Surgical Distal Outflow Occlusion for the Treatment of Complex Intracranial Aneurysms: Experience With 18 Cases

Eric S. Nussbaum, MD

National Brain Aneurysm Center at the John Nasseff Neuroscience Institute, Allina Health, United Hospital, St. Paul, Minnesota

Correspondence:
Eric S. Nussbaum, MD,
3033 Excelsior Blvd., Suite 403,
Minneapolis, MN 55416.
E-mail: lnussbaum@comcast.net

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BACKGROUND: Selected intracranial aneurysms still require parent artery occlusion. Although such occlusion is usually performed proximal to the aneurysm, in rare instances, it may be difficult or impossible to access the proximal parent artery.

OBJECTIVE: To describe the use of parent artery sacrifice distal to the aneurysm (distal outflow occlusion) in the management of complex aneurysms not amenable to standard microsurgical or endovascular therapy.

METHODS: We reviewed a comprehensive database of intracranial aneurysms evaluated between 1997 and 2013. Hospital records, neuroimaging studies, operative reports, and outpatient clinic notes were examined for all patients treated with distal outflow occlusion.

RESULTS: Eighteen patients (11 women, 7 men; ages 28-69 years) underwent surgical distal outflow occlusion. Eight (44%) underwent concomitant distal revascularization. Intraoperative and delayed postoperative angiography was performed in every case. Nine presented with acute subarachnoid hemorrhage, 1 had a remote bleeding episode. The remaining lesions were unruptured; 3 were discovered incidentally, 3 had symptomatic cerebral edema, 1 had transient ischemic attacks, and 1 had cranial neuropathy. The average follow-up period was 6.5 years; no patient was lost to follow-up review. Two aneurysms required delayed endovascular treatment. Overall, 16 patients achieved a good outcome, 1 had moderate disability, and 1 died.

CONCLUSION: We describe our experience with distal outflow occlusion in the treatment of complex aneurysms not amenable to primary clip reconstruction or endovascular therapy. This technique has been described in very limited fashion in the past and may be particularly useful for patients requiring parent artery occlusion when proximal occlusion is challenging or impossible.

KEY WORDS: Aneurysm, Brain, Bypass, Dissection, Sacrifice, Stroke

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Although most intracranial aneurysms can be treated microsurgically with primary clip reconstruction or endovascularly with stents and/or coils, more complex lesions including dissecting, giant, and fusiform aneurysms often require creative options such as parent artery occlusion and possibly distal revascularization.[1-14] For those patients undergoing parent artery occlusion, arterial sacrifice proximal to the aneurysm has represented the traditional therapeutic option of choice.[15-21] Nevertheless, we have encountered selected cases in which proximal occlusion has been difficult from an anatomic perspective. In these instances, we have used parent artery occlusion distal to the aneurysm. This report details our experience with the technique of distal outflow occlusion, a measure that has been described previously in only very limited fashion.

ABBREVIATIONS: PICA, posterior inferior cerebellar artery; **SAH,** subarachnoid hemorrhage

METHODS

From July 1997 to June 2013, more than 2250 aneurysms were treated using open microsurgical techniques at our center. Of these, 18 aneurysms were treated using occlusion of the parent artery distal to the

aneurysm itself (distal outflow occlusion). We retrospectively reviewed the records of these patients to better understand the results obtained using this technique. Hospital records, neuroimaging studies, operative reports, and follow-up clinic notes were available in all cases. The 17 patients who survived were followed for 30 to 104 months; average follow-up was 6.5 years.

All patients underwent surgical exploration of their aneurysms using mild hypothermia (34°C) and barbiturate anesthesia. After microsurgical exposure to assess the possibility of primary clipping, a decision was made to proceed with distal occlusion in these instances. Distal revascularization was performed in 8 cases (44%) using the superficial temporal artery (6 cases) or a saphenous vein graft (2 cases). Intraoperative angiography and delayed postoperative angiography were performed in every case.

RESULTS

The demographics of the patients treated along with aneurysm locations, complications, and results are shown in the Table. Involved locations included the posterior inferior cerebellar artery (PICA) in 6 patients, basilar trunk in 2, M1 segment of the middle cerebral artery in 2, A1 segment of the anterior cerebral artery in 2, paraclinoid internal carotid artery in 2, supraclinoid internal carotid artery in 2, A2 segment of the anterior cerebral artery in 1, and cavernous internal carotid artery in 1 case. There were 7 men and 11 women. Ages ranged from 28 to 69 years.

