Neurobehavioural disturbances, rehabilitation outcome, and lesion site in patients after rupture and repair of anterior communicating artery aneurysm

Stefanie Böttger, Mario Prosiegel, Hans-Jakob Steiger, Alexander Yassouridis

Abstract

***Objective*—To determine: (1) patterns of cognitive and psychiatric dysfunction; (2) those neurobehavioural parameters which mostly influence disability in activities of daily living (ADL) and handicap in occupational and psychosocial activities, and (3) underlying neuroanatomical pathology in patients after rupture and repair of anterior communicating artery (ACoA) aneurysm.**

***Methods*—30 patients were extensively examined by means of a comprehensive battery of neuropsychological tests, by rating of psychopathological symptoms, and by use of the functional independence measure (FIM), Glasgow outcome scale (GOS), and MRI.**

***Results and conclusions*—(1) Three main groups were characterised by primary impairment of memory, executive functions, or of attentional performance. Within these main groups, specific patterns were identified relating to extent of primary dysfunction and associated disorders. The variety of neuropsychological disturbances is in contradiction to the existence of an "ACoA syndrome" as an entity. (2) Rehabilitation outcome proved to be mostly associated with both memory and attentional performance. (3) In neuropathological terms, lesions of the medial septum and nucleus of the diagonal band of Broca (MS/ndbB) were closely associated with memory deficits and prefrontal lesions were associated with attentional, executive, and psychopathological dysfunctions. At the same time, bilateral lesions were associated with severe disturbances. The type and severity of the above mentioned deficits were independent of the side of lesion in unilateral cases, of rectus gyrus resection, and of the Hunt and Hess grading system.**

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Keywords: anterior communicating artery (ACoA) aneurysm; neurobehavioural disturbances; rehabilitation outcome

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After rupture and repair of anterior communicating artery (ACoA) aneurysms patients In the past were often reported as having severe cognitive deficits and psychopathological symptoms which remained permanent in many cases. In the meantime reports accumulated about only mild to moderate deficits and good recovery in many patients. This change was interpreted as a consequence of improved preoperative, intraoperative, and postoperative management in modern neurosurgery.[1]

Accordingly, studies carried out on patients with ACoA published hitherto can be divided into two categories. In earlier studies a chronic mental disorder was described showing the main symptoms of severe amnesia, personality changes, and confabulation. The deficits were compared with the Wernicke-Korsakoff syndrome and they were given the term "ACoA syndrome".[2–6]

Recent studies no longer characterised the "ACoA syndrome" exclusively as a global amnesic syndrome. They differ in three respects from the earlier studies: Firstly, a differentiation was made between a mild and severe degree of memory deficit and personality change.[7] Secondly, studies aimed to achieve a qualitative in depth analysis of memory deficits. In this context one special subject of interest in patients with ACoA was the learning of contextual information.[8 9] In particular, the linking together of target information with spatial location or time tag was found to be severely impaired in patients with memory disorders. Another special subject of interest in patients with AcoA was their sensitivity to proactive interference when learning two or more sets of information.[10] Another focus of interest was the preservation of priming and skill learning.[11] Thirdly, other cognitive impairments were also considered, especially frontal dysfunctions, mainly in the form of concept formation deficiency and perseverative responding.[12–14]

It is necessary to emphasise that an assessment of the total range of attentional processes has not been adequately performed to date. General screening of concentration disclosed either normal attentional performance or deficiency of complex attentional processes.[1 3 7 14 15] Furthermore, rehabilitation outcome of patients with ACoA has not been studied at all.

In the present study, therefore, we looked for connections between neuropsychological test results and the underlying neuroanatomical pathology on the one hand and outcome in terms of activities of daily living as well as of occupational and psychosocial activities on the other. In doing so, the levels of impairment, disability, and handicap, as defined by the

Table 1 Characteristics of sample (n=30)

Case No	Sex	Age at onset	SAH grade	Site of lesion		Resection of rectus gyrus	Months after injury	
				MS/ndbB	Prefrontal		Cognitive testing	Outcome rating
1	F	65	IV	Bil	Bil	Y	8	20
2	F	66	V	R	Bil	N	8	38
3	F	68	III	R	R	N	5	15
4	F	68	III	R	–	Y	4	5
5	F	37	IV	R	R	Y	3	5
6	F	62	II	R	R	N	7	19
7	F	49	IV	R	–	Y	10	24
8	F	48	V	L	Bil	N	5	19
9	F	52	III	L	R	Y	5	29
10	M	72	II	L	L	Y	2	9
11	F	47	IV	L	L	N	4	7
12	F	60	III	Bil	L	N	9	10
13	F	56	IV	L	–	Y	3	5
14	M	59	II	R	R	N	5	16
15	M	52	III	–	L	N	5	30
16	M	38	II	L	R	N	6	18
17	F	61	IV	L	L	Y	3	25
18	M	55	I	L	L	Y	5	7
19	M	37	II	L	Bil	Y	4	6
20	F	60	II	–	–	Y	8	28
21	M	66	II	–	–	Y	5	10
22	F	46	II	R	R	Y	2	15
23	F	39	IV	L	–	N	4	7
24	M	39	IV	L	–	N	2	5
25	M	59	II	Bil	–	N	13	13
26	F	68	IV	R	–	Y	2	6
27	M	54	I	R	–	Y	3	7
28	M	40	II	–	–	Y	2	5
29	F	55	II	–	–	Y	2	5
30	M	51	IV	L	Bil	N	8	10

L=left sided lesion; R=right sided lesion; Bil=bilateral lesion; −=no detectable lesion; Y=rectus gyrus resection; N=no rectus gyrus resection; SAH=subarachnoid haemorrhage.

