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# Clinical presentation and management of giant anterior communicating artery region aneurysms

**STEPHEN P. LOWNIE, M.D., F.R.C.S.(C), CHARLES G. DRAKE, M.D., F.R.C.S.(C), SYDNEY J. PEERLESS, M.D., F.R.C.S.(C), GARY G. FERGUSON, M.D., F.R.C.S.(C), AND DAVID M. PELZ, M.D., F.R.C.P.(C),**

*Departments of Clinical Neurological Sciences (Neurosurgery) and Diagnostic Radiology (Neuroradiology), University of Western Ontario, London, Ontario, Canada*

*Object.* The authors reviewed their 20-year experience with giant anterior communicating artery aneurysms to correlate aneurysm size with clinical presentation and to analyze treatment methods.

*Methods.* In 18 patients, visual and cognitive impairment were quantitated and clinical outcome was categorized according to the Rankin scale. Statistical analysis was performed using Fisher's exact test.

*Conclusions.* At least 3.5 cm of aneurysm mass effect was required to produce dementia in the patient ($p = 0.0004$). Dementia was usually caused by direct brain compression by the aneurysm rather than by hydrocephalus. Optic apparatus compression occurred with smaller aneurysms (2.7–3.2 cm) when they pointed inferiorly.

Aneurysm neck clipping was possible in half of the cases. Special techniques, including temporary clipping, evacuation of intraluminal thrombus, tandem and/or fenestrated clipping, and clip reconstruction were often required. Occlusion of or injury to the anterior cerebral artery (ACA) was the main cause of poor outcome or death.

Proximal ACA occlusion, even of dominant $A_1$ segments with small or no contralateral $A_1$ artery, was an effective treatment alternative and was well tolerated as a result of excellent leptomeningeal collateral circulation.

**KEY WORDS • anterior communicating artery aneurysm • giant aneurysm • aneurysm size • mass effect • microsurgery • detachable coil occlusion**

COMPARED with their high frequency among small or large intracranial aneurysms, giant aneurysms of the ACoA are uncommon.[5,50,56,58] When such aneurysms do attain a giant size, compression of the basal forebrain or optic apparatus results. With their midline location and associated septal perforating arteries, together with a dual arterial inflow and outflow at the ACoA complex, these giant aneurysms can present a particular challenge to management strategies. We review our experience treating these giant aneurysms at a single center over a 20-year period.

## Clinical Material and Methods

### *Data Collection and Analysis*

For all patients found to harbor a giant ACA aneurysm, clinical records were reviewed to determine patient age and sex and the grade of subarachnoid hemorrhage according to the World Federation of Neurosurgical Societies scale.[15] Symptoms and signs of mass effect were documented. Visual acuity, visual fields, and funduscopic examinations were performed. Cognitive impairment was quantitated using the Folstein minimental status test.[21] Axial CT or MR imaging or angiographic studies corrected for magnification[22] were used to confirm giant ($\geq 2.5$ cm) aneurysm size. Aneurysm size and direction of growth were correlated with the patient's clinical presentation. The presence or absence of hydrocephalus and the response to CSF shunting were noted. Approach to treatment, anesthetic management, surgical and/or endovascular techniques, and complications were reviewed. Clinical outcome was categorized according to the Rankin disability scale as excellent (Grade 0 or 1), good (Grade 2), poor (Grades 3–5), or dead. Statistical analysis was performed using commercially available software (Graphpad Instat; GraphPad Software, Inc., San Diego, CA) to create $2 \times 2$ contingency tables based on Fisher's exact test.

## Results

### *Summary of Cases*

During the period from 1975 to 1996, we treated 29 giant aneurysms of the ACA. Of these, five aneurysms of the dis-

*Abbreviations used in this paper:* ACA = anterior cerebral artery; ACoA = anterior communicating artery; AVM = arteriovenous malformation; CSF = cerebrospinal fluid; CT = computerized tomography; GDC = Guglielmi detachable coil; ICA = internal carotid artery; MCA = middle cerebral artery; MR = magnetic resonance.

TABLE 1
*Characteristics of 18 patients with giant ACA region aneurysms**

| Case No. | Age (yrs), Sex | WFNS Grade | Mass Effect | Aneurysm Verifications, Size (cm) | Aneurysm Location | Thrombus | Hydrocephalus | | | Aneurysm Treatment | Long-Term Rankin Grade |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Present | Shunted | Improved | | |
| 1 | 54, M | 0 | brain | angio, 5.0 | ACoA | yes | yes | yes | yes | $A_1$ tourniquet | 1 |
| 2 | 56, M | 0 | brain | angio, 3.5 | ACoA | no | no | no | NA | AVM only | dead |
| 3 | 28, F | 1 | none | CT, 3.0 | bilat $A_1$ | yes | no | no | NA | wrapped in gauze & plastic | unknown |
| 4 | 50, M | 2 | optic apparatus | angio, 2.6 | ACoA | yes | no | no | NA | neck clipping | 1 |
| 5 | 49, F | 0 | optic apparatus | CT, 2.7 | distal $A_1$ | yes | no | no | NA | neck clipping | dead |
| 6 | 41, M | 0 | optic apparatus | angio, 2.7 | ACoA | yes | no | no | NA | neck clipping | 1 |
| 7 | 42, M | 1 | none | angio/CT, 2.5 | ACoA | no | no | no | NA | lt $A_1$ clipping & later trapping | 1 |
| 8 | 25, M | 0 | optic apparatus | angio/CT, 3.2 | distal $A_1$ | yes | no | no | NA | $A_1$ tourniquet & later trapping | 1 |
| 9 | 69, M | 2 | none | CT, 3.0 | ACoA | yes | no | no | NA | explored only | unknown |
| 10 | 7, M | 0 | none | skull x-ray, 3.2 | fusiform rt $A_1$–$A_2$ | yes | no | no | NA | $A_1$ clipping | 0 |
| 11 | 52, M | 1 | none | CT, 2.5 | ACoA | no | no | no | NA | neck clipping | 0 |
| 12 | 54, M | 1 | none | CT, 2.7 | proximal $A_2$ | yes | no | no | NA | $A_2$ tourniquet & later trapping | 1 |
| 13 | 76, M | 1 | none | 3.5 | ACoA | yes | no | no | NA | neck clipping | 4 |
| 14 | 66, M | 0 | brain | 5 | ACoA | yes | yes | yes | no | neck clipping | 3 |
| 15 | 41, M | 1 | none | angio, 2.5 | ACoA | no | no | no | NA | neck clipping | 0 |
| 16 | 72, F | 0 | brain | MR/CT, 3.5 | ACoA | yes | yes | postop | yes | GDC | 1 |
| 17 | 65, F | 0 | brain | MR/CT, 5.0 | ACoA | yes | yes | yes | no | $A_1$ balloon | 2 |
| 18 | 65, M | 0 | brain | MR, 5.0 | ACoA | yes | no | no | NA | antihypertension medication | 2 |

* Angio = angiography; NA = not applicable; WFNS = World Federation of Neurosurgical Societies.

tal $A_2$ (pericallosal) segment and two aneurysms of the very proximal $A_1$ segment were excluded from the present study. Four other aneurysms were excluded because, on review of CT or MR images, their actual diameter was found to be slightly smaller than 2.5 cm. Eighteen cases fulfilled the criteria for giant ACoA region aneurysms (Table 1).

There were 14 male and four female patients who ranged in age from 7 to 76 years (mean 51 years). Nine patients (50%) presented with mass effect alone, which involved the brain in six cases and the optic apparatus in three. Eight patients presented with subarachnoid hemorrhage, one of whom also had mass effect on the optic apparatus. In the remaining patient, the aneurysm was discovered as a calcified lesion on skull x-ray films.

