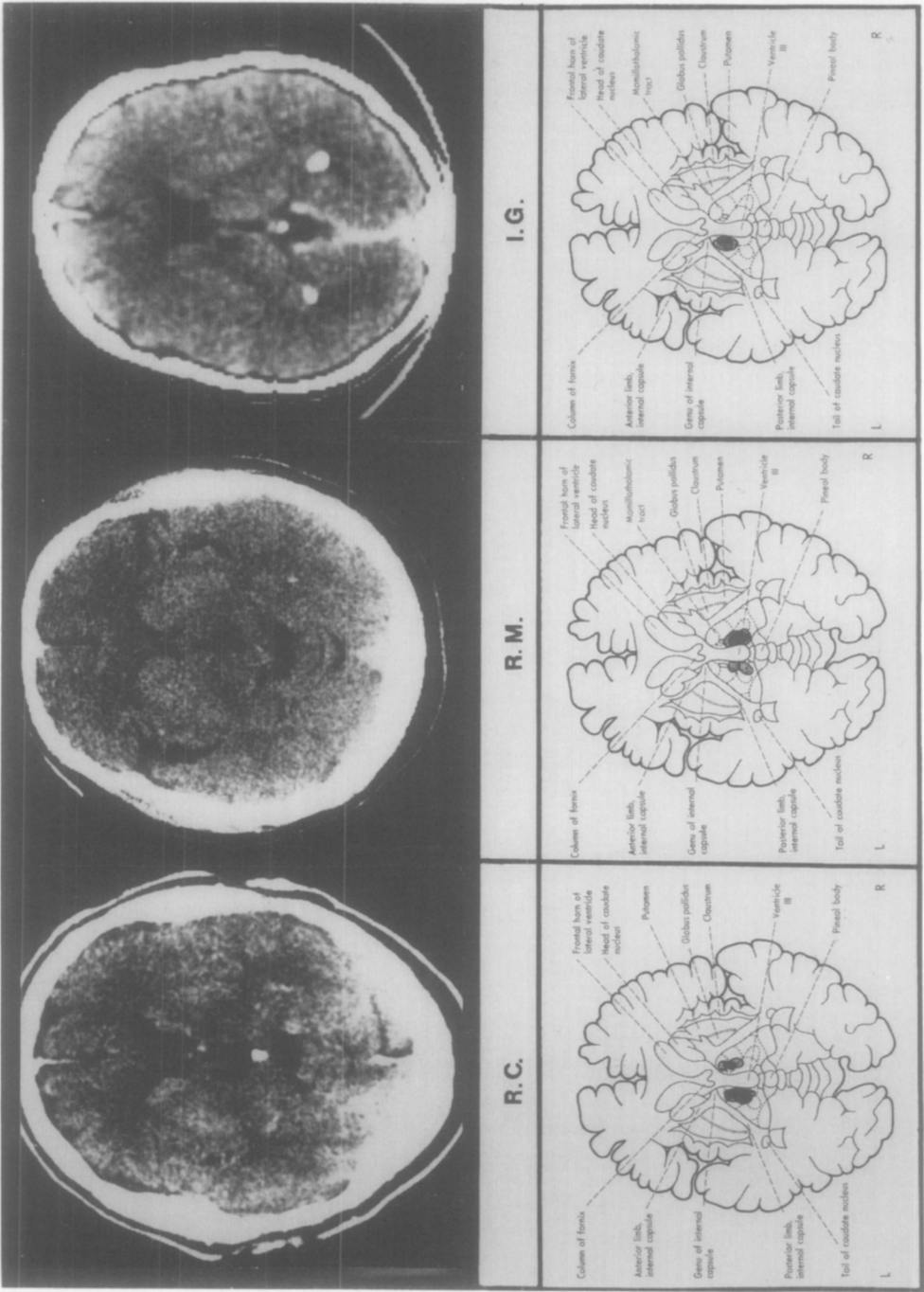
*Case 2.* This 51-yr-old gentleman (R.M.) with mild untreated hypertension presented with coma, resolving into hypersomnolence, disorientation, inappropriate behavior, and both anterograde and retrograde amnesia. Within 2 days, he was alert and oriented, but slow to respond, with an obvious memory deficit. He eventually returned to work. A CT scan (see Fig. 1) done 19 months after admission revealed a discrete low density lesion in the right paramedian thalamus. A coronal view suggested a barely discernible hypodense area in the left thalamus.

*Case 3.* This 70-yr-old right-handed female (I.G.) was in good health until December 5, 1981. She had had one episode of vertigo a year previously for which she saw a neurologist. A CT scan at that time was normal. On the day of the present admission, she felt unwell. She was unable to follow a recipe to bake a cake. Later her husband noted that she had trouble finding words and was using inappropriate words. On admission to hospital, her blood pressure was 170/90, but later 130/60. Heart rate and rhythm were normal but she had an early systolic click. No bruits were heard in the neck. She appeared confused, said she was in a hospital in another city and gave the year initially as 1941 but later corrected herself. The neurological examination was otherwise normal.

A CT scan completed 48 hr after the onset of symptoms revealed a low density lesion in the left thalamus adjacent to the genu of the internal capsule. Four days later, the lesion was not seen on a contrast enhanced study but reappeared on a subsequent scan and was still noted 1 year later (see Fig. 1). The EEG was normal and cerebral angiography did

TABLE 1  
DEMOGRAPHIC DATA OF THREE PATIENTS WITH PARAMEDIAN THALAMIC INFARCTIONS

Patient	Sex	Age	Handedness	Education	Present employment	Previous employment	Assessment dates
1. R.C.	M	54	R	1 year university	Disability	Business analyst; civil servant	0-3 weeks; 6, 15, 24 months
2. R.M.	M	51	R	5	Truck driver	Truck driver	0-2 weeks; 10, 27 months
3. I.G.	F	70	R	B.A.	Retired	School principal	0-1 week; 7, 9 months



not show any occlusive disease. Both posterior cerebral arteries were noted to arise mainly from the posterior communicating arteries. Echocardiogram demonstrated no mitral valve prolapse. On discharge from hospital, she returned to her regular schedule.

### *Neurological Investigations*

The neurological symptoms and investigations are summarized in Table 2. Part A (top) outlines the presenting and early states; Part B (bottom) summarizes the chronic stage.