Twelve aneurysms were treated with occlusion of the parent artery immediately distal to the aneurysm, whereas 6 patients with PICA aneurysms underwent remote distal outflow occlusion.[21] These PICA aneurysms included 1 patient with a giant aneurysm of the PICA origin and 5 patients with dissecting aneurysms located distal to the PICA origin but proximal to the telovelotonsillar segment. Ten aneurysms had ruptured; 9 presented with acute subarachnoid hemorrhage (SAH), and 1 had headaches and a remote history of bleeding. One of these patients had initially presented with a brainstem stroke and was treated with antiplatelet therapy only to return months later with an SAH (Patient 3). Eight patients had unruptured aneurysms. Of these, 3 had symptomatic cerebral edema with headaches and/or confusion, 1 had transient ischemic events, 1 had cranial neuropathy, and 3 were discovered incidentally.

Eight patients with aneurysms involving the internal carotid artery, the M1 segment of the middle cerebral artery, or the A2 segment of the anterior cerebral artery underwent distal revascularization at the same setting as the occlusion procedure. The only patient in this series who experienced a distal ischemic injury underwent a remote distal outflow occlusion of the PICA and an asymptomatic, small, peripheral cerebellar infarct identified on routine postoperative MR imaging developed.

No patients experienced bleeding or rebleeding from their aneurysms after distal occlusion. No patient experienced an immediate perioperative neurological complication related to the occlusion. In 1 patient with an A1 segment aneurysm, the aneurysm failed to thrombose, and in a second patient with a dissecting M1 aneurysm, the aneurysm nearly thrombosed but continued to enlarge on serial imaging studies. In both cases, endovascular treatment of the aneurysm consisting of coil embolization of the aneurysm sac or its inflow zone was performed. In these treatment failures, we noted the absence of a normal parent artery proximal to the fusiform aneurysm as a common possible underlying cause (Patient 2).

Overall, 16 of the 18 patients (89%) made a good functional recovery, regaining full independence. Three required placement of a ventriculoperitoneal shunt, and 1 patient with a giant cavernous aneurysm had worsening of her preoperative headache syndrome that persisted for several months after surgery. This may have been related to thrombosis of the aneurysm producing local increased mass effect, which gradually improved over time. One patient with a large dissecting basilar aneurysm presented with a brainstem stroke. He was managed with antiplatelet therapy but returned months later with severe headache, additional brainstem ischemia, and an SAH (Patient 3). He was left with significant disability from his brainstem stroke necessitating assistance with daily activities, but the patient lives at home with his family. Unfortunately, the patient with the dissecting M1 aneurysm requiring delayed endovascular retreatment 2 months after his initial surgery experienced an arterial perforation during the coiling procedure, was left in poor condition after urgent craniotomy for hematoma evacuation, and eventually died.

ILLUSTRATIVE CASES

Patient 1

Severe headaches developed in this 45-year-old woman followed by transient loss of consciousness. She was transferred to our facility and was found to be somnolent but able to follow commands without focal neurological deficit. Computed tomography scan of the brain revealed a significant SAH with blood in front of the brainstem. Cerebral angiography demonstrated a large aneurysm of the midbasilar artery with a very broad neck (Figure 1). The aneurysm was not thought to be amenable to endovascular therapy, and microsurgical exploration was recommended.

The aneurysm was exposed via a combined subtemporal-presigmoid approach. At the time of surgery, a thick clot was evacuated from the prepontine cistern, and the basilar artery was identified and dissected at the level of the aneurysm. The aneurysm incorporated a significant portion of the artery, and several large perforating arteries were found to arise from the aneurysm itself just beyond the aneurysm "neck." At best, it was thought that reconstruction would require leaving a portion of the aneurysm open to avoid perforator compromise. It was difficult to access the basilar artery below the aneurysm due to local mass effect, and a decision was made to apply a temporary clip across the basilar artery just distal to the aneurysm using intraoperative monitoring of motor and sensory evoked potentials and brainstem auditory evoked responses.

The monitoring remained at baseline for 10 minutes while intraoperative angiography was performed, demonstrating significant slowing of flow within the aneurysm sac, and excellent filling

TABLE. Demographic Factors, Presenting Features, Locations, and Results in 18 Patients Treated With Distal Parent Artery Occlusion for Their Intracranial Aneurysms[a]

