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Language and Cognitive Deficits Resulting from Medial and Dorsolateral Frontal Lobe Lesions*

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Summary. A total of 36 patients with chronic unilateral circumscribed medial and dorsolateral frontal lobe lesions were investigated with a range of neuropsychological tests. Lateralized deficits in tasks depending on language functions were found with dorsolateral but not with medial lesions. The specific role of Broca's area could not be confirmed. Lesions of the supplementary motor area led to mild deficits in tests of "concept formation". Frontomedial lesions situated more deeply in the interhemispheric fissure resulted in memory deficits. The results are discussed on the basis of recent neurophysiological theories of brain function.

Key words: Frontal lobe – Supplementary motor area – Language – Cognition – Memory

Introduction

Disturbances in delayed response tasks resulting from frontal ablations in monkeys were reported by Jacobson in 1935. These findings have been corroborated by numerous studies which have shown that circumscribed dorsolateral frontal lesions in animals lead to specific behavioral alterations (for a review, see Fuster 1980). For the species "man" it is widely accepted that separable clinical syndromes result from orbitofrontal and dorsolateral frontal lesions (Kleist 1934; Luria 1969). Whereas orbitofrontal lesions frequently produce modifications in the affective and emotional sphere, dorsolateral lesions are reported to result in specific cognitive deficits (Milner 1963, 1971; Damasio 1979). Language disturbances have been reported with left frontal lesions in two areas: Broca's area in the foot of the second and third frontal convolution and the supplementary motor area (SMA) on the medial surface of the brain (Brickner 1940, Penfield and Roberts 1959; Rubens 1975; Racy et al. 1979). Furthermore, it has

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been shown (Tonkonogy and Goodglass, 1981) that for the lesion of Broca's area the size of the damage was an important factor which determined the degree and duration of the resulting language deficit.

There is conclusive evidence that the SMA is important for any "intended" activity. For instance Goldberg et al. (1981) reported a contralateral "alien hand" sign to occur with SMA lesions. Also readiness potential studies (Deecke and Kornhuber 1978; Grözinger et al. 1979) have shown that medial frontal structures are active earlier than other regions of the brain for speech and voluntary finger movements. These results have also been supported by single cell recordings (Brinkman and Porter 1979) and regional cerebral blood flow studies which have demonstrated high SMA activity during speech and voluntary movements (Ingvar and Schwartz 1974; Roland et al. 1980).

These findings have lead to the conclusions that the SMA is either one of the ways that channel drive, will and planning into action (Kornhuber 1980a) or else that it is the liason area of the brain for intentions (Eccles 1982).

Clinical evidence for the postulated specific role of the SMA is scarce. The most common clinical findings with lesions involving the SMA are a transient stage of apathy, akinesia, lack of drive and motivation and also disturbances of speech similar to transcortical motor aphasia (Laplane et al. 1977; Jonas 1981) which are probably related to the lack of drive and motivation. Clinical evidence of the effects of lower frontomedial lesions of the cingulate and percingulate cortex is even more scarce, although it has been suggested that they lead to memory deficits (Kornhuber 1973) and hemineglect (Heilman and Valenstein 1972).

One major problem when investigating the effects of frontal lobe lesions in humans has been the determination of the extent and nature of the lesion. This has been greatly facilitated in recent years by the computerized tomography (CT) scan, although this method shows only the extent of necrosis or removed tissue and not the extent of impaired function (Metter et al. 1981).

The present study was designed to answer the following questions:

1) Do specific deficits occur with medial frontal lobe lesions which do not occur with dorsolateral lesions?

2) Are there differential effects according to the laterality of the lesions?

3) Are there differential effects resulting from SMA and other medial frontal lobe lesions?

Orbitofrontal lesions were not included in this study, because frequently these lesions are associated with disturbances of affect, which were considered to be difficult to control when using tests of language and cognitive functions. For this reason we also excluded subjects who showed psychiatric symptoms.

Patients

Thirty-six patients with unilateral circumscribed medial or dorsolateral frontal lobe lesions were investigated. All patients were right-handed native German speakers between 30 and 74 years of age. In all cases the lesions had been verified by CT scans using a 256×256 pixel matrix. Subjects whose clinical history, neurological examination or CT suggested multiple

Table 1. Etiology of frontal lobe lesions

Etiology	All left lesions	Dorsolateral incl./excl. Broca's area		Medial incl./excl. SMA		All right lesions	Dorsolat.	Medial incl./excl. SMA		
Ischemia	12	4	2	5	1	8	3	1	4	20
Hemorrhage	0	0	0	0	0	1	0	0	1	1
Operation	4	1	1	0	2	6	3	3	0	10
Trauma	3	1	2	0	0	2	1	1	0	5
n	19	6	5	5	3	17	7	5	5	36