World Health Organisation, were taken into consideration.[16 17]

Patients and methods

Thirty patients after rupture and repair of ACoA aneurysm were included in the study. On admission, standard neuropsychological testing disclosed neurobehavioural deficits of different quality and degree. Therapy was stopped when neurobehavioural dysfunction showed no further improvement. Comprehensive neuropsychological testing took place shortly before discharge.

The mean age of the patients (18 women and 12 men) was 54.3 (range: 37 to 72)years. The severity of subarachnoid haemorrhage was rated using the Hunt and Hess scale.[18] Aneurysms were normally clipped via a right craniotomy. A left sided approach was used only when the aneurysmal neck and the feeding arteries were more easily accessible from the left. Resection of part of the rectus gyrus was performed in 17 patients with high positioned aneurysms. Postsurgery MRI showed whether patients had lesions in the so called septohippocampal system, especially in the medial septum/nucleus of the diagonal band of Broca (MS/ndbB) complex. The lesion extent of each patient was transferred to the appropriate reference section of the Talairach and Tournoux atlas corresponding to the area immediately anterior to the optic chiasma containing the septum pellucidum, septal nuclei, diagonal band nuclei, area subcallosa, diagonal band of Broca, nucleus accumbens, putamen, and head of the caudate nucleus.[19 20] One of the limitations in terms of anatomical assessment was that the clips produced artefacts so the lesions could only be established with a high degree of probability but not with absolute certainty.

The neuropsychological study was conducted in two phases. The appendices contain detailed information on the comprehensive test battery used to assess the neurobehavioural impairments at discharge two to 13 months after surgery (mean duration: 5.2 months). In addition, psychopathological symptoms such as confabulation, anosognosia, apathy, and affective disorder were rated on the basis of therapists' findings. The second part of the neuropsychological study was conducted between five and 38 months after surgery (mean duration 13.9 months). Patients and their relatives were contacted by phone. The functional independence measure (FIM) was used for rating the outcome in terms of activities of daily living and the Glasgow outcome scale (GOS) was used for rating the outcome with regard to occupational and psychosocial activities.[48 49]

Table 1 shows sample characteristics (sex, age at onset, grade of subarachnoid haemorrhage, lesion site, and resection of rectus gyrus, as well as time interval between brain lesions and cognitive testing and brain lesions and outcome rating).

DATA ANALYSES

The sample size (n=30) was small relative to the number of test variables. Therefore, to avoid singularities and biases in the parameter estimations statistical analysis was performed with only five summary indices for the cognitive domains of orientation, memory, learning, attention, and executive functions and four summary indices for the psychopathological symptoms. Both these indices were defined as

follows: Firstly, each single test result was rated as: (0) normal, (1) mildly impaired (scores are below the 25th percentile), (2) moderately impaired (scores are below the 10th percentile), and (3) severely impaired (scores are below the 5th percentile or the test could not be performed because of too serious impairment). Secondly, within each cognitive domain the mean of these rating scores over all corresponding tests was computed as the summary index. Data were subsequently transformed into z-scores to approach normal distribution and homogeneity in the data sample.

Agglomerative hierarchical cluster analysis was performed to identify patient groups with specific patterns of cognitive and psychopathological disorders. The squared Euclidean distance and the method of average linkage between groups were used to decide which cases or clusters should be combined at each step.[50 51]

Multiple regression analysis was performed to determine whether a linear association exists between rehabilitation outcome and the aforementioned summary indices and, if so, to what extent cognitive, psychological, and neuroanatomical pathology might influence the rehabilitation outcome as measured by the GOS. The association between the ordinal scaled variables of the FIM and GOS scales was tested for significance with the Spearman rank correlation coefficient.

Contingency tables with Pearson χ^2 and Somers' d statistics were used to analyse the association between neuropsychological and psychopathological disturbances on the one hand and neuropathological findings expressed as categorical data on the other (for Pearson χ^2 statistics MS/ndbB and prefrontal lesions: 0=no lesion, 1=left-sided, 2=right-sided and 3=bilateral lesion; rectus gyrus: 0=no resection, 1=resection; Hunt and Hess: graded from 1 to 5; for Somers' d statistics MS/ndbB and prefrontal lesions were recoded into three classes: 0=no lesion, 1/2=lesion on one side only, 3=bilateral lesion). The Somers' d statistic is a measure of association for a contingency table of ordinal variables.[52 53] It is appropriate for the asymmetric case in which one variable is considered to be dependent and the other the predictor or explanatory variable. For tables larger than 3×3 and/or with low (n<5) absolute frequencies in the cells, the p values of the χ^2 statistics were calculated by Monte Carlo simulations. Exact p values are in such cases difficult to compute, whereas the asymptotic p values carry no probabilistic guarantee whatsoever as to their accuracy. Monte Carlo simulations are available within the heading "exact tests" of SPSS/PC for WINDOWS.