Aneurysm size ranged from 2.5 to 5 cm (mean 3.4 cm). Thirteen aneurysms were located at the ACoA complex; two were on the distal $A_1$ segment; one involved both distal $A_1$ segments; one was on the proximal $A_2$ segment; and one was a fusiform $A_1$–$A_2$ aneurysm (Fig. 1).

Significant intraluminal thrombus was observed in 14 aneurysms. The four aneurysms without thrombus included the two smallest and two located on proximal feeding arteries supplying AVMs.

In seven patients the aneurysm was treated by clipping its neck. Proximal $A_1$ or $A_2$ occlusion, created using a tourniquet, clip, or balloon, was used in another six patients; three of these patients had aneurysms that were subsequently trapped. Guglielmi detachable coils were used in one patient after neck clipping failed. One aneurysm could only be wrapped. In one patient, an associated large AVM was treated initially in the hopes of inducing aneurysm thrombosis by flow reduction. One patient underwent surgical exploration; however, clipping was not possible and nothing further was done. One patient declined surgery after experiencing cognitive improvement in response to antihypertension medication. Clinical outcomes were excellent in 10 cases, good in two, poor in two, unknown in two, and two patients died.

### *Mass Effect on the Optic Apparatus*

In four cases in which there was optic nerve or chiasm compression, one in which the patient presented with hemorrhage, the mean aneurysm size was 2.8 cm (range 2.7–3.2 cm). Three of these patients presented with visual loss, two with intact aneurysms, and one with a remote rupture. In another, preoperative visual assessment was hampered by the patient's drowsiness; however, at surgery compression of the left optic nerve and optic chiasm was observed. All patients had a bilateral reduction in visual acuity, but the reduction was usually much greater in one eye (Table 2). Three patients also exhibited visual field defects: in one case, bitemporal hemianopsia; and in the other two cases, a dense central scotoma in one eye with an incongruous hemianopsia in the opposite visual field. One patient experienced anosmia in addition to the visual disturbance.

### *Mass Effect on the Brain*

Six patients presented with dementia. Symptoms consisted of inattentiveness, confusion, lack of interest, forgetfulness, and inappropriate behavior. These symptoms had been present from 3 months to 5 years before diagnosis. Examination revealed disorientation, hesitant speech, poor concentration, flattened affect, and profound memory impairment. One patient had begun to chew tobacco, drink rum with his coffee, and steal items from a variety store. Another wandered about his house, leaving cigarettes burning in ashtrays; he had a pseudobulbar affect with outbursts of laughing and crying. Profound lethargy was seen in only one patient, whose aneurysm also had caused hydrocephalus; he improved after shunt placement.

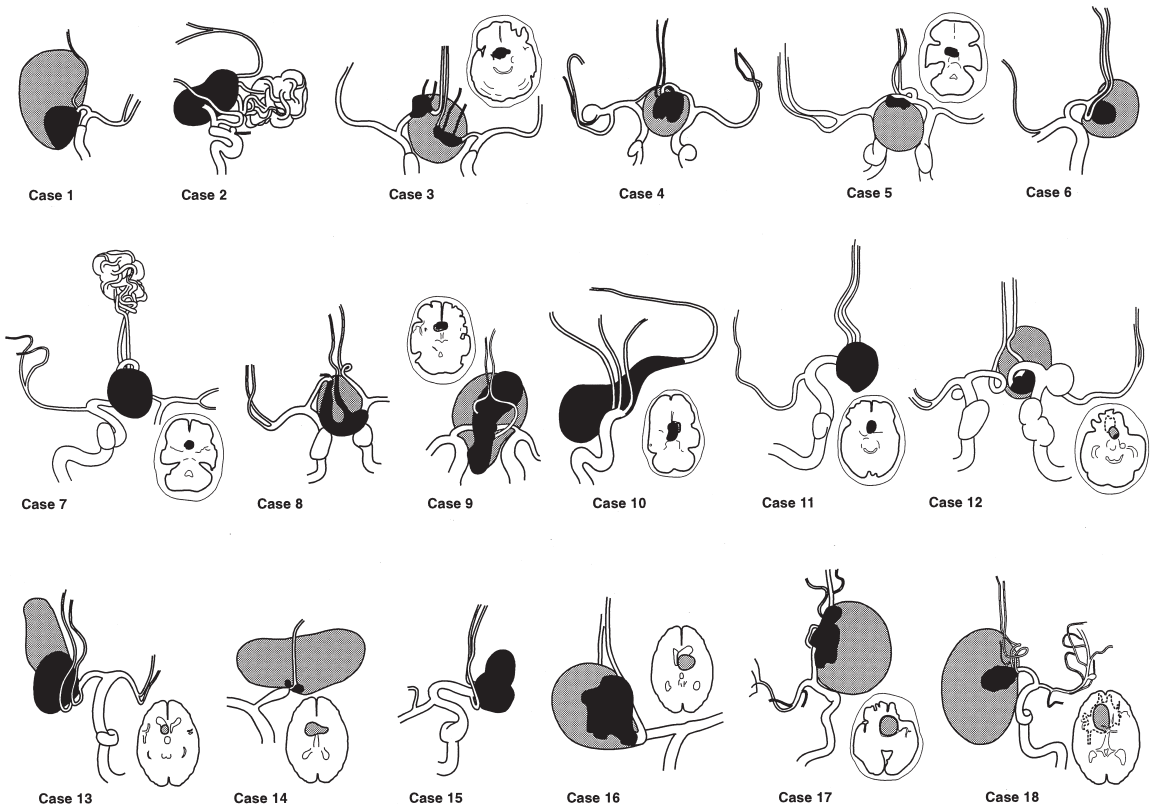


FIG. 1. Angiographic and CT scan tracings with clinical details in 18 cases of giant ACoA region aneurysms. Regions shaded *black* indicate the aneurysm lumen; *gray* shading denotes intraluminal aneurysm thrombus. See Table 1 for more detailed information on each case.

Four of the six patients with dementia also experienced gait disturbance. Two had experienced falling and one was unable to sit up in bed without falling backward. Examination showed slow shuffling steps or an inability to walk in tandem. Bladder and bowel incontinence was a late development in one patient. No patient with dementia had visual impairment or anosmia. Computerized tomography scans demonstrated hydrocephalus in three patients, which may have contributed to the cognitive impairment, although only one patient improved significantly after shunt placement.

*Aneurysm Size and Clinical Presentation*

The mean aneurysm size in the group with dementia was 4.5 cm. Eleven patients harbored aneurysms smaller than 3.5 cm in diameter, none of whom presented with cerebral mass effect. Of the remaining seven patients, whose aneurysms ranged between 3.5 and 5 cm in diameter, six exhibited dementia due to mass effect. The seventh harbored a 3.5-cm aneurysm, but the sac projected as a tubular structure into the lateral ventricle (Case 13; Fig. 1). In cases in which the aneurysm was 3.5 cm or larger, there was a strong relationship with a clinical presentation of dementia ($p = 0.0004$, two-sided test).

*Cerebral Angiography and Clinical Presentation*

In nine of 18 patients there was an asymmetrical circle of Willis, with a dominant $A_1$ segment filling both ACAs. All aneurysms were located at the bifurcation of this $A_1$ segment; in other words, they were typical ACoA aneurysms. Among these nine patients were five of the six who presented with dementia due to mass effect. In the other nine patients the circle of Willis was symmetrical, with $A_1$ segments of equal or similar size. Only one of these patients had dementia ($p = 0.13$, two-sided test). Of those aneurysms with symmetrical $A_1$ segments, two were distal $A_1$ saccular aneurysms, one was a proximal $A_2$ aneurysm, and one was a fusiform $A_1$–$A_2$ aneurysm. Two others were peculiar aneurysms, involving both distal $A_1$ arteries in one case and arising on a blown-out ACoA in the other. Two other aneurysms were accompanied by bilateral large $A_1$ arteries supplying AVMs distal to the aneurysms (Cases 2 and 7). Only one case with symmetrical $A_1$ segments proved to be a typical ACoA (Case 4); it appeared preoperatively to be a distal left $A_1$ aneurysm, but, following neck clipping, the angiogram revealed that the aneurysm had originated at the ACoA complex, which had been displaced to the left side.

