Journal of Clinical and Experimental Neuropsychology

ISSN: 0168-8634 (Print) (Online) Journal homepage: http://www.tandfonline.com/loi/ncen19

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To cite this article: John Deluca (1992) Cognitive dysfunction after aneurysm of the anterior communicating artery, Journal of Clinical and Experimental Neuropsychology, 14:6, 924-934, DOI: 10.1080/01688639208402544

To link to this article: http://dx.doi.org/10.1080/01688639208402544

Journal of Clinical and Experimental Neuropsychology
1992, Vol. 14, No. 6, pp. 924-934

0168-8634/92/1406-0924$3.00

Cognitive Dysfunction After Aneurysm of the Anterior Communicating Artery*

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ABSTRACT

The present study examined the nature of the amnestic syndrome following aneurysm of the anterior communicating artery (ACoA) in humans. Eleven ACoA and 13 subjects with intracranial hemorrhages (ICH) elsewhere in the brain were administered a battery of standard neuropsychological tests. The ACoA group performed significantly worse than the ICH controls on tests of delayed verbal memory and on the Wisconsin Card Sorting Test, despite significantly higher Full Scale IQ. No significant differences were observed between groups on tests of immediate recall, attention and concentration, and visuo-spatial functions, although the ACoA group tended to perform better on many of these tests. The results do not support the hypothesis that the cognitive impairments observed following ACoA aneurysm are the result of diffuse cortical damage. The role of specific anterior cerebral structures in defining the "ACOA syndrome" are discussed.

Neurobehavioral impairments following Anterior Communicating Artery (ACoA) aneurysm have been reported for at least three decades. The most salient and consistent neuropsychologic deficits reported include: memory defects (e.g., Alexander & Freedman, 1984; Damasio, Graff-Radford, Eslinger, Damasio, & Kassel, 1985; Lindqvist & Norlen, 1966; Logue, Durward, Pratt, Piercy & Nixon, 1968; Phillips, Sangalang, & Sterns, 1987; Talland, Sweet, & Ballentine, 1967; Vilkki, 1985; Volpe & Hirst, 1983); personality changes (e.g., Alexander & Freedman, 1984; Lindqvist & Norlen, 1966; Logue et al., 1968; Okawa, Maeda, Nukui, & Kawafuchi, 1980; Steinman & Bigler, 1986; Storey, 1970); and confabulation (Damasio et al., 1985; DeLuca & Cicerone, 1991; Kapur & Coughlan, 1980; Stuss, Alexander, Lieberman, & Levine, 1978; Vilkki, 1985). This cluster of neurobehavioral impairments has been called the "ACoA syndrome" (e.g., Alexander & Freedman, 1984) or referred to as "Korsakoff-like" in nature (e.g., Volpe & Hirst, 1983).

* I am grateful to Dr. Larry R. Squire for his comments on an earlier version of this manuscript. I also thank Dr. Keith D. Cicerone for his encouragement throughout this project.
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Accepted for publication: February 27, 1992.

However amnesia, confabulation, and personality changes are not always observed together. For instance, patients with significant memory impairments may or may not confabulate (e.g., Vilkki, 1985), while other patients may confabulate but not be "globally amnestic" (Kapur & Coughlan, 1980). Such heterogeneity in behavioral presentations has led to the suggestion that these neurobehavioral symptoms may have different underlying neuropathological mechanisms (DeLuca, 1990).

Neurobehavioral functions other than amnesia, confabulation, and personality changes have received relatively less attention by investigators. Examination of this literature reveals several inconsistencies. For instance, some authors have presented data suggesting intact concept formation (e.g., Volpe & Hirst, 1983) while others report significant impairments (e.g., Steinman & Bigler, 1986). Similar inconsistencies have been reported for verbal fluency and visuo-spatial functioning.

Discrepancies in describing the neurobehavioral consequences of ACoA aneurysm are, in large part, due to several methodological limitations that make cross-study comparisons difficult. One significant limitation concerns time at testing. For instance, some reports examined ACoA patients during acute recovery (e.g., Gade, 1982; Richardson, 1989) while others studied patients one or more years postinjury (e.g., Desantis et al., 1989; Laiacona et al., 1989; Volpe & Hirst, 1983).

Another significant limitation related to cognitive sequelae following ACoA aneurysm concerns the method of investigation. A large number of reports in the literature have been in the form of case studies, presumably because patients presented with a specific disorder (e.g., amnesia) or displayed a curious presentation. However, these case report findings may not represent the clinical presentation of most ACoA patients.

Lastly, the most serious methodological flaw found in most of the ACoA studies concerns the lack of a control group in the design. The majority of reports in the literature suffer from this methodological limitation. Because of the lack of a control group, it is not clear whether the cognitive sequelae observed in ACoA patients are specific to aneurysms at the ACoA or to a more general reason, such as aneurysms at the Circle of Willis, or to generalized cognitive debilitation, or to diffuse cerebral damage (cf., Ljunggren, Sonesson, Saveland, & Brandt, 1985; Richardson, 1989).

In addition to issues of defining an "ACoA syndrome" and methodological difficulties in the literature, a third issue in understanding cognitive impairments following ACoA aneurysm concerns the mechanism(s) underlying the observed memory impairments. There are two general hypothesis that address mechanism: (1) the focal lesion hypothesis, and (2) the diffuse cerebral injury hypothesis.

The focal lesion hypothesis is generally supported by the case study literature on ACoA aneurysm. For instance, Damasio et al. (1985) hypothesized that amnesia was associated with infarct of the basal forebrain, an area whose blood is supplied by perforating branches of the ACoA (Crowell & Morawetz, 1977; Dunker &

Harris, 1976). Damasio et al. presented five amnesic patients with basal forebrain infarcts, but with areas traditionally associated with amnesia spared (i.e., medial temporal region, dorsomedial nucleus of the thalamus). More recent case reports also present neurobehavioral evidence supporting a focal lesion hypothesis implicating basal forebrain involvement in amnesia (Morris, Heilman, & Bowers, 1991; Phillips et al., 1987). Recent group studies also provide some evidence supporting a focal lesion interpretation (DeLuca & Cicerone, 1991; Larsson et al., 1989). Taken together, these data support the hypothesis that the amnesia frequently observed following ACoA aneurysm is a result of a focal infarct of the basal forebrain.

The second hypothesis regarding the mechanism of amnesia following ACoA aneurysm suggests that impairments are a result of diffuse cerebral involvement. Four recent group studies provide support for this contention. Richardson (1989) compared verbal memory performance among several groups of patients with aneurysms at four cerebral sites, at roughly six weeks and six months post discharge from the acute hospital. Richardson found no differences in memory performance across the four aneurysm locations and concluded that subarachnoid hemorrhage results in diffuse cerebral damage. Ljunggren et al. (1985) examined 40 patients 14 months to 7 years post aneurysmal subarachnoid hemorrhage from either the ACoA, middle cerebral, or internal carotid arteries. They concluded that the pattern of neuropsychological deficits were consistent with a "diffuse subarachnoid hemorrhage induced encephalopathy".

DeSantis et al. (1989) compared ACoA, posterior communicating artery, and middle cerebral artery aneurysm groups on performance on a composite neuropsychological score (i.e., the number of neuropsychological tests performed in the "pathological" range). These authors reported no significant differences on the global composite score across aneurysm sites. However, use of a global composite score of neuropsychological deficits may have masked potential group differences by "canceling out" the effects of one variable vs another.

Furthermore, the considerable heterogeneity in the time at testing of subjects in the DeSantis study (ranging from 7 to 115 months postsurgery) may have masked potential group differences. In a second report published by this group studying ACoA patients from 7 to 101 months postinjury, Laiacona et al. (1989) reported a wide range of neuropsychological impairments, some not consistent with that expected from a focal lesion hypothesis. These authors concluded that "... the cause of this deficit is to be sought in some kind of 'toxic' or diffuse brain impairment consequent upon the subarachnoid bleeding, or in the surgical intervention" (p. 271).

The present study was designed to overcome many of the methodological flaws outlined above in examining cognitive dysfunction following aneurysms of the ACoA, and to address the issue of the focal versus diffuse neuropathologic mechanism. The study was specifically designed to test the hypothesis that patients with ACoA aneurysm display a pattern of cognitive dysfunction that is distinct from that observed in patients with other intracranial hemorrhages. Patients with

ACoA aneurysms as well as patients with hemorrhages elsewhere in the brain were administered a battery of neuropsychological tests to examine differences in cognitive functioning between the two groups. It was reasoned that, if the ACoA group displayed a focal pattern of cerebral damage, this group should display impairments only in memory and on tests of frontal-lobe integrity. However, if cognitive impairments resulted from diffuse damage, the two groups should not differ in performance on the neuropsychological tests.

METHOD

Subjects

Twenty-four patients with intracranial hemorrhages served as subjects. Eleven subjects (7 male and 4 female) sustained aneurysms of the ACoA requiring a craniotomy for surgical repair (i.e., clipping of the aneurysm). Ten of these subjects were consecutive admissions to an inpatient brain trauma unit and were tested 1 to 4 months postinjury. The other subject was a referral to an outpatient facility (the only ACoA admitted to this facility during the course of the study) and was 11 months postinjury at the time of testing. One additional ACoA subject was not included in the present study because the patient did not present with any symptoms typically associated with the "ACoA syndrome" (i.e., amnesia, confabulation, personality change). This ACoA patient will be the focus of a future paper. The mean age of the ACoA group was 50.2 (SD = 10.4, range = 33-68) and the mean years of education was 13.4 (SD = 2.4, range = 11-18). Table 1 summarizes the age, etiology, and CT findings for the ACoA group.

In addition, 13 subjects (4 male and 9 female) with intracranial hemorrhages in locations other than the ACoA were included as control subjects. All control subjects were consecutive admissions to an inpatient brain-trauma unit and tested 1 to 4 months postinjury. Table 2

Table 1. A summary of the most relevant neurologic data among the twelve ACoA subjects.

	Age	Etiology	CT Scan
1.	49	ACoA aneurysm	Infarct/edema R basal frontal
2.	33	ACoA aneurysm	L caudate and frontal midconvexity infarct
3.	45	ACoA aneurysm	R frontal-paricta subdural hygroma
4.	48	ACoA aneurysm	Small bifrontal subdural hygromas
5.	60	ACoA aneurysm	Dilation of frontal horns, third and lateral ventricles, severe hydrocephalus
6.	58	ACoA aneurysm	Mild ventricular dilation
7.	40	ACoA aneurysm	N/A
8.	44	ACoA aneurysm	R caudate and midline frontal infarct
9.	61	ACoA aneurysm	Subfrontal infarct adjacent to the region of clipping
10.	46	ACoA aneurysm	Infarct in inferior left frontal lobe and caudate nucleus
11.	68	ACoA aneurysm	Decreased attenuation in low left frontal region extending into the anterior corpus callosum

L - Left
R - Right
N/A - Not Available

Table 2. Summary of the most relevant neurologic data among the other ICH subjects.

Age		Etiology	CT Scan
1.	61	Cerebellar hematoma	L cerebellar encephalomalacia, small L occipital and R parietal infarcts
2.	28	R ICA aneurysm	Large R temporal-parietal infarct, early midline shift to R
3.	57	L MCA intracranial hemorrhage	L MCA hemorrhage with mass effect, L basal ganglia infarct
4.	61	R PCoA aneurysm	R occipital low attenuation subdural with mild R to L shift
5.	36	L frontal AVM from proximal ACA branches	Encephalomalacia in frontal midline structures bilaterally, L>R
6.	26	L parietal hemorrhage; history of AVM bleed	L parietal hemorrhage with mild mass effect
7.	51	L MCA, temporal hemorrhage	L temporal infarct, post-op changes
8.	41	R cerebellar hemorrhage	Cerebellar hemorrhage extending into 4th ventricle, marked dilation of posterior lateral ventricles
9.	51	R PICA aneurysm	SAH, early severe hydrocephalus
10.	25	R temporal AVM rupture	R posterior temporal hemorrhage with midline shift
11.	69	R PCoA aneurysm	Bilateral posterior temporal infarcts
12.	41	R PCoA	N/A
13.	52	L carotid/ opthalmic artery aneurysm	L parietal intracerebral hemorrhage

L - Left
R - Right
ICA - Internal Carotid Artery
MCA - Middle Cerebral Artery
PCoA - Posterior Communicating Artery
AVM - Arteriovenous Malformation
ACA - Anterior Cerebral Artery
SAH - Subarachnoid Hemorrhage
PICA - Posterior Inferior Cerebellar Artery
N/A - Not Available

displays the age, etiology, and CT scan information for the control subjects (referred to as the other intracranial hemorrhage control group; other ICH group). The mean age of the other ICH group was 46.1 (SD = 14.4, range = 25-69), and mean years of education was 13.7 (SD = 2.1, range = 11-16). While the mean age of the ACoA group was significantly higher than the other ICH group ($t = 2.79$, $p < .02$), no statistical difference was observed between groups in mean years of education.

Procedure

All subjects were administered an extensive neuropsychological battery. Testing commenced after subjects had cleared from a "confusional" state, and were oriented to person, place, month, and year on two consecutive sessions. Tests consisted of the following: *Intelligence*, WAIS-R (Wechsler, 1981); *memory*, Wechsler Memory Scale (WMS) (Wechsler & Stone, 1945); *attention and concentration*, digit span, and mental control; *visuo- spatial functions*, Judgment of Line Orientation (Benton, Hamsher, Varney, & Spreen, 1983); and *concept formation*, Wisconsin Card Sorting Test (WCST) (Heaton, 1981), and the Category Test (DeFilippis & McCampbell, 1979). Due to a variety of reasons (e.g., discharge from hospital), a full battery could not be administered to all subjects. All tests were scored in accordance with standard published procedures.

Hypothesis and Data Analysis

Mean differences between the ACoA and the other ICH groups on neuropsychological tests were analyzed using the *t* test. Based on the ACoA literature, certain a priori predictions were tested. It was hypothesized that ACoA subjects would perform significantly worse than the other ICH group on tests of memory (particularly delayed recall) and on the WCST. Thus, one-tailed *t* tests were conducted on the the WCST and WMS dependent measures. The secondary hypothesis in the present study concerned ruling out the influence of general cognitive debilitation in explaining the memory and frontal test results. To this end, it was predicted that the ACoA group would not perform worse than the other ICH group on tests of attention/concentration, intelligence, and visuo-spatial functions. Thus, these data were analyzed using two-tailed *t* tests.

RESULTS

Neuropsychological test results are presented in Table 3. With respect to memory functions, immediate recall on both logical memory (LM) and visual reproduction (VR), as well as performance on paired associate learning (PAL) did not differ significantly between the ACoA and other ICH groups. However, the ACoA group recalled significantly less material following a 30-min delay than did the other ICH group on LM (i.e., LMD). In fact, all 11 ACoA subjects scored a zero on LM delayed; most of them forgot that the stories had even been presented.

ACoA subjects emitted significantly more perseverative responses on the WCST than did subjects in the other ICH group. The number of categories achieved on the WCST was also significantly reduced among the ACoA group relative to the other ICH condition. In contrast, the two groups did not differ significantly in the number of errors on the Category Test.

Full Scale IQ was significantly higher in the ACoA group relative to controls. In addition, subjects in the ACoA group performed slightly better compared to the other ICH group on tests of visuo-spatial functions (Judgment of Line Orientation), and attention and concentration (mental control and digit span). While in some cases the data only approached statistical significance, of substantive importance is the fact that in all cases, the ACoA group performed better than did the ICH controls.

Table 3. Psychometric performance by ACoA and other ICH subjects.

TEST	ACoA			ICH group			*t*	
	M	*SEM*	*n*	*M*	*SEM*	*n*		
WCST Categ	1.0	0.6	11	2.8	0.8	10	1.90	p<.037*
WCST Persev	72.0	8.0	11	37.9	8.5	10	2.91	p<.005*
LM	6.1	0.7	11	6.2	0.9	13	0.12	p<.452*
LMD	0.0	0.0	11	3.0	1.0	13	2.70	p<.007*
VR	6.5	0.7	11	7.6	0.7	13	1.12	p<.138*
VRD	1.3	0.7	11	2.4	0.9	13	0.90	p<.188*
PAL	9.2	0.7	11	9.3	1.0	13	0.06	p<.476*
Digit Span	11.2	0.7	10	10.2	0.4	12	1.38	p<.183
MC	7.6	0.5	10	6.5	0.5	13	1.53	p<.141
Category test	97.2	6.5	11	96.2	11.5	11	0.08	p<.940
FSIQ	97.9	3.3	11	87.7	3.3	11	2.19	p<.040
Judg. Line	24.6	1.4	8	18.1	2.8	10	1.92	p<.073

Abbreviations
SEM = Standard error of the mean
WCST = Wisconsin Card Sorting Test
Categ = Categories
Persev = Perseverations
LM & LMD = Logical Memory & Logical Memory Delay
VR & VRD = Visual Reproduction & Visual Reproduction Delay
PAL = Paired Associate Learning
FSIQ = Full Scale IQ
MC = Mental Control
* One-tailed tests. All other contrasts were two-tailed.

DISCUSSION

The results of the present investigation are consistent with those of previous studies that have demonstrated significant memory impairment following ACoA aneurysm. However, on tests of immediate verbal recall (i.e., Logical Memory, Paired Associate Learning), the ACoA group did not differ significantly from a control group of patients with intracranial hemorrhages in locations other than the ACoA. In fact, in several ACoA patients, immediate verbal recall was performed within normal limits.

The characteristic that clearly differentiated the ACoA group from the other ICH group was performance on tests of delayed verbal recall. Further, during the delayed recall trial, most ACoA subjects could not even recall that the material had even been presented earlier. This latter experience was observed in only one subject in the other ICH group, the only subject with documented frontal-lobe involvement (see Table 2, subject 5). Thus, while impaired verbal memory performance is a common sequelae following brain injury, the primary feature that

differentiates ACoA patients from other patients with intracranial hemorrhages lies in defective performance on delayed recall.

The present finding of significantly impaired delayed verbal memory functions following ACoA aneurysm is in agreement with the work of Larsson et al., (1989), but in marked contrast to the report by Richardson (1989). Richardson compared verbal memory performance among several groups of patients with aneurysms at various intracranial sites, including ACoA patients. Comparing patients with aneurysms of the anterior cerebral arteries, internal carotid arteries, middle cerebral arteries, and vertebral and basilar arteries, Richardson found no significant group differences on a verbal memory test adapted from an experimental investigation on mental imagery. However, there are a number of important methodological differences between the present study and that of Richardson (1989) that could account for the discrepancies.

First, the Richardson study examined patients consecutively discharged from a neurosurgical unit as opposed to the present study which evaluated consecutive admissions to an inpatient rehabilitation unit. Consecutive discharges from a neurosurgical unit will include a heterogeneous group of patients with respect to postoperative cognitive sequelae. Cognitive involvement postsurgery may range from significant amnesia to patients who are relatively unimpaired (Sengupta, Chiu & Brierly, 1975). The use in the present study of admissions to a rehabilitation facility increases the homogeneity of the groups studied because only patients with significant neurobehavioral deficits are admitted to such a facility.

Second, in the Richardson study, ACoA aneurysm patients were not directly compared to patients with aneurysm elsewhere in the brain on memory performance. Rather, ACoA subjects were incorporated into a group which included aneurysms of the anterior cerebral artery, once again perhaps masking any potential effects specific to ACoA patients. Thirdly, the Richardson study did not explicitly examine delayed recall. In the present study this was found to constitute the best differentiation between ACoA subjects and subjects with hemorrhages elsewhere in the brain.

In the present study, the mean number of perseverative responses on the WCST was significantly elevated, and mean number of categories achieved was significantly reduced in the ACoA group relative to the ICH controls. These group differences in performance on the WCST may reflect increased involvement of the frontal lobes among the ACoA subjects relative to controls. Virtually all ACoA subjects in the present study displayed CT scan documented frontal involvement (see Table 1), while documented frontal involvement in the other ICH group was only observed in one subject (Table 2, subject 5). Additionally, the fact that both groups did not differ in performance on the Category Test, a test sensitive to generalized cerebral damage (Boyle, 1988; Russel, Neuringer, & Goldstein, 1970), further strengthens the hypothesis of increased frontal system involvement of ACoA subjects relative to controls. Involvement of frontal structures in ACoA subjects has been well documented (Alexander & Freedman, 1984; Steinman & Bigler, 1986). Such frontal infarcts have been implicated in the

significant personality changes (e.g., Alexander & Freedman, 1984; Damasio et al., 1985; Steinman & Bigler, 1986) and confabulation (e.g., DeLuca & Cicerone, 1991; Kapur & Coughlan, 1980; Stuss et al., 1978) observed following ACoA aneurysm.

With respect to other areas of cognition, Full Scale IQ was significantly higher among ACoA subjects, and the ACoA group performed relatively better than the other ICH group on tests of attention and concentration (i.e., Digit Span and Mental Control), and visuo-spatial functions (Judgment of Line Orientation). These results support the notion that the cognitive impairments observed in the ACoA group (i.e., amnesia, confabulation, performance on "frontal" tests) are not simply manifestations of general cognitive debilitation (e.g., delirium, confusional state, diffuse encephalopathy) (DeLuca & Cicerone, 1991). Rather, these findings, in conjunction with the memory and frontal data reported above, likely reflect involvement of specific anterior cerebral structures.

A number of authors have speculated on the specific anterior cerebral structures responsible for aspects of the "ACoA syndrome". Recent theories have implicated structures perfused by the ACoA and anterior cerebral artery. For instance, Damasio et al. (1985) hypothesized that amnesia is associated with infarct of the basal forebrain. These authors presented several amnesic patients with basal forebrain infarcts, but with areas traditionally associated with amnesia spared (i.e., medial temporal region and medial thalamus). They hypothesize that, because of the bi-directional communication between the basal forebrain and hippocampus, amnesia from ACoA aneurysm results from disruption of medial temporal function secondary to the basal forebrain lesion. Direct support for the basal forebrain hypothesis was presented by Phillips et al. (1987) who reported postmortem evidence of destruction of only basal forebrain structures as well as portions of the anterior limb of the internal capsule and globus pallidus in an ACoA amnestic patient.

In the present study, marked amnesia was observed among ACoA patients despite no evidence on CT of diencephalic or medial temporal involvement. A number of recent reports have emerged in the literature documenting amnesia without involvement of the "traditional" cerebral structures (Berti, Arienta, & Papagano, 1990; Dusoir, Kapur, Byrnes, McKinstry, & Hoare, 1990; Morris et al., 1991; and Phillips et al., 1987). It is likely that the amnesia observed among the ACoA subjects in the present study is a manifestation of basal forebrain dysfunction. However, it should be clear that there are no group studies to date that have definitively confirmed the relationship between amnesia and the basal forebrain among ACoA patients. Such studies are clearly one direction for future research.

In summary, the results of the present study support a "focal lesion" model of the neuropathologic mechanism underlying the observed cognitive impairments following ACoA aneurysm. A specific amnesia for delayed verbal memory was observed in the ACoA group relative to controls. Full Scale IQ was significantly higher in the ACoA group relative to controls. No statistically significant differ-

ences between groups were observed in attention and concentration, and visuospatial ability, although a trend toward better performance in the ACoA group was observed. While it is likely that amnesia following ACoA aneurysm is a manifestation of basal forebrain involvement, a definitive relationship is yet to be established. Clarification of the relationship between amnesia and basal forebrain impairment is clearly an area for future research. One avenue for the future is the study of amnesic and nonamnesic ACoA patients. Documenting differences in cerebral involvement between these two groups is an important test of the basal forebrain hypothesis of memory impairment following ACoA aneurysm.

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