For inferential statistics an α=0.05 was accepted as a nominal level of significance. To keep the type I error<0.05 all tests were performed at a reduced level of significance (adjusted α according to the Bonferroni procedure).

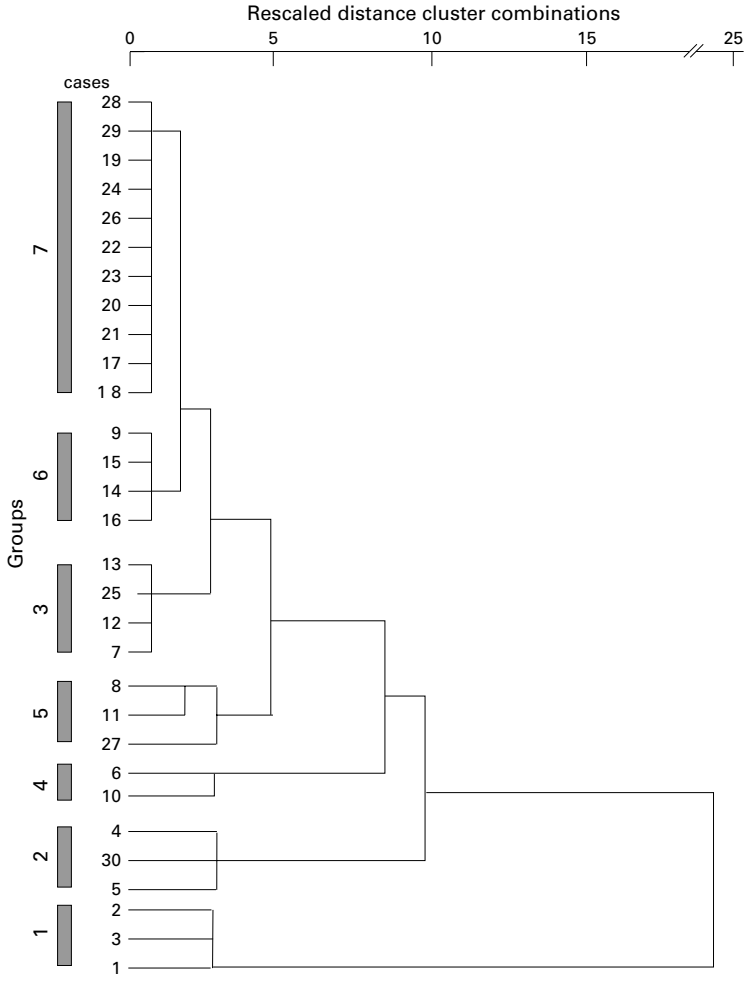

Figure 1 Dendrogram of cluster analysis using average linkage between groups. Vertical lines denote joined clusters whereas their corresponding positions on the distance scale at the top indicate the distances at which clusters were joined.

Results

The degree of impairment in memory, learning, attention, and executive functions ranged from severe, moderate, or slight dysfunctions in some patients with ACoA to nearly normal ability in others. The detailed frequency distributions for each test are presented in the appendices.

Appendices 1 and 2 show that the following areas were particularly poor within the domain of memory and learning: the processes of encoding information (that is, working memory for complex material (appendix 1, test 5)), short term recall of non-verbal material without convenient context (appendix 1, test 9) as well as learning sets of discrete verbal information (appendix 2, tests 3 and 4). Memory spans (appendix 1, tests 2 and 3) and remote and implicit memory (appendix 1, tests 11, 12, and 13) were normal in most patients.

Appendix 3 indicates that only few patients showed no impairment in each test examining attentional processes. If there were deficits in selective attention (appendix 3, tests 2 and 3), they occurred in most cases in the form of increased interference. Information processing speed in the case of complex requirements (appendix 3, test 1), divided attention (appendix 3, tests 5 and 6) as well as sustained attention (appendix 3, test 8) were often reduced.