The CT scans (top) and the schematic localizations (bottom) are presented in Fig. 1. For each patient, the level best demonstrating the lesion was selected. For purposes of localization, a horizontal brain section from a CT scan atlas (DeArmond, Fusco, & Dewey, 1976, p. 28) was selected as the diagram most representative of all three scans. For patient 2 (R.M.), an additional coronal cut was used to approximate the left thalamic lesion. For each CT scan, two neurologists (A.G., R.N.) independently outlined the lesion on a schematic drawing. These two independent approximations were then overlapped (darker area) to yield the composite schematic localization on the bottom of Fig. 1, and indicate close agreement in localization. In Fig. 2, the level of the horizontal view and the thalamic area, in enlarged format without lesion demarcation, are presented to facilitate localization in Fig. 1.

The schematic diagram based on analysis of the CT scans suggests the following localized maximum thalamic involvement. Involvement of other proximal areas, such as the internal medullary lamina and thalamic inflow and outflow pathways cannot be definitely ruled out due to the limitations of volumetric analyses of CT scan, especially for subcortical structures.

1. Dorsomedial nucleus: This nucleus is damaged in all three patients. In patient R.C., there is maximum left DM involvement, and apparently only minimal right DM nucleus damage. In patient R.M., the dorsomedial pathology appears more bilateral, with maximum involvement on the right. Patient I.G. has unilateral left damage, the DM involvement apparent only at the rostral tip of the nucleus.

2. Mamillothalamic tracts: In all three patients, the mamillothalamic tract, coursing between three thalamic regions (dorsomedial, ventral anterior, and ventral lateral) en route from the mamillary bodies to the thalamus appears to be involved, either bilaterally (R.C.), or unilaterally (R.M.—on the right; I.G.—on the left).

3. Ventral anterior thalamic nucleus: The ventral anterior thalamic nucleus appears to be involved bilaterally in patient R.C. and unilaterally on the left for patient I.G. Patient R.M. has no evidence of ventral anterior involvement.

4. Ventral lateral thalamic nucleus: The involvement of this nucleus is variable. In all patients, there is some involvement. For example, in patient R.C. there is minimal bilateral involvement. In patient R.M. there is minimal suggestion of involvement of this nucleus. In patient I.G., there is evidence of some unilateral left ventral lateral involvement.

5. Centromedian, ventral posterolateral, and pulvinar have little if any evidence of pathology. There is a suggestion of minimal involvement of the centromedian nucleus in patients R.C. and R.M.

Involvement of each of these brain areas, in each of the three patients, is schematically represented in Table 3, using a rating of 0 to 5 to provide an approximation of zero to maximum involvement.

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FIG. 1. CT scan and schematic representation of localization of lesions in the three patients with paramedian thalamic infarcts. The top half illustrates the actual CT scan results; the lower half depicts the location of the lesion according to a CT scan atlas. The procedure is described in the text. The left thalamic lesion in patient R.M. is a barely discernible hypodense area seen only on a coronal cut, suggesting that there is primarily right paramedian thalamic involvement in this patient.

TABLE 2  
NEUROLOGICAL SYMPTOMS AND INVESTIGATIONS

	Arousal	General physical, neurological	Cranial nerves	Mental status	EEG
1. R.C.	Coma→Stupor→ Fluctuating levels of consciousness, hypersomnolence	Bilateral asterixis; normal	A. Presenting and early findings Absence of voluntary vertical gaze & oculocephalic movements	Not testable→ drifting attention, confabulation	Diffuse slowing, with minimal $\alpha$ in arousal
2. R.M.	Coma→ Hypersomnolence	Early incontinence; normal	Eye movement limitation	Slow mentation, confusion	Diffuse slowing
3. I.G.	Confusion	Normal	Normal	Confusion	Normal
1. R.C.	Alert, apathy, abulia	Normal	B. Chronic state Minimal vertical saccadic gaze deficit	Severe memory, initiation deficit	
2. R.M.	Normal	Normal	Normal	Limited memory impairment	
3. I.G.	Normal	Normal	Normal	Limited memory disturbance	

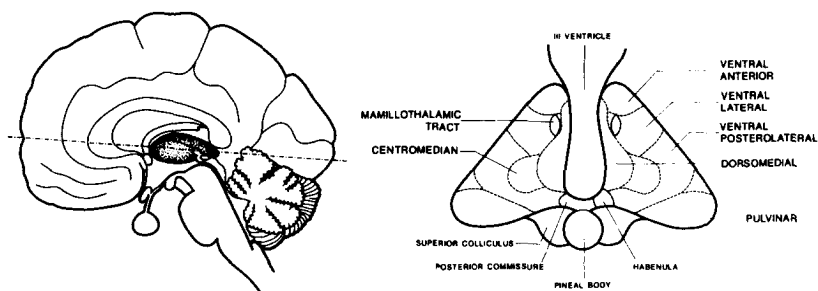


FIG. 2. On the left, the level of the horizontal cut is shown (DeArmond et al., 1976). On the right, a schematic blowup of the paramedian thalamic area is illustrated, for more precise lesion localization of the areas depicted in Fig. 1. See Table 3.

### *Neuropsychological Investigations*

Commonly used clinical tests are summarized in Table 4. Experimenter-devised procedures are described below. The latter were all administered after the patient had stabilized (after 6 months).

### *Learning of Spatial Mazes*

Following Milner (1964), two simplified manual forms were devised (see Fig. 3). For each, holes were drilled in a wooden board at equal intervals for either a  $10 \times 10$  or  $4 \times 4$  grid. The subject was told that there was one correct path leading from the bottom left to the top right. The subject had to learn the path by trial and error by placing pegs in the holes. Feedback was given for each response. If an error was made, all pegs were removed, and the patient restarted the test. Because of the patients' difficulty (particularly R.C.) in successfully completing the  $10 \times 10$  grid, the  $4 \times 4$  grid was used for comparison across all three patients. Thirteen trials were administered to each patient; the score was the number of correct pegs placed in each trial. The patients were compared to three normal control subjects of the same mean age and educational level.

### *Memory Encoding and Retrieval*

Two different conditions were used. In the free encoding condition, a list of 12 words, 3 words from four different semantic categories, were presented at a rate of 1 every 2 sec

TABLE 3  
LOCALIZATION OF LESION IN THE THREE SUBJECTS ACCORDING TO BRAIN STRUCTURE

Patient:	R.C.		R.M.		I.G.	
	L	R	L	R	L	R
Mammillothalamic tract	5	3	0	2	5	0
Dorsomedial	5	1	2	5	1	0
Ventral anterior	1	2	0	0	2	0
Ventral lateral	2	1	1	1	1	0
Ventral posterolateral	0	0	0	0	0	0
Pulvinar	0	0	0	0	0	0
Centromedian	1	0	2	2	0	0

Note. 0 = normal; 5 = maximum involvement.



