

# A VERBAL LONG TERM MEMORY DEFICIT IN FRONTAL LOBE DAMAGED PATIENTS

**Wolfgang Jetter, Ulrich Poser, Robert B. Freeman, Jr., and Hans J.  
Markowitsch**

(Department of Psychology, University of Konstanz, and Clinics Dr.  
Schmieder, Allensbach)

## INTRODUCTION

The prefrontal cortex is probably the most frequently investigated cortical area in neuropsychology. This can be seen in the numerous surveys on its anatomy and roles in behavior, one of the most recent ones being from Stuss and Benson (1984). But in spite of this fact a good deal of uncertainty still exists as to what the principal functions are for this area of the brain, an uncertainty which is probably due to the variety of possible causes of frontal damage (tumor, trauma, vascular damage, lobectomy) (Poeck, 1982), to the involvement of the frontal lobes in a number of quite different functions, as well as to the possibility of dividing the frontal lobes into several subregions on the basis of cytoarchitecture, hodology, and behavior (Markowitsch, 1986). After reviewing the literature on the frontal lobe, Stuss and Benson (1984) extracted six specific prefrontal functions, but they were unsure whether these functions were "facets of one main deficit or based on functions of specific location" (p. 23).

The present investigation was directed to the question of whether or not mnemonic functions are altered following frontal lobe injury. The role of the frontal lobes in memory processes has been a matter of dispute for a long time (Feuchtwanger, 1923; Jacobsen, 1935) and has led to the development of a number of specific test situations both for animals and humans (surveys in Damasio, 1979; Fuster, 1980; Luria, 1976, 1980; Markowitsch and Pritzel, 1977; Rosenkilde, 1983; Rozin, 1976; Warren and Akert, 1964). Admittedly, human case reports are rare in which frontal lobe damage resulted in Korsakoff-like amnesic syndromes (e.g., Pfeifer, 1910), although even Hécaen and Albert (1978) mention such cases. The majority of them are probably caused by brain damage beyond that of the frontal lobes. Nevertheless, a number of cases which mani-

fested memory-related defects have been described (e.g., Lewinsohn et al., 1972; Ruff and Volpe, 1981), many of which might, however, be interpreted as secondary to nonmnemonic deficits (Luria, 1980; Milner, 1982; Stuss and Benson, 1984; Stuss et al., 1982, Walsh, 1978). (In 1923 Feuchtwanger already made the important distinction between primary and secondary disturbances following frontal lobe damage.) Some exceptions, however, can be listed: Arnold (1984) made a strong argument for an involvement of the (dorsolateral) prefrontal cortex in motor memory and of the (posterior) orbitofrontal cortex in so-called affective memory. Risse and co-workers (1984) observed severe impairments in the acquisition and long-term retention of a verbal list-learning task in patients with inferior frontal lobe damage and Wallesch et al. (1983) found deficits in pair associate learning, digit span, sequential concept formation and in the Benton test in patients with deeper frontomedial lesions. In a recent article on amnesia, Moscovitch (1982) also emphasized the memory-related role of the frontal lobe and provided a list of memory disorders which are common to both Korsakoff amnesics and frontal lobe damaged patients (increased susceptibility to interference, poor memory for temporal order, poor short term memory, difficulty in using imagery mnemonics, poor release from proactive interference; his Table 16.1).

The intimate relations between the mediodorsal nucleus of the thalamus and the prefrontal cortex (Markowitsch, 1982, 1986) may provide a further line of argumentation for a memory-related role of the frontal lobes, as has been indicated in numerous case reports in which damage to the mediodorsal nucleus resulted in a profound and lasting anterograde amnesic state (Cramon and Eilert, 1979; Mair et al., 1979; Markowitsch, 1982; Schott et al., 1980; Squire and Moore, 1979; Squire and Slater, 1978; Victor et al., 1971; Winocur et al., 1984).

In order to clarify somewhat more the possible role of the frontal lobe in the long term processing of information, we used three forms of a verbal retention test, free recall, cued recall, and recognition of words which had been learned 15 min. or 1 day before. The patients tested had left, right, or bilateral damage to the frontal lobes.