Appendix 3 also shows, that within the domain of executive functions, about half of

Table 2 Patterns of cognitive and psychopathological dysfunctions at discharge; functional, occupational, and psychosocial outcome at least five months after injury

Case No	*Orientation*	*Attention*	*Memory*	*Learning*	*Planning*	*Confabulation*	*Anosognosia*	*Apathy*	*Affect*	*FIM*	*GOS*
Group 7:											
28	–	*	–	–	–	–	–	–	–	126	5
29	–	*	*	–	–	–	–	–	–	124	4
19	–	*	–	–	–	–	–	–	–	125	5
24	–	–	–	–	–	–	–	–	–	126	5
26	–	*	–	–	–	–	–	–	–	126	5
22	–	*	–	–	–	–	–	–	–	125	5
23	–	–	–	–	–	–	–	–	–	124	5
20	–	*	–	–	–	–	–	–	–	125	5
21	–	–	–	–	–	–	–	–	–	126	5
17	–	*	–	–	*	–	–	–	–	125	4
18	–	*	–	–	*	–	–	–	–	125	4
Group 6:											
9	–	**	–	*	*	–	–	–	–	124	4
15	–	**	–	*	–	–	–	–	–	125	4
14	–	**	–	*	–	–	–	–	–	125	4
16	–	**	–	–	*	–	–	–	–	125	4
Group 3:											
13	–	*	*	**	–	–	–	–	–	123	4
25	–	*	*	**	–	–	–	–	–	124	4
12	–	*	**	**	*	–	–	–	–	118	4
7	–	*	**	**	*	–	–	–	–	116	4
Group 5:											
8	–	**	–	**	**	–	–	*	*	124	4
11	–	*	–	*	*	–	–	*	*	124	5
27	–	**	*	*	–	–	–	*	*	124	4
Group 4:											
6	–	***	*	**	***	–	–	***	–	103	4
10	–	**	*	*	**	–	–	**	–	123	4
Group 2:											
4	**	**	***	***	**	–	–	–	**	114	3
30	*	**	**	**	**	*	–	*	*	104	3
5	*	**	**	**	**	–	–	–	**	119	3
Group 1:											
3	***	**	***	***	***	***	**	–	*	114	3
2	***	***	***	***	***	***	***	–	–	103	3
1	***	***	***	***	***	***	***	***	–	32	3

***severe impairment; **moderate impairment; *slight impairment; −=no impairment; FIM=functional independence measure; GOS=Glasgow outcome scale.

the patients had a reduction of cognitive flexibility (appendix 3, test 10). In addition, they showed an inadequate overview of relevant conditions, a loss of concept formation, and a need for more time to finish the planning task and the Tower of Hanoi puzzle (appendix 3, tests 11 and 12), as a result of attentional and working memory dysfunctions. Verbal fluency (appendix 3, test 13) was diminished in many patients, whereas intelligence (appendix 3, tests 14 and 15) was spared in most.

Figure 1 shows the dendrogram of a cluster analysis of all 30 patients using the five summary indices for orientation, memory, learning, attention, and executive functions as well as the four rating indices for the psychopathological symptoms (confabulation, anosognosia, apathy, and affective disorder). If point 4 on the distance scale of the dendrogram is used as a cut off point then five groups with specific patterns of cognitive and psychopathological dysfunctions can be differentiated. One of these can further be divided into three subgroups with specific patterns, so that altogether seven groups with characteristic patterns can be established.

Table 2 provides a summary of these seven patterns. Groups 1, 2, and 3 form a main pattern of primary memory impairment. In the first group, patients 1–3 were disoriented in nearly all modalities. Furthermore, they could not remember or learn anything. Therefore, attentional and problem solving processes could not be investigated by tests. All these patients showed a striking tendency towards confabulation and were characterised by severe anosognosia. Patients 4, 5, and 30 in the second group differed from the first group in so far as their orientation deficits were ascribed to memory disturbances and because they were able to learn and to remember several new items of information. These patients were aware of their cognitive dysfunctions and had affective disorders such as depression, anxiety, and low self confidence. Patients in the third group (7, 12, 13, 25) were fully oriented in all modalities. They were characterised by slight to moderate memory and learning dysfunctions. As a consequence of these disturbances, problem solving and attentional processes were reduced slightly. In these patients no psychopathological symptoms were found.

Groups 4 and 5 form a main pattern of primary impairment of executive and attentional functions. The patients in the fourth group (6, 10) were characterised by moderate to severe apathy, moderate to severe attentional and problem solving impairment, and only slight impairment in memory. The patients in the fifth group (8, 11, 27) showed similar disturbances, but to a lesser degree.

Groups 6 and 7 form a main pattern of primary attention impairment. In the sixth group (patients 9, 14–16) the main symptom was a moderate attentional dysfunction. As a result of this disturbance learning or problem solving processes were reduced to a slight degree. In the seventh group the main symptom was a slight attentional deficit (patients 17–20, 22, 26, 28, 29), mostly without dysfunctions in other cognitive domains.