TABLE 4  
NEUROPSYCHOLOGICAL PROCEDURES

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- A. Attention, mental control
    - 1. Arousal level (Plum & Posner, 1980).
    - 2. Digit span.
    - 3. Wechsler Memory Scale (WMS) mental control (Wechsler, 1945).
    - 4. Trail Making Test (Army Individual Test Battery, 1944).
  - B. Speech and language
    - 1. Boston Diagnostic Aphasia Examination (BDAE) fluency and content (Goodglass & Kaplan, 1972).
    - 2. Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1983).
    - 3. F-A-S verbal fluency (Spreen & Benton, 1969).
    - 4. Western Aphasia Battery (Kertesz, 1982).
  - C. Cognitive tests
    - 1. Wechsler Adult Intelligence Scale (WAIS) (Wechsler, 1955).
  - D. Planning and executive control functions
    - 1. Wisconsin Card Sorting Test (WCST) (Grant & Berg, 1948).
    - 2. Porteus Maze (Porteus, 1933/1965).
    - 3. Color-Form Sorting (Goldstein & Scheerer, 1941).
  - E. Memory
    - 1. Wechsler Memory Scale (WMS) (Wechsler, 1945).
    - 2. Benton Visual Retention Test (Benton, 1974).
    - 3. Three word memory.
    - 4. Consonant Trigrams (Brown, 1958; Peterson & Peterson, 1959).
    - 5. Retrograde Amnesia Test (Ottawa General Hospital French Version, developed by SLR).
  - F. Visual-spatial
    - 1. WAIS Block Design, Object Assembly
    - 2. WMS Copy
    - 3. BDAE Parietal Lobe Drawings
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with immediate free recall required. The patient was then told what the four categories were, and recall was again requested (category recall). In condition two, the two types of recall were identical. Before the presentation of the list of 12 different words from four other semantic categories, however, the patient was informed what the four categories were (category encoding). Comparisons were possible between type of recall within and across the two acquisition conditions. On this test, the three patients were compared to 12 normal subjects (age = 58.9 (6.9); education = 14.3 years (2.3)).

### *Retrograde Amnesia*

One patient (R.C.) was administered a series of measures of retrograde amnesia. All tests were devised, since no valid test existed for a French Canadian population. Twenty-two items constituted a measure of autobiographical remote memory covering work, school history, family history, and personal preferences. Knowledge of generic facts was assessed by the WAIS-R information subtest, and 10 additional questions (where does the sun rise? what is the color of snow?). Ten questions addressed memory of singular facts (who sculpted the Pieta and painted the Sistene chapel?; who invented the airplane?). Finally, 50 questions spanning five decades from 1930 to 1980 were presented, based on expected knowledge for an educated individual with a French-Canadian background. Spontaneous recall and multiple choice responses were requested. Three normal control subjects of the same approximate age (range 52-57), education (completed high school to 3 years university),

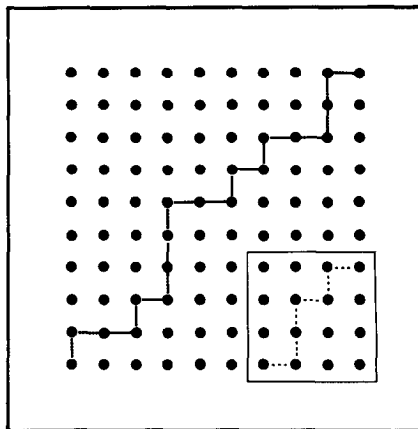


FIG. 3. The dotted path depicts the simplified route the patients had to learn in a trial and error fashion, moving from bottom left to top right. The inset illustrates the very reduced  $4 \times 4$  grid and route.

cultural background (all French-Canadian), and socioeconomic status (upper middle-class) were tested for comparison.

## RESULTS

### *General Neuropsychological Tests*

Data gathered are described below, and summarized in Table 5.

### *General Observations, Arousal, Attention*

Two (R.C., R.M.) of the three patients presented with coma, evolving into hypersomnolence and fluctuating levels of consciousness. Patient I.G. presented with confusion, evolving into an unconcerned state, with elements of denial. In the chronic (see Table 5 for definition) state, all three were alert, pleasant, and cooperative. R.M. had returned to work, with some lack of insight. I.G. was somewhat distractible, anxious, with a tendency to minimize any residual deficits. R.C. remained at home, apathetic, unaware, unconcerned, distractible, slow in response, with flat affect, good humor, occasionally aggressive outbursts, and little spontaneous initiation. His wife, however, noted improvement in daily activities 3 years postonset. The patient took limited walks by himself and became involved in at least minimal social interaction.

Attentional capabilities as measured by digit span (see Table 5), remained relatively stable after the acute period. In the early stage, other clinical tests used to measure attention capabilities revealed a tendency for errors and slowness in response. All patients, particularly R.C., continued to have extreme difficulty with the Trail Making Test, particularly in Part B, which requires alternating between numbers and letters (1-A-2-

TABLE 5  
SUMMARY OF NEUROPSYCHOLOGICAL RESULTS IN THE ACUTE AND CHRONIC STAGES FOR THE THREE PATIENTS

	Acute <sup>a</sup>			Chronic <sup>b</sup>		
	R.C.	R.M.	I.G.	R.C.	R.M.	I.G.
Attention						
Digit Span						
FWD	5	5	5	5	7	5
BWD	4	4	3	5	5	3
Mental Ctl						
20-1	37"(2)"		17"	10"	15"	12"
1-4-7	56"(1)		22"(1)	9"	24"	19"
Trail making						
A	300**		55	244"	85"	33"
B	300**		300**	300**	285"	163"
Language						
Speech	Low vol., no spontaneous output	Low vol.; normal	Normal	Low vol., increased output	Normal	Normal
Content	Confab., persev., redupl.	Confab., persev., redupl. paramnesia	Confab., persev., redupl. paramnesia	Mild confab.	Normal	Normal
Repetition						
Comprehension	8.8/10	10/10	10/10	10/10	10/10	10/10
BNT <sup>c</sup>	7.5/10		9.3/10	9.7/10	10/10	10/10
Word generation	5/18	20/40	38/84	10/18	29/40	68/84
General intelligence <sup>d</sup>	0		7	2	4	11
WAIS						
VIQ	83		89	103		111
Info	6		13	10		15
Comp	5		6	10	5	11
Arith	8		10	11	7	12
Simil	6		7	9	3	12
D. span	7		8	6	6	12
Voc	9		14	13		17