Eight aneurysms demonstrated significant growth in an upward direction. Six of these aneurysms presented with a brain mass effect. In four cases there was a primary downward orientation. These cases were associated with compression of the optic apparatus. In three of these cases there was also significant upward displacement of the proximal ACAs.

*Surgical Management*

Four patients experienced hydrocephalus due to mass

TABLE 2

*Published cases of unruptured giant ACA region aneurysms with mass effect on optic apparatus**

| Authors & Year | Aneurysm Verification | Size (cm) | Visual Acuity (OD, OS) | Visual Fields | Fundi |
|---|---|---|---|---|---|
| Fouché, 1931 | autopsy | NA | CF, 6/18 | OD central scotoma | rt optic pallor |
| Jefferson, 1937 | autopsy | 4 | NLP, 6/18 | bitemporal hemianopsia | NA |
| Wakeley, 1937 | autopsy | NA | NA | bitemporal hemianopsia | bilat optic atrophy |
| Walsh & King, 1942† | autopsy | 3 | 20/50, 10/200 | bitemporal field defects | NA |
| Jefferson, 1953 | angio | NA | NA | bitemporal hemianopsia | NA |
| Norlén & Barnum, 1953† | angio | NA | NA | bitemporal hemianopsia | NA |
| Norlén & Olivecrona, 1953 | angio | grape ~2.5 | near blindness | bitemporal hemianopsia | NA |
| Wappenschmidt & Bettag, 1960 | angio | walnut ~3 | 5/70, LP only | bitemporal hemianopsia | early papilledema |
| Van't Hoff, et al., 1961 | angio | NA | CF, CF | bitemporal field defects | bilat optic pallor |
| Gilman, et al., 1962 | angio | 4.5 | 20/200, NLP | OD temporal defect | lt optic pallor |
| Heiskanen & Nikki, 1962 | autopsy | 3 | OD reduced | OD nasal hemianopsia | NA |
| Odom, 1964 | angio | 3.5 | CF, 20/100 | bitemporal hemianopsia | rt optic atrophy |
| Cullen, et al., 1966 | angio | NA | 6/18, NLP | OD temporal hemianopsia | NA |
| Bull, 1969† | angio | 3 | CF, CF | bitemporal hemianopsia | bilat optic pallor |
| Lavyne, et al., 1978 | angio | 3 | 20/400, LP only | bitemporal hemianopsia | lt optic atrophy |
| Peiris & Ross Russell, 1980‡ | angio | NA | 6/18, 6/6 | OD central scotoma | NA |
| | angio | NA | 6/9, 6/60 | bitemporal hemianopsia§ | NA |
| | angio | NA | 6/9, 6/60 | OS temporal hemianopsia§ | NA |
| Højer-Pedersen & Haase, 1981 | angio | 2.5 | 20/80, 20/80 | bitemporal hemianopsia | bilat optic pallor |
| Pia & Zierski, 1982 | angio | NA | NA | superior temporal quadrant defect | NA |
| Yaşargil, 1984 | angio | ~3 | NA | bitemporal hemianopsia | optic atrophy |
| Maurice-Williams, 1987 | angio | 3 | OS NLP | NA | lt optic atrophy |
| Soloman, et al., 1991 | angio | 3.5 | OD reduced | NA | NA |
| Vargas, et al., 1994 | angio | NA | 20/20, 20/200 | OS hemianopsia | NA |
| present series | | | | | |
| Case 5 | angio | 2.7 | OD CF | lt incongruous hemianopsia | no atrophy |
| Case 6 | angio | ~2.7 | OS NLP | bitemporal hemianopsia | NA |
| Case 8 | angio | 3.2 | 20/200, NLP | fields full in seeing eye | NA |

* CF = counting fingers; LP = light perception; NA = not available; NLP = no light perception; OD = right eye; OS = left eye; ~ = approximately.

† The original surgical procedures on which these data are based were performed by Dandy, Krayenbuhl, and McKissock, respectively.

‡ Two cases in this series are not included.

§ Patient had more visual field changes. See article by Peiris and Ross Russell.

effect. All underwent ventriculoperitoneal shunt placement; in two, shunts were placed in both lateral ventricles. Of the three patients in whom shunts were placed before direct aneurysm treatment, only one exhibited significant improvement.

Surgical exploration was undertaken in the majority of cases to determine if the aneurysm neck could be clipped. Exceptions to this principle included one case in which an AVM was excised first, one in which the fusiform nature of the aneurysm warranted proximal $A_1$ occlusion, one in which the $A_1$ was deliberately occluded with a balloon, and one case in which the patient refused surgery. Surgical adjunctive therapies included use of mannitol during the craniotomy and lumbar CSF drainage after dural opening. These treatments resulted in a slack brain, minimizing brain retraction pressure. Barbiturate- and/or hypothermia-induced cerebral protective measures were not used. Before 1982, hypotension was commonly induced, initially by using intravenous agents and, subsequently, by using isoflurane. Mean arterial pressures of 45 to 60 mm Hg were well tolerated for 1 or 2 hours and lowered to 30 mm Hg for 8 minutes in one case. After 1982, the use of controlled hypotension largely gave way to temporary clipping of one or both $A_1$ segments. Temporary clips were applied for periods lasting between 1 and 5 minutes, usually no more than 4 minutes, with at least 4 minutes of recirculation time between applications. Up to five periods of temporary clipping were required in this series. Complete temporary trapping was not used.

The surgical approach consisted most often of a frontotemporal craniotomy or, occasionally, a bifrontal flap. Transsylvian, subfrontal, and interhemispheric arachnoidal approaches were used, often in combination, to visualize the arterial supply. The exposure was enhanced on a few occasions by partial subpial removal of the gyrus rectus. In one case, separate right-sided subfrontal and left-sided transsylvian approaches allowed exposure and clipping of the left $A_1$ segment; at the third surgical venture an interhemispheric approach permitted trapping (Case 7).

### *Aneurysm Neck Clipping*

Neck clipping was possible in seven of 14 attempts. These aneurysms were all between 2.5 and 3.5 cm in diameter, with the exception of one 5-cm aneurysm that was largely thrombosed. The initial attempt at clipping frequently led to the clip's sliding down and off the neck with occlusion of the $A_1$ segment, the ACoA, or the adjacent $A_2$ segment. In two cases, forceps or a hemostat were used to create a space for clip application. Opening of the sac and evacuation of thrombus was required in two others. Ultrasonic aspiration of thrombus and temporary $A_1$ clipping were important adjunctive treatments in the clipping of a

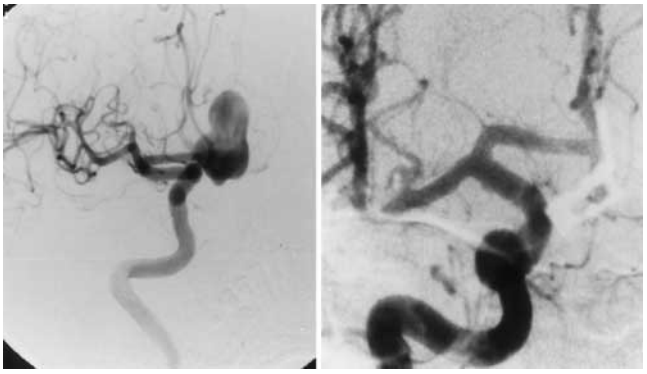

FIG. 2. Case 15. *Left:* Angiogram obtained with right ICA injection (right anterior oblique projection). *Right:* Postoperative right ICA angiogram (anterior projection) demonstrating that the aneurysm has been occluded using two fenestrated Drake–Sugita clips.