Cognitive and psychopathological indicators	MS/ndbB	p Value	Prefrontal	p Value	Rectus gyrus	p Value	Hunt and Hess	p Value
Orientation	0: 5, 11, 6, 2 1: 1 2: 2 3: 2, 1	NS	0: 12, 5, 5, 2 1: 1 2: 1 3: 1, 2	NS	0: 10, 14 1: 1 2: 2 3: 2, 1	NS	1: 2, 11, 3, 7, 1 2: 1 3: 1, 1 4: 1, 1 5: 1	NS
Attention	0: 1: 4, 7, 3, 2 2: 1, 5, 5 3: 2, 1	NS	0: 1: 10, 4, 1, 1 2: 3, 1, 5, 2 3: 1, 2	*	0: 1 1: 5, 10 2: 6, 5 3: 2, 1	NS	1: 2: 1, 7, 1, 7 3: 1, 3, 4, 2, 1 4: 1, 1 5:	NS
Memory	0: 5, 9, 3 1: 2, 2, 2 2: 1, 2 3: 3, 1	*	0: 9, 4, 2, 2 1: 2, 1, 3 2: 1, 1 3: 1, 2	NS	0: 6, 10 1: 4, 3 2: 1, 2 3: 2, 2	NS	1: 2, 8, 1, 5, 1 2: 3, 2, 1 3: 3 4: 2, 1, 1 5:	NS
Learning	0: 4, 6, 3 1: 1, 3, 1 2: 3, 4, 2 3: 2, 1	NS	0: 7, 2, 3, 1 1: 2, 2, 1 2: 4, 1, 2, 2 3: 1, 2	NS	0: 4, 9 1: 2, 3 2: 5, 4 3: 2, 1	NS	1: 1, 8, 4 2: 1, 1, 2, 1 3: 2, 2, 4, 1 4: 1, 1 5:	NS
Executive function	0: 5, 4, 4, 2 1: 4, 1 2: 4, 2 3: 3, 1	NS	0: 11, 1, 2, 1 1: 3, 1 2: 1, 1, 2, 2 3: 2, 2	*	0: 6, 9 1: 1, 4 2: 3, 3 3: 3, 1	NS	1: 1, 8, 2, 4 2: 1, 3 3: 2, 1, 2, 1 4: 1, 1, 1, 1 5:	NS
Confabulation	0: 5, 11, 8, 2 1: 1 2: 3: 2, 1	NS	0: 13, 5, 6, 2 1: 1 2: 3: 1, 2	*	0: 10, 16 1: 1 2: 2, 1	NS	1: 2, 11, 4, 8, 1 2: 1 3: 1, 1, 1 4: 5:	NS
Anosognosia	0: 5, 12, 8, 2 1: 2: 1 3: 1, 1	NS	0: 13, 5, 6, 3 1: 1 2: 2 3:	*	0: 11, 16 1: 1 2: 1, 1 3:	NS	1: 2, 11, 4, 9, 1 2: 1 3: 1, 1 4: 5:	NS
Apathy	0: 5, 8, 8, 2 1: 3, 1 2: 1 3: 1, 1	NS	0: 13, 3, 5, 2 1: 1, 1, 2 2: 1 3: 1, 1	NS	0: 8, 15 1: 4 2: 1 3: 1, 1	NS	1: 2, 8, 5, 7, 1 2: 1, 2, 1 3: 1 4: 1 5:	NS
Affect	0: 5, 9, 6, 3 1: 3, 3 2: 1 3:	NS	0: 11, 4, 5, 3 1: 2, 1, 1, 2 2: 1 3:	NS	0: 9, 14 1: 4, 2 2: 1 3:	NS	1: 1, 11, 3, 7, 1 2: 1, 2, 2, 1 3: 1 4: 5:	NS

* $p < 0.05$.

Figure 2 Frequency distributions of the cognitive and psychopathological indices within the various categories of the variables MS/ndbB and prefrontal lesions, rectus gyrus resection, and Hunt and Hess grading system. The results of the corresponding tests of independence performed with the Pearson χ^2 statistic are also included.

Table 2 also displays the scores obtained by each patient using the FIM and the GOS. Spearman rank correlation between the GOS and FIM scores was highly significant ($r=0.80$; $p<0.05$). The GOS demonstrated "severe handicap" (score 3) for patients with severe and moderate memory disorders, "moderate handicap" (score 4) for patients with slight memory disorders, with executive dysfunctions and with moderate attentional disturbances, and "good recovery" for patients with only slight attentional disturbances.

Based on the transformed data a multiple regression analysis showed that memory (β coefficient=−0.508, $p<0.05$) and attentional processes (β coefficient=−0.459, $p<0.05$) were good predictors for outcome as measured by the GOS, whereas executive dysfunctions, psychopathology, and neuropathology were not able to predict the outcome. The amount of variance explained by the regression model was about 75%.

