CASE STUDY

Recovery of memory and executive function following anterior communicating artery aneurysm rupture

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Abstract

We studied the recovery of memory and executive function in 10 patients following anterior communicating artery aneurysm (ACoA) rupture and repair. Patients were tested at 2 consecutive points in time following surgery (approximately at 2 and 3 months). At the first testing, the patients divided into 2 groups based on the severity of impairment on executive measures. Both groups had severe anterograde amnesia, but only patients with severe executive impairments had retrograde amnesia with a temporal gradient. At second testing, both groups had persistent severe anterograde amnesia. The dysexecutive group showed significant improvement in executive deficits and in retrograde amnesia, with attenuation of the temporal gradient. Patients with more severe executive impairments had more extensive bilateral frontal lesions than other patients. These results suggest that the cognitive profile following ACoA rupture changes with time. Time postonset following aneurysm rupture and lesion site are both critical for defining the neuropsychological profile, and determining the underlying cognitive mechanisms in this neurological disorder. (JINS, 1996, 2, 565-570.)

Keywords: Anterior communicating artery aneurysm, Amnesia, Executive function, Recovery

INTRODUCTION

The profile of cognitive impairment following ACoA rupture and repair consists of a variable degree of amnesia, executive function impairment, and confabulation (Alexander & Freedman, 1984). Several investigators have explored specific aspects of this cognitive impairment in detail in small groups of patients. For example, Irle et al. (1992) described a spectrum of anterograde amnesia in ACoA patients ranging from a severe deficit comparable to that seen in global amnesics (i.e., Korsakoff’s syndrome) to no memory deficit at all. Likewise, Gade et al. (1990) described a pattern of retrograde amnesia in ACoA patients as a group that was similar to Korsakoff’s syndrome. They observed a temporal gradient of remote recall with information from earlier decades being retrieved better than information from more recent decades. The executive function impairments commonly present following ACoA rupture have only been characterized in a few patients (Shoqeirat et al., 1990; Parkin et al., 1994), and a clear understanding has not emerged. Many, but not all, patients with ACoA rupture confabulate, but in those who do, confabulation ranges from spontaneous and fantastic to confabulations that are only obtained when provoked (Fischer et al., 1995).

Most investigations of the “ACoA syndrome” have studied patients who were selected because they exhibited cognitive deficits. Studies of unselected ACoA rupture patients have, however, found persistent cognitive deficits present...
in a large percentage of patients (Laiacona et al., 1989; Stenhouse et al., 1991). All studies have found a variable neuropsychological profile without a clear relationship between the cognitive profile and the site of brain damage. Thus, the effects of vasospasm or a “toxic” etiology have been put forward as explanations for the cognitive impairments, but these potential etiologies are unlikely. For example, 3 of 6 patients in one study with patients who exhibited global cognitive impairments had no or mild vasospasm (Stenhouse et al., 1991). Moreover, the exact nature of a possible “toxin” has not been elucidated (Laiacona et al., 1989).

Several factors contribute to the difficulty in uncovering clinicanoatomic relations in ACoA patients. First, most studies have not grouped patients according to lesion site, including patients with a mixture of lesions, or even no lesions at all (Stenhouse et al., 1991). Second, time postonset of aneurysm rupture is not controlled; patients have been tested at widely varying points during their recovery (Laiacona et al., 1989; Stenhouse et al., 1991; Irle et al., 1992). Finally, lesion site may not have an unvarying relationship with deficit. Different lesion sites may produce different deficits that also have differing potentials for recovery. We have demonstrated such an interaction for lesion site, confabulation, and time postonset in ACoA patient (Fischer et al., 1995). In this study, it was our hypothesis that there are similar interactions for executive function impairment, lesion site and time postonset; and for retrograde amnesia, executive function impairments and time postonset in these patients. Only longitudinal assessment of ACoA patients will characterize the cognitive profile of this neurological disorder and the relationship of cognitive deficits to lesion sites. To our knowledge, there have been no reports that evaluate the effect of recovery of specific cognitive functions in this patient group.