5-cm diameter aneurysm. Tandem clipping, useful in two cases, was performed by placing a fenestrated Drake–Sugita clip and then applying a second clip across the fenestration of the first one. In another case, the thrombosed portion of the aneurysm neck was isolated in the fenestration while occluding the adjacent open neck. In one patient (Case 15), reconstruction of the ACoA complex was accomplished by placing the apertures of two right-angled Drake–Sugita clips around the ACoA and right $A_2$ segment (Fig. 2).

*Unclippable Aneurysms: Proximal ACA Occlusion*

Seven patients harbored aneurysms that could not be clipped. Five of these aneurysms had no discernible neck at surgery. In one case, the left frontopolar artery arose from the aneurysm 1 cm away from the emergence of the left $A_2$ artery, which was 0.5 cm away from the entry point of the left $A_1$ segment. In another case, the aneurysm grew out of the ACoA itself, whereas the $A_1$–$A_2$ junctions were separate from the aneurysm's origin. In a third case both $A_1$ segments and the left $A_2$ were incorporated into the sac. An attempt at clip application resulted in a dangerous occlusive stenosis of the left $A_2$ segment in one case. In another, the clips slipped down to occlude the left $A_1$ and $A_2$ because of the thickness and calcification of the neck with a separate origin of the $A_2$ off the aneurysm sac.

In patients with unclippable aneurysms, proximal ACA occlusion was considered next. In our first case it was performed by application of a tourniquet to the left $A_1$ segment proximal to the recurrent artery of Heubner. The tourniquet was tightened while the patient was awake in the angiography suite. Results of angiography studies revealed that the aneurysm was filling to only one sixth of its former size from the opposite ACA. The patient remained well for more than 10 years. After this experience, $A_1$ tourniquet occlusion was used in another case after surgery elsewhere had resulted in inadvertent occlusion of the ACoA. Complete occlusion was tolerated and exuberant filling of the isolated $A_2$ segment from retrograde leptomeningeal collateral vessels was observed angiographically. The aneurysm was later evacuated and trapped. The patient's optic nerve compression had not worsened following tourniquet occlusion; however, the visual deficits did improve with the evacuation. In two other cases, retrograde leptomeningeal collateral flow was sufficient to sustain the territory of a single ACA following tourniquet or clip occlusion.

*Unclippable Aneurysms: Alternative Techniques*

As an alternative to the tourniquet, a small detachable balloon was used for testing and for permanent occlusion of the $A_1$ segment that provided the sole supply to a giant ACoA aneurysm. Excellent retrograde filling of both ACA territories was seen after occlusion of the solitary $A_1$ segment, the opposite $A_1$ segment not being visible on angiography. Delayed surgical evacuation was required (Fig. 3).

When both ACAs provided supply to an unclippable aneurysm, the sac was wrapped, the ACoA segment was trapped, or nothing was done. In one case in which neck clipping failed, endovascular saccular occlusion was performed using GDCs, with excellent long-term results despite a wide aneurysm neck (Fig. 4).

*Complications of Surgery or Endovascular Treatment*

Surgical complications occurred in two patients. In one, bleeding from the right $A_1$ segment developed during dissection at the aneurysm neck, which was easily controlled. However, with the cessation of induced hypotension, the pinhole enlarged to a rent and it was necessary to place an encircling Sundt clip on the terminal ICA. The patient died of elevated intracranial pressure.

The other surgical complication also involved a difficult dissection during which a rent developed in the right $A_2$ origin. This was controlled by placing oxycellulose in the hole and holding it in place with a fenestrated clip. The postoperative angiogram confirmed complete occlusion of the aneurysm, but the left $A_2$ was also occluded.

A perioperative death occurred early in the series in a patient with an associated large AVM and a giant aneurysm, which caused a severe and rapidly progressive dementia. It was hoped that excision of the AVM might diminish the flow through the aneurysm sufficiently to induce thrombosis. Five days postoperatively the patient experienced a seizure followed by coma. Results of angiography studies revealed complete excision of the AVM, thrombosis of 75% of the aneurysm, and good filling of both ACAs. Autopsy revealed a huge rupture site at the posterosuperior aspect of the aneurysm, with intracerebral and intraventricular hemorrhage.

Alternate treatment methods were also associated with complications. None resulted in a poor outcome or death. In one case, after tightening the tourniquet the aneurysm neck was torn, resulting in an acute subdural hematoma. The clot was evacuated and the aneurysm was trapped, opened, and evacuated.

Unilateral leg weakness occurred 2 days after placement of GDCs in one case. Angiography demonstrated additional thrombosis in the aneurysm, but a probable small embolus in the left ACA was manifested by slow flow in the distal branches. The patient's weakness improved, but still causes some limitation 4 years later.

In the case in which the balloon was used, a safety balloon ruptured, resulting in a tiny metallic balloon marker embolizing to a distal MCA branch without clinical consequence.