Figure 2 presents a summary of associations between neuroanatomical and neurobehavioural variables. There was no significant association between the Hunt and Hess grading

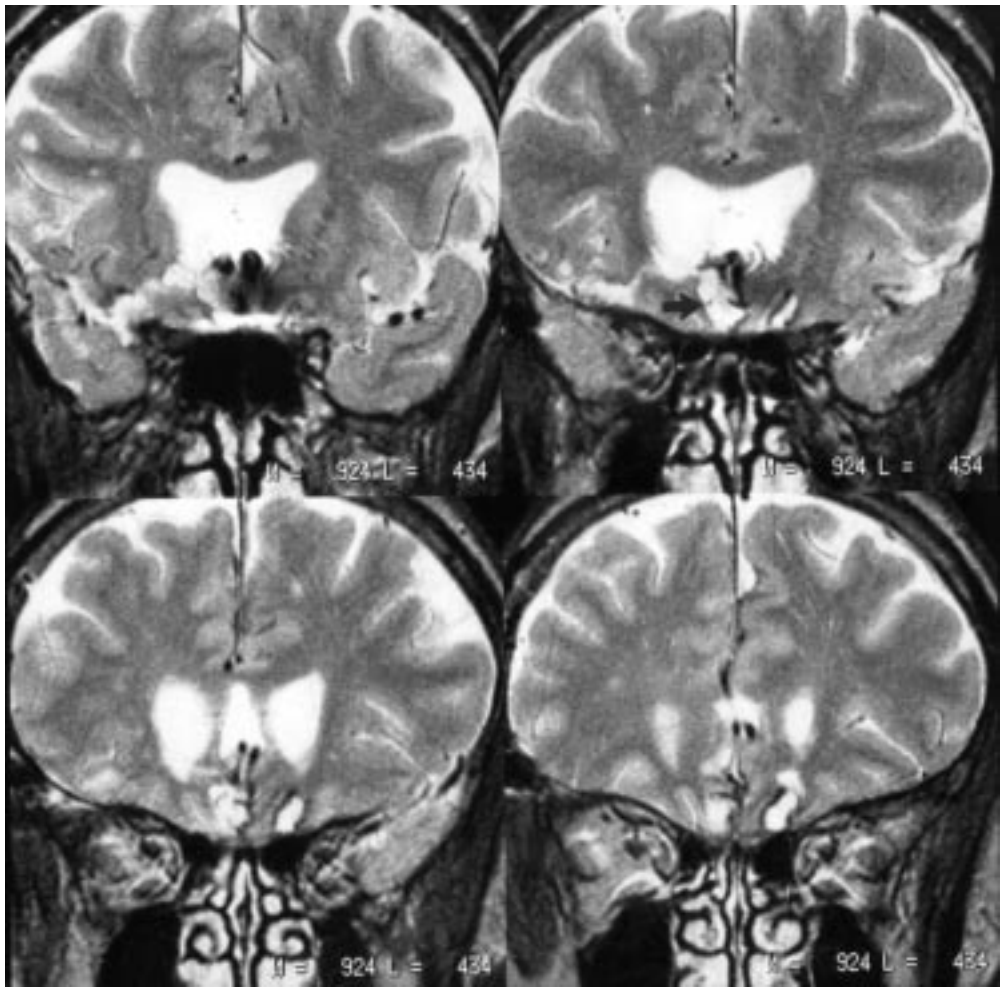

Figure 3 T2 weighted MRI of patient 4: coronal sections show (especially in the top right—black arrow —and bottom left) involvement of the right sided MS/ndbB complex as well as bilateral lesions of the rectus gyrus together with the clip artefact.

system or the resection of the rectus gyrus and any of the neurobehavioural variables. There was a significant association between the occurrence of lesions of the MS/ndbB complex and memory deficits (Pearson χ^2 statistic 16.74; $p<0.05$) in so far as in the case of bilateral lesions these deficits were more profound than in the case of unilateral ones (Somers' $d=0.503$; $p<0.05$). On the other hand, lesion side had no significant effect on the severity of these dysfunctions. There was also a significant association between the occurrence of prefrontal lesions and deficits of attention, executive dysfunctions, confabulations, and anosognosia (corresponding values of the Pearson χ^2 statistics 14.85, 21.20, 13.05, 13.05; p values<0.05). Bilateral lesions led to more severe deficits than unilateral ones (corresponding Somers' d values for attention, executive functions, confabulations, and anosognosia 0.491, 0.548, 0.571, 0.476; p values<0.05). Again, lesion side had no significant effect on the severity of these dysfunctions. Apathy seemed to be only marginally influenced by prefrontal lesions and rectus gyrus resection, because the p values of the corresponding Pearson χ^2 statistics were close to the level of significance (0.074 and 0.053, respectively). Figure 3 shows brain MRI of patient 4 with a right sided lesion of the MS/ndbB complex.

Discussion

First of all it has to be emphasised that our patient sample is not generally representative of all patients after rupture and repair of ACoA aneurysms, as only those patients were included who were admitted to a rehabilitation unit because of neurobehavioural symptoms or complaints.

Our findings on the domain of memory and learning are consistent with those of other investigators. For storage processes long term memory is more impaired than short term memory.[3 6 15] For retrieval processes free recall is more affected than cued recall or recognition.[2–4 11] In the temporal dimension anterograde amnesia is much more pronounced than retrograde, memory spans are preserved, and, finally, implicit memory and procedural learning are undisturbed in most patients.[2 5 6 11] For deficits of source memory or sequencing which were reported to be typical for frontal lobe lesions we found such dysfunctions but they were not generally predominant.[35 54]

For encoding processes—that is, working memory and learning—current models for

neuronal information processing seem to be of special interest. Working memory means simultaneous storage as well as processing, integration or manipulation of a set of single items of information for a short time and with limited capacity, whereas learning is the processing of more complex information by repetition and the use of strategies over a longer period and with large capacity.[55 56] Prerequisites for these processes are control mechanisms and executive functions belonging to the "supervisory attentional system".[57] The basis for such complex cognitive functions are neuronal networks consisting of small and large neuronal units interacting intensively with each other.[58–60] Lesions of the MS/ndbB complex in patients with ACoA affect an important interface between the hippocampus and the ascending reticular activating system.[61] Therefore, it is not surprising that complex cognitive abilities such as working memory and learning are particularly often impaired in these patients.

For attentional functions, the findings of the present study point to the fact that slight impairment within this cognitive domain occurs often. If there are deficits in selective attention, they occur as increased interference in most cases. Deficient attentional inhibitory mechanisms were reported in another study also.[10] Furthermore, the results of the present study very often showed a reduced information processing speed (especially in the case of complex requirements), poor divided attention ability, and a reduction of sustained attention and increased distractibility. Altogether only three of the 30 patients with ACoA had normal attentional functions in all investigated aspects.

Within the domain of executive functions, our findings of a reduced cognitive flexibility are consistent with those of others.[8 9 12 15] In addition, the ability to coordinate plans and to develop strategies for problem solving was impaired, manifest as a loss of concept formation, an inadequate grasp of essential items of information, and an increase in the time required to perform tasks. The secondary causes of such deficits were memory and attention dysfunctions. As a last separate test result verbal fluency deserves comment. In the present study as well as in many others verbal fluency often proved to be impaired.[5 6 8 10 13] In one study, reduced retrieval of verbal items from semantic memory was the only remaining deficit of patients with AcoA.[62]

Neuropsychological (dys)functions should not be considered in isolation, but in reciprocal interactions. This was made clear by the results of a cluster analysis. Seven specific patterns of cognitive and psychopathological dysfunctions could be differentiated. These impairment patterns fit in well with those differentiations described in previous studies, but they expand the wide range of cognitive dysfunctions especially for attentional impairment and the interrelation between disorders of individual cognitive components.[7 12–14]

For the outcome results it has to be emphasised that, besides memory, attention processes were especially good predictors. That mainly working memory was affected indicates that attentional disturbances are associated to a high degree with this brain function.

The pivotal role of the MS/ndbB complex in memory/learning processes was shown for the first time in humans by the well known postmortem study of Phillips *et al*.[5] Our findings of a significant association between the occurrence of lesions of the MS/ndbB complex and memory deficits confirm the fact that this basal forebrain region is an important interface within the so called septohippocampal system. Lesion side did not influence the severity of neuropsychological dysfunctions even in relation to memory deficits as opposed to diencephalic or mediotemporal amnesia, in which left sided affections produce more severe dysfunctions.[59] Perhaps this lack of lateralisation in the case of basal forebrain lesions might be explained by the fact that the main output fibres project via the fornix to the ipsilateral and via the fornix commissure to the contralateral hippocampus also. Nevertheless, bilateral forebrain lesions do produce more severe memory deficits than unilateral ones. The correlation between prefrontal lesions and deficits of attention, executive dysfunctions, and psychopathological disturbances was expected because of the well known role of this heteromodal association cortex in respect of these brain functions.[63] We could not confirm the results of the study of Irle *et al* who reported that only combined lesions of the basal forebrain (or basal forebrain and ventral frontal cortex) and striatum were associated with severe memory deficits.[7] In our patients striatal lesions played no part in this respect. Our finding that resection of the rectus gyrus did not influence outcome is in accordance with a recent study.[64]

The question whether or not hydrocephalus or vasospasm had any impact on the patients studied can be answered as follows. In four cases with hydrocephalus this complication was immediately recognised and cured by shunt implantation. Therefore, hydrocephalus can be excluded as a long lasting cause of neurobehavioural deficits in our patient sample. The issue of vasospasm was not considered in this study, as we were only interested in the lesion site responsible for certain deficits irrespective of the cause of the lesions. It may well be possible that, for example, some of the MS/NdbB complex lesions were caused by vasospasm. It is almost impossible to differentiate whether such lesions result from vasospasm or tearing during the neurosurgical procedure.

In summary, based on our neurobehavioural and neuropathological findings, the legitimacy of the concept of an "ACoA syndrome" has to be denied. There are various patterns of neuropsychological impairment. This has implications for rehabilitation, in so far as the planning and selecting of specific therapeutic techniques need to be carried out according to the underlying patterns of cognitive disturbance and the psychosocial background. In this context, it must be emphasised that patients with only minor deficits, but complex occupational or social demands require individually adapted—tailor made—rehabilitative approaches.

Appendices

Appendix 1: Results of cognitive testing: orientation and memory (n=30 ACoA patients)

Cognitive test/scale	*Patients n≥25%ile/ without impairment (n)*	*Patients 25%ile>n≥ 10%ile/ slight impairment (n)*	*Patients 10%ile>n≥ 5%ile/ moderate impairment (n)*	*Patients n<5%ile/ severe impairment (n)*
Orientation:				
Questionnaire[21]	24	2	1	3
Memory span/working memory:				
Digit span[22]:				
Forward	26	2	1	1
Backward	22	4	2	2
Corsi block tapping test[26]:				
Forward	25	3	1	1
Backward	21	4	2	3
Word list[23]	22	3	2	3
Reading span[24 25]	16	6	4	4
Short and long term memory:				
Short story[27]:				
Immediate recall	20	4	3	3
Recall after 48 hours	18	5	3	4
Long story[23]:				
Immediate recall	19	4	3	4
Recall after 48 hours	17	6	3	4
Recognition memory test/faces[28]	19	5	3	3
Complex figure test[29]:				
Copy	21	4	2	3
Immediate free recall	16	5	5	4
Prospective memory:				
Agreements[23]	17	5	4	4
Remote memory:				
Autobiographical memory[23]	26	2	1	1
Famous faces (modified)[23]:				
Free recall	24	2	1	3
Cued recall	26	1	0	3
Implicit memory:				
Incomplete pictures test[30]	24	2	1	3

ACoA=Anterior communicating artery aneurysm.

Appendix 2: Results of cognitive testing: learning (n=30 ACoA patients)

Cognitive test/scale	*Patients n≥25%ile/ without impairment (n)*	*Patients 25%ile>n≥ 10%ile/ slight impairment (n)*	*Patients 10%ile>n≥ 5%ile/ moderate impairment (n)*	*Patients n<5%ile/ severe impairment (n)*
Associates:				
Learning of word paired associates[23]	16	5	6	3
Learning of face-name paired associates[23]	15	3	8	4
Separated informations:				
Selective reminding test[31]:				
Long term storage	13	3	6	8
Consistent long term recall	12	3	5	10
California verbal learning test (German version)[32]:				
Trial 5	14	2	8	6
Short delay/free recall	13	5	6	6
Short delay/cued recall	17	4	4	5
Long delay/free recall	15	5	4	6
Long delay/cued recall	17	2	5	6
Long delay/recognition	18	3	4	5
Recurring figures test[33]	14	4	7	5
Spatial positions:				
7/24 Spatial recall test[31]:				
Recall (trial 1 to 5)	14	4	8	4
Recall after interference	15	5	6	4
Delayed recall after 20 minutes	16	4	6	4
Contextual information:				
Recall, recognition, and spatial localisation of objects (modified)[34]:				
Recall of objects (trial 1 to 4)	16	4	6	4
Recognition of objects (trial 1 to 4)	18	5	3	4
Localisation of objects (trial 1 to 4)	16	3	7	4
Delayed recall of objects (after 24 hours)	15	6	5	4
Delayed recognition of objects (after 24 hours)	18	4	4	4
Delayed localisation of objects (after 24 hours)	15	7	4	4
Recognition and temporal order of faces (modified)[35]:				
Recognition of faces (trial 1 to 4)	21	2	3	4
Order of faces (trial 1 to 4)	15	4	7	4
Delayed recognition of faces (after 24 hours)	20	3	3	4
Delayed order of faces (after 24 hours)	14	5	7	4

ACoA=Anterior communicating artery aneurysm.

Appendix 3: Results of cognitive testing: attention and executive functions (n=30 ACoA patients)

Cognitive test/scale	*Patients n≥25%ile/ without impairment (n)*	*Patients 25%ile>n≥ 10%ile/ slight impairment (n)*	*Patients 10%ile>n≥ 5%ile/ moderate impairment (n)*	*Patients n<5%ile/ severe impairment (n)*
Attention:				
Trail making test/part A[26 36 37]	14	6	4	6
Go-/No-Go test[38]:				
Reaction time	19	6	2	3
Error rate	20	5	2	3
Stroop test (German version)[39 40]:				
Reaction time	16	7	4	3
Error rate	18	7	2	3
Trail making test/part B[26]:				
Reaction time	12	6	4	8
Error rate	14	6	3	7
Divided attention test[38]:				
Reaction time	6	6	6	12
Error rate	9	6	5	10
Paced auditory serial addition task[31]:				
Easy	8	4	6	12
Hard	3	5	7	15
Vigilance test[38]:				
Reaction time	17	5	4	4
Error rate	12	6	5	7
Sustained attention[41]	6	10	8	6
Distractibility[41]	11	10	4	5
Executive functions:				
Planning and problem solving:				
Modified card sorting test[42]	14	8	4	4
Planning task[43]	14	7	5	4
Tower of Hanoi puzzle[44]	15	9	2	4
Verbal fluency:				
Controlled oral word association test[31]	8	9	11	2
Intelligence:				
WAIS-R (German version)[45 46]	25	1	1	3
Raven progressive matrices[47]	25	1	1	3

ACoA=Anterior communicating artery aneurysm.

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