SMA: supplementary motor area

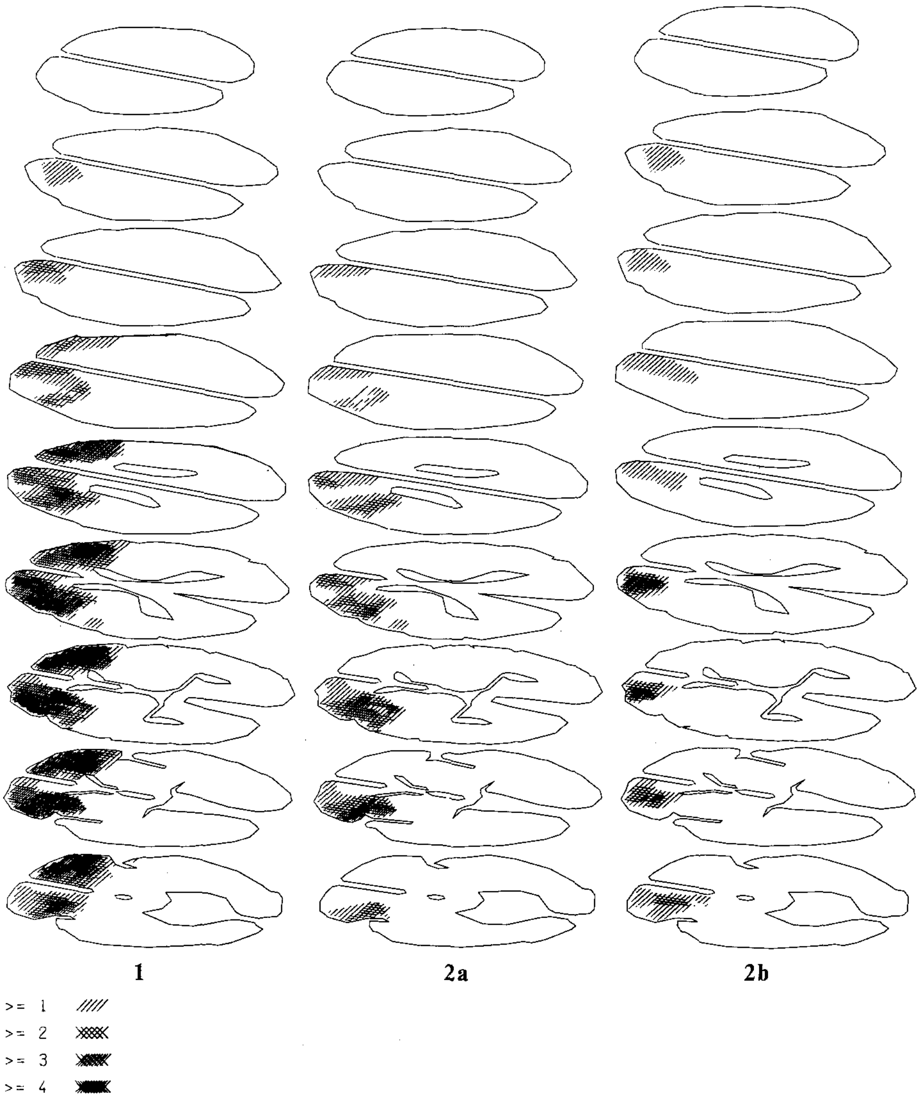

Fig. 1. Superimposed plot of 11 left and 7 right predominantly dorsolateral frontal lesions.

Fig. 2. (a) Superimposed plot of 6 dorsolateral frontal lesions including Broca's area, **(b)** Superimposed plot of 5 left dorsolateral frontal lesions not including Broca's area. Regions of overlap between **(a)** and **(b)** concerning Broca's area are due to individual differences in skull configuration

lesions were not included. Also excluded were patients with cerebral atrophy or psychiatric disorders. Of the patients 20 had suffered from ischemic infarctions, 1 from a cerebral hemorrhage, 5 from cerebral contusions and 10 had been operated on for brain or meningeal tumours (Table 1); 17 patients had a contralateral (hemi-)paresis, and 4 subjects suffered from infrequent epileptic fits. All patients were able to walk and all were alert and cooperative.

The control subjects for the neuropsychological assessment were 14 patients with retrorolandic lesions of similar size and aetiology who did not show significant aphasia.