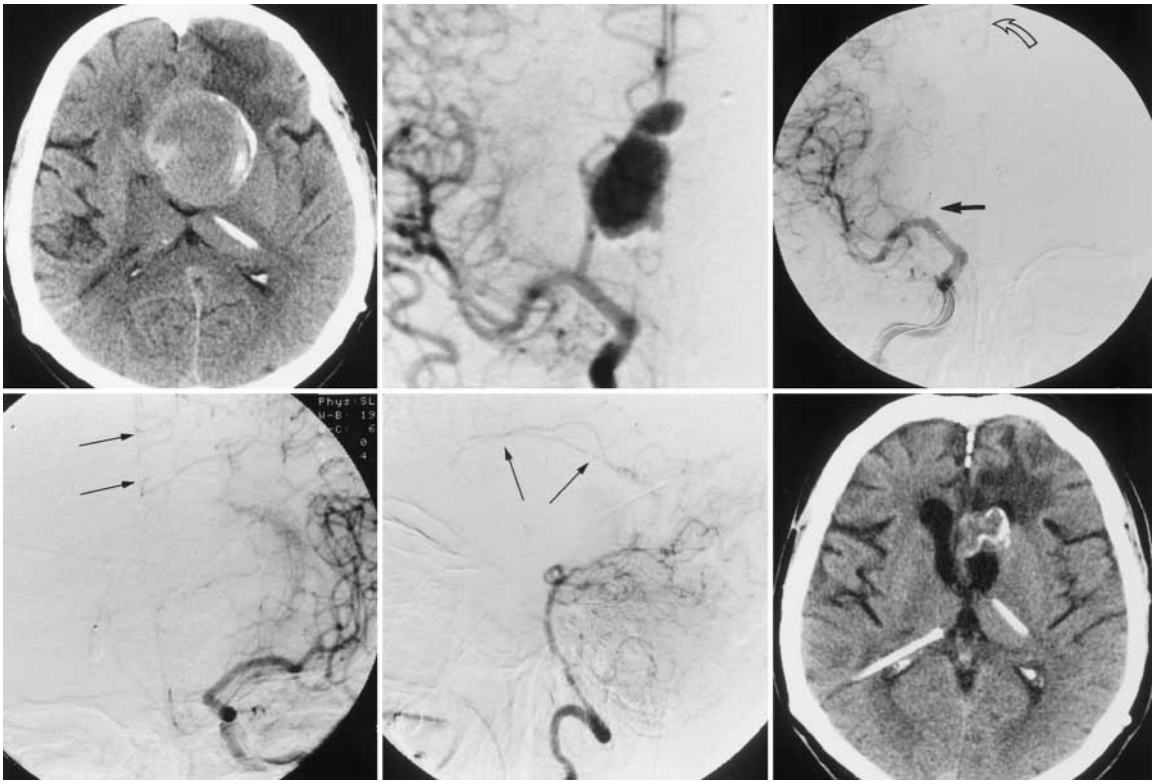

FIG. 3. Case 17. *Upper Left:* Nonenhanced CT scan revealing a large mass near the interhemispheric fissure with rim calcification and overlying left frontal lobe hypodensity. *Upper Center:* Right common carotid artery angiogram (anterior projection) demonstrating that the right $A_1$ segment is bowed vertically. Beyond the aneurysm, the $A_2$ segments of both hemispheres are filled from the dominant right ACA. *Upper Right:* Right ICA angiogram (anteroposterior view) confirming that the right $A_1$ segment has been occluded by a detachable latex balloon *(straight arrow)*. Leptomeningeal collateral flow is seen extending from the right MCA territory to the right ACA territory *(curved arrow)*. *Lower Left:* Left ICA angiogram (anteroposterior view) obtained after detachment of the balloon. The absent left $A_1$ is noted. An exuberant collateral vessel is seen extending from the MCA to the left ACA *(arrows)*. *Lower Center:* Left vertebral artery angiogram (lateral projection). Leptomeningeal collateral vessel passes from the posterior cerebral artery branches to the middle and anterior segments of the pericallosal arteries *(arrows)*. *Lower Right:* Follow-up nonenhanced CT scan obtained 27 months after treatment. Note the marked reduction in the size of the mass.

*Patient Outcome*

Postoperative angiograms confirmed complete aneurysm occlusion in 13 patients. Angiography demonstrated in two cases (Cases 1 and 2) that the majority of the aneurysm had thrombosed and in one case that there was no change in an aneurysm that had been wrapped; two patients did not undergo repeated angiography.

Visual acuity was improved in one patient but unchanged in the other two. In Case 8, visual acuity improved in the left eye from blindness to the ability to count fingers and from 20/200 to 20/50 in the right eye.

Cognitive function improved significantly in four patients but showed only minimal improvement in one. In one case shunt placement resulted in remarkable improvement and, after tourniquet occlusion, the patient remained well for more than 10 years. In another case, left ACA infarction occurred following surgical exploration performed elsewhere. After surgical evacuation and clipping of the aneurysm, follow-up neuropsychological testing demonstrated improved attention and improved auditory comprehension, but the patient remained emotionally inappropriate and confabulatory. In the case in which GDCs were used, the aneurysm reduced in size very gradually from 3.5 cm initially to 3 cm at 15 months and 1.9 cm at 32 months (Fig. 4), and the patient's dementia resolved over a period of 1 year. The patient in whom a balloon was used had a minimental status score of 17 of 30, but improved significantly to 26 of 30 by 5 months after balloon $A_1$ occlusion and evacuation of the sac. One patient exhibited a remarkable degree of improvement in response to a course of antihypertension medication alone. Over 4 months this patient's minimental status scores improved from 21 to 28 of 30. Four years later the dementia returned with an increase in blood pressure, but the patient again improved with the use of an additional antihypertension agent. Magnetic resonance imaging revealed little change in the giant thrombosed mass, with surrounding edema restricted to the right frontal lobe.

## Discussion

Compared with their high frequency among small (< 12 mm) or large (12–24 mm) intracranial aneurysms, giant ACoA aneurysms are uncommon.[5,50,56,58] The reason

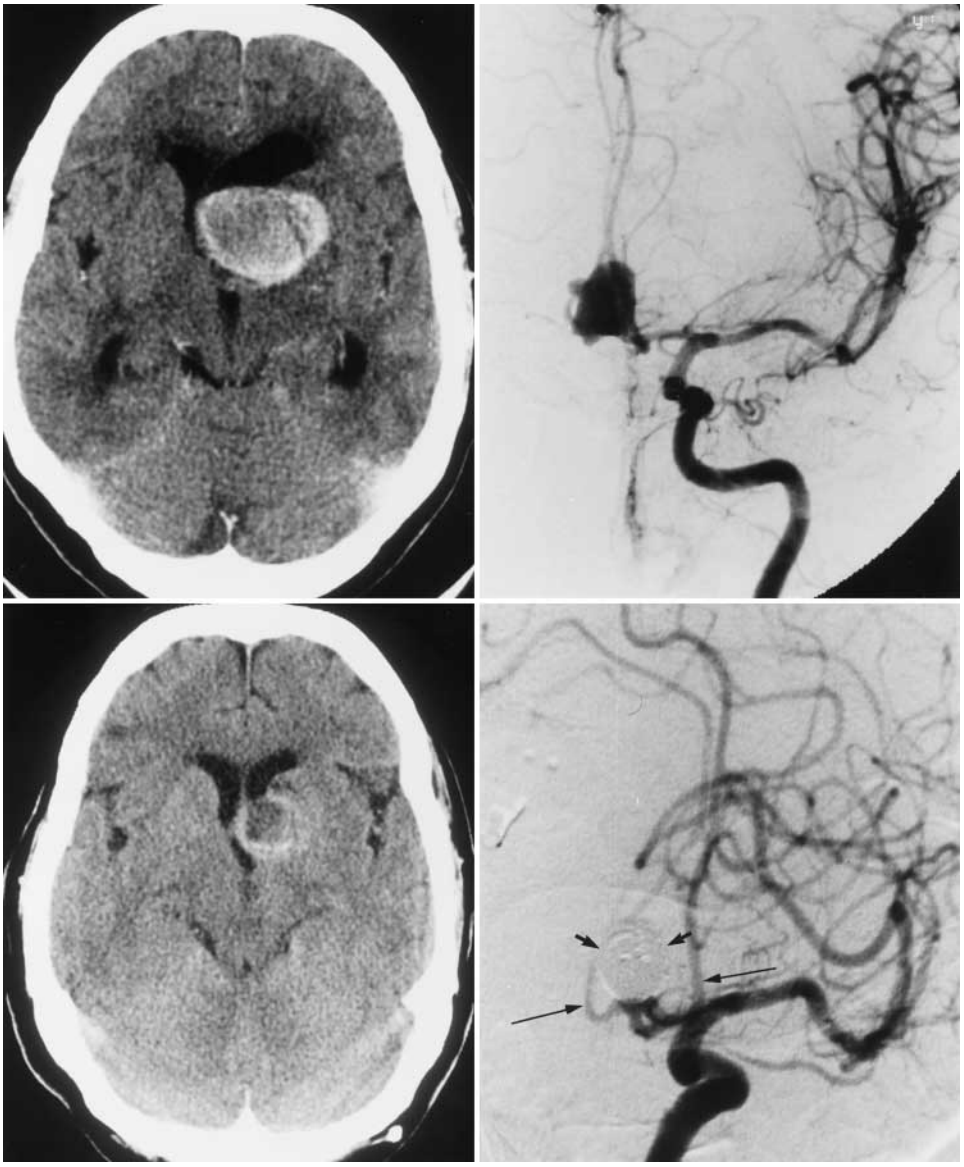

FIG. 4. Case 16. *Upper Left:* Contrast-enhanced CT scan revealing a large mass in the left caudate region with involvement of the left lateral ventricle and displacement of the septum pellucidum to the right side. *Upper Right:* Left ICA angiogram (anterior projection). *Lower Left:* Contrast-enhanced CT scan obtained 3.5 years after treatment. Note the marked reduction in the size of the mass. *Lower Right:* Left ICA angiogram (oblique projection) obtained 3.5 years after treatment. The wide-necked aneurysm remains completely occluded by GDCs *(short-tailed arrows)*, and both $A_2$ segments fill well *(long-tailed arrows)*.

for this relative rarity is unknown; however, it may be due to a higher rate of rupture among smaller ACoA aneurysms.

Before the introduction of cerebral angiography, giant ACoA aneurysms were identified either at autopsy following fatal rupture or during surgery for a presumed suprasellar meningioma.[3,23] Jefferson[31] was likely the first to consider the diagnosis of a giant ACoA region aneurysm preoperatively and to confirm the diagnosis at surgery. A large suprasellar mass was found and punctured with a needle, but nothing could be withdrawn. After incision and curettage, there was a "rush of bright blood," which was arrested by packing the front wall of the aneurysm with muscle. The patient died of rebleeding 18 months later.