Visual-spatial				Verbal				Memory			
WAIS	PIQ	WISC	WMS	WISC	PIQ	WISC	WMS	WISC	PIQ	WISC	WMS
	0	0	55		71	102	81		71	102	101
Digit symbol.	0	0	3		3	7	2		3	7	5
Pict. comp.	0	0			4	9	3		4	9	8
Block des.	0	0	2		5	7	5		5	7	9
Pict. arr.	0	0			0	6	6		0	6	7
Obj. ass.	0	0			3	7	7		3	7	10
WMS copy (14)	5	8			9	12	12		9	12	12
Memory											13
WMS											
MQ	55						81				84
Info	3						2				6
Orien	4.1						5				4
Stories											
Immed.	1.5						6				7
Delayed	0						6				8
Paired assoc.											
Easy immed.	1/18	0/18					2/18				14/18
Delay	0/6	5/6					0/18				6/6
Hard immed.	0	0					0				3
Delay	0	0					0				0
Drawings											
Immed.	0	2					9				4
Delay	0	3					10				4
Benton VRT											4/10
3 word	0/3	1/3					0/3				3/3

**Note.** Absent data indicates test was not administered.

<sup>a</sup> Acute, maximum results in the period 0-3 weeks after infarct.

<sup>b</sup> Chronic, maximum results in the period 6–12 months after onset.

<sup>c</sup> Number in brackets reflects the number of errors. Score is the time required to completion.

<sup>d</sup> BNT, Boston Naming Test.

\* Scale scores—a score of 10 for these WAIS subtests would be average.

<sup>f</sup> For the WMS, delayed recall was approximately 30 min.

\* Maximum time allotted.

B-3-. . . ). Patient R.C. could never successfully complete this task. Despite continuing impairment, the overall results indicate an evolving improvement in the attentive abilities required by this task.

### *Speech and Language*

Two of the three (R.C., R.M.) had diminished voice volume in the acute stages, which remained permanent for R.C. Neither had had laryngeal intubation. None revealed dysarthria or stuttering. Perseveration was common.

Although all had language disturbance, it is uncertain whether they can be called "aphasic." Repetition in the acute period was impaired only with longer phrases, and might be attributed to attentional or memory problems. There was no repetition deficit in the chronic stage. Comprehension was impaired only for sequences or complex ideational questions. Naming was essentially perfect on the simpler Western Aphasia Battery subtest for all three patients. However, a naming deficit was identified by the Boston Naming Test. Patient R.M.'s performance was primarily characterized by perceptual errors and educational deficiencies ("I don't know"). In the chronic stage, a mild to moderate naming deficit remained, patient R.C. being more severely impaired. His performance significantly improved with phonetic cues. Except for R.M., errors were not easily classifiable. Some were perseveration, others nonaphasic misnaming, and others paraphasias. Word list generation (number of words generated in 1 min beginning with a specific letter) was impaired for R.C. and R.M. in the chronic stage. The patient with unilateral left pathology did not reveal persistent impairment on this test.

### *General Intelligence*

Although the cognitive assessment was not equivalent for all three patients, there was a consistent trend for impaired functioning soon after onset, with improvement over time. Patient R.M., with a Grade 5 education, appeared to have premorbid limited intellectual abilities. Patient I.G., the retired school principal, had a definite improvement from low average to high average functioning. While overall verbal I.Q. abilities increased from 83 to 103 for patient R.C., suggesting normal functioning in the chronic state, this likely does not reflect his premorbid abilities. This conclusion is based on the intertest scatter, superior score (13) on the test with the highest correlation with overall I.Q. (vocabulary), and knowledge of his previous occupation. In summary, however, verbal cognitive abilities, even with bilateral paramedian infarct, recovered to at least normal levels.

### *Planning and Executive Abilities*

In the early stage, all three revealed confabulation and reduplicative paramnesia of place. Because of overall differences in level of functioning, examination of planning and executive abilities was completed at different levels for each patient. Patient I.G., at both the acute and chronic stages, despite her high I.Q., had severely impaired performance on the Wisconsin Card Sorting Test (only two sorting categories), and the Porteus Maze (test age of 10/17). R.C. and R.M. were administered the simple color-form sorting task, requiring one shift of concept: R.M., despite his lower I.Q., completed the task successfully, while R.C. formed one concept, and continued to persevere this. He was unable to shift.

### *Visual-Spatial Functions*

Visual-spatial functions were assessed by the Performance subtests of the WAIS (particularly Block Design), related drawings from the Boston Diagnostic Aphasia Examination (BDAE) Parietal Lobe Battery (performed to command and copy), and the copy of the Wechsler Memory Scale (WMS) figures. Patient I.G., with the unilateral left lesion, was only minimally deficient on simple drawings in the acute stage. While her WAIS performance I.Q. was 102 at this time, results were mildly beneath expected levels. This was confirmed by her above-average results once recovered. The other two patients, with documented right thalamic infarcts, had more severe deficits, never recovering to expected levels. Impairment was most striking in complex tasks and in the spontaneous reproduction of houses, cubes, etc. Copy of simple designs (WMS) was the least affected, suggesting that elementary visual-perceptual functions were reasonably intact in all three patients.

### *Memory*

Functioning was impaired in all three patients, particularly in the acute stages. While recovery did occur, two facts were evident in the chronic stage. First, the patient with bilateral damage had persistent amnesia for both verbal and nonverbal material. Tactile memory was also severely impaired. Despite repeated assessments, he never remembered having seen the examiner before, or having undergone any tests. Even after 3 years, he was disoriented for time, indicating he could never learn the day, date, or year. Second, there appeared to be hemispheric asymmetry of memory functions in the two patients who had predominantly left or right unilateral pathology: R.M., with the predominant right thalamic lesion, was relatively impaired with nonverbal material; I.G., with the left thalamic lesion, had pronounced difficulty with verbal information (see Fig. 4).

The results of the  $4 \times 4$  maze learning test for the two separate testing sessions are illustrated in Fig. 5. Two observations are readily made.