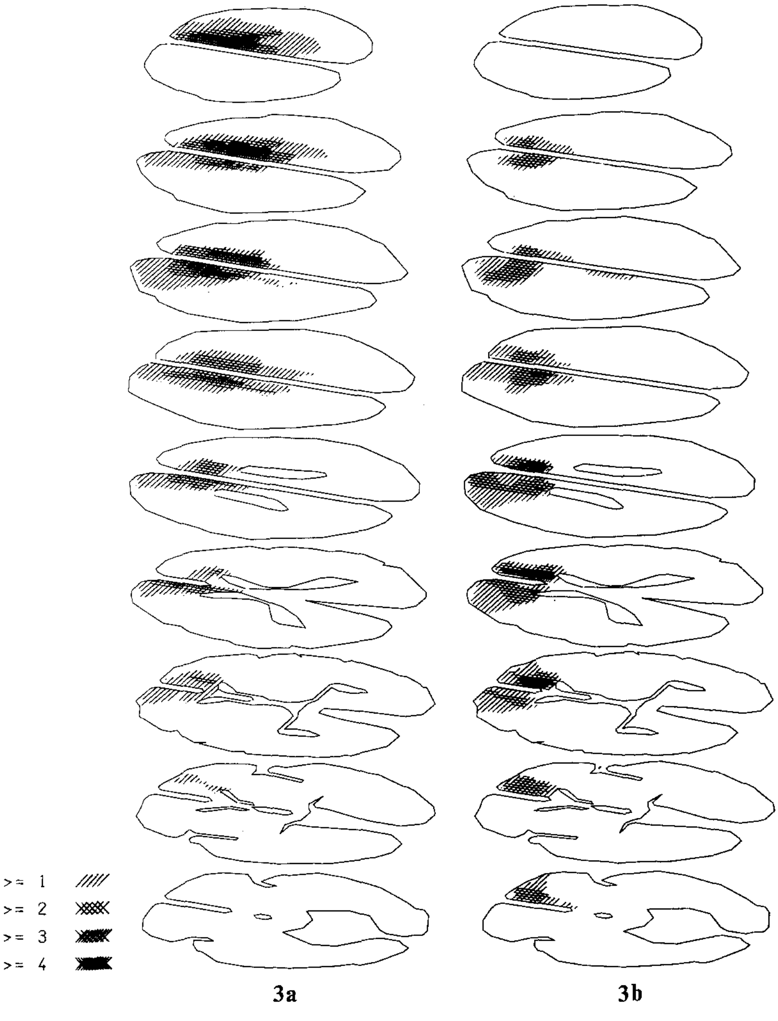

Fig. 3. (a) Superimposed plot of 5 left and 5 right frontomedial lesions including the SMA. **(b)** Superimposed plot of 3 left and 5 right frontomedial lesions not including the SMA. The location of the SMA was defined by the criteria given by Jonas (1981). Regions of overlap between **(a)** and **(b)** are due to individual differences in skull configuration

The CT scans were evaluated by the method of Blunk et al. (1981) which was extended to include high frontoparietal slices. The method requires transcription of the CT scans to standard matrices. By counting the number of standard matrix pixels included by a lesion over all slices the volume of the lesion as a percentage of the hemisphere volume between the cuts 0°–10° and 0°–2° of Matsui and Hirano (1978) can be estimated.

It was of utmost importance for this study to localize the SMA in the CT scans. We decided to apply the coordinates of Jonas (1981), which are based on the drawings of Penfield and Roberts (1959), to the intact hemisphere and construct a mirror image on the damaged.

Figures 1 to 3 show superimposed plots of the patients' lesions. Figure 3 indicates that the majority of those medial lesions which did not include the SMA were situated more deeply in the interhemispheric fissure.

Methods

The subjects were tested in two to four sessions more than 6 months after the lesions had occurred. Assessment was discontinued if any sign of fatigue occurred.

The following tests were used:

A) Language Assessment

– the Aachener Aphasia Test (Huber et al. 1983). This aphasia battery consists of the subtests spontaneous speech, which is recorded in a semistandardized interview and rated according to well defined criteria on six point scales on the following parameters: communication, articulation and prosody, automatized speech, semantic, phonemic and syntactic structure, repetition of sounds, words and sentences (50 items), naming of objects, colours and action pictures (40 items), written language (writing to dictation, "scrabble" to dictation, and reading aloud), language comprehension (40 spoken and written items), and the revised Token Test of Orgass (1976). Only correct responses were scored.

– several subtests of the Ulm Department of Neurology Test of Aphasia (UTA, in preparation, partly published in Brunner et al. 1982). This latter test assesses more specific lexical functions: superonyms (the subject has to name a common category in response to a picture of at least eight items of the category. Six stimulus cards were given), antonyms and synonyms (10 items each), usage association of word-pairs ("pepper and"...-"salt", 10 items) according to Wyke (1966), and production of words/min by phonemic association (words starting with the letter "B") and by lexical association (animal names). The last task is similar to that used by Newcombe (1969).

The spontaneous speech production of the patients was transcribed fully and analysed linguistically. The following parameters were used in this study: syllables/min, semantic paraphasias/min, phonemic paraphasias/100 syllables, errors of inflection and other grammatical errors/min.

B) Neuropsychological Assessment

– the German version of the Wechsler Adult Intelligence Scale (HAWIE, Wechsler et al. 1964)
– the Standard Progressive Matrices A-C (Raven 1956)
– the Visual Retention Test (Benton 1972)
– a pair associate learning of 10 pairs of related and unrelated words and three trials similar to that used in the Wechsler Memory Scale (Wechsler 1945).
– the Sequential Concept Formation (Talland 1965). In this test the subjects had to learn the regular color sequence of a deck of playing cards. We scored the number of correct responses in 72 trials (a score of 36 corresponds to chance level). The procedure is described in detail in Wallesch et al. (1983).
– translation of 13 sentences containing a figurative idiomatic expression, which the subject was required to put into his own words. Only correct descriptions of the hidden abstract meaning were scored.
– the Wisconsin Card Sorting (Grant and Berg 1948) with the instruction and scoring criteria of Wallesch et al. (1983).
– the Mental Folding Test from the BET (Schmale and Schmidtke 1966), a complex three dimensional visuoconstructive multiple choice task, which requires no movement of the patient.
– the Trail Making Test (Armitage 1946).

This range thus included tests of intelligence (HAWIE, Progressive Matrices), of verbal and nonverbal short-time retention (Digit Span, Benton Test), of learning within a span of minutes (pair associate learning, Sequential Concept Formation), of categorisation and abstraction (translation of idiomatic expressions, HAWIE Similarities, Wisconsin Card Sorting), and of spatial abilities (Block Design, Mental Folding, Trail Making Test). As we intended to cover an inventory of higher mental functions, the range resembles an extended version of routine neuropsychological assessment. A more specific approach was not considered adequate, as the hypotheses and findings concerning the role of the medial frontal lobes are highly divergent.