**METHODS**

**Research Participants**

Ten consecutive patients admitted to Braintree Rehabilitation Hospital who had had rupture and repair of an anterior communicating aneurysm were studied. There were 4 men and 6 women with a mean age of 47 years (range 27–66) and 14 years of education (range 9–20). All patients were right-handed.

**Evaluation**

Each patient underwent a comprehensive neuropsychological evaluation:

1. **General intellectual functioning**: Wechsler Adult Intelligence Scale—Revised (Wechsler, 1981)

2. **Executive functions**: backward digit span of the Wechsler Memory Scale—Revised (Wechsler, 1987), FAS test (Benton, 1968), Wisconsin Card Sorting Test (Heaton, 1981), and the Trail Making Test part B (Reitan, 1958)

3. **Anterograde amnesia**: supraspan list learning test [first testing, California Verbal Learning Test (Delis et al., 1987); second testing, Rey Auditory Verbal Learning Test (Lezak, 1983)]


All scores obtained in patients were compared to published normative values (Spray & Strauss, 1991) except for the Famous Faces Test in which normative values were obtained from 10 age- 

**RESULTS**

Analysis of patients’ performance on tasks of executive function during early testing revealed two distinct groups. Patients in Group 1 (n = 5) were severely impaired (> 2 SDs from published normative means) on at least three of four executive measures (dysexecutive group). In contrast, patients in Group 2 (n = 5), performed within normal limits on at least three of four measures (see Table 1). There were no significant differences on the remaining measures between these patient groups in age (t = 1.49, df = 8), education (t = 0.88, df = 8), general intellectual functioning [VIQ: (t = .66); PIQ: (t = 1.93, df = 8)], or the number of days between initial and later testing (t = .77, df = 8).

**First Evaluation (Table 1)**

Both groups demonstrated severe deficits in new learning as evidenced by their supraspan list learning. There was no difference between groups in the percentage of items recalled during Trial 5 (t = .83, df = 8), during delayed free recall after distraction (t = 1.73, df = 8), or the percentage lost from Trial 5 to the delay (t = .23, df = 8). Only Group 1 showed evidence of retrograde amnesia during recall on the Famous Faces Test as compared to controls (t = 2.11, df = 13, p < .002). Regression analyses of the percentage of faces correctly recalled across decades from the 1950s through the 1980s was conducted. A significant negative
Table 1. Demographic characteristics and neuropsychological performance between two groups of ACoA patients

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group 1 (dysexecutive)</th>
<th>Group 2 (normal)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time 1</td>
<td>Time 2</td>
<td>Time 1</td>
</tr>
<tr>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>Age</td>
<td>52.4</td>
<td>7.7</td>
</tr>
<tr>
<td>Education</td>
<td>12.6</td>
<td>2.0</td>
</tr>
<tr>
<td>WAIS-R VIQ</td>
<td>96.6</td>
<td>10.1</td>
</tr>
<tr>
<td>WAIS-R PIQ</td>
<td>89.6</td>
<td>5.9</td>
</tr>
<tr>
<td>Digits backward</td>
<td>2.6</td>
<td>1.7</td>
</tr>
<tr>
<td>WCST (categories)</td>
<td>1.2</td>
<td>2.2</td>
</tr>
<tr>
<td>Verbal fluency (words)</td>
<td>13.8</td>
<td>3.3</td>
</tr>
<tr>
<td>Trails B (s)</td>
<td>132.8</td>
<td>105.7</td>
</tr>
<tr>
<td>Supraspan Trial 5</td>
<td>44.0%</td>
<td>12.1</td>
</tr>
<tr>
<td>Supraspan delayed recall</td>
<td>0.0%</td>
<td>0.0</td>
</tr>
<tr>
<td>Supraspan Trial 5—delay</td>
<td>44.0%</td>
<td>12.1</td>
</tr>
<tr>
<td>Famous faces total recall</td>
<td>44.8%</td>
<td>21.0</td>
</tr>
</tbody>
</table>

A linear relationship was revealed for Group 1 ($r = -.71$, $p < .01$) but not for Group 2. The negative correlation in Group 1 is consistent with the existence of a temporal gradient (Figure 1).