*Visual Loss Caused by Mass Effect on the Optic Apparatus*

Jefferson[31,32] recognized that aneurysms arising from the ACoA complex might compress the optic chiasm from above or extend over the anterior edge of the chiasm and compress the optic nerves from below. Compression from above would cause a visual field disturbance that initially would affect the inferior quadrants, whereas compression from below would mimic a pituitary adenoma.[29,66]

Including three in our series, there have been 27 reported cases of direct compression of the optic nerves and/or chiasm by giant ACoA region aneurysms verified by autopsy or cerebral angiography (Table 2).[8–10,23,25,27,29,31,32,36,39,44–47,49,54,61,62,65–67,69] Other reports[1,8,10,11,17,19,33,41,42,47] lack such verification or are insufficiently detailed. The majority of

TABLE 3
*Published cases in which unruptured giant ACA region aneurysms caused dementia**

| Authors & Year | Aneurysm Verification | Size (cm) | Hydro-cephalus | Cognitive Features | Other Features | Time |
|---|---|---|---|---|---|---|
| Gilman, et al., 1962 | angio | 4.5 | NI | slowness, memory loss, disheveledness | visual loss, anosmia, seizures, hypopituitarism | 23 yrs |
| Morley & Barr, 1969 | angio | NI | NI | dementia | episodic catatonic schizophrenia | 14 yrs |
| Maxwell & Chou, 1977 | angio | ~5.5 | yes | confusion, severe memory loss | headache, gait disturbance, urinary symptoms | 2 mos |
| | angio | ~5 | yes | confusion, memory loss, slovenliness | headaches, seizures, gait & urinary symptoms | 6 mos |
| Hosobuchi, 1979 | angio | NI | NI | progressive dementia | none | NI |
| | angio | NI | yes | progressive dementia | none | NI |
| Peiris & Ross Russell, 1980 | angio | NI | NI | dementia, personality change | visual loss | NI |
| Yaşargil, 1984 | angio | ~3 | NI | slowness, memory loss, emotional lability | headaches, visual loss | NI |
| Maurice-Williams, 1987 | angio | 3 | NI | withdrawal, dementia | depression, visual loss, anosmia | 2 yrs |
| Bokemeyer, et al., 1990 | autopsy | 7.5 | yes | confusion, personality change, memory loss | gait disturbance, incontinence, optic disc pallor | 12 yrs |
| present series | | | | | | |
| Case 1 | angio | 5 | yes | inappropriateness, memory loss | lethargy, gait disturbance | 9 mos |
| Case 2 | autopsy | 3.5 | no | inattentiveness, memory loss | gait disturbance | 3 mos |
| Case 14 | angio | 5 | yes | confusion, memory loss, inappropriateness | seizure | 4 yrs |
| Case 16 | angio | 3.5 | yes | slowness, confusion, memory loss | gait disturbance | 3 mos |
| Case 17 | angio | 5 | yes | apathy, disinterest, memory loss | depression, gait disturbance, bowel incontinence | 5 yrs |
| Case 18 | angio | 5 | no | disinterest, memory loss, inappropriateness | none | 2 yrs |

* NI = not indicated; time = time course before diagnosis.

cases displayed asymmetrical reduction in visual acuity, usually with profound loss in one eye and moderate-to-no loss in the other.[47] Visual fields commonly demonstrated bitemporal hemianopsia. Some reports provide a description of a gradual progression of visual field defects culminating in a complete bitemporal hemianopsia before definitive diagnosis.[8,31,32,36,61,65]

The average size at which ACoA aneurysms cause compression of the optic apparatus is 3.1 cm (Table 2). Previous aneurysm rupture—either remote[32,61] or recent[9,29]—may be a factor, as occurred in five cases. Visual loss has been reported with ACoA aneurysms smaller than 2.5 cm, but the inciting factor has been the impact of aneurysm rupture, often causing sudden monocular blindness.[44,52] Only one report exists of a subgiant aneurysm causing chiasmal compression without rupture.[2]

Aneurysms situated proximally on the ACA have more often been associated with a homonymous hemianopsia due to lateral chiasmal compression[12,34,63] than with the bitemporal features observed with ACoA region sacs.

### *Dementia Caused by Mass Effect on the Brain*

Dementia is uncommon in cases of unruptured giant intracranial aneurysms at any location.[8,14,30,43,58] In most cases it occurs as a result of obstructive hydrocephalus. Of the aneurysm locations predisposing to this, the basilar bifurcation is the most common.[8,14,53] Aneurysms at that location reach a size of 3 to 3.5 cm before hydrocephalus occurs.[8,53] At the ICA bifurcation and ACoA region, the diameter at presentation has often been considerably larger, from 5 to 7.5 cm.[5,7,8,40]

Even in reported series of aneurysms located in the ACoA region, dementia has received little mention.[37,51] Ten verified cases obtained from the literature are detailed in Table 3 in addition to the six cases in our series.[6,25,30,39,40,43,47,49] Of the 12 cases in which the exact size of the aneurysm was known, the average size was 4.6 cm with a range of 3 to 7.5 cm (measured at autopsy). Of the 10 cases in which ventricular size was indicated, hydrocephalus was manifest in eight.

Dementia with gait disturbance and, in some cases, bladder or bowel incontinence has been the dominant clinical picture. It has been contended that urinary symptoms antedate cognitive impairment by several months in patients with such aneurysms.[40] This is not supported by the autopsy case of a 7.5-cm aneurysm[6] or by those cases in the present series.

### *Surgical Treatment: Early Experiences*

Aneurysm wrapping is not effective. After wrapping, fatal rupture has been seen only 6 hours postoperatively[27] and up to 7 months after gauze and acrylic–reinforced wrapping.[68] Early attempts at trapping and excision were also dismal, with septal perforator injury and dementia.[8,32]

### *Surgical Treatment: Proximal Arterial Occlusion*

In the past, carotid artery ligation in the neck was used safely and effectively to treat giant ACoA aneurysms.[8,44,46] Significant recovery of visual acuity and even bitemporal hemianopsia was observed in cases of 3- to 3.5-cm-diameter aneurysms. Dott[13] extended the concept to intracranial ACA occlusion in 1944. Its use for giant sacs is not commonly reported;[38] only one case report prior to 1979 has been cited.[24] Odom and colleagues[46,59] tried to restrict the supply to ACoA aneurysms by ligating one of the ACAs and performing gradual occlusion of the opposite common carotid artery. No report exists of this method being used for a giant aneurysm.

Our experience has indicated that in the presence of a giant aneurysm, the potential for robust leptomeningeal collateral vessels following therapeutic $A_1$ or $A_2$ artery occlusion is high.[16] In this series, an $A_1$ or $A_2$ tourniquet (three cases), $A_1$ clip (two cases), and $A_1$ balloon (one case) were

all used without ACA infarction. In the three aneurysms presenting with mass effect, two required delayed surgical evacuation for worsening mass effect. Most significantly, in the case treated with balloon $A_1$ occlusion there was no angiographic evidence of a contralateral $A_1$ artery, yet the occlusion was tolerated. Bilateral leptomeningeal collateral vessels from both the MCA and posterior cerebral artery territories supported the distal ACA territories retrogradely to the subcallosal region (Fig. 3). We have found no published report in which this degree of collateral circulation following what amounts to bilateral ACA occlusion has been described. Frank E. Nulsen (personal communication, year unavailable) once successfully ligated both ACA $A_1$ segments without mishap.

*Surgical Treatment: Neck Occlusion*

A previous study concerning the treatment of 335 giant anterior circulation aneurysms showed that neck clipping was possible in 62% of the cases.[16] In particular, carotid ophthalmic artery (76%) and carotid communicating artery (79%) aneurysms were often treated in this way, whereas carotid bifurcation artery (23%), MCA (47%), and ACA (47%) aneurysms were less responsive to this treatment modality. Temporary trapping and suction decompression techniques[4,20] have been useful for giant aneurysms located below the carotid artery bifurcation.

In this series, neck clipping was successful in only seven of 14 attempts. The size of the aneurysm sac was a factor, as was the anatomy of the aneurysm neck. In two cases, at least 1 cm of separation existed between the entrance to the parent artery and its branches. Intraoperative clip slippage precluded neck clipping in two patients. The evacuation of intraluminal thrombus sometimes proved critical, with the important adjunctive treatment of ultrasonic aspiration.

Fifty cases have been reported of successful neck clipping or ligation of giant ACoA aneurysms.[19,26,28–30,33,35–37,40,45,54–57,60,67,69] The value of thrombus evacuation and temporary $A_1$ clipping has been emphasized.[40] Even temporary trapping of both $A_1$ and $A_2$ segments to allow safe bloodless removal of aneurysm contents has been used, with the duration of the trapping procedure ranging from 16 to 60 minutes.[35] However, others have used proximal $A_1$ occlusion alone with thrombectomy at the aneurysm base to minimize the temporary occlusion time.[55,58] We too have preferred proximal temporary occlusion over trapping, largely because of concern for the septal perforating arteries and their vulnerability to complete ischemia.

The early experience of using hypothermic circulatory arrest for treating giant ACoA aneurysms was unfavorable, resulting in death due to stroke or during rewarming.[9,60] However, more recent results have been encouraging, although still associated with ACA occlusion and infarction.[33,54]

*Endovascular Treatment*

Detachable balloon therapy of giant aneurysms has largely been used for occlusion of the internal carotid, vertebral, and basilar arteries. It can be performed at the $A_1$ segment of the ACA if the vessel is sufficiently long. Stroke may occur as a result of inadequate collateral circulation or direct occlusion of a medial lenticulostriate origin. However, as is the case with the tourniquet method, balloon therapy permits a test occlusion of the vessel in the awake patient before permanent occlusion.

Enthusiasm has waned for balloon deposition of hydroxyethylmethacrylate within aneurysms. Vargas and associates[62] reported the deflation and migration out of a giant ACoA aneurysm of a hydroxyethylmethacrylate-filled balloon, which lodged at the ICA bifurcation, causing permanent aphasia and hemiplegia, despite emergency balloon embolectomy.

Detachable coil occlusion of giant aneurysms with a neck wider than 4 mm is associated with delayed coil compaction and additional aneurysm enlargement.[18] Also, the frequency of thromboembolic ischemia during coil embolization may be higher with wide-necked aneurysms.[48,64] Nonetheless, detachable coils represent a useful alternative for unclippable aneurysms. The ability of coils to reduce aneurysm mass effect cannot be predicted, but appears to be long lasting in medium-term follow-up studies.

## Conclusions

In cases of giant ACoA aneurysms, at least 3.5 cm of aneurysm mass effect is required to cause dementia. Dementia is usually caused by direct brain compression by an aneurysm rather than hydrocephalus. Optic apparatus compression occurs with smaller aneurysms (2.7–3.2 cm) that are pointing inferiorly.

Neck clipping was possible in half of the cases in this series. Special techniques, including temporary clipping, evacuation of intraluminal thrombus, tandem and/or fenestrated clipping, and clip reconstruction were often required. Anterior cerebral artery occlusion or injury due to neck clipping was the main cause of poor outcome or death in this series.

Proximal ACA occlusion, even of dominant $A_1$ segments with only a small or no contralateral $A_1$ artery, was an effective treatment alternative and was well tolerated because of excellent leptomeningeal collateral circulation.

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This is the last paper submitted to the *Journal of Neurosurgery* in which Charles G. Drake, M.D., F.R.C.S.(C), is listed as a coauthor. Doctor Drake died in 1998.

### References

1. Alajouanine T, Nehlil J, Billet R: Anévrysme de la communicante antérieure avec hématome basilaire calcifié et porencéphalie frontale. **Rev Neurol 92:**397–401, 1955
2. Aoki N: Partially thrombosed aneurysm presenting as the sudden onset of bitemporal hemianopsia. **Neurosurgery 22:** 564–566, 1988
3. Bagley C Jr: Blood in the cerebrospinal fluid. Resultant functional and organic alterations in the central nervous system. B. Clinical data. **Arch Surg 17:**39–81, 1928
4. Batjer HH, Samson DS: Retrograde suction decompression of giant paraclinoidal aneurysms. Technical note. **J Neurosurg 73:**305–306, 1990
5. Beadles CF: Aneurysms of the larger cerebral arteries. **Brain 30:**285–336, 1907

6. Bokemeyer C, Frank B, Brandis A, et al: Giant aneurysm causing frontal lobe syndrome. **J Neurol 237:**47–50, 1990
7. Bramwell B: Two enormous intra-cranial aneurysms. **Edinb Med J 32:**911–922, 1887
8. Bull J: Massive aneurysms at the base of the brain. **Brain 92:** 535–570, 1969
9. Cullen JF, Haining WM, Crombie AL: Cerebral aneurysms presenting with visual field defects. **Br J Ophthalmol 50:** 251–256, 1966
10. Dandy WE: Aneurysm of the anterior cerebral artery. **JAMA 119:**1253–1254, 1942
11. Dandy WE: **Intracranial Arterial Aneurysms.** Ithaca, NY: Comstock, 1944
12. Doran S, Leibrock L, Mawk J, et al: Therapy of giant intracranial aneurysms in the elderly. **Nebr Med J 75:**165–170, 1990
13. Dott NM: Intracranial aneurysmal formations. **Clin Neurosurg 16:**1–16, 1969
14. Drake CG: Giant intracranial aneurysms: experience with surgical treatment in 174 patients. **Clin Neurosurg 26:**12–95, 1979
15. Drake CG: Report of World Federation of Neurological Surgeons Committee on a universal subarachnoid hemorrhage grading scale. **J Neurosurg 68:**985–986, 1988 (Letter)
16. Drake CG, Peerless SJ, Ferguson GG: Hunterian proximal arterial occlusion for giant aneurysms of the carotid circulation. **J Neurosurg 81:**656–665, 1994
17. Durston JHJ, Parsons-Smith BG: Blindness due to aneurysm of anterior communicating artery. With recovery following carotid ligation. **Br J Ophthalmol 54:**170–176, 1970
18. Fernandez Zubillaga AH, Guglielmi G, Viñuela F, et al: Endovascular occlusion of intracranial aneurysms with electrically detachable coils: correlation of aneurysm neck size and treatment results. **AJNR 15:**815–820, 1994
19. Fisher A, Cooper RL: On the visual disturbances associated with massive basal aneurysms. **Proc Aust Assoc Neurol 13:** 97–107, 1976
20. Flamm ES: Suction decompression of aneurysms. Technical note. **J Neurosurg 54:**275–276, 1981
21. Folstein MF, Folstein SE, McHugh PR: "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. **J Psychiatr Res 12:**189–198, 1975
22. Forbes G, Fox AJ, Huston J III, et al: Interobserver variability in angiographic measurement and morphologic characterization of intracranial aneurysms: a report from the International Study of Unruptured Intracranial Aneurysms. **AJNR 17:** 1407–1415, 1996
23. Fouché CH: Aneurysm of the anterior communicating artery of the brain. **Proc R Soc Med 24:**1471–1472, 1931
24. Fox JL: **Intracranial Aneurysms.** New York: Springer-Verlag, 1983, Vol 3, pp 1351–1352
25. Gilman S, Braverman LE, Starr A, et al: Intracranial aneurysm causing panhypopituitarism, blindness, seizures and dementia. **Ann Intern Med 57:**639–646, 1962
26. Hamburger C, Schönberger J, Lange M: Management and prognosis of intracranial giant aneurysms. A report on 58 cases. **Neurosurg Rev 15:**97–103, 1992
27. Heiskanen O, Nikki P: Large intracranial aneurysms. **Acta Neurol Scand 38:**195–208, 1962
28. Heros RC: Giant aneurysms, in Ojemann RG, Ogilvy CS, Crowell RM, et al (eds): **Surgical Management of Neurovascular Disease, ed 3.** Baltimore: Williams & Wilkins, 1995, pp 323–367
29. Højer-Pedersen E, Haase J: Giant anterior communicating artery aneurysm with bitemporal hemianopsia: case report. **Neurosurgery 8:**703–706, 1981
30. Hosobuchi Y: Direct surgical treatment of giant intracranial aneurysms. **J Neurosurg 51:**743–756, 1979
31. Jefferson G: Compression of the chiasma, optic nerves, and optic tracts by intracranial aneurysms. **Brain 60:**444–497, 1937
32. Jefferson G: Further concerning compression of the optic pathways by intracranial aneurysms. **Clin Neurosurg 1:**55–103, 1953
33. Jolin Å, Edén E, Berggren H, et al: Management of a giant intracranial aneurysm using surface-heparinized extracorporeal circulation and controlled deep hypothermic low flow perfusion. A case report. **Acta Anaesthesiol Scand 37:**756–760, 1993
34. Klemme RM, Woolsey RD: Suprasellar aneurysm. Report of a case, with recovery. **Arch Neurol Psychiatry 47:**662–666, 1942
35. Kodama N, Suzuki J: Surgical treatment of giant aneurysms. **Neurosurg Rev 5:**155–160, 1982
36. Lavyne MH, Kleefield J, Davis KR, et al: Giant intracranial aneurysms of the anterior circulation: clinical characteristics and diagnosis by computed tomography. **Neurosurgery 3:** 356–363, 1978
37. Lawton MT, Spetzler RF: Surgical management of giant intracranial aneurysms: experience with 171 patients. **Clin Neurosurg 42:**245–266, 1995
38. Logue V: Surgery in spontaneous subarachnoid haemorrhage. Operative treatment of aneurysms on the anterior cerebral and anterior communicating artery. **Br Med J 1:**473–479, 1956
39. Maurice-Williams RS: **Subarachnoid Haemorrhage: Aneurysms and Vascular Malformations of the Central Nervous System.** Bristol: Wright, 1987, p 112
40. Maxwell RE, Chou SN: Aneurysmal tumors of the basifrontal region. **J Neurosurg 46:**438–445, 1977
41. Miller NR: **Walsh and Hoyt's Clinical Neuro-Ophthalmology, ed 4.** Baltimore: Williams & Wilkins, 1991, pp 2044–2047
42. Mitchell SW: Aneurism of an anomalous artery causing anteroposterior division of the chiasm of the optic nerves and producing bitemporal hemianopsia. **J Nerv Ment Dis 16:**44–62, 1889
43. Morley TP, Barr HWK: Giant intracranial aneurysms: diagnosis, course, and management. **Clin Neurosurg 16:**73–94, 1969
44. Norlén G, Barnum AS: Surgical treatment of aneurysms of the anterior communicating artery. **J Neurosurg 10:**634–650, 1953
45. Norlén G, Olivecrona H: The treatment of aneurysms of the circle of Willis. **J Neurosurg 10:**404–415, 1953
46. Odom GL: Ophthalmic involvement in neurological vascular lesions. **Neuroophthalmology 1:**228–231, 1964
47. Peiris JB, Ross Russell RWR: Giant aneurysms of the carotid system presenting as visual field defect. **J Neurol Neurosurg Psychiatry 43:**1053–1064, 1980
48. Pelz DM, Lownie SP, Fox AJ: Thromboembolic events associated with the treatment of cerebral aneurysms with Guglielmi detachable coils. **AJNR 19:**1541–1547, 1998
49. Pia HW, Zierski J: Giant cerebral aneurysms. **Neurosurg Rev 5:**117–148, 1982
50. Rosta L, Battaglia R, Pasqualin A, et al: Italian cooperative study on giant intracranial aneurysms: 2. Radiological data. **Acta Neurochir Suppl 42:**53–59, 1988
51. Sarwar M, Batnitzky S, Schechter MM: Tumorous aneurysms. **Neuroradiology 12:**79–97, 1976
52. Schisano G: Large anterior communicating artery aneurysm with temporal hemianopsia. Case report. **J Neurosurg Sci 29:** 267–271, 1985
53. Soeur M, Brihaye J, Moerman C: Pseudo-tumoural aneurysm in the third ventricle: report of three cases. **Acta Neurochir 45:** 247–258, 1979
54. Soloman RA, Smith CR, Raps EC, et al: Deep hypothermic circulatory arrest for the management of complex anterior and posterior circulation aneurysms. **Neurosurgery 29:**732–738, 1991
55. Sundt TM Jr: **Surgical Techniques for Saccular and Giant Intracranial Aneurysms.** Baltimore: Williams & Wilkins, 1990
56. Sundt TM Jr, Piepgras DG: Surgical approach to giant intracranial aneurysms. Operative experience with 80 cases. **J Neurosurg 51:**731–742, 1979
57. Suzuki J, Sato T: Direct surgical treatment of giant intracranial aneurysms. **Neurol Res 2:**305–325, 1980
58. Symon L: Surgical experiences with giant intracranial aneurysms. **Acta Neurochir 118:**53–58, 1992

59. Tindall GT, Kapp J, Odom GL, et al: A combined technique for treating certain aneurysms of the anterior communicating artery. **J Neurosurg 33:**41–47, 1970
60. Uihlein A, Theye RA, Dawson B, et al: The use of profound hypothermia, extracorporeal circulation and total circulatory arrest for an intracranial aneurysm. Preliminary report with reports of cases. **Proc Mayo Clin 35:**567–576, 1960
61. Van't Hoff W, Hornabrook RW, Marks V: Hypopituitarism associated with intracranial aneurysms. **Br Med J 2:** 1190–1194, 1961
62. Vargas ME, Kupersmith MJ, Setton A, et al: Endovascular treatment of giant aneurysms which cause visual loss. **Ophthalmology 101:**1091–1098, 1994
63. Versavel M, Witmer JP, Matricali B: Giant aneurysm arising from the anterior cerebral artery and causing an isolated homonymous hemianopsia. **Neurosurgery 22:**560–563, 1988
64. Viñuela F, Duckwiler G, Mawad M: Guglielmi detachable coil embolization of acute intracranial aneurysm: perioperative anatomical and clinical outcome in 403 patients. **J Neurosurg 86:** 475–482, 1997
65. Wakeley CPG: The surgery of the parapituitary region. **M Press 194:**337–346, 1937
66. Walsh FB, King AB: Ocular signs of intracranial saccular aneurysms. Experimental work on collateral circulation through the ophthalmic artery. **Arch Ophthalmol 27:**1–33, 1942
67. Wappenschmidt J, Bettag W: [An aneurysm simulating a suprasellar space-occupying process and its treatment.] **Neurochirurgia 3:**193–202, 1960 (Ger)
68. Whittle IR, Dorsch NW, Besser M: Giant intracranial aneurysms: diagnosis, management, and outcome. **Surg Neurol 21:** 218–230, 1984
69. Yaşargil MG: **Microneurosurgery. Vol 2: Clinical Considerations, Surgery of the Intracranial Aneurysms and Results.** New York: Thieme Stratton, 1984, p 198

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*Address reprint requests to:* Stephen P. Lownie, M.D., F.R.C.S.(C), London Health Sciences Centre, University Campus, 339 Windermere Road, London, Ontario N6A 5A5, Canada. email: lownies@lhsc.on.ca.