The results were analysed using the Mann and Whitney *U*-test and the Kruskal-Wallis analysis of variance (Siegel 1956). In the context of this study, these tests cannot be regarded as tests of "significance" in the strict sense as we investigated a large number of variables with limited numbers of observations. The comparisons were therefore not independent. The *P*-values given in the tables are thus only comparative measures of the relative sizes of differences.

Results

A) Language Assessment

a) Effects of Localisation (Table 2). As the frontomedial and frontodorsal lesion groups were not comparable in age, subjects of extreme ages were excluded in order to make the groups comparable. In Table 2, the laterality of the lesions is not considered, but a comparison performed between lesions including the SMA and other frontomedial and dorsolateral lesions. There were no significant differences between the groups. The results do not support the existence of qualitatively different functions between medial and lateral regions of the frontal lobes. But, as laterality was not taken into consideration, specific deficits might have been blurred by its effects.

b) Effects of Laterality in Dorsolateral Lesions (Table 3). Right and left dorsolateral lesions, the latter divided into those which included and those which did not include Broca's area, were made comparable (by excluding patients on the basis of age and lesion size). Patients with left dorsolateral lesions showed deficits in both tests of written language and in the production of synonyms. The only major difference between the performance of patients who had lesions in Broca's area and other left frontodorsal lesions occurred with the number of inflectional errors in spontaneous speech. On the whole, neither the results obtained in patients with Broca's area lesions nor with other left frontodorsal lesions showed evidence of a systemic disturbance of language. The effects of laterality on language tasks in patients with chronic lesions confined to the left dorsolateral frontal lobe appear to be rather discrete.

c) Effects of Laterality in Medial Lesions (Table 4). Left and right frontomedial lesions were not fully comparable. The right-sided lesions were larger. Patients with right SMA lesions were older and patients with other right frontomedial lesions younger than patients with left-sided lesions. The results of the language assessment were almost identical between the groups. None of the patients showed aphasic symptoms.

B) Neuropsychological Assessment

Patients with frontal lobe lesions could not be discriminated by the tests used in this study from a group of nonaphasic patients with retrorolandic lesions (Table 5).

Table 2. Assessment of language functions in frontal lobe lesions of different localization. Laterality of the lesions is not considered. The groups were made comparable for age by exclusion of patients of extreme ages. No comparison proved significant by Kruskal-Wallis analyses of variance

Localization	Dorsolateral		Frontomedial excl. SMA		Frontomedial incl. SMA	
	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD
n	8		7		7	
Age	56.6	6.3	55.0	11.3	62.7	7.5
Lesion size (%)	2.9	1.6	3.2	1.6	2.9	2.4
Left/right ratio	5/3		3/4		4/3	
AAT:						
Communication	4.9	0.4	5.0	0.0	4.9	0.4
Articulation and prosody	4.8	0.7	4.9	0.4	4.9	0.4
Semantic structure	4.5	0.5	4.7	0.5	4.6	0.7
Phonemic structure	5.0	0.0	5.0	0.0	5.0	0.0
Syntax	4.2	1.0	4.3	0.8	4.0	0.8
Visual confrontation naming (%)	96.7	4.0	96.7	5.7	98.1	3.2
Naming of sceneries (%)	90.4	3.3	81.4	15.0	87.5	13.0
Repetition (%)	95.8	7.0	98.6	2.7	99.0	2.6
Writing to dictation (%)	95.0	7.7	92.2	12.3	90.0	25.7
Speech comprehension (%)	82.5	21.0	87.5	19.0	80.7	26.5
Script comprehension (%)	90.0	9.5	86.8	17.0	76.9	23.0
Token Test, errors	0.0	0.0	0.1	0.4	0.6	1.4
UTA:						
Usage Association (1/ items)	6.5	1.4	6.4	1.9	6.9	1.7
Superonyms (6 items)	5.6	0.5	5.3	1.1	5.5	1.1
Synonyms (10 items)	6.6	2.4	7.3	2.1	7.9	1.8
Antonyms (10 items)	9.1	1.2	9.0	1.5	9.6	0.5
Verbal fluency "B"	6.8	4.6	9.4	5.9	11.1	4.2
Verbal fluency "animals"	13.9	6.8	11.1	3.6	14.0	5.0
Analysis of spontaneous speech:						
Syllables/min	173.6	39.6	193.8	20.2	173.9	43.6
Semantic paraphasias/min	0.0	0.0	0.0	0.0	0.0	0.0
Phonemic paraphasias/100 syll.	0.1	0.2	0.2	0.2	0.0	0.0
Errors of inflexion/min	4.1	6.7	5.1	3.7	4.2	2.8
Other grammatical errors/min	3.2	2.8	5.4	3.9	4.0	3.2

a) Effects of Localisation (Table 6). The patient groups were again made comparable equating their age by excluding patients of extreme ages. The dorsolateral group was found to be not balanced for laterality of the lesions. Patients with dorsolateral lesions did better than both medial groups in the Sequential Concept Formation and the Picture Arrangement. Their performance was never markedly inferior to that of patients with frontomedial lesions.