**Second Evaluation (Table 1)**

In Group 1 (dysexecutive group), performance improved on all executive measures from the first to the second evaluation. On two of these measures, the extent of improvement was statistically significant (FAS: $t = 2.68$, $df = 4$, $p < .05$; Trails B: $t = 2.86$, $df = 4$, $p < .05$). No significant change was seen on these measures in Group 2. The amount of improvement across executive measures between the first and second evaluation was calculated. Group 1 improved an average of 84.1%, whereas the improvement in Group 2 was only 10.2%. Performance in Group 1 remained impaired on executive measures compared to Group 2, but this reached significance for only the Trail Making test ($t = 2.37$, $df = 8$, $p < .05$). The degree of anterograde amnesia remained severe in both groups. There was no significant improvement noted between testing during Trial 5 learning (Group 1: $t = .31$, $df = 4$; Group 2: $t = 1.90$, $df = 4$); delayed free recall (Group 1: $t = 2.38$, $df = 4$; Group 2: $t = .58$, $df = 4$); or the number of items lost from Trial 5 to the delay (Group 1: $t = 1.33$, $df = 4$; Group 2: $t = 1.37$, $df = 4$). In Group 1, improvement was noted in the severity of retrograde amnesia ($t = 3.21$, $df = 4$, $p = .06$) but performance was still impaired compared to controls ($t = 2.11$, $df = 13$, $p < .05$). Regression analyses revealed that the pattern of retrograde loss in this group no longer showed a significant linear relationship ($r = .31$). This finding is consistent with an attenuation of the temporal gradient in the dysexecutive group (Figure 1).

Lesion analysis revealed that Group 1 patients had bilateral medial frontal lesions including the anterior cingulate and corpus callosum, as well as striatal (caudate) and basal forebrain damage (Figure 2). Patients in Group 2 had lesions limited to basal forebrain (Figure 3) except for two patients who also had small orbital frontal lesions. The lesion profile for each patient is presented in Table 2.

**DISCUSSION**

Following rupture and repair of anterior communicating aneurysms, our patients showed varying degrees of cognitive dysfunction in the domains of memory and executive functions. All patients exhibited significant anterograde amnesia that persisted over time. In the early stage following...
surgery (1-2 months), two distinct groups could be identified, based on the magnitude of executive function impairment. Further analysis indicated that this executive impairment correlated with extent of frontal lobe damage. Dysexecutive patients had extensive bilateral frontal lesions; patients who performed relatively normally on executive measures did not have significant frontal lesions. Only patients with executive function impairments had significant difficulty recalling remote information, and a significant temporal gradient was observed in their pattern of recall (poorer recall in more recent decades). Dysexecutive patients (Group 1) showed significant improvement in executive function within the next month. Some improvement might have been due to practice effect (two testings, 1 month apart). Group 2, however, had only modest improvement, suggesting practice effects are small. Furthermore, the dysexecutive patients were and remained densely amnesic, so that no explicit benefit of practice could be exploited. These patients also showed improvement in recalling information from more recent decades, thereby flattening the slope of their retrograde deficit. This change could not be due to practice.

Studying the recovery of cognitive function after an acute brain insult provides a unique opportunity to gain insight regarding interactions among different cognitive processes. For example, the temporally graded retrograde amnesia that was observed only in the dysexecutive group supports the hypothesis that this pattern of retrograde amnesia may be related to the combination of executive and memory impairments. This finding is consistent with the common observation of a temporally graded retrograde amnesia in

### Table 2. Lesion sites for all patients

<table>
<thead>
<tr>
<th>Group</th>
<th>Patient</th>
<th>Frontal (L/R)</th>
<th>Striatal (L/R)</th>
<th>Basal forebrain</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (dysexecutive)</td>
<td>1</td>
<td>+ ++/ +</td>
<td>+/0</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>+ ++/ +</td>
<td>0/ +</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>+ ++/ +</td>
<td>0/ +</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>+ ++/ +</td>
<td>+/0</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>0/ +</td>
<td>+/0</td>
<td>+</td>
</tr>
<tr>
<td>2 (normal)</td>
<td>6</td>
<td>+ / +</td>
<td>0/ +</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>+ /0</td>
<td>0/ +</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>0/ 0</td>
<td>0/ +</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>0/ 0</td>
<td>0/ +</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>0/ 0</td>
<td>0/ 0</td>
<td>0/ 0</td>
</tr>
</tbody>
</table>

For frontal lesions, 0 = no lesion, + = 25%, ++ = 50%. For striatal and basal forebrain lesions, 0 = absent, + = present.

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Fig. 2. MRI scan of a patient in Group 1 demonstrating lesions in the medial frontal lobes, basal forebrain (septum) and striatum (caudate).

Fig. 3. MRI scan of a patient in Group 2 demonstrating a lesion in the basal forebrain (septum).
Korsakoff's syndrome (Albert et al., 1979), another disorder in which patients have both executive and memory impairments (Butters & Stuss, 1989). Improvement in our patients' retrograde amnesia was concurrent with improvement in executive function, despite a persistent anterograde memory deficit. Impairment in strategies for retrieving remote information may contribute to their impaired performance. Gade (Gade & Mortensen, 1990) also found a temporal gradient pattern in remote recall in a large group of ACoA patients. Their patients had significant anterograde memory impairments, but they were not tested on executive measures assessing frontal lobe function. Since lesion site was not reported either, it also cannot be determined whether their group of patients showed a similar relationship between retrograde amnesia, executive function impairment, and lesion site. Most of their patients were tested approximately 2 years after injury, suggesting that retrograde amnesia can persist. The mechanisms underlying persistent retrograde amnesia in ACoA patients remain unclear, although persistent impairment in executive function would seem the likely cause.

All of our patients had severe anterograde amnesia that persisted up to 4 months following aneurysm rupture. In a study by Irle (1992), anterograde amnesia was investigated in a large group of ACoA patients who were selected because of lesions identified in the ventromedial frontal lobe or striatum. They found a clear relationship between lesion site and severity of amnesia: Patients with combined lesions in the basal forebrain and striatum, or basal forebrain, striatum and frontal lobes, had a severe memory deficit, whereas patients with lesions in the basal forebrain or striatum alone showed essentially no deficit. Our findings are not consistent with these data since several of our patients had lesions restricted to the basal forebrain, yet had severe anterograde amnesia. Since the patients in Irle et al.'s study were evaluated at times much later postonset (1–5 years after surgery) than our patients, it seems possible that anterograde amnesia continues to recover, especially in patients with less extensive lesions.

Executive function has been investigated in detail in only a few studies of ACoA patients. When assessed, patients have shown impairments on tests thought to be sensitive to frontal lobe function. In one study, a single patient was studied 3 years after aneurysm rupture (Parkin et al., 1994) and in another, the time of testing was not reported (Shoqeirat al., 1990). Neither study reported details about the lesion site in their patients. Similar to the studies of memory functioning in ACoA patients, it is apparent that impairments in executive function can also persist. The factors that lead to poor recovery of executive function in ACoA patients have not been elucidated, but large frontal lesions are the likely cause in most patients.

Our study demonstrates that the cognitive profile following ACoA rupture changes with time. During the first 3 months following aneurysm rupture, there can be significant recovery in specific cognitive functions such as executive function, whereas others, such as anterograde memory, can remain quite impaired. Although the number of patients in our study is small, there appears to be a relationship between lesion site, time of testing after injury, and the pattern of cognitive deficits. It will be crucial for future neuropsychological studies of ACoA patients to account for both recovery and lesion site when investigating the cognitive and behavioral profiles of the ACoA syndrome.

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REFERENCES