Age, y/Sex	Presentation	Aneurysm Location, Size	Revascularization	Complications	Outcome, GOS Score	F/U, y
39, M	SAH, HH I	PICA,[b] small, dissecting	—	None	5	6
44, M	SAH, HH IV	PICA,[b] large, dissecting	—	None	5	6.5
34, F	SAH, HH IV	PICA,[b] small, dissecting	—	Hydro, VP shunt	5	7.5
43, M	SAH, HH II	PICA,[b] small, dissecting	—	Small cerebellar infarct, VP shunt	5	8
69, F	TIA	PICA (origin), giant	—	None	5	7
47, F	Remote SAH, HA	PICA,[b] large, dissecting	—	None	5	6.5
56, M	Brainstem stroke, then SAH, HH I	Basilar trunk, large, dissecting	—	Significant deficit from presenting stroke	3	8.5
45, F	SAH, HH III	Basilar trunk, large	—	None	5	7
41, F	SAH, HH I	M1, giant, dissecting	STA-MCA	None	5	3
46, M	SAH, HH III	M1, large, dissecting	STA-MCA	Required delayed endovascular tx, endovascular perforation	1	—
39, F	Incidental	A1, small	—	Required delayed endovascular tx	5	5.5
55, F	Incidental	A1, small	—	None	5	8
62, F	SAH, HH II	Paraclinoid, giant, dissecting	STA-MCA	Hydro, VP shunt	5	7.5
28, M	HA, cerebral edema	Paraclinoid, large	ECA-ICA, SVG	None	5	8.5
61, F	HA, cerebral edema	Supraclinoid ICA, giant	STA-MCA	None	5	7.5
36, F	Incidental	Supraclinoid ICA, giant	STA-MCA	None	5	4.5
55, M	HA, Confusion, cerebral edema	A2, giant	STA-ACA, SVG	None	5	7
59, F	HA, Cranial neuropathy	Cavernous, giant	STA-MCA	Worsened HAs	5	2.5

[a]GOS, Glasgow Outcome Score; F/U, follow-up; SAH, subarachnoid hemorrhage; HH, Hunt-Hess; PICA, posterior inferior cerebellar artery; Hydro, hydrocephalus; VP, ventriculoperitoneal; TIA, transient ischemic attack; HA, headache; ICA, internal carotid artery; STA-MCA, superficial temporal artery-middle cerebral artery; tx, treatment; ECA-ICA, external carotid artery-internal carotid artery; SVG, saphenous vein graft; STA-ACA, superficial temporal artery-anterior cerebral artery.
[b]Dissecting peripheral PICA aneurysm located distal to the PICA origin but proximal to the telovelotonsillar segment.

of the upper basilar artery through the posterior communicating arteries. The temporary clip was replaced with a permanent clip, and the patient awoke promptly from anesthesia without focal deficit. Mild vasospasm developed, but otherwise she had an uncomplicated postoperative course and made a complete recovery. Postoperative angiography at 24 hours, 1 week, and 1 year demonstrated stable occlusion of the aneurysm with excellent filling of the upper basilar artery from posterior communicating artery supply. The patient remained neurologically intact 5.5 years after the event.

Patient 2

A 39-year-old woman presented with vertigo and unsteadiness and was found to have a roughly 6-mm fusiform aneurysm involving the A1 segment of the anterior cerebral artery. The patient had a strong family history of SAH, and surgical exploration was recommended. At the time of surgery, a truly fusiform aneurysm involving the entire circumference of the artery was encountered. Because both A2 vessels filled well from the contralateral side, a decision was made to sacrifice the ipsilateral A1 segment to treat the aneurysm. There was effectively no normal A1 segment proximal to the aneurysm, so a clip was placed across the A1 just distal to the aneurysm (Figure 2). Intraoperative angiography showed slow filling of the lesion with normal filling of the distal ACA territories bilaterally from the opposite ICA.

The patient tolerated the procedure without complication, but follow-up angiography obtained 6 weeks later revealed brisk filling of the aneurysm. In effect, it appeared that the aneurysm had been converted into a "sidewall"-type lesion arising off the distal ICA where it turned to become the M1 segment of the middle cerebral artery.

Angiography was repeated at 3 months, and the aneurysm was unchanged. A decision was made to fill the aneurysm sac with coils. The patient tolerated the procedure without incident and has been followed for 6 years since that time without complications or evidence of aneurysm recurrence. Repeat angiography 2 years after the coiling procedure showed no evidence of aneurysm recurrence.