Medial lesions including the SMA led to low scores in the Block Design, Wechsler Similarities and Picture Arrangement. Lower frontomedial lesions excluding the SMA resulted in low scores in pair associate learning, Benton Test

Table 3. Assessment of language functions. Dorsolateral frontal lesions. The groups were made comparable by exclusion of subjects of extreme ages and lesion sizes. Probabilities below 0.05 (*U*-Test) are indicated by *arrows*

Localization	Right dorsolateral		All left dorsolateral		Broca's area		Other left dorsolateral	
	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD
n	7		8		4		4	
Age	45.6	13.2	46.8	11.9	49.5	14.2	44.0	10.6
Lesion size (%)	3.0	1.6	2.6	1.2	3.2	0.7	2.0	1.4
AAT:								
Communication	5.0	0.0	4.9	0.4	0.5	0.0	4.8	0.5
Articulation and prosody	4.7	0.8	4.6	0.7	4.8	0.5	4.5	1.0
Semantic structure	4.6	0.8	4.2	0.8	4.2	0.5	4.2	0.5
Phonemic structure	5.0	0.0	4.9	0.4	4.8	0.5	5.0	0.0
Syntax	4.4	1.1	4.0	0.9	4.2	0.5	3.8	0.3
Visual confront. naming (%)	97.0	3.2	97.1	3.8	95.3	4.3	98.9	2.2
Naming of sceneries (%)	93.8	5.0	92.5	5.3	91.7	6.3	93.3	4.7
Repetition (%)	98.2	3.2	96.1	6.9	97.2	2.5	95.0	10.0
Writing to dictation (%)	100.0	0.0 ←	→ 94.3	8.0	95.6	2.0	93.3	11.3
Speech comprehension (%)	96.5	5.0	79.4	22.0	72.5	27.0	86.2	18.0
Script comprehension (%)	98.6	4.0 ←	→ 88.2	9.0	85.0	7.0	91.2	11.0
Token Test, errors	0.0	0.0	0.2	0.7	0.5	1.0	0.0	0.0
UTA:								
Usage Association (10 items)	6.9	1.3	6.4	1.4	6.2	1.3	6.5	1.7
Superonyms (6 items)	6.0	0.0 ←	→ 5.5	0.5	5.5	0.6	5.5	0.6
Synonyms (10 items)	8.3	1.3	6.9	2.7	6.2	1.3	7.5	3.8
Antonyms (10 items)	9.6	0.8	8.9	1.2	8.2	1.3	9.5	1.0
Verbal fluency "B"	10.3	4.3	6.9	5.3	4.0	3.6	9.8	5.5
Verbal fluency "animals"	20.7	9.2	13.8	5.6	12.5	5.4	15.0	6.2
Analysis of spontaneous speech:								
Syllables/min	171.9	41.4	165.0	29.8	176.8	28.5	153.2	29.8
Semantic paraphasias/min	0.1	0.4	0.0	0.0	0.0	0.0	0.0	0.0
Phonemic paraphasias/100 syll.	0.0	0.0	0.2	0.3	0.3	0.3	0.1	0.2
Errors of inflexion/min	2.1	2.3	4.0	6.8	7.8	8.4	0.2	0.5
Other grammatical errors/min	3.2	2.3	3.2	2.9	2.8	1.6	3.4	3.6

Table 4. Assessment of language functions. Frontomedial lesions. No left/right comparison proved significant by the *U*-test

Localization	All frontomedial				Including SMA			
	Left		Right		Left		Right	
	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD
n	8		9		5		4[a]	
Age	61.5	6.2	57.7	14.8	61.4	8.1	69.8	5.9
Lesion size (%)	2.6	2.2	3.3	1.4	2.3	2.5	3.3	1.3
AAT:								
Communication	4.9	0.4	4.7	0.5	4.8	0.4	4.2	0.5
Articulation and prosody	4.8	0.5	4.9	0.3	4.8	0.4	4.8	0.5
Semantic structure	4.5	0.8	4.3	0.5	4.4	0.9	4.0	0.0
Phonemic structure	5.0	0.0	5.0	0.0	5.0	0.0	5.0	0.0
Syntax	4.0	0.5	4.1	0.6	3.8	0.4	4.0	0.0
Visual confrontation naming (%)	94.4	7.9	92.8	7.8	94.4	8.3	90.6	7.6
Naming of sceneries (%)	82.5	10.3	77.8	18.0	87.7	8.3	71.7	19.7
Repetition (%)	99.6	0.8	98.0	2.6	99.7	0.6	97.8	2.2
Writing to dictation (%)	78.9	39.3	76.7	31.5	73.0	30.5	87.5	15.0
Speech comprehension (%)	81.2	21.5	79.5	23.4	79.0	27.5	78.8	13.0
Script comprehension (%)	76.9	24.0	76.7	31.5	73.0	30.5	87.5	15.0
Token Test, errors	0.5	1.4	0.2	0.4	0.8	1.8	0.2	1.5
UTA:								
Usage Association (10 items)	6.6	0.9	6.9	1.8	7.0	0.7	6.5	0.6
Superonyms (6 items)	4.9	1.4	4.9	1.3	5.0	1.4	4.5	1.3
Synonyms (10 items)	6.5	2.5	7.0	1.4	6.8	2.7	6.2	0.5
Antonyms (10 items)	8.9	1.4	9.0	1.5	9.4	0.5	9.0	1.4
Verbal fluency "B"	7.0	3.7	9.0	5.1	8.2	3.6	6.5	2.5
Verbal fluency "animals"	13.1	4.9	10.2	3.3	14.8	5.4	9.5	1.0
Analysis of spontaneous speech:								
Syllables/min	170.0	45.8	180.2	37.9	153.5	49.8	155.0	35.8
Semantic paraphasias/min	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Phonemic paraphasias/100 syll.	0.1	0.2	0.0	0.1	0.0	0.0	0.0	0.0
Errors of inflexion/min	3.9	4.7	4.1	3.8	3.2	3.1	3.5	4.5
Other grammatical errors/min	5.3	4.1	4.3	2.8	4.8	3.6	2.8	2.3

[a] One subject with a right frontomedial lesion including the SMA was not completely assessed and therefore excluded.

Table 5. Neuropsychological assessment. Comparison of unilateral frontal lesions with unilateral, nonaphasic retrorolandic lesions. No difference proved significant by the *U*-test

Localization	Frontal		Retrorolandic	
	$\bar{x}$	SD	$\bar{x}$	SD
n	23		14	
Age	45.3	11.8	45.9	15.3
Left/right ratio	12/11		7/7	
Tests of intelligence:				
Wechsler Full Scale IQ	96.1	12.3	97.6	10.9
Wechsler Verbal IQ	95.0	11.0	96.1	13.7
Wechsler Performance IQ	97.5	13.8	100.4	10.9
Progressive Matrices A-C	24.3	4.6	24.9	5.0
Tests of memory functions:				
Digit Span	8.3	2.7	8.4	2.7
Benton Test, no. of correct responses	4.8	2.5	5.0	1.3
– No. of errors	8.1	4.9	7.9	2.5
Sequential Concept Formation	48.4	12.6	50.4	11.4
Tests of categorisation and abstraction:				
Similarities	9.4	1.8	8.9	2.0
Wisconsin Card Sorting				
No. of correct responses	76.0	19.2	75.8	16.3
Criteria achieved	3.8	2.2	3.8	1.6
Perseverative errors	23.4	13.6	23.8	15.8
Tests of spatial abilities:				
Block Design	8.4	2.2	9.1	2.5
Mental Folding (no. correct)	12.3	5.0	14.5	4.7
Trail Making Test (s)	55.0	27.6	57.5	23.8
Other tests of cognitive functions:				
Arithmetic	7.7	2.4	9.0	2.9
Picture Arrangement	8.6	2.7	8.9	2.8

and the Wechsler Digit Span. They performed better than both other groups in the Wisconsin Card Sorting test.

On the whole again no specific deficit could be demonstrated with dorsolateral lesions. An analysis of the tasks in which patients with SMA lesions exhibited albeit mild deficits, might suggest an underlying common dysfunction. In all of these tasks (Sequential Concept Formation, Similarities, Wisconsin Card Sorting, Block Design, and Picture Arrangement) one common aspect is involved: to construct general concepts from given pieces of information. On the other hand, patients with deeper frontomedial lesions showed deficits involving different types of memory function (pair associate learning, Digit Span, Benton Test, Sequential Concept Formation).

b) Effects of Laterality in Dorsolateral Lesions (Table 7). Except for the Wisconsin Card Sorting test, patients with left dorsolateral lesions performed worse than

Table 6. Neuropsychological assessment in frontal lobe lesions of different localization. Laterality of the lesion is not considered. The groups were made comparable for age by exclusion of patients of extreme ages. Kruskal-Wallis analysis of variance

Localization	Dorsolateral		Frontomedial excl. SMA incl. SMA		Frontomedial		P
	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD	
n	12		7		8		
Age	56.8	7.7	55.0	11.3	61.9	7.4	
Lesion size (%)	2.6	1.5	3.2	1.6	2.7	2.3	
Left/right ratio	8/4		3/4		4/4		
Tests of intelligence:							
Wechsler Full Scale IQ	97.4	8.9	93.6	9.1	94.0	13.3	
Wechsler Verbal IQ	96.3	8.8	93.0	7.9	98.8	10.2	
Wechsler Performance IQ	98.8	10.6	93.7	11.4	88.4	15.2	
Progressive Matrices A-C	23.2	3.7	19.6	5.0	20.9	5.5	
Tests of memory functions:							
Pair Associate Learning	11.0	4.5	9.4	6.3	14.1	4.9	
Digit Span	7.8	2.2	7.1	2.5	8.9	2.1	
Benton Test, no. of correct responses	4.5	1.8	3.7	2.2	4.4	2.7	
– No. of errors	8.0	3.6	11.1	4.1	10.1	6.2	
Sequential Concept Formation	50.8	11.7	43.4	11.7	45.2	11.8	<0.05
Tests of categorisation and abstraction:							
Translation of idiom. express.	8.5	2.9	8.0	2.4	10.4	1.7	
Similarities	8.8	1.9	8.9	2.3	7.6	2.3	
Wisconsin Card Sorting							
No. of correct responses	66.6	20.2	68.6	18.5	68.1	21.8	
Criteria achieved	2.8	2.1	3.6	1.1	2.8	2.2	
Perseverative errors	31.8	14.8	26.3	21.5	27.9	17.2	
Tests of spatial abilities:							
Block Design	8.8	1.8	8.1	2.1	5.4	2.9	<0.05
Mental Folding (no. correct)	11.5	4.6	13.1	3.6	11.9	6.3	
Trail Making Test (s)	54.7	20.7	58.6	20.1	64.8	25.1	
Other tests of cognitive functions:							
Arithmetic	7.2	1.8	6.7	2.1	7.9	2.7	
Picture Arrangement	8.8	2.2	7.4	2.3	6.8	3.4	