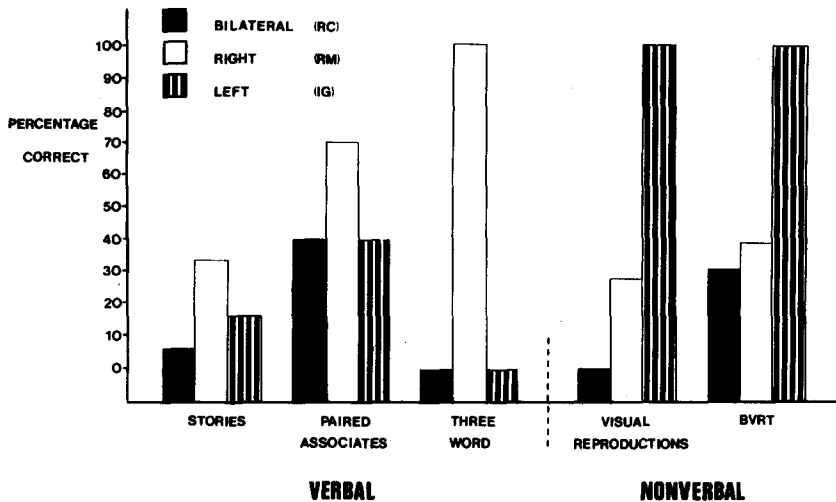


FIG. 4. Delayed recall on verbal and nonverbal measures of memory for all three patients with thalamic lesions (bilateral, primarily right, and unilateral left) during the chronic testing stage. Stories, paired associates, and visual reproductions are derived from the Wechsler Memory Scale. BVRT refers to the Benton Visual Retention Test, multiple choice format. For the three-word test, the patient is asked to recall three words after a 5-min delay.

First, the two patients with "unilateral" (unilateral left; primarily right) lesions both eventually learned the task on the first session, with greater success on the second testing session. Patient R.C., on the other hand, never learned the task. His performance deteriorated over time, his best results generally occurring on the earlier trials during both the first and second visits. Second, the learning (or "forgetting") curve for each patient frequently showed to a greater or lesser degree a "scaloping" effect, with good performance on one trial altering with poor performance on a subsequent trial.

On the memory encoding test, all three patients were inferior to the normal control subjects under all conditions (see Fig. 6). There was little evidence of consistent differences among the three patients in their ability to use category cues for either recall or encoding. All patients not only failed to benefit from category recall, but deteriorated in the number of words recalled. The two patients with more prominent left thalamic damage benefited at least slightly from category encoding, while the patient with primarily right thalamic infarction did not improve.

Retrograde amnesia tests were completed only for subject R.C., approximately 3 years postonset. Knowledge of generic facts was 100%. Knowledge of singular facts was at the 50th percentile for the WAIS information subtest (scale score of 10; equivalent to I.Q. of 100), and

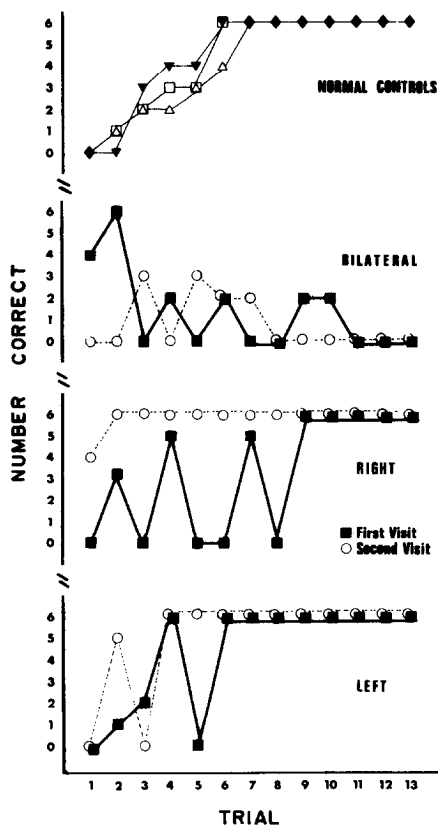


Fig. 5. First and second testing sessions results on the simplified maze learning test for all three patients are illustrated. A total of six correct moves are possible for each trial. Thirteen trials were given to each patient. The results of one testing of three normal control subjects are illustrated on top for comparison.

60% correct for our 10 questions. He was unable to indicate who painted the Sistine chapel (although he knew what it was), who invented the airplane, or what kind of artist Rembrandt was. Twenty-two questions of autobiographical remote memory (verified by his wife) were 55% correct. There therefore appears to be a personal remote memory loss. A temporal gradient cannot be claimed, although descriptively the deficit appears more severe in the years proximal to the injury, with at least a patchy loss extending for many years prior.

The results for the verbal retrograde amnesia test for both spontaneous recall and recognition are illustrated in Fig. 7. Spontaneously, R.C. could recall only such information as the Prime Minister of England (Thatcher), the birth place of a Prime Minister of Quebec in the 1950s, Jesse Owens, and the name of the Dionne quintuplets. As indicated by the figure, the



		MEMORY ENCODING			
		B	R	L	NC
FREE ENCODING	FREE RECALL	3	3	3	8
	CATEGORY RECALL	0	1	1	8
CATEGORY ENCODING	FREE RECALL	4	3	6	9
	CATEGORY RECALL	2	2	0	8

FIG. 6. Illustration of the results on the memory encoding tests. The results for the normal control subjects on this task are derived from Lafèche (1987).

recognition procedure improved performance to the level of spontaneous recall of the control subjects. However, there obviously remains a retrograde amnesia in comparison to the recognition results for the normal control subjects. With the exception of the improved performance in the 1940s, R.C.'s profile appears to be relatively constant across decades. This may be an artifact of test construction.

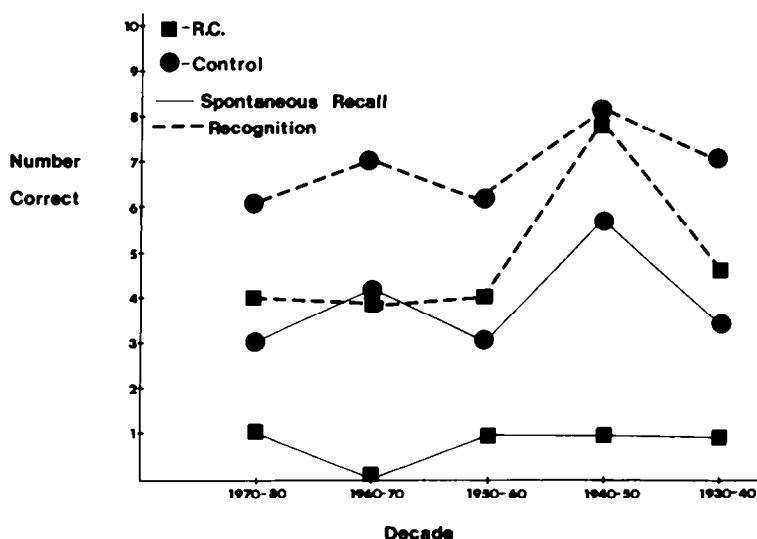


FIG. 7. Results on the verbal Remote Memory Test for both spontaneous recall and recognition. The "control" profile is the mean of three neurologically normal individuals matched in age, education, cultural background, and socioeconomic status to patient R.C.

## DISCUSSION

### *General Discussion*

Our results confirm that midline thalamic infarcts, especially when bilateral, cause a memory impairment. Our patients were ideal for study of clinico-anatomical correlations since they showed no clinical or CT evidence for brain lesions outside the thalamus. Whether the amnesia occurs as an isolated disorder or part of a more global dementia associated with thalamic pathology is open to question (Castaigne et al., 1966, 1981; Chassagnon, Boucher, Tommasi, Bianchi, & Moene, 1969; Cheek & Taveras, 1966; Smyth & Stern, 1938; Stern, 1939). Changes in higher mental functions other than amnesia were noted in our patients. A disorder in arousal and consciousness was prominent, particularly in the early stages. This observation has been reported previously in patients with thalamic lesions, with suggestions of disruption in subthalamic areas, ascending noradrenergic pathways, and/or the mesencephalic-diencephalic junction associated with the reticular activating system (Castaigne et al., 1981; Cox, 1937; Graff-Radford, Eslinger, Damasio, & Yamada, 1984; Graff-Radford, Damasio, Yamada, Eslinger, & Damasio, 1985; Magoun, 1958; Mills & Swanson, 1978; Teuber et al., 1968; Von Cramon, Hebel, & Schuri, 1985). In the chronic stage, this is frequently manifested as a lethargic, apathetic state, possibly with excessive sleepiness. Closely allied with the disorder in arousal is the reported attentional impairment (Archer, Ilinsky, Goldfader, & Smith, 1981; Schott et al., 1980). This may not be evident on simple tests such as digit span, or mental control tasks. Certain characteristics appear similar to the selective attention or executive control disorder of frontal lobe patients (Lezak, 1983; Luria, 1980, 1973; Stuss & Benson, 1984, 1986).

In the acute stage, all three had alterations in personality, mood, and awareness, including confabulation, reduplicative paramnesia, apathy, and unconcern. The personality change appears to be permanent only in patient R.C. Several years postonset, he remains unconcerned, unaware, jovial, and lacking any spontaneous drive or initiation. This apathy extends to activities of daily living. Other studies have reported similar findings in the acute or chronic stages (Graff-Radford et al., 1984; Mills & Swanson, 1978; Swanson & Schmidley, 1985; Teuber et al., 1968). Many of the personality characteristics resemble those described after focal frontal or frontal-limbic system damage (Damasio, 1979; Eslinger & Damasio, 1985; Stuss & Benson, 1984, 1986). The type of personality change after thalamic damage may be dependent on lesion location (Graff-Radford et al., 1984): anterior thalamic lesions, interrupting connections to the cingulate gyrus, could result in withdrawal and akinesia; DM lesions, affecting projections to the frontal (orbitofrontal) cortex, may cause euphoria and lack of insight.

Visual-perceptual and visual-constructive disorders were found in all three patients in the early stages. The relatively intact abilities for our patient with unilateral left pathology in the chronic stage replicates previous reports (Speedie & Heilman, 1982; Squire & Moore, 1979; Teuber et al., 1968). In contrast, Graff-Radford et al. (1984) reported visual-spatial impairment in 4/5 thalamic patients, including those with apparent unilateral left thalamic lesions. Only R.C. (bilateral) and R.M. (primarily right) revealed persistent problems, but only with more complex tasks such as block design. Whether the deficit was a premorbid limitation, as appears likely in part in the case of R.M., a reflection of lack of initiation, an impairment in attention (Graff-Radford et al., 1985; Henderson, Alexander, & Naeser, 1982), or an organizational, conceptual deficit similar to those found in patients with focal frontal pathology (Luria, 1980, 1973; Stuss & Benson, 1986) cannot be readily determined.

All three patients had a language disturbance that was difficult to characterize. The language functioning in our patients, typified by our patient with bilateral lesions, may be summarized as follows: poor initiation of speech with a general poverty of output that occasionally was contaminated by confabulation and lack of monitoring of output; fluctuation and variability in performance; relatively intact repetition and comprehension; and naming problems, apparently characterized more by perceptual errors, nonaphasic misnaming, intrusions, perseverations, and confabulation than by phonemic and semantic paraphasias. A deficit in word list generation has been considered a generic sign of aphasia, diffuse brain dysfunction (Benson, 1979), and focal frontal system lesions (Milner, 1964) including frontal white matter, supplementary motor area, or cingulate gyrus pathology (Damasio & Van Hoesen, 1980; Freedman, Alexander, & Naeser, 1984; Kertesz, 1979; Sanides, 1970; Stuss & Benson, 1986). In most of these cases, the deficit is one of initiation of speech rather than a true language impairment. It is noteworthy that the patient with the unilateral left thalamic lesion does not have significantly deficient verbal fluency.

Although language deficits have been reported after focal thalamic lesions (Archer et al., 1981; Bell, 1968; Barraquer-Bordas, Illa, Escartin, Rusalleda, & Marti-Vilalta, 1981; Mohr, Watters, & Duncan, 1975; Walshe, Davis, & Fisher, 1977), there appears to be lesion specificity, with more deficits reported after pulvinar, posterolateral, and ventrolateral thalamic nuclear damage (Brown, Riklin, & Waltz, 1971; Crosson, 1984; Davous et al., 1984; Eidelberg & Galaburda, 1982; Graff-Radford & Damasio, 1984; Graff-Radford et al., 1984, 1985; Ojemann, 1977; Ojemann, Fedio, & Van Buren, 1968; Van Buren & Burke, 1969; Vilkki & Laitinen, 1976). In general, these are areas that have reciprocal involvement with a cortical language area (Crosson, 1984; Eidelberg & Galaburda, 1982; Graff-Radford et al., 1984). In other patients with thalamic lesions, the disturbances in

language (similar to our patients) have been ascribed to alterations in alerting or in a gate controlling access (Fedio & Van Buren, 1975; Mateer & Ojemann, 1983; Ojemann, 1977); a deficit in the integration and monitoring of language (Penfield & Roberts, 1959); impaired arousal and vigilance in a system closely allied with the dominant hemisphere language system (Luria, 1977); and interaction with other functions such as attention, concentration, and memory (Castaigne et al., 1981; Choi, Sudarsky, Schachter, Biber, & Burke, 1983; Reynolds, Turner, Harris, Ojemann, & Davis, 1979; Speedie & Heilman, 1982), at least in the early stages (Graff-Radford & Damasio, 1984; Graff-Radford et al., 1984). The language impairment in our patients may be attributed to multiple dysfunctions including alteration in alerting, arousal, and monitoring.

While the diencephalic lesions temporarily disrupt performance on I.Q. tests (WAIS), there is a recovery to average or premorbid levels (Mills & Swanson, 1978; Squire & Moore, 1979; Teuber et al., 1968; Winocur, Oxbury, Roberts, Agnetti, & Davis, 1984). In particular, verbal I.Q. skills are within the normal range in the chronic stage, even for the patient with bilateral DM damage. It is likely that this does not reflect this patient's premorbid abilities. It is uncertain if the deficits that are observed are primarily cognitive in nature or reflect indirect effects on behavior due to apathy or lack of organization. Lesion localization may also play a role, with greater cognitive impairment with damage in caudal and/or lateral areas (Graff-Radford et al., 1985; Jurko & Andy, 1973; Wallesch, Kornhuber, Kunz, & Brunner, 1983).

It has been proposed that thalamic lesions may cause dementia (e.g., Castaigne et al., 1966; Chassagnon et al., 1969). In some cases small unilateral lesions result in general but not severe impairment of cognitive functions (Wallesch et al., 1983). Graff-Radford and colleagues (1984) proposed that patients with left but not right thalamic lesions meet the DSM criteria for dementia. Our "unilateral" patients had impairments other than memory, but these were not severe, and the disorder is considered limited to memory. Considering as well their return to a relatively normal life, we conclude that unilateral focal diencephalic lesions do not result in dementia (Speedie & Heilman, 1982, 1983; Squire & Moore, 1979).

Patient R.C. meets the criteria for dementia, in that he has impaired functioning in several higher mental functions. Just as clearly, his dementia has characteristics unlike those described for patients with Alzheimer's disease (Cummings & Benson, 1983). With the exception of the amnesia, the striking slowness in rate of information processing; the absence of apraxia, agnosia, or a true aphasia; the average I.Q. with impairment in using his acquired knowledge; the breakdown in organization of new or complex material; the striking inertia and apathy coupled with unconcern and a jovial attitude have all been described as characteristics of "sub-

cortical dementia" (Albert, 1978; Albert, Feldman, & Willis, 1974; Benson, 1983; McHugh & Folstein, 1975). Many of the symptoms described in our patient(s) are those reported after frontal and/or anterior limbic damage (Damasio, 1979; Poirier, Barbizet, Gaston, & Meyrignac, 1983; Stuss & Benson, 1986). In this respect, the proposed terms fronto-subcortical, frontal system, or frontal-limbic syndrome appear more appropriate (Albert, 1978; Freedman & Albert, 1985).

The severe memory impairment, however, is unlike that described under the generic heading of "subcortical dementia." The syndrome of paramedian thalamic infarct in our patients has its own symptom complex, characterized by a severe memory disorder and frontal-limbic behavioral changes. These deficits may occur with either unilateral or bilateral lesions depending on the size and location of the damage.

### *Memory*

We have no ready explanation for the deterioration in performance on the memory encoding test when given category names. A lack of accessibility to the stored information (Tulving & Madigan, 1970) does not seem feasible to explain the rapid loss of information that had already been recalled. Not only is the effectiveness of cueing related to the memory capacity of the subject tested (Tulving & Pearlstone, 1966), but additional influences such as inability to maintain working memory in the face of interference may be a factor.

### *Laterality*

Studies of patients with unilateral thalamic infarction have led to the proposal that hemispheric specialization extends to the thalamus (Squire & Moore, 1979; Teuber et al., 1968). In general, left unilateral lesions resulted in verbal memory problems, while right led to nonverbal memory deficits (Cappa & Vignolo, 1979; Choi et al., 1983; Jurko & Andy, 1973; Michel, Laurent, Foyatier, Blanc, & Portafaix, 1982; Reynolds et al., 1979; Speedie & Heilman, 1982, 1983; Squire, 1982; Squire & Moore, 1979; Squire & Slater, 1978; Vilkki, 1978). In some cases, unilateral left thalamic dysfunction has been reported as causing verbal memory deficit or disorientation, with right-sided lesions showing little evidence of any deficit (Choi et al., 1983; Graff-Radford et al., 1985). Others report that left-sided thalamic lesions appear to have a more devastating result, affecting both verbal and nonverbal memory and/or causing "dementia," while unilateral right damage causes only a questionable nonverbal deficit (Alexander & LoVerme, 1980; Graff-Radford et al., 1984, 1985; Squire, 1982; Squire & Slater, 1978). Finally, Wallesch et al. (1983) did not demonstrate a laterality effect for patients with long-standing unilateral thalamic lesions, suggesting that differences in the time of assessment was relevant.

Our results indicate right-left asymmetries at the level of the thalamus (see Fig. 4). Testing completed 1 to 2 years following the acute event suggested a continuing asymmetry. Hemispheric differences were most apparent only with relatively simple tests; with increasing complexity, the deficit became more general. At a certain level of complexity, non-specific functions of the thalamus, such as alerting, vigilance, or directed attention (Brown, 1974; Luria, 1977; Riklan & Levita, 1969, 1970; Wallesch et al., 1983), may obscure hemispheric differences. Patient R.M., with the predominant right thalamic involvement, did have difficulty on verbal tests such as the paired associates. His minimal left thalamic involvement may be a factor, as may be his lower intellectual abilities.

### *The Neuroanatomy of Memory*

*Anterograde vs. retrograde memory disorders in diencephalic amnesia.* Separate roles have been proposed for bitemporal and diencephalic regions in memory functions (Huppert & Piercy, 1978; Squire, 1981). In these claims, the bitemporal region has been suggested as being involved in faulty information storage, while diencephalic zones are hypothesized as being involved in the encoding operations, the acquisition of information. Most reports indicate "good" to normal memory for remote events after acute diencephalic dysfunction (as opposed to Korsakoff's disease in which a notable retrograde amnesia is demonstrated) (Michel et al., 1982; Schott et al., 1980; Von Cramon & Eilert, 1979; Winocur et al., 1984), with anterograde memory primarily or exclusively affected. The predominance of anterograde over retrograde amnesia has been interpreted as lending support to the idea that diencephalic amnesia is mainly a learning deficit, a failure in the initial stages of information processing, rather than a retrieval deficit (Winocur et al., 1984).

In contrast, two patients with lesions apparently limited to the diencephalic regions appear to have a severe and extensive remote memory loss in addition to the anterograde amnesia. Patient A.V. (Butters, 1984), with a paramedian glioma, had a severe and extensive retrograde amnesia, with no temporal gradient; our patient R.C. has a pronounced remote memory loss, again with no apparent temporal gradient for historical facts, but a possible gradient for personal events. Our patient R.C. had isolated bilateral diencephalic damage secondary to infarction and demonstrated a moderately severe retrograde amnesia.

Diencephalic pathology can cause both an impairment in acquisition as well as a remote memory deficit. Both of these paramedian thalamic patients (A.V., R.C.) appear similar in their memory functions to patient DRB (Damasio, Eslinger, Damasio, Van Hoesen, & Cornell, 1985), who had herpes simplex encephalitis. Damasio and colleagues argued that a combination of basal forebrain, lateral temporal, and insular damage were necessary for major retrograde amnesia. Our findings suggest that, while

focal limited DM damage may not cause severe retrograde amnesia, extension into very proximal corresponding regions or systems may result in severe remote memory loss in addition to anterograde amnesia. Our patient had bilateral mamillothalamic tract, dorsomedial nucleus, and anterior nucleus damage.

*Additional influences on memory functioning.* To establish primacy of one brain region for memory, it is necessary to demonstrate amnesia with lesions limited to that area (Butters, 1984). With this precondition, it is doubtful that human research will unequivocally unravel the role of different diencephalic areas in memory. In addition to limitations of *in vivo* analysis of structural pathology, the two diencephalic systems involved in memory are intimately linked. As illustrated in Figs. 1 and 2, DM nucleus is immediately adjacent to the anterior nucleus and mamillothalamic tract, the outflow of the MB. Involvement of fibers of passage surrounding the lesion site must be considered (Aggleton & Mishkin, 1983). In addition to physical proximity, they are usually fed by the same vascular system (Castaigne et al., 1981). Finally, Speedie & Heilman (1982, 1983), based on Valenstein and Nauta (1959) and Meibach and Siegel (1977), suggest that the DM receives input from the MB medial limbic system. While our quantitative structural analysis would appear to implicate the mamillothalamic tract to a greater extent (Von Cramon et al., 1985), it appears extremely difficult to selectively damage either the DM nucleus or the mamillothalamic tract output of the MB. Moreover, we are unable to negate possible involvement of other adjoining regions such as the subthalamic zones.

We propose that different brain regions in each of the two systems contribute to overall memory functioning in a specific way (Aggleton & Mishkin, 1983; Huppert & Piercy, 1979; Squire, 1981). First, based on previous animal and human research, both the DM nucleus and MB systems (including the mamillothalamic tract and anterior nuclei) contribute to memory, the severity of the disorder depending on the amount of damage, with maximum impairment if both systems are implicated (Aggleton & Mishkin, 1983; Thomas & Gash, 1985; Zola-Morgan & Squire, 1985). Second, in addition to the memory functions, each of these systems likely adds a unique contribution, based on its own role in behavior as well as its connections with more anterior brain regions. Involvement of the DM nucleus may be responsible for the "frontal lobe" component observed in the behavior. Much of the prefrontal cortex is defined by projections from DM (Goldman, 1979; Nauta, 1971). Many prefrontal signs can be replicated with focal DM pathology (Alexander & Fuster, 1973; Fuster & Alexander, 1973; Kolb, 1977; Schulman, 1964; Speedie & Heilman, 1982, 1983). This concept would also be compatible with the suggested frontal lobe or frontal system dysfunction (among other problems) observed in Korsakoff patients (Butters, 1984; Moscovitch,

1982; Oscar-Berman, 1980). The ventral-anterior, and the surrounding reticular nuclei, also have important frontal and limbic connections (Akert, 1964; Goldman, 1979; Nauta, 1971), possibly contributing to the arousal, mood, motivational, and attentional characteristics of behavior (Cummings & Benson, 1984; Scheibel, 1980; Scheibel & Scheibel, 1967). The medial limbic circuit, including the mamillary bodies, anterior thalamic nuclei, frontal connections, and linkage with the brain stem reticular core (Nauta, 1958), is theoretically implicated in activating and feedback control mechanisms (Livingston & Escobar, 1973). Both the DM nucleus and MB appear to be involved in memory as well as other functions associated with the frontal-subcortical executive and arousal systems and limbic motivational/emotional activities.

It may be hypothesized that impairment of these supplementary functions is sufficient to explain the memory deficit. Our results on the maze test (a "scalloping" effect, with an error occurring most often on the very first trial) suggested that motivational and/or attentional problems influenced the learning process. However, a true memory deficit, including left-right asymmetry, was revealed on memory tasks that were relatively simple and/or on tasks that required immediate recall without additional attentional demands.

### *Prognosis*

We have had the opportunity to follow our three patients for varying durations up to 3 years. Previous reports have been conflicting, with evidence of persistent deficits, full recovery, and partial recovery (Mills & Swanson, 1978; Schott et al., 1980; Swanson & Schmidley, 1985). In our cases there was a progressive and rapid improvement in the first 6 months, with some stabilization of performance between 6 and 12 months. Deficits demonstrated at this time appear to be relatively permanent (Aggleton & Mishkin, 1983; Castaigne et al., 1981; Michel et al., 1982; Zola-Morgan & Squire, 1985). In the case of R.C., the persistent impairment includes both anterograde and retrograde amnesia.

### *Summary*

Even small infarcts in the paramedian thalamic area can cause devastating neurobehavioral results. Although the primary deficit is in memory, these patients have serious problems with motivation, arousal, initiation, attention, and executive functions. The memory deficits reflect hemispheric specialization. Damage to the thalamus can cause persistent impairment in a number of higher mental functions.

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