Patient 3

A 56-year-old man presented with sudden-onset hemiparesis and imbalance. He was found to have ischemic injury to the pons

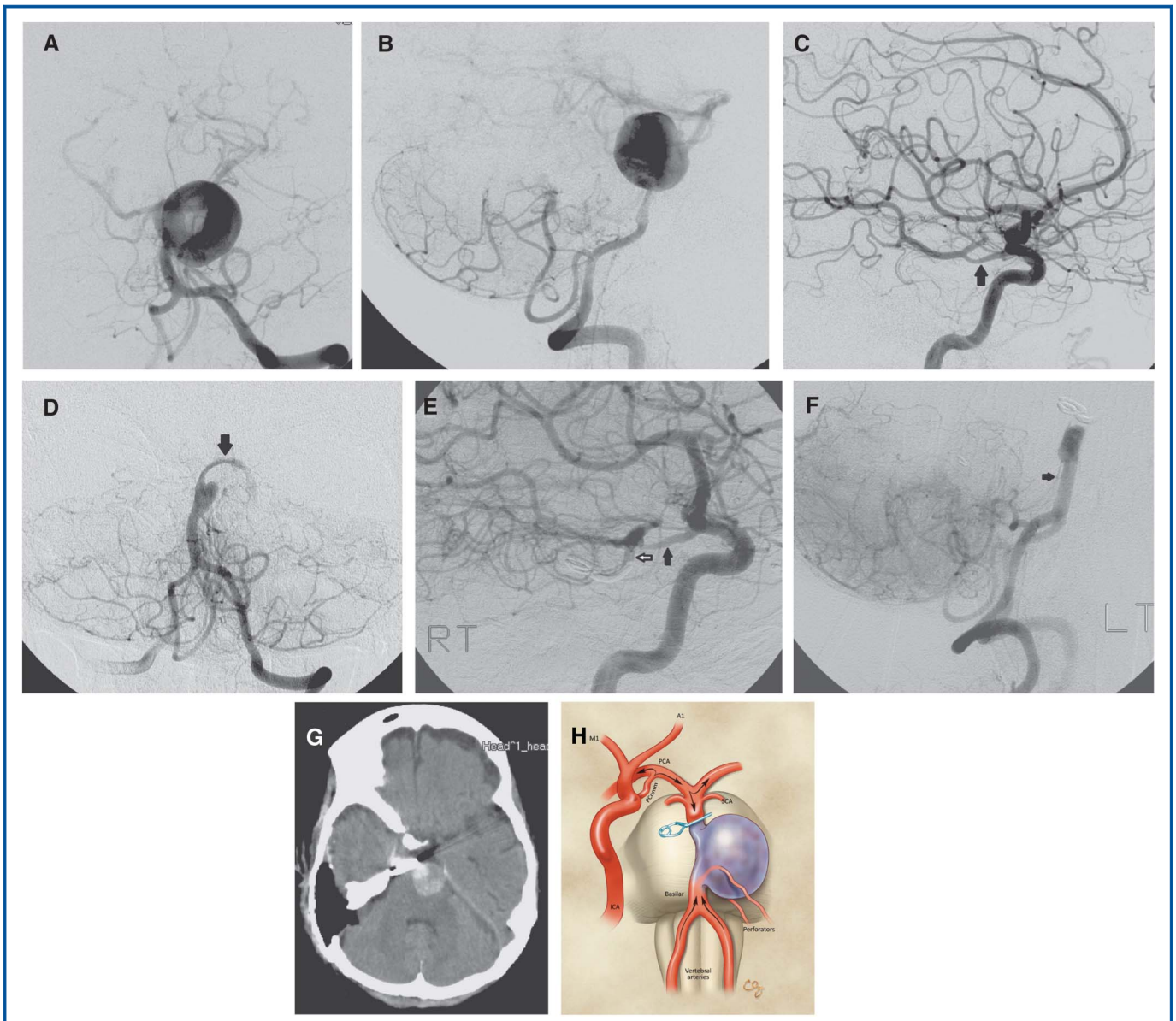

FIGURE 1. *Anteroposterior (A) and lateral (B) vertebral arteriographic images demonstrate a large midbasilar aneurysm in a patient presenting with acute subarachnoid hemorrhage. C, a lateral arteriographic image of the internal carotid artery (ICA) reveals a generous posterior communicating artery (arrow). D, an anteroposterior vertebral arteriogram obtained on the first postoperative day shows immediate significant thrombosis of the aneurysm with a thin rim (arrow) of contrast filling the dome of the aneurysm. E, postoperative lateral ICA injection shows the posterior communicating artery (black arrow) filling the upper basilar artery (white arrow) back down to the level of the clip. F, a delayed postoperative arteriogram obtained 1 month after surgery reveals progressive occlusion of the aneurysm. Note the long perforators (arrow) continuing to fill and trail down from the base of the aneurysm. G, postoperative axial computed tomography scan shows the thrombosed aneurysm without evidence of brainstem ischemia. H, artist's illustration depicts the large thrombosed aneurysm with filling of the upper basilar artery through the posterior communicating artery and filling of the perforators that arise at the base of the aneurysm through a trickle of persistent proximal flow. PCA, posterior cerebral artery; PComm, posterior communicating artery; SCA, superior cerebellar artery.*

and a large dissecting aneurysm of the basilar artery (Figure 3). He was admitted to the hospital, and aspirin therapy was instituted. His condition stabilized, and he was discharged to home on aspirin therapy with 3-month a follow-up magnetic resonance imaging study planned. Of note, the patients' brother had experienced an almost identical lesion. Two months later, the patient experienced a sudden severe headache, and a small amount of blood was noted on a computed tomography scan in the vicinity of the aneurysm. His baseline hemiparesis worsened acutely, and significant incoordination and vertigo developed.