## MATERIALS AND METHOD

### *Subjects*

Twenty-eight brain damaged patients participated in the study. All of them were hospitalized during the time of investigation. Half of the patients had lesions within the frontal lobes (group F); the other half had brain lesions outside this area (group NF), consisting of patients with temporal, parietal, and occipital brain damage. This group was used to control for the factor 'localization of damage', that is, it was introduced to control for the variables brain lesions and

hospitalization. In group F only patients were included who lacked aphasic symptoms or motor disturbances. In group NF aphasic disturbances could not be totally excluded, especially as patients with left temporal brain damage were included. The etiology of the sample was rather homogeneous: 93% of the cases had traumatic brain lesions, 85% of which had a contusio cerebri and 15% open brain damage. In two patients of group F brain damage was of cerebro-vascular nature. Neurological diagnoses were based on computer tomographic scans in all cases. Table I gives a survey of several patient variables.

During the first meeting all patients in the ward were told that we planned an investigation on memory disturbances resulting from brain damage. (When asked about their mnemonic abilities all admitted having problems with memory.) As these disturbances were the most obvious handicaps of the majority of the patients, most of those who were asked to participate agreed to do so. All participants behaved cooperatively and were well motivated.

### *Tests and Procedure*

Three lists of words with 16 nouns each were used. The words of each of the three lists originated from four different categories, each category being represented by four words. Within each list the sequence of words was random. As different categories were used for each list, the total stimulus material consisted of 12 different semantic categories. Categories were clothing, jobs, body parts, and weather for list I, animals, cities, buildings, and transportation vehicles for list II, countries, food products, musical instruments, and sport activities for list III. All three lists of words had the same degree of difficulty; they were adapted for the German language and culture from Battig and Montague (1969).

### *Experimental design*

In order to operationalize 'remembrance' and 'retrieval' (1) free recall, (2) cued recall, and (3) recognition procedures were employed: In the 'free recall' test patients had to write the words they remembered, in the 'cued recall' test those semantic categories, which the words belonged to, were provided, and in the 'recognition' test the patients received a list of words, containing the 16 stimulus words together with an additional 16 distracting words (randomly distributed). The distracting words came from the same kinds of words as the original list. Tests were performed 15 min. and 24 h after the original learning.

Each patient participated in the four testing sessions during four consecutive days. During each of the first three days one of the three lists of words had to be learned. In the learning phase a 16-word list was presented two times for one min each. Both learning phases were followed by a short check of remembrance. The first test started 15 min. after the end of the second learning phase. Table II gives a survey of the time sequence.

A semantic encoding instruction was given, that is, the patient was told that the words could be categorized. The sequence of presentation of the words was varied randomly to minimize possible side effects such as proactive interference, practice, and sequence effects.

TABLE I  
Survey of Patient Variables

Patient No.	Frontal Group				Patient No.	Non-frontal Group					
	Sex	Age	Chronicity (in years)	IQ		CT-diagnosis	Sex	Age	Chronicity (in years)	IQ	CT-diagnosis
1	M	24	2.66	94	Contusio cerebri right frontal	15	F	42	2.58	112	Contusio cerebri left temporal
2	M	35	1.25	106	Contusio cerebri bifrontal	16	M	34	2.66	112	Contusio cerebri left parietal
3	M	34	4	100	Open skull trauma right frontal	17	M	21	4.74	91	Contusio cerebri left temporal
4	M	42	1.08	92	Ischemic infarct left frontal	18	M	19	0.75	103	Contusio cerebri right temporal
5	M	43	0.66	96	Contusio cerebri right frontal	19	M	19	2.42	124	Contusio cerebri right temporal
6	M	38	0.42	102	Contusio cerebri left frontal	20	M	62	1.33	124	Open skull trauma left parieto-occipital
7	F	44	4.66	100	Contusio cerebri left frontal	21	M	23	3.92	100	Contusio cerebri left temporal
8	M	23	1.66	118	Open skull trauma bifrontal	22	M	41	3.50	106	Contusio cerebri left temporal
9	M	32	1.42	102	Aneurysmic bleeding bifrontal	23	M	23	0.75	90	Contusio cerebri left parietal
10	F	40	3.92	100	Contusio cerebri right frontal	24	M	29	2.75	100	Contusio cerebri left parietal
11	F	27	1.83	112	Contusio cerebri right frontal	25	F	31	1.33	98	Contusio cerebri right parieto-occipital
12	F	52	3.75	103	Contusio cerebri bifrontal	26	F	24	0.75	80	Contusio cerebri right temporo-occipital
13	F	25	3.41	79	Open skull trauma bifrontal	27	M	25	0.50	115	Contusio cerebri left temporo-occipital
14	M	46	0.92	145	Contusio cerebri left frontal	28	M	45	5.50	100	Contusio cerebri left temporal
Means		36	2.26	103.5				31	2.39	103.9	

TABLE II  
*Sequence and Kind of Word List Testing*

Word list	Time of testing	
	15 min.	1 day
List I	free recall	cued recall
List II	cued recall	recognition
List III	recognition	free recall

TABLE III  
*Time Sequence of the Tests Presented*

Days	Experimental tests
1	Learning of List I in 2 sessions; free recall after 15 min.
2	Cued recall test of List I; learning of List II; cued recall test of List II after 15 min.
3	Recognition test of List II (after 1 day); learning of List III; recognition test of List III after 15 min.
4	Free recall test of List III; discussion of the procedure and results with the subjects

## RESULTS

### *Learning performance*

The two 16-word lists (which were presented twice in succession to the subjects and which were each followed by a free recall test after one minute) revealed a rather similar performance between the two groups of patients (2-factor analysis of variance;  $F = 0.42$ ; d.f. = 1, 26;  $p > 0.05$ ) (Figure 1). The increase from the first to the second session was similar as well for both groups ( $F = 1.95$ ; d.f. = 1, 26;  $p > 0.05$ ), the increase between the first and second session being highly significant ( $F = 158.25$ ; d.f. = 1, 26;  $p < 0.01$ ).

### *Retention performance*

The average values of correctly reproduced and recognized words are presented in Figure 2. Recognition performance was calculated according to the formula of Warrington and Weiskrantz (1974) (no. = number):

$$\frac{(\text{no. of all words produced}) \times (\text{no. of correctly identified words} - \text{no. of false positives})}{(\text{no. of all words produced}) - (\text{no. of false positives})}$$

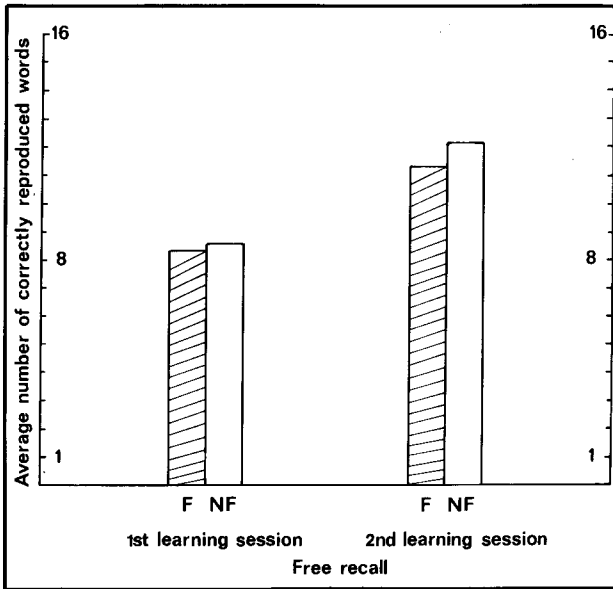


Fig. 1 — Reproduction performance of groups F and NF during the first (left two columns) and second (right two columns) learning test under free recall conditions.

A three factor analysis of variance revealed no main difference between the two groups ( $F=2.86$ ;  $d.f.=1$ ;  $p>0.05$ ), but a significant difference between time of testing ( $F=58.64$ ;  $d.f.=1, 26$ ;  $p<0.01$ ), and methods of testing ( $F=107.13$ ;  $d.f.=1, 26$ ;  $p<0.01$ ). Between groups and testing methods, and time of testing and testing methods, significant interactions were obtained (groups  $\times$  testing methods:  $F=8.48$ ;  $d.f.=2, 52$ ;  $p<0.01$ ; time of testing  $\times$  testing methods:  $F=7.20$ ;  $d.f.=2, 52$ ;  $p<0.01$ ).

The principal result, however, was the significant interaction between

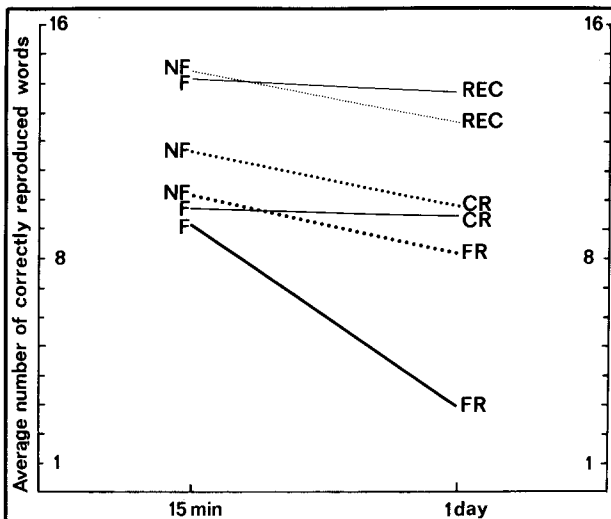


Fig. 2 — Memory performance of groups F and NF during free recall (FR), cued recall (CR), and recognition (REC).

group, time of testing, and method of testing ( $F=6.12$ ;  $d.f.=2, 52$ ;  $p<0.01$ ). Application of the Scheffé test revealed a number of significant ( $p<0.01$ ) differences: A drastic drop in performance of freely recalled words was observed in the frontal group when testing took place after 15 min. and 1 day (difference: 6.21 words); furthermore, group F reproduced significantly fewer words (difference: 5.21 words) after one day than group NF did. And lastly, the frontal group produced significantly fewer words (difference: 6.36 words) under the free-recall than under the cued recall condition after one day.

The most significant difference, however, was that observed in group F between free recall and recognition after one day (difference 9.85 words). In the comparison of these two tests group NF as well reproduced fewer words during free recall (difference: 2.74).

Spearman rank correlations did not reveal significant relations between IQ and performance. In general no differences were observed between left, right, or bifrontal damage (Table IV), though the right frontal damaged subjects tended to be somewhat inferior to the other groups during cued recall and the patients with bifrontal damage somewhat inferior to patients with unilateral damage during the free recall condition after one day.

### *Reproduction of categories*

As the strongest difference between the two groups of patients occurred under the free recall condition with testing after 24 hours, the question was addressed as to whether this deficit of group F was paralleled by a smaller number of reproduced categories.

Tulving and Pearlstone (1966) defined category reproduction as the number of categories from which at least one word is reproduced (p. 386). Indeed, between the two groups no significant differences were found under the 15 min. free recall condition (3.62 vs. 2.86 categories for groups F and NF, resp.), while under the one-day free recall condition, group NF

TABLE IV

*Medians of Correctly Reproduced/Reidentified Words for Patients with Right (R), Left (L), or Bifrontal (B) Damage under the Different Testing Conditions*

Frontal group	Recognition		Cued recall		Free recall	
	15 min.	1 day	15 min.	1 day	15 min.	1 day
R	14	12.8	7	7	7	4
L	16	14	12.5	10.5	10	3.5
B	14.3	13.4	11	11	7.5	0

produced significantly more categories than group F (3.21 vs. 1.64 categories;  $t = 4.03$ , d.f. = 13,  $p < 0.05$ )

### *Categorical clustering*

Because persons usually have the tendency to cluster words according to categories under conditions of free recall (Bousfield and Bousfield, 1966), a phenomenon which is interpreted as indicating a semantic organization or as a "deeper" encoding (Craik and Lockhart, 1972), we analyzed the tendency for categorial clustering according to Rubin and Butters' (1981) formula

$$cc = \frac{\sum_{i=1}^4 SCR_i}{n - k}$$

with  $cc$  = categorial clustering,  $SCR_i$  being the number of reproduced words of category  $i$  which are followed by at least one word of the same category,  $n$  the total number of correctly reproduced words, and  $k$  the number of correctly reproduced categories. (Optimal clustering results in a value of 1, no clustering in a 0). Table V presents the mean values of categorial clustering for both groups of patients in the two acquisition tests and in the free recall tests performed 15 min. and 1 day after the second acquisition test. A two-factor analysis of variance resulted in insignificant effects throughout (group:  $F = 1.17$ ; d.f. = 1, 26;  $p > 0.05$ ; time of testing:  $F = 1.03$ ; d.f. = 3, 78;  $p > 0.05$ ; group  $\times$  time of testing:  $F = 2.52$ ; d.f. = 3, 78;  $p > 0.05$ ), that is, the two groups did not differ in their use of categorial clustering.

That each of the two groups did apply clustering was revealed by the values obtained with the formula of Bousfield (1966)

$$E_{(cc)} = \frac{\frac{\sum m_i^2}{\sum m_i} - 1}{n - k}.$$

In this formula  $E_{(cc)}$  is the expected value for categorial clustering,  $m_i$  the number of reproduced words from category  $i$ ,  $n$  the total number of correctly reproduced words, and  $k$  the number of the correctly reproduced categories. The means obtained by using this formula are given in Table V



TABLE V

*Categorical Clustering During Acquisition and Free Recall*

Group	First acquisition test	Second acquisition test	Free recall tests	
			15 min.	1 day
Group F	0.37 (0.37)	0.76 (0.35)	0.79 (0.29)	0.72 (0.53)
Group NF	0.75 (0.37)	0.78 (0.28)	0.79 (0.30)	0.83 (0.36)

The obtained mean values are given without parentheses; the expected values [ $E_{cc}$ ], calculated according to a modified formula of Bousfield and Bousfield (1966), are given in parentheses.

(values in parentheses). A comparison of the observed and expected means in Table V (t-tests) revealed that both of the groups demonstrated significant clustering. One can therefore assume that the randomly presented words were subjected to semantic analysis (semantic encoding).

*Intrusion errors*

A susceptibility to being distracted is among the most prominent symptoms of frontal lobe damaged patients (e.g., Luria, 1969; Milner, 1974). To determine the proportion of interference phenomena within the impaired reproduction performance of frontal lobe damaged patients during free recall after one day, the protocols were screened for errors of intrusion. (Intrusions are defined as such words which were learned in prior word lists, but are reproduced in later lists and which are thus errors.) When considering the number of intrusion errors, no differences were observed between the two groups (t-test;  $t=0.2$ , d.f. = 13,  $p>0.05$ ). However, when the number of intrusion errors was compared to the correctly reproduced words of each group, group F obtained a value of 0.81 intrusions, while group NF had a value of only 0.27.

## DISCUSSION

*A defective long term memory in frontal lobe damaged patients?*

The main results of our investigation are the similar performance of both groups of patients under the recognition and cued recall conditions, and the drastically inferior performance of the frontal group under the free recall condition after one day. Surprisingly, no specific relations between side of frontal damage or between unilateral versus bilateral frontal damage could be established.

The fact that patients of group F performed at a level comparable to that of group NF under the cued recall and recognition conditions argues against an interpretation of the free recall deficit being due to reduced encoding or to an inferior level of processing. That semantic categorization was used similarly in both groups of patients is a further argument against an inferior encoding of information in group F. Consequently, we suggest the existence of a kind of retrieval deficit for group F. However, this suggestion has to be qualified further, as the behavior of group F during cued recall was indistinguishable from that of group NF. The crucial difference between free recall and cued recall seems to lie in the fact that the subjects have to generate their own categories internally in the free recall condition, while they are free of this work in cued recall. Though the number of reproduced categories is related to the number of words reproduced on the whole, the fact that the frontal damaged patients (compared to the others) reproduced words from significantly fewer categories during free recall after one day, gives further support for the interpretation that the impaired ability to generate adequate retrieval cues following a delay of one day (but not following a delay of 15 min.) is a long term memory related defect in the frontal lobe damaged group.

A related observation was recently made by Petrides and Milner (1982). These authors found that patients with damage to the frontal lobe were impaired in their ability to "self-order" tasks, that is to organize and carry out a sequence of responses. This impairment was consistently found for all four tasks administered (2 verbal, 2 nonverbal tests) in subjects with damage to the left frontal lobe, but for the two nonverbal tests only in patients with right frontal damage.

#### *Alternative explanation of the deficit*

Though it is tempting to consider the observed deficit of the frontal group during free recall after one day as a retrieval deficit (caused by a reduced ability to generate appropriate categorial cues), other interpretations of the impaired performance can be offered as well, which are more conservative and less memory-related: The primary deficits of subjects with damage to the frontal convexity which are most widely accepted concern spontaneity and active attention (Choroschko, 1923; Damasio, 1979; Faust, 1955, 1960; Feuchtwanger, 1923; Forster, 1919; Homskaya, 1973; Kleist, 1934; Ruffin, 1939). Denny-Brown (1951), in reviewing Feuchtwanger's (1923) data, wrote about frontal lobe damaged patients: "Inability to concentrate, lack of motivation and initiative were primary disorders. Lack of spontaneity, with variable changes in motivation were closely related" (p. 65). Faust (1955) defined this symptom of frontal lobe pathology as a lack of drive in the presence of external "arousability"

("Antriebschwäche bei vorhandener Fremderregbarkeit"), or, in 1960, as a lack of self-determined initiative ("Mangel an Eigeninitiative"). Frontal lobe damaged patients fail to define their goals appropriately ('to program themselves'), or — when they do succeed — they fail to carry out behavior as planned (Damasio, 1979; Pribram, 1975).

Seen in the light of the statements made above we can interpret the observed deficit in our frontal lobe damaged patients under the condition of free recall after one day as a reduced ability to generate appropriate cues (e.g., to categorize semantically), caused by a lack of initiative and concentration and/or by a limited use of possibly available cues (cf. Cicerone et al., 1983). Although this interpretation appears plausible, it probably does not explain all of the deficits as the frontal lobe damaged patients would then most probably have shown a significant deficit already under the free recall condition after 15 min. As this was not the case (cf. Figure 2), and as the motivational level of the patients appeared to be high in general, we favor an interpretation of the deficit during free recall after one day as related to both attentive *and* long term memory disturbances. Within this view we would attribute the problems in attention and drive to the prefrontal cortex, and the mnemonic components to the mediodorsal nucleus, which most likely was at least partly degenerated in each of our cases with frontal lobe pathology. It should be remarked, however, that because trauma was the most frequent etiology in our subjects, this might have led in several of the patients to a more extensive neuropathology than is suggested by the outcome of the CT scans, with perhaps some involvement of the cerebral white matter and/or ischemic necrosis.

#### ABSTRACT

The retention performance for learned words was compared in two groups of cortically damaged patients: A group of 14 patients with uni- or bilateral damage to the frontal lobes (group F), and a group of 14 patients with postrolandic damage. The patients learned three lists of words each of which had to be reproduced after 15 min. and after 1 day: one list under free recall, one under cued recall, and one under a recognition condition. While the performance of the two groups of patients was similar under all three conditions when tested after 15 min., group F was significantly inferior in the one day free recall retention test. We interpret this deficit as related in part to the classic "frontal" symptomatology (reduced attention and lack of initiative, drive, and concentration), and in part to a distinct disturbance of long term memory. We base this conclusion on the similar performance levels of frontal and non-frontal patients under all other conditions of testing, in particular under the free recall condition after the 15 min. delay. Reasons are given why attentive and drive related components might be mediated by prefrontal cortical neurons themselves, while mnemonic components might be mediated by mediodorsal thalamic neurons projecting to the prefrontal cortex. No or only minor differences could be established between the side of damage or between uni- versus bilateral frontal damage.

## REFERENCES

- ARNOLD, M.B. *Memory and the Brain*. Hillsdale, N.J.: Erlbaum, 1984.
- BATTIG, W.F., and MONTAGUE, W.E. Category norms for verbal items in 56 categories: A replication and extension of the Connecticut category norms. *Journal of Experimental Psychology Monograph*, 80 (No. 3, Pt.2): 1-46, 1969.
- BOUSFIELD, A.K., and BOUSFIELD, W.A. Measurement of clustering and of sequential constancies in repeated free recall. *Psychological Reports*, 19: 935-942, 1966.
- BOUSFIELD, W.A. The occurrence of clustering in the recall of randomly arranged associates. *Journal of General Psychology*, 49: 229-240, 1966.
- CHOROSCHKO, W.K. Die Stirnlappen des Gehirns in funktioneller Beziehung. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, 83: 291-302, 1923.
- CICERONE, K.D., LAZAR, R.M., and SHAPIRO, W.R. Effects of frontal lobe lesions on hypothesis sampling during concept formation. *Neuropsychologia*, 21: 513-524, 1983.
- CRAIK, F.I.M., and LOCKHART, R.S. Levels of processing: A framework for memory research. *Journal of Verbal Learning and Verbal Behavior*, 11: 671-684, 1972.
- CRAMON, D. von, and EILERT, P. Ein Beitrag zum amnestischen Syndrom des Menschen. *Nervenarzt*, 50: 643-648, 1979.
- DAMASIO, A. The frontal lobes. In K.M. Heilman and E. Valenstein (Eds.), *Clinical Neuropsychology*. New York: University of Oxford Press, 1979, pp. 360-412.
- DENNY-BROWN, D. The frontal lobes and their functions. In A. Feiling (Ed.), *Modern Trends in Neurology*. London: Hoeber-Harper, 1951, pp. 13-89.
- FAUST, C. Zur Symptomatik frischer und alter Stirnhirnverletzungen. *Archiv für Psychiatrie und Nervenkrankheiten*, 193: 78-97, 1955.
- FAUST, C. Die psychischen Störungen nach Hirntraumen: Akute traumatische psychische Spätfolgen nach Hirnverletzungen. In H.W. Gruhle, R. Jung, W. Mayer-Gross and M. Müller (Eds.), *Psychiatrie der Gegenwart. Forschung und Praxis, Vol. II: Klinische Psychiatrie*. Berlin: Springer, 1960, pp. 552-645.
- FEUCHTWANGER, E. *Die Funktionen des Stirnhirns*. Berlin: Springer, 1923.
- FORSTER, E. Agrammatismus (erschwerter Satzfindung) und Mangel an Antrieb nach Stirnhirnverletzung. *Monatsschrift für Psychiatrie und Neurologie*, 46: 1-43, 1919.
- FUSTER, J.M. *The Prefrontal Cortex*. New York: Raven Press, 1980.
- HÉCAEN, H., and ALBERT, M.L. *Human Neuropsychology*. New York: Wiley, 1978.
- HOMSKAYA, E.D. The human frontal lobes and their role in the organization of activity. *Acta Neurobiologiae Experimentalis*, 33: 509-522, 1973.
- JACOBSEN, C.F. Functions of the frontal association area in primates. *Archives of Neurology and Psychiatry*, 33: 558-569, 1935.
- KLEIST K. Kriegsverletzungen des Gehirns in ihrer Bedeutung für die Hirnlokalisation und Hirnpathologie. In K. Bonhoeffer (Ed.), *Handbuch der ärztlichen Erfahrungen im Weltkriege 1914/1918, Vol. IV: Geistes- und Nervenkrankheiten*. Leipzig: Barth, 1934, pp. 343-1393.
- LEWINSOHN, P.M., ZIELER, R.E., LIBET, J., EGEGERG, S., and NIELSON, G. A comparison between frontal and nonfrontal right- and left-hemisphere brain-damaged patients. *Journal of Comparative and Physiological Psychology*, 81: 248-255, 1972.
- LURIA, A.R. Frontal lobe syndromes. In P.J. Vinken and G.W. Bruyn (Eds.), *Handbook of Clinical Neurology, Vol. 2*. Amsterdam: North-Holland, 1969, pp. 725-768.
- LURIA, A.R. *The Neuropsychology of Memory*. New York: Halstead Press, 1976.
- LURIA, A.R. *Higher Cortical Functions in Man* (2nd ed.). New York: Basic Books, 1980.
- MAIR, W.G.P., WARRINGTON, E.K., and WEISKRANTZ, L. Memory disorder in Korsakoff's psychosis: A neuropsychological and neuropathological investigation of two cases. *Brain*, 102: 749-783, 1979.
- MARKOWITSCH, H.J. The thalamic mediodorsal nucleus and memory: A critical evaluation of studies in animals and man. *Neuroscience and Biobehavioral Reviews*, 6: 351-380, 1982.

- MARKOWITSCH, H.J. Anatomical and functional organization of the primate prefrontal cortical system. In H.D. Steklis (Ed.), *Comparative Primate Biology, Vol. IV: The Neurosciences*. New York: Alan R. Liss, 1986, in press.
- MARKOWITSCH, H.J., and PRITZEL, M. Comparative analysis of prefrontal learning functions in rats, cats, and monkeys. *Psychological Bulletin*, 84: 817-837, 1977.
- MILNER, B. Some effects of frontal lobectomy in man. In J.M. Warren and K. Akert (Eds.), *The Frontal Granular Cortex and Behavior*. New York: McGraw-Hill, 1964, pp. 312-334.
- MILNER, B. Hemispheric specialization: Scope and limits. In F.O. Schmitt and F.G. Worden (Eds.), *The Neurosciences: Third Study Program*. New York: MIT Press, 1974, pp. 75-89.
- MILNER, B. Some cognitive effects of frontal-lobe lesions in man. *Philosophical Transactions of the Royal Society (London)*, 298 B: 221-226, 1982.
- MOSCOVITCH, M. Multiple dissociations of functions in amnesia. In L.S. Cermak (Ed.), *Human Memory and Amnesia*. Hillsdale, N.J.: Erlbaum, 1982, pp. 337-370.
- PETRIDES, M., and MILNER, B. Deficits on subject-ordered tasks after frontal- and temporal-lobe lesions in man. *Neuropsychologia*, 20: 249-262, 1982.
- PFEIFER, B. Psychische Störungen bei Hirntraumen. *Archiv für Psychiatrie und Nervenkrankheiten*, 47: 548-738, 1910.
- POECK, K. Störungen von Antrieb und Affektivität. In K. Poeck (Ed.), *Klinische Neuropsychologie*. Stuttgart: Thieme, 1982, pp. 198-203.
- PRIBRAM, K.H. The primate frontal cortex: Progress report 1975. *Acta Neurobiologiae Experimentalis*, 35: 609-625, 1975.
- RISSE, G.L., RUBENS, A.B., and JORDAN, L.S. Disturbances of long term memory in aphasic patients. *Brain*, 107: 604-617, 1984.
- ROSENKILDE, C.E. Functions of the prefrontal cortex. *Acta Physiologica Scandinavica, Suppl. 514*: 1-58, 1983.
- ROZIN, P. The psychobiological approach to human memory. In M.R. Rosenzweig and E.L. Bennet (Eds.), *Neural Mechanisms of Learning and Memory*. Cambridge, M.A.: MIT Press, 1976, pp. 3-46.
- RUBIN, D.C., and BUTTERS, N. Clustering of alcoholic Korsakoff patients. *Neuropsychologia*, 19: 137-140, 1981.
- RUFF, R.L., and VOLPE, B.T. Environmental reduplication associated with right frontal and parietal lobe injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, 44: 382-386, 1981.
- RUFFIN, H. Stirnhirnsymptomatologie und Stirnhirnsyndrome. *Fortschritte der Neurologie und Psychiatrie*, 11, 34-81, 1939.
- SCHOTT, B., MAUGUIERE, F., LAURENT, B., SERCLERAT, O., and FISCHER, C.L. Amnésie thalamique. *Revue Neurologique*, 136: 117-130, 1980.
- SQUIRE, L.R., and MOORE, R.Y. Dorsal thalamic lesion in a noted case of chronic memory dysfunction. *Annals of Neurology*, 6: 503-506, 1979.
- SQUIRE, L.R., and SLATER, P.C. Anterograde and retrograde memory impairment in chronic amnesia. *Neuropsychologia*, 16: 313-322, 1978.
- STUSS, D.T., and BENSON, D.F. Neuropsychological studies of the frontal lobes. *Psychological Bulletin*, 95: 3-28, 1984.
- STUSS, D.T., BENSON, D.F., KAPLAN, E.F., WEIR, W.S., NAESER, M.A., LIEBERMAN, T., and FERRILL, D. The involvement of orbitofrontal cerebrum in cognitive tasks. *Neuropsychologia*, 21: 235-248, 1983.
- STUSS, D.T., KAPLAN, E.F., BENSON, D.F., WEIR, W.S., CHIULLI, S., and SARAZIN, F.F. Evidence for the involvement of orbitofrontal cortex in memory functions: an interference effect. *Journal of Comparative and Physiological Psychology*, 96: 913-925, 1982.
- TULVING, E., and PEARLSTONE, Z. Availability versus accessibility of information in memory for words. *Journal of Verbal Learning and Verbal Behavior*, 5: 381-391, 1966.
- VICTOR, M., ADAMS, R.D., and COLLINS, G.H. *The Wernicke-Korsakoff Syndrome*. Oxford: Blackwell, 1971.

- WALLESCH, C.W., KORNHUBER, H.H., KUNZ, T., and BRUNNER, R.J. Neuropsychological deficits associated with small unilateral thalamic lesions. *Brain*, 106: 141-152, 1983.
- WALSH, K.W. *Neuropsychology. A Clinical Approach*. New York: Churchill Livingstone, 1978.
- WARREN, J.M., and AKERT, K. (Eds.) *The Frontal Granular Cortex and Behavior*. New York: McGraw-Hill, 1964.
- WARRINGTON, E.K., and WEISKRANTZ, L. The effect of prior learning on subsequent retention in amnesic patients. *Neuropsychologia*, 12: 419-428, 1974.
- WINOCUR, G., OXBURY, S., ROBERTS, R., AGNETTI, V., and DAVIS, C. Amnesia in a patient with bilateral lesions to the thalamus. *Neuropsychologia*, 22: 123-143, 1984.

Dr. Hans Markowitsch, Department of Psychology, University of Konstanz, P.O. Box 5560, D-7750 Konstanz, Federal Republic of Germany