Table 7. Neuropsychological assessment. Dorsolateral frontal lesions. Probabilities below 0.05 (*U*-test) are indicated by *arrows*

Localization	Left		Right	
	$\bar{x}$	SD	$\bar{x}$	SD
n	11		7	
Age	50.9	12.6	45.6	13.4
Lesion size (%)	2.4	1.2	2.9	1.4
Tests of intelligence:				
Wechsler Full Scale IQ	94.0	9.2	99.0	10.8
Wechsler Verbal IQ	93.5	10.2	95.5	7.9
Wechsler Performance IQ	95.2	9.6	102.4	14.0
Progressive Matrices A–C	23.2	3.8	25.9	4.2
Tests of memory function:				
Pair Associate Learning	11.9	5.6	15.4	4.0
Digit Span	7.4	1.9	8.6	2.2
Benton Test, no. of correct responses	4.5	2.3	5.2	1.0
- No. of errors	8.3	4.2	6.5	1.9
Sequential Concept Formation	50.0	11.9	52.8	12.8
Tests of categorization and abstraction:				
Translation of idiomatic expressions	7.0	3.0 ←	→ 11.0	1.4
Similarities	8.2	1.9 ←	→ 10.1	0.8
Wisconsin Card Sorting				
No. of correct responses	74.4	18.3	71.4	22.4
Criteria achieved	4.1	1.9	3.0	2.6
Perseverative errors	27.2	12.4	23.8	16.9
Tests of spatial abilities:				
Block Design	8.3	1.2	9.2	2.3
Mental Folding (no. correct)	11.3	5.0	13.0	5.6
Trail Making Test (s)	60.1	17.6	42.2	17.1
Other tests of cognitive functions:				
Arithmetic	7.5	2.2	7.5	1.8
Picture Arrangement	8.5	2.8	9.2	2.5

patients with right-sided lesions, even when their scores were corrected for age (in the Wechsler scale IQs). The differences in two verbal tests of abstraction (Similarities and translation of idiomatic expressions) reached the 5% level. Thus, similar to the language assessment, the neuropsychological tests give some evidence of a lateralisation of the deficits with dorsolateral lesions, but, as in the language tests, the degree of lateralisation appears to be rather small.

c) Effects of Laterality in Medial Lesions (Table 8). Patients with right frontomedial lesions did worse than those with left lesions in the Block Design and in the Picture Arrangement. The differences however were not statistically significant. In the other tests only unremarkable differences occurred. If those lesions which involved the SMA were considered separately, patients with right-sided lesions did worse in all tests, but they were an older group of subjects.

Table 8. Neuropsychological assessment. Frontomedial lesions. Probabilities below 0.05 (U-test) are indicated by *arrows*

Localization	All frontomedial				Including SMA			
	Left		Right		Left		Right	
	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD
n	8		9[a]		5		5	
Age	61.5	6.2	59.4	13.3	61.4	8.1	67.0	8.0
Lesion size (%)	2.6	2.2	3.1	1.6	2.6	2.2	2.9	1.6
Tests of intelligence:								
Wechsler Full Scale IQ	94.9	11.6	91.1	10.3	95.5	13.9	89.2	10.7
Wechsler Verbal IQ	96.6	9.8	96.1	8.6	100.6	10.4	96.8	8.4
Wechsler Performance IQ	92.2	14.4	85.7	13.5	89.6	15.9	81.0	14.3
Progressive Matrices A–C	18.8	5.3	20.1	5.5	20.4	6.0	18.4	6.0
Tests of memory functions:								
Pair Associate Learning	9.2	7.2	9.1	6.1	12.6	6.9	6.0	5.4
Digit Span	8.2	2.3	7.7	2.4	9.2	2.2	7.8	1.8
Benton Test, no. of correct responses	4.0	2.9	3.6	2.1	4.4	3.6	3.2	1.3
– No. of errors	10.5	6.8	12.4	4.7	10.4	8.8	13.2	3.8
Sequential Concept Formation	43.6	11.7	45.2	10.4	46.6	14.2	43.8	6.8
Tests of categorization and abstraction:								
Translation of idiom. express.	8.9	2.7	8.0	2.7	10.6	1.7 ←	→ 6.5	3.1
Similarities	7.9	1.9	8.2	2.7	8.2	1.9	6.8	2.6
Wisconsin Card Sorting								
No. of correct responses	69.0	20.4	68.1	17.8	72.4	25.2	64.6	13.0
Criteria achieved	3.2	2.0	2.9	1.5	3.4	2.6	2.0	0.7
Perseverative errors	25.4	21.2	28.8	15.8	24.2	21.4	31.2	10.8
Tests of spatial abilities:								
Block Design	6.9	2.8	5.6	3.2	5.8	2.7	3.8	2.9
Mental Folding (no. correct)	13.0	4.4	12.0	5.9	13.0	5.8	10.3	8.0
Trail Making Test (s)	59.5	11.3	63.1	28.9	53.3	11.5	76.3	32.2
Other tests of cognitive functions:								
Arithmetic	7.5	3.3	7.3	2.3	8.6	3.6	7.2	2.8
Picture Arrangement	8.0	3.0	6.2	2.6	8.2	3.8	5.2	2.5

[a] One subject with a right frontomedial lesion not including the SMA was not completely assessed and therefore excluded.

Discussion

This study produced mostly negative results. Several arguments can be put forward to explain our failure to demonstrate clear cut deficits:

1) It is a well established fact that even patients with marked frontal pathology may perform well in intelligence tests like the Wechsler scale (Damasio 1979). Tests which are widely considered to be specific for cognitive impairments due to frontal lobe lesions may also not always be appropriate.

2) The lesions were unilateral and chronic, thus allowing for functional compensation to occur (Butters et al. 1974; Damasio 1979).

3) The dorsolateral frontal lobe with the exception of Broca's area was treated in this study as if it was a functionally homogenous structure, which is certainly not the case. Therefore, effects of damage to functional subunits could have escaped detection by the grouping of the subjects.

4) Patients with obvious changes of personality, affect and emotion were excluded. Thus one important aspect of the "frontal lobe syndrome" (for critical discussion of that term, see Damasio 1979) has not been investigated.

The lateralisation of language functions as well as deficts in tasks requiring abstraction and categorisation are supposed to be well established facts with dorsolateral frontal lesions (for a review, see Hecaen and Albert 1975). The results of our study emphasize the ability of the frontal lobes to compensate the effects of unilateral lesions in a chronic stage.

To claim a few "significant" results among many negative ones poses problems of interpretation. If the data of this study are considered in their context, three main observations emerge: (1) There is some degree of lateralisation for language deficits with dorsolateral lesions. (2) The medial lesions classified as non-SMA lead to deficits of memory and learning. (3) SMA lesions resulted in mild impairments in tests of concept formation. On the other hand, no clear-cut observations can be made concerning the role of the medial frontal cortex in language functions.

Language disorders resulting from lesions of the SMA have frequently been reported (for a review, see Jonas 1981). The rapid recovery from the effects of unilateral SMA lesions has been explained on the basis of a complete take over of functions by the contralateral SMA (Eccles 1982). The two SMAs probably work in cooperation in tasks requiring initiation and organisation of intentional acts. The bilateral projection of the SMA to the neostriatum might constitute the anatomical substrate (Wiesendanger et al. 1973). The basal ganglia, of which the neostriatum is an integral part, are supposed to be crucial structures for the generation of complex programs (Kornhuber 1974, 1977) and have been shown to participate in language functions (Kornhuber et al. 1979; Brunner et al. 1982). The mild impairments sometimes seen in cases of bilateral lesions of the SMA (one of our patients complete a teacher's training after a bilateral SMA lesion due to a shell fragment injury) raise doubts on whether Eccles (1982) hypothesis is tenable. Kornhuber (1980a), on the other hand, considers the juxtalimbic supplementary area to be one of a number of channels from the substrates of drives, will and planning towards action. A model of a multiple representation of functions (Kornhuber 1980b) would explain a lack of severe deficits even with bilateral SMA lesions.

In this study we found no convincing evidence for a lateralisation of the deficits resulting from frontomedial lesions in the chronic stage. Our findings rather support equipotentiality. The deficits found with medial lesions of either hemisphere demonstrate that there is no complete take over of functions.

On the basis of the deficit in tests requiring concept formation exhibited by patients with SMA lesions, it is speculated but requires further confirmation that nonmotor aspects of planned and goal-directed intelligent behaviour are also affected by such a lesion.

The participation of parts of the frontomedial cortex in memory functions was further confirmed by the results of our study. Neither effects of laterality nor specific deficits of single functions of memory and learning emerged. The deficits appear to be supramodal and, as far as can be judged from the tests used, independent of the time structure of the task. The deficits could be explained by a theory advanced by Kornhuber (1973), which suggests that the act of memorizing information relevant by the subject's intention depends on the frontomedial cortex' participation in the limbic system and its connections with the memory loop via anterior thalamic nuclei. Supporting this theory are the findings of similar impairments with rostral thalamic lesions (Wallesch et al. 1983).

As already reported in a number of other studies, superficial lesions of Broca's area lead to lasting aphasia only in exceptional cases (Mohr et al. 1978; Kornhuber et al. 1979; Brunner et al. 1982). On the other hand, a participation of Broca's area in language activities can be demonstrated by regional cerebral blood flow (Ingvar and Schwartz 1974). Tonkonogy and Goodglass (1981) pointed out that the extent of a Broca's area lesion is an important factor in the effect of a permanent aphasia. Brunner et al. (1982) proposed a compensation of language deficits resulting from Broca's area, basal ganglia and SMA lesions among these structures. Our findings are similarly in favour of a high degree of functional plasticity within the frontal lobes.

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