Hunterian proximal arterial occlusion for giant aneurysms of the carotid circulation

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Hunterian proximal artery occlusion was used in the treatment of 160 of 335 patients harboring giant aneurysms of the anterior circulation. One hundred and thirty-three of these aneurysms arose from the internal carotid arteries, 20 from the middle cerebral arteries, and seven from the anterior cerebral arteries. Ninety percent of the patients had satisfactory outcomes. The safety of internal carotid artery occlusion has been greatly enhanced by preoperative flow studies and by test occlusion with an intracarotid balloon to identify those patients who require preliminary extracranial-to-intracranial bypass, which was used in all of the middle cerebral occlusions. The anterior cerebral artery had magnificent leptomeningeal collateral flow that prevented infarction even without cross flow. Obliteration of the aneurysm by thrombosis was complete, or nearly so, in all but four patients whose treatment was completed. Analysis of poor outcome in 16 patients revealed that hemodynamic ischemic infarction was known to occur after only two of the carotid occlusions.

KEY WORDS • giant aneurysm • carotid artery occlusion • extracranial-to-intracranial bypass procedure • outcome

Hunterian proximal arterial occlusion has been performed on every major intracranial artery presenting to our institution, mostly for giant aneurysms considered otherwise inoperable at the time, but occasionally as an alternative that has been deemed safer.

The origin, size, and shape of giant aneurysms and the extent of their mural thrombosis were described and illustrated by tracings in our early experience. There have been no significant changes since then except for the recognition of a significant number of nonatherosclerotic fusiform aneurysms in 15% of that series. These occurred in a younger age group and although some may have arisen from the expansion of the base of saccular aneurysms, most were presumed to have had their origin in an arterial wall disorder of unknown nature.

Clinical Material and Methods

Since we began performing these operations in 1961, our surgical experience now includes 723 patients with giant aneurysms of which 335 were on the anterior circulation (Table 1).Nearly one-half of the giant anterior aneurysms (47%) were treated by clipping the neck of the sac (62%, excluding the 82 infraclinoid aneurysms) but only 31% of the giant sacs that arose from the posterior circulation were clipped (Table 2). For most of the remaining operations, proximal occlusion was used in 48% of the anterior and in 61% of the posterior aneurysms (Table 1). This paper will be restricted to our experience with proximal occlusion of giant aneurysms on the carotid circulation. Because trapping is merely an extension of the Hunterian principle, this method has been included. The incidence of proximal occlusion has not changed over the years except in regard to giant carotid ophthalmic aneurysms, most of which can now be clipped.

Proximal carotid occlusion was used for all giant petrous and cavernous aneurysms discussed in this study. Most giant intracranial saccular and all obviously fusiform aneurysms demand preoperative consideration to determine whether proximal occlusion is the only reasonable alternative to clipping. Virtually all of the intracranial giant anterior saccular aneurysms were explored first to determine whether neck clipping was feasible and whether the patients were prepared for the possibility of proximal occlusion.
sion with or without bypass. The results for all methods are included in Table 3.

Outcomes other than morbidity that occurred in the hospital were often incompletely followed because of the large referral of patients from distant or overseas units. Whenever follow-up examination or control angiography permitted, the recovery of vision or ocular muscle paresis and the degree of thrombosis of the sac were recorded.

**Carotid Occlusion**

In the 1950's, Sir Geoffrey Jefferson described carotid occlusion as the sheet anchor for the treatment of carotid aneurysms (G. Jefferson, unpublished observation, 1958). Its former wide use has been abandoned for surgery on ordinary smaller aneurysms since the advent of microsurgery and microtechniques and tools, but carotid occlusion continues to play an important role in certain giant carotid aneurysms. The danger of cerebral ischemia and infarction after carotid occlusion has been greatly reduced by decreasing the risk of thromboembolism and by preoperative studies to determine the potential for collateral flow, which, if inadequate, can be supplemented by surgical arterial bypass.

Thromboembolism has been reduced by performing abrupt rather than gradual occlusion of the proximal vessel and by doing so as close to the origin of the sac as possible to minimize the volume of the isolated segment and eliminate flow into it. Since 1978, the use of the detachable balloon, the administration of acetylsalicylic acid, and the assurance of adequate hydration have all been thought to be important in minimizing the risk of an embolus migrating from the thrombosing sac.

The preoperative studies used to evaluate collateral flow have included angiographic demonstration of cross flow from the opposite carotid and ipsilateral posterior communicating artery (PCoA) to visualize the collateral flow at the circle of Willis; test occlusion of the carotid artery with a temporary, proximal balloon in the conscious patient; and cerebral blood flow studies using xenon-131 without and then with temporary ipsilateral carotid compression or balloon occlusion. When this information reveals marginal or low collateral potential, an extracranial to intracranial (EC-IC) bypass should be performed before the internal carotid artery (ICA) is occluded, using the superficial temporal artery or interpositional vein graft, if necessary. Miller, et al., believed that carotid occlusion was safe if the cerebral blood flow during temporary occlusion was in the range of 20 to 40 ml/100 gm/min and flow reduction was less than 25%. Our inclination has been to use EC-IC bypass if the flow was below 35 ml/100 gm/min. Before the bypass era, ICA occlusion was done gradually, using the Selverstone clamp placed on its proximal cervical segment to test the collateral supply of the communicating artery. Except for four early cases of carotid cavernous aneurysms in which the ICA was abruptly ligated with heavy silk or umbilical tape, the clamp

<table>
<thead>
<tr>
<th>Location of Aneurysm</th>
<th>No. of Cases</th>
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**TABLE 2**

Methods of treating giant anterior aneurysms*

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* MCA = middle cerebral artery; ACA = anterior cerebral artery.

**TABLE 3**

Results of treating giant anterior aneurysms*

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* MCA = middle cerebral artery; ACA = anterior cerebral artery.
was used in the first 45 carotid aneurysms in our series. Balloon occlusion was first used in our institution in 1978, and we first used the microtourniquet in 1975, again as a means of reversible testing of the adequacy of the natural or surgical collateral and as a method to more precisely occlude an intracranial vessel and spare local branches and perforators. An EC-IC bypass was considered to be essential for all middle cerebral artery (MCA) occlusions or isolation of the aneurysm. The microtourniquet proved invaluable in assessing the patency and efficiency of the collateral artery while the patient was awake. In contrast, bypass was not considered essential for giant anterior cerebral artery (ACA) aneurysms, and reliance was placed on temporary, reversible tourniquet occlusion of the proximal ACA segment.

We have continued to perform ICA occlusion because of the new measures for safety that can be employed and our experience with too many incomplete aneurysm thromboses and recurrences seen after common carotid artery occlusion. In this series, carotid occlusion for carotid aneurysms was attempted in 133 patients using internal carotid occlusion in 131 and common carotid occlusion in two. Two proximal middle cerebral aneurysms were also treated by intracranial ICA occlusion.

**Giant Petrous and Cavernous Carotid Aneurysms**

We have treated five giant petrous and 77 cavernous carotid aneurysms via ICA occlusion. Three of the five petrous aneurysms probably were infectious in origin, resulting from carotid arteritis. Two patients had pre-existing chronic middle ear infection and apical petrositis; in the other patient, who had only otitis media, the “tumor” that was seen behind the tympanum was biopsied and the bleeding led to an angiogram and diagnosis of a giant petrous aneurysm. At least two of the cavernous aneurysms were known to arise from smaller saccular aneurysms, an origin presumed to be common to most. One case involved both the petrous and cavernous carotid arteries. Three other cases were associated with and had probably caused carotid-cavernous fistulae.

**Treatment and Outcome.** With the exception of two early patients in whom the common carotid artery was occluded, all remaining giant petrous and cavernous carotid aneurysms in our series have been treated by ICA occlusion. In earlier years occlusion was performed with a Selverstone clamp, occasionally by ligation, but more recently and in larger numbers by detached balloons. In one patient, a persistently flowing trigeminal artery also had to be clipped intracranially as it entered the clivus when it continued to fill the carotid siphon and the aneurysm above the balloon occlusion. In this series of 82 patients, there was only one poor result and one death, both in the cavernous group and both occurring after the cervical ICA had been clamped. The patient with the poor outcome depl-
Proximal arterial occlusion for giant aneurysms

oped a sudden contralateral hemiplegia and aphasia 12 hours after closure of the Selverstone clamp, which resulted from a large embolus that arose from the thrombosing aneurysm that occluded her carotid bifurcation. The death resulted from an acute subdural hematoma that developed after the bypass had been disrupted; the patient had been receiving heparin for postoperative transient ischemic attacks 5 days after closing the carotid clamp. The patient died despite evacuation of the clot and repair of the bypass. All of the other 14 bypasses remained open.

Four patients had sudden transient ischemic episodes after carotid occlusion, which almost certainly resulted from small emboli emerging from the thrombosing aneurysm, but they experienced no persistent deficit. In several patients, a more slowly developing hemiparesis and/or dysphasia, thought to be due to insufficient collateral flow, was promptly reversed with temporary use of volume expansion and/or hypertension for a few days to 1 week.

Complete thrombosis of the aneurysm occurred in the 80 cases with control angiography, and there were no known recurrences. A marked increase in ophthalmic paresis, sometimes to complete ophthalmoplegia, occurred in six patients after carotid occlusion. Transient retro-orbital discomfort was common. This complaint must be the result of a slight enlargement of the aneurysm from swelling of the fresh thrombus. In all but one patient, whose ophthalmoplegia remained complete, there was early recovery to the preoperative state or better. On long-term follow-up examination, it was noted that recovery of preoperative ophthalmoplegia was complete in eight cases, improved in six, and unchanged in six others.

Four of the cavernous aneurysms were trapped at subsequent craniotomy by intracranial carotid clipping. Two of these procedures were performed to relieve optic nerve compression and one to prevent thromboembolism. The fourth patient had had eight balloons detached in the aneurysm with near but incomplete obliteration. He returned 5 weeks later with complete ophthalmoplegia and a blind eye. Remarkably, an abscess had developed within the aneurysm from swelling of the fresh thrombus. In all but one patient, whose ophthalmoplegia remained complete, there was early recovery to the preoperative state or better. On long-term follow-up examination, it was noted that recovery of preoperative ophthalmoplegia was complete in eight cases, improved in six, and unchanged in six others.

Fig. 1. Angiograms in a 58-year-old woman showing a giant, partially thrombosed carotid ophthalmic aneurysm (right), and the middle cerebral artery occluded by an embolus from the thrombosing aneurysm (left).

Summary. The success rate of ICA occlusion for giant petrous and cavernous aneurysms, particularly with the use of detached balloons and a bypass procedure when necessary, must be contrasted with the radical procedures used by skull-base surgeons who employ clipping, even excising of these masses by use of interpositional grafts to retain or restore carotid artery continuity. Although there has been some argument against sacrificing a carotid artery for the long-term future of the patient, we doubt that this notion is often sufficient for most patients to choose to face the risks of intracavernous procedures. Moreover, in our series, no new aneurysm has been known to develop on the contralateral carotid artery and an existing untreated aneurysm was not known to have enlarged.

Giant Carotid Ophthalmic and Paraophthalmic Aneurysms

We treated 109 giant carotid ophthalmic and paraophthalmic aneurysms, and used ICA occlusion in 23, most of these early in the series (Table 4).

Treatment and Outcome. The carotid artery was occluded by Selverstone clamp in 14 patients or ligation in three patients, and all three of the poor outcomes occurred in this group. The first patient rebled above a severe clamp stenosis that was used to prevent rebleeding for a delayed intracranial operation and died. Another patient, who had a functioning bypass, suffered a major hemisphere infarction 19 hours after carotid artery occlusion from a middle cerebral embolus that formed in the thrombosing aneurysm (Fig. 1). In the third patient, a portion of the ICA was avulsed from its origin when the stem was detached from the closed Selverstone clamp. She remained well with intermittent finger occlusion on the incision until an attempt at repair of the artery was made and resulted in occlusion of the common and external carotid arteries, hemiplegia, and aphasia from loss of external carotid collateral supply.

Outcome. All six patients in whom detached balloon occlusion was performed fared well, although a 67-year-old woman developed a paresis of the opposite foot that recovered completely after 1 month.

Two of the 11 bypasses failed. One interpositional vein graft thrombosed, but the patient tolerated detached balloon occlusion. The other patient would tolerate only a severe clamp stenosis and the lower third of the aneurysm remained open; however, she has remained well for 10 years during follow-up study.

Five of these patients had an additional intracranial
carotid clipping to trap the aneurysm, three to relieve optiocochiasmatic compression and two to prevent embolism. In one of the latter patients, the balloon detached in the carotid artery, and migrated into and floated in the aneurysm; the other patient had an additional intracranial carotid clipping to prevent further thromboembolism that had already occluded the MCA.

Thrombosis in the aneurysm was known to be complete in 11 patients, nearly so in five, and incomplete in one other patient in whom the bypass (failed and who would tolerate only a severe clamp stenosis. The small portions remaining open at the aneurysmal necks of the incomplete cases filled either from the ophthalmic arteries or retrogradely from the PCoA's. In none of the incomplete thromboses is the aneurysm known to have recurred.

Significant recovery of vision occurred in five patients, but there was little change in the other five known cases.

Giant Carotid Communicating Aneurysms

Most giant carotid communicating aneurysms can be clipped; in only seven of our 39 patients was ICA occlusion used (Table 4).

Treatment and Outcome. There were no poor outcomes; one man was already hemiplegic after an angiogram in the referring institution, but he nevertheless desired repair of his aneurysm.

In three patients, an EC-IC bypass procedure was deemed essential and each remained patent. One was still intolerant of complete occlusion, but his aneurysm thrombosed completely above a severe clamp stenosis.

Four of the seven patients had complete thrombosis of their aneurysm and another nearly so. Giant carotid communicating aneurysms in the other two patients with partial thrombosis and slow filling of the lower sac from ophthalmic arteries or PCoA's have not enlarged over 6 to 9 years.

Ipsilateral severe visual loss in one patient recovered dramatically to reading newsprint over a lower altitudinal defect, but another patient developed immediate and unexplained severe visual loss in the ipsilateral eye.

Summary. Carotid occlusion for giant carotid ophthalmic and communicating aneurysms will seldom be needed in the future, for most can now be clipped using the tandem method. This is particularly true for treating aneurysms in the ophthalmic artery using the technique described by Batjer and Samson to collapse the sac for more complete dissection of the neck and clipping.

Giant Carotid Bifurcation Aneurysms

Thirty patients with giant bulbous carotid bifurcation aneurysms were treated in our series. Of these, only seven of the aneurysms could be clipped for fear of kinking the origin of the A_1 or M_1 segment or both. Two early cases were only explored: one patient died from massive edema and the other refused balloon carotid occlusion.

Treatment and Outcome. Twenty-one patients were treated by carotid occlusion in the neck or intracranially when the A_1 segment had to be occluded to prevent cross flow. Two of these patients had giant aneurysms treated by trapping of the carotid bifurcation. An EC-IC bypass was used in 16 patients (Table 4).

The effect of ICA occlusion in the aneurysmal neck or in its petrous segment is jeopardized by continued filling of the sac retrogradely and through cross flow from the ophthalmic, anterior, and/or PCoA's. Of the 11 patients in whom only cervical ICA occlusion was attempted (five in whom the clamp was used, six the balloon), it is remarkable that in eight patients in whom cross flow existed and ICA occlusion alone was completed in the neck by clamp (two) or balloon (six), thrombosis in the sac was complete in six and nearly complete in another. The other patient with 60% thrombosis remains well under observation for 7 years.

One patient with balloon occlusion had had the A_1 segment clipped at the time of exploration and another patient, in whom cross flow still filled the aneurysm, had the A_1 subsequently occluded by tourniquet. Both patients had complete thrombosis of their aneurysms.

Of the five clamp placements, in only three was the carotid artery occluded with complete thrombosis. One of these, a 21-year-old woman with lupus erythematosus had no cross flow because of spontaneous A_1 occlusion (Fig. 2). After undergoing a bypass procedure, she tolerated complete ICA clamp occlusion.

Both poor outcomes in this group of 13 patients with extracranial carotid occlusion occurred in the clamp group and in neither was the carotid permanently occluded. The first example of such a patient, whose operation took place prior to the bypass era, had a large intracerebral hemorrhage after the opening of the clamp for a hemiplegia that developed 10 hours after the occlusion. The other patient died from rebleeding when clamp closure was delayed for 1 month to allow maturation of the feeble bypass graft.

In six patients, vigorous cross flow from anterior communicating arteries (ACoA's) and/or PCoA's demonstrated at angiography made ICA occlusion alone unlikely to be successful. In the first patient with cross filling from both sources, two tourniquets were placed before the bypass was done, one on the A_1 segment near its origin from the aneurysm and the other on the terminal carotid artery above the origin of the choroidal artery. This placement allowed temporary testing of complete isolation of the aneurysm and the whole MCA from either carotid artery flow. The patient tolerated this isolation and complete thrombosis of the aneurysm occurred. There was good retrograde filling of the M_1 segment, and the lenticulostriates from the bypass and the tourniquets were trimmed and buried on the second day.

As a result of the success of this outcome, the
A1 segment was clipped in the next three patients, and tourniquet occlusion only of the terminal carotid artery was used with functioning bypasses (Fig. 3). All patients had good outcomes with complete thrombosis of their aneurysms.

The fifth patient with a 5-cm aneurysm and increasing hemiparesis had the A1 segment clipped, but it was not possible to place the tourniquet on the carotid artery beyond the choroidal artery, which arose near the base of the sac. Although the bypass graft was strong, to ensure choroidal filling the snare was placed and subsequently closed on the carotid artery below the PCoA. The aneurysm thrombosed except for a 1-cm portion at the neck that filled feebly retrograde from the PCoA and also filled the choroidal artery. Although there was dramatic improvement of the hemiparesis after 2 months, the refilling of the neck enlarged to 2 cm. At re-exploration, another tourniquet was placed on the carotid artery above the origin of the PCoA. Occlusion of the carotid artery here was uneventful, even though the anterior choroidal artery was no longer seen and the aneurysm underwent complete thrombosis.

The sixth patient had good cross flows, but without filling of the aneurysm; ICA clamp occlusion in the neck was followed by refilling of the sac retrogradely from the PCoA and caused recurrent headache. Two years later, tourniquet occlusion of the terminal carotid was tolerated without a bypass graft. Although the dome of the aneurysm thrombosed, there was now some cross filling from the ACoA, but the patient has remained symptom-free for 9 years.

In two other patients with bypass grafts, the aneurysm was trapped with terminal carotid artery and proximal A1 and M1 clipping. The first was done when the aneurysm was torn at its neck, but nevertheless had an excellent outcome. The other patient with a huge aneurysm became drowsy and hemiparetic after terminal carotid artery clipping. After the first successful trapping, it was believed that trapping and evacuation of this mass might stop his decline. After initial improvement, he deteriorated after 24 hours and died from massive brain swelling.

All 17 of the completed ICA occlusions, with or without A1 occlusion and bypass, had a good outcome, and all but one case resulted in complete or near-complete thrombosis of the aneurysm. Remarkable, too, is that one of the two trappings was uneventful despite the potential of ganglionic infarction from perforating vessels that usually arise from the bifurcation region.

**Summary.** Thrombosis in an aneurysm becomes most complete when no or tiny branches arise from the artery between the ICA occlusion site and the
aneurysm, such as that seen in cavernous aneurysms. Retrograde flow from the ophthalmic, and/or PCoA’s, or ACoA’s tends to keep at least part of the supraclini- 
doid aneurysms open and also enhances the danger of thromboembolism from the thrombosing sac by this in and out flow. Even so, it is remarkable how few of the giant supraclinoid carotid aneurysms’ partial filling continued above ICA occlusion alone; in addition, continued enlargement or bleeding is not known to have occurred.

Giant Middle Cerebral Aneurysms

Middle cerebral artery occlusion was safely employed in 26 cases of giant middle cerebral aneurysm in our series, although other techniques had been used. In 29 of 55 giant middle cerebral aneurysms, neck clipping seemed impossible. Before use of the bypass technique, six of these aneurysms were wrapped with cotton (one) or cotton gauze (five); and four of the patients had good outcomes over long periods of time, including a 16-year-old boy who was already hemiplegic, and another patient whose aneurysm doubled in size despite use of a plastic restraint. Two patients had poor outcomes from early rebleeding. Three early cases were only explored when the superficial temporal artery was deemed too small for an effective bypass.

Treatment and Outcome. The remaining 20 patients accepted proximal occlusion treatment (Table 5). Two of the giant middle cerebral aneurysms on M1 branches were trapped and excised without deficit. One, in the right hemisphere, arose from an enlarged temporal branch feeding a sylvian arteriovenous malformation. The other, arising from the left MCA, enlarged despite being massively thrombosed, and had caused a dementing dysphasic syndrome. Both patients had excellent outcomes.

Internal carotid artery occlusion was used in two patients who had huge proximal middle cerebral aneurysms. A 35-year-old man with an 8-cm aneurysm, already hemiplegic and aphasic, had intractable headache. As there was no demonstrable filling of the MCA branches, the terminal carotid artery was clipped intracranially and the sac evacuated. His pain was thus relieved and, remarkably, he recovered useful function of his right leg and some speech. In another patient with a large fusiform aneurysm of the terminal carotid artery and MCA but no M1 segment, balloon occlusion in the carotid siphon after bypass was followed by uneventful and complete occlusion of the aneurysm.

In 16 patients, the only reasonable alternative was to try to occlude the MCA proximal to the aneurysm in the presence of an EC-IC bypass. In 1975, a tourniquet, which had been developed for basilar artery occlusion or stenosis, was used to occlude the MCA while testing the collateral flow of the bypass when the patient was awake. The first patient had only transient ischemia reversed with hypertension and her aneurysm thrombosed completely. Encouraged by this outcome, attempts were made at M1 occlusion in the presence of a bypass in 15 more patients with giant middle cerebral aneurysms. In one patient who was already hemiplegic, the M1 segment was occluded using a clip at the time of the bypass procedure. The tourniquet was used in 15 patients overall and was placed on the MCA beyond the lenticulostrate perforating vessels in 10 but proximal to them in five.

Twelve of the 15 patients who underwent bypass and M1 tourniquet occlusions had very successful outcomes, including two patients who needed further treatment. One 13-year-old girl became stuporous with mass effect after M1 occlusion, but she recovered completely after reopening the craniotomy, and trapping and evacuating the aneurysm. The other patient, a 55-year-old man, had had his common carotid artery clamp closed 13 years before; subsequently, declining vision in the left eye led to the discovery of the mass. For a year after a tourniquet occlusion of the M1 segment, he improved but only to decline again with a temporal hemianopsia in the right eye as well. Restudy showed that the aneurysm had enlarged enormously because the tourniquet had “slipped” partially, thus allowing a refilling of the sac and occluding the vein bypass. Two years after the first occlusion was performed, the M1 segment was reoccluded with a clip and the medial portion of the aneurysm was evacuated. There was excellent collateral filling of M2 branches and although the patient’s field defect recovered on the right, he had only light perception in the left eye.

Three of the patients with tourniquets had poor outcomes. One, a 64-year-old man with a huge aneurysm, awakened from anesthesia with a hemiplegia after mere placement of the tourniquet. Closure of the tourniquet, followed by trapping and evacuation was not helpful. It was presumed that he had a lenticulostrate infarction as a result of the exploration. Another patient, a woman who was 46 years of age, tolerated M1 occlusion for only 4 hours before becoming hemiplegic. After emergency opening of the snare and then creation of a severe M1 stenosis that she tolerated, this intact aneurysm ruptured fatally 2 days

Table 5

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* E = excellent; G = good; P = poor; D = deceased; ( ) = no. of patients who had a bypass procedure; MCA = middle cerebral artery.
† Preoperative hemiplegia was noted.
‡ Four bypasses not patent, one was feeble.
later. The third poor outcome was in a patient who tolerated M1 occlusion for 1 week before becoming hemiparetic and dysphasic from several M2 branch occlusions due to embolism from the thrombosing aneurysm; recovery was incomplete.

Six patients were at first intolerant of complete occlusion. Two needed only drug-induced hypertension for several days before weaning. Four tolerated a severe M1 stenosis and in three of these cases, a complete occlusion was tolerated 1 day, 12 days, and 6 months later, respectively. The other aneurysm mentioned above burst beyond the stenosis 2 days later and the patient died.

In one patient with a 4-cm fusiform aneurysm, the lenticulostriate vessels arose from a smaller proximal dilatation. A deliberate, severe stenosis was created just proximal to this dilatation to preserve the flow in these vessels. Remarkably, the rest of the aneurysm thrombosed completely with no ill effect (Fig. 4).

Summary. This unique experience illustrates that the MCA can be occluded safely in most patients in the presence of a bypass graft, and can even be totally isolated by terminal carotid and A1 occlusion. By adding the 14 patients with M1 isolation by the A1 segment and terminal carotid artery occlusion to the 16 M1 occlusions, there are 30 cases of MCA occlusion with only four poor outcomes, a mere two of which were clearly related to bypass inefficiency.

What is more remarkable is that in four patients, the bypass did not remain open postoperatively and in one it was feeble, yet there was no hemiplegia. This resulted from the presence of luxuriant leptomeningeal collateral flow from anterior and posterior cerebral arteries that filled M1 branches retrogradely, even back to the thrombosed aneurysm (Fig. 5).

Furthermore, there must have been spontaneous collateral flow to the lenticulostriate territory in three of the five fusiform aneurysms that became thrombosed but from which these vessels must have arisen, because these patients had no infarction.

Giant Anterior Cerebral Aneurysms

Anterior cerebral artery occlusion was successfully employed in 5 cases of giant anterior cerebral aneurysm. Only nine of 19 giant anterior cerebral aneurysms in our series could be clipped, three of these after preliminary evacuation of the sac was undertaken. In seven cases, occlusion of the A1 or A2 segment was performed because, remarkably, each aneurysm was fusiform in nature (Fig. 6). Three aneurysms arose from the A1 segment near but not at its origin. In two, there was no cross flow, in one of these because a surgeon had somehow clipped the ACoA instead of the aneurysm. Four arose at or near the origin of the A2 segment.

Treatment and Outcome. Not knowing the potential for the A1 or A2 segment natural collateral flow, tourniquet occlusion of either the A1 or A2 in the awake patient was used in five of the seven cases. Even though the A1 or A2 segment or both were completely isolated from ACoA cross flow, no infarction occurred in either territory. Five procedures (three at A1 and two at A2) had excellent outcomes because of hitherto unknown luxuriant leptomeningeal collateral flow from MCA’s and PCA’s that filled A1 branches and even the A2 back to the site of the thrombosed aneurysms (Fig. 6). It seems that the A2 segment, like the P2 segment, has this potential, at least in the presence of a giant anterior cerebral aneurysm. Because of the presence of this collateral flow in one patient, simple clip occlusion of the A2 segment was deemed safe.
and proved uneventful. The prevalence and extent of this collateral flow makes any surgical bypass to the A2 segment seem unnecessary when the ACA needs to be clipped proximal to a giant aneurysm. One patient with severely impaired bilateral vision had dramatic recovery of sight after evacuation of the thrombosed sac.

There was one poor outcome and one death. One 12-year-old boy, Grade V from a subarachnoid hemorrhage, did not survive a proximal A2 clip. The other, a 32-year-old man, progressed to a vegetative state from the presence of severe bifrontal edema that existed preoperatively and which became massive despite a large frontal bone decompression (Table 6).

Discussion

Hunterian proximal arterial occlusion of cerebral arteries that give rise to giant aneurysms, when tolerated, is a very effective therapy. With the use of EC-IC bypass, the safety of carotid artery and even MCA occlusion or isolation in preventing major hemispheric ischemia has improved dramatically.

Another feature that emerges from this experience has been the recognition of the extent and functional significance of collateral circulation other than that seen in the circle of Willis upon which surgeons have relied in the past. Collateral circulation exists to a surprising degree between all major arterial systems above and even below the tentorium. The extent of its incidence and degree are enough to question the need for a bypass prior to undertaking anterior and posterior cerebral artery occlusion. Although occasionally exuberant, the leptomeningeal collateral flow to middle cerebral branches must be marginal enough in most patients to demand a preliminary bypass.

Although not usually seen in angiograms, collateral flow must exist in some patients between perforating arteries in deeper regions, such as lenticulostriate, choroidal, and pontine areas, as evidenced by the absence of a deep central infarction following thrombotic occlusion of some fusiform aneurysms from which these vessels were seen to arise.

Obliteration of the aneurysm is completed by thrombosis after proximal occlusion in most instances, except a few in which substantial collateral flow intervenes between the occlusion site and the aneurysm. This exception pertains mostly to aneurysms that occur around the basilar bifurcation. Surprisingly, in only four of 51 supraclinoid carotid aneurysms was the degree of thrombosis unsatisfactory. However, in this arterial segment, there was the opportunity to exclude ophthalmic, posterior, and/or anterior communicating collateral flow by performing intervening occlusions with bypass when necessary.

The causes of poor outcome are summarized in Table 7. Major persisting hemodynamic infarction occurred only in two patients, in both cases after carotid occlusion. One of these patients, who had tolerated 2 days of clamp occlusion of the ICA, only became hemiplegic after common and external carotid occlusion was attempted to repair the carotid avulsion that resulted from the removal of the clamp stem. The patient had been obviously dependent on the external carotid collateral flow. The three patients with preexisting hemiplegia are included in the poor results because it is not known whether they would have tolerated proximal occlusion.

<table>
<thead>
<tr>
<th>Surgical Technique</th>
<th>No. of Cases</th>
<th>E</th>
<th>G</th>
<th>P</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1 occlusion</td>
<td>3</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A2 occlusion</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>7</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

* E = excellent; G = good; P = poor; D = deceased.
† Grade V subarachnoid hemorrhage.
Temporary reversible ischemia occurred during six of the 13 MCA occlusions by tourniquet, and all but one patient tolerated complete M1 occlusion eventually, usually within 1 week. In one unfortunate patient, the aneurysm ruptured beyond a severe stenosis 2 days later and before complete occlusion could be reattempted.

Three patients developed persisting hemiplegias from the emboli that emerge from the thrombosing aneurysm, which will remain an infrequent but persisting problem when the aneurysm is not trapped. Aspirin has been used routinely after carotid artery occlusion, but one patient, for whom heparin was prescribed for repeated transient ischemic attacks from presumed thromboembolism, succumbed to an acute subdural hematoma from disruption of her bypass. Another patient with a huge fusiform aneurysm had a presumed lenticulostriate infarction from mere exploration and placement of an open tourniquet. Tragically, three patients died from aneurysm rupture before the artery could be occluded. Massive brain swelling from pre-existing edema but without known infarction resulted in one death and one vegetative state.

Good clinical outcomes occurred in 84% of these anterior aneurysms including patients in poor condition preoperatively. These results seem reasonable considering the poor outcome to be expected when giant aneurysms are untreated.

References


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