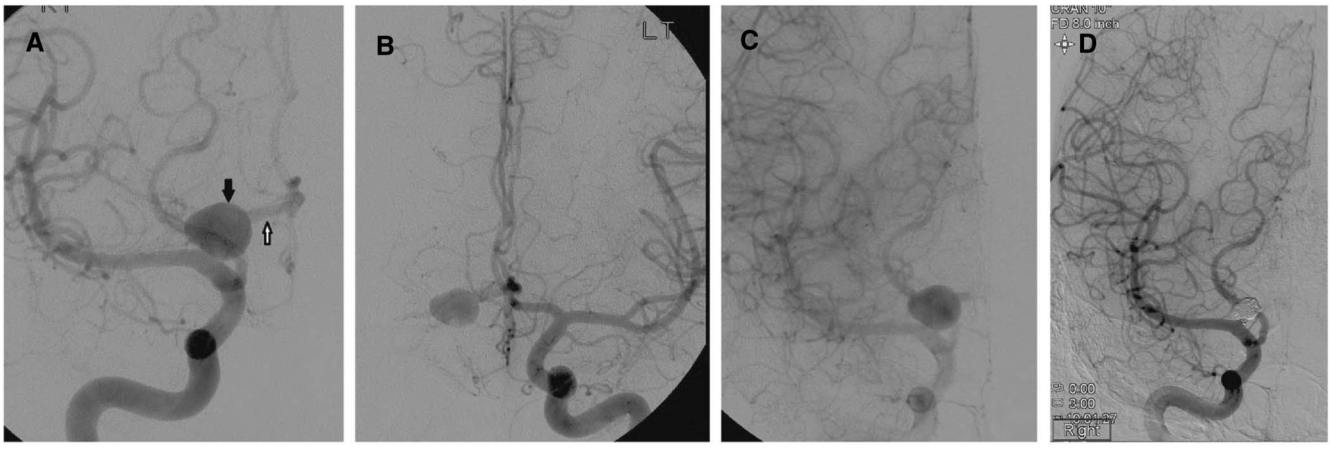

FIGURE 2. **A**, *anteroposterior image of an internal carotid arteriogram demonstrates a fusiform aneurysm (black arrow) involving the proximal aspect of the A1 segment without a meaningful length of normal A1 proximal to the lesion. The distal A1 (white arrow) fills the anterior communicating artery.* **B**, *left internal carotid artery arteriographic image demonstrates filling of both A2 vessels through the left A1 segment.* **C**, *postoperative arteriogram performed 1 day after surgery reveals stasis of contrast within the aneurysm after clip occlusion of the A1 segment distal to the aneurysm.* **D**, *when the aneurysm failed to thrombose on serial follow-up angiography, the aneurysm was treated with endovascular coiling, resulting in long-term stable occlusion of the aneurysm.*

Surgical treatment was offered, and the patient underwent a pterional craniotomy with orbitozygomatic osteotomy. The aneurysm wall was noted to be yellow-white and atheromatous with some thinner red areas (Figure 4). The lesion was not thought to be amenable to direct reconstruction, and the patient underwent occlusion of the upper basilar artery with intraoperative somatosensory and motor evoked potential and brainstem auditory evoked response monitoring. Intraoperative angiography demonstrated normal filling of the upper basilar system through generous posterior communicating arteries. The patient tolerated the procedure well, but was left with a significant hemiparesis and incoordination. He has been followed for 8 years without further incident. He lives at home but requires assistance with daily living activities.

DISCUSSION

Although most intracranial aneurysms are amenable to primary clip reconstruction or endovascular therapy with coils and/or stents, there remains a select group of complex, giant, or fusiform lesions that are best treated using vascular sacrifice with or without distal revascularization.[8,11-14,16] Such sacrifice can be performed using proximal occlusion, trapping the aneurysmal segment, or distal outflow occlusion. In our experience, we have rarely found trapping to be necessary, as most lesions do not require complete exclusion from the circulation, and most intracranial arteries include perforator-bearing segments that are jeopardized by trapping.[11-14] In most instances, proximal occlusion is feasible and adequate to treat the aneurysm.[1,2,17] Nevertheless, in rare cases, such proximal occlusion may be difficult due to anatomic considerations. In such cases, distal outflow occlusion may represent a reasonable option that has been described in only very limited fashion in the past.

Rationale for Distal Outflow Occlusion

The idea than an intradural aneurysm could be safely treated by distal occlusion was first suggested to the author by Dr Robert Spetzler during a conversation regarding the treatment of a complex posterior circulation aneurysm (personal communication, 2001). Initially, the idea of occluding the parent artery distal to an aneurysm seemed ill-advised, particularly in the setting of a recent rupture. On careful consideration, however, it became clear that outflow occlusion would produce an immediate diminution in the flow through the occluded artery and would be unlikely to result in bleeding from the aneurysm. Horowitz et al[22] used the Bernoulli equation to develop an elegant theoretical model predicting the intraluminal pressure changes after a distal occlusion. They found that the changes in pressure resulting from distal occlusion were less than those induced by normal daily activities, suggesting that distal occlusion would not be expected to increase the risk of aneurysm rupture or rerupture. Although the intra-aneurysmal pressure could increase momentarily, it should rapidly diminish thereafter. Despite this, we suspect that discomfort with the idea of suddenly occluding the outflow of an aneurysm has limited the use of this technique.

In our first case, which was a ruptured dissecting aneurysm of the PICA, we occluded the distal PICA with some measure of trepidation, initially expecting to encounter an obvious increase in turgor within the aneurysm, possibly manifested by immediate bleeding. Instead, the intraoperative findings were surprisingly unimpressive, and after watching for several minutes without

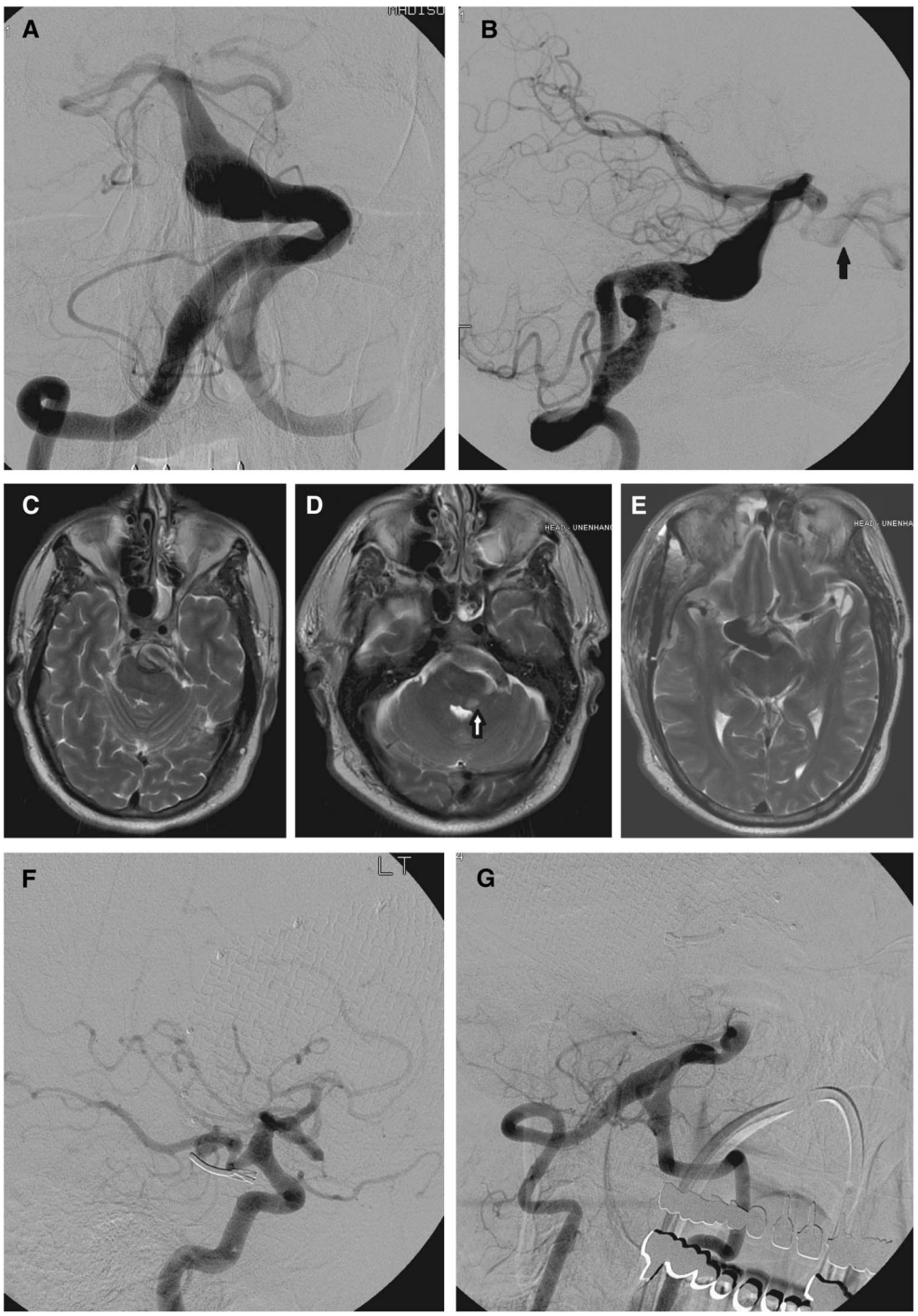

FIGURE 3. *Preoperative anteroposterior (A) and lateral (B) vertebral arteriographic images demonstrate a fusiform basilar aneurysm in a patient presenting with brainstem ischemia and subsequent subarachnoid hemorrhage. The black arrow demonstrates filling of the internal carotid artery (ICA) through a competent posterior communicating artery. Preoperative (C) and postoperative (D, E) axial magnetic resonance images demonstrate the large aneurysm compressing the brainstem and limited ischemic injury to the pons (arrow). F, delayed postoperative arteriographic image of the ICA (obtained 1 year after surgery) reveals excellent filling of the upper basilar artery through the posterior communicating artery. G, corresponding vertebral arteriographic image shows retrograde thrombosis of the basilar artery without evidence of aneurysm filling.*

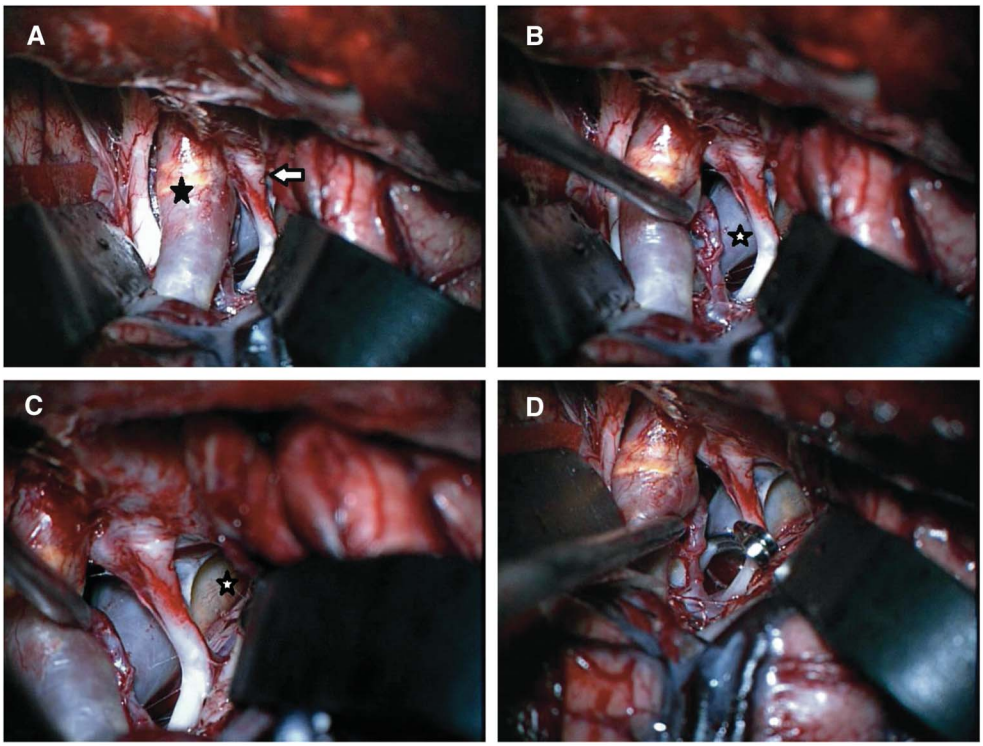

FIGURE 4. **A**, *an intraoperative photomicrographic image obtained after pterional exposure with orbitozygomatic osteotomy reveals the moderately dilated internal carotid artery (ICA) (black star) and the third cranial nerve (white arrow) just lateral to it.* **B**, *by retracting the ICA, the upper basilar artery (white star) is exposed in the space between the ICA and the third cranial nerve.* **C**, *further retraction reveals the basilar trunk aneurysm (white star), which appears white and abnormal.* **D**, *a clip has been used to occlude the upper basilar artery distal to the aneurysm but below the superior cerebellar arteries.*

incident, we simply replaced the temporary clip with a permanent one. In fact, this has been our experience in all treated cases.

Isolated case reports have described the use of distal occlusion to treat giant, partially thrombosed, M1 segment aneurysms.[22-24] In these cases, either a proximal occlusion or trapping of the lesion had been planned preoperatively. During surgery, however, proximal occlusion was found to be unacceptably dangerous or was left as a possible future option, and extracranial-intracranial bypass with distal occlusion alone was followed by rapid aneurysm thrombosis.[22-24]

We initially used distal occlusion in the unique setting of ruptured dissecting aneurysms of the PICA. For these lesions, we introduced the idea of remote distal outflow occlusion, occluding the PICA beyond the tonsillar loop and thus beyond the last critical brainstem perforator and also well beyond (remote from) the aneurysm itself.[12] In theory, it was thought that this should allow for a small amount of continued flow through the proximal PICA to irrigate critical brainstem perforators while decreasing flow so significantly that the dissection should heal without rebleeding. In practice, this worked well, and our initial experience was described in a previous report.[12] We have used distal occlusion immediately beyond the aneurysmal segment in other cases, and our complete experience with such occlusions forms the basis of this report.

Lessons Learned Over Time

In our experience, distal outflow occlusion, either immediately beyond the aneurysm or "remote" from it, has been extremely safe with no instance of early or delayed rebleeding in a series of predominantly ruptured aneurysms. In particular, we have never encountered immediate bleeding from the aneurysm sac, a possibility that had been particularly concerning early in our experience. At the time of surgery, one can watch the flow diminish rapidly within the aneurysm either by direct visual inspection in thin-walled lesions or with intraoperative angiography for thicker walled aneurysms.

We have encountered 2 patients whose aneurysms were not controlled using distal outflow occlusion. In the instance detailed above in patient 2, we attempted to treat a fusiform A1 aneurysm arising along the A1 segment at the level of the internal carotid artery bifurcation with no normal A1 proximal to the aneurysm

and found that the aneurysm did not thrombose. Instead, it appeared that the distal occlusion "created" a true sidewall aneurysm with flow continuing from the distal internal carotid artery into the M1 segment, keeping the aneurysm open. It is possible that the lack of a length of normal parent artery proximal to the aneurysm may have led to this treatment failure.

Our other treatment failure occurred in a giant dissecting M1 segment aneurysm. In this case, there was limited flow into the aneurysm after distal occlusion, and although the aneurysm nearly thrombosed on follow-up angiography, serial magnetic resonance imaging demonstrated continued growth of the aneurysm as additional clot layered within the dissected aneurysm wall. This necessitated additional endovascular treatment. In this particular case, there was again almost no normal parent artery proximal to the aneurysm as the aneurysm began just beyond the origin of the M1 segment, and it is possible that one of the reasons the distal occlusion failed was because of this anatomic configuration. In addition, similar dissecting aneurysms have been very challenging to control in our experience and have, at times, required complete exclusion from the circulation to achieve long-term stability.

Based on our experience, we believe that if parent artery occlusion is chosen to treat an aneurysm, it likely matters little whether the occlusion is performed proximal or distal to the aneurysm with regard to the aneurysms' likelihood of thrombosis in response to the occlusion. The site of occlusion should be determined by the adequacy of the collateral supply that can be augmented by revascularization as needed, by the location of nearby perforating arteries, and by the ease of access to the proximal and distal parent artery. As noted previously, those lesions with little or no parent artery proximal to the aneurysm and patients with giant dissecting aneurysms require close follow-up to ensure proper occlusion of their aneurysms.

Limitations

It should be noted that distal outflow occlusion does not definitively exclude the involved segment from the arterial circulation and thus cannot completely eliminate the potential for repeat hemorrhage.[25,26] Nevertheless, no aneurysm in our series rebled during the period of follow-up, and those that enlarged or failed to thrombose were subsequently treated endovascularly. In general, parent artery occlusion carries some risk of proximal or distal thrombus formation within the occluded artery that could result in symptomatic ischemic injury, although this has not happened in our experience. In addition, parent artery sacrifice by definition places the distal arterial territory at risk of hypoperfusion, and we have used distal revascularization liberally in our series whenever adequacy of distal collateral circulation was a concern. Finally, this study spans more than a decade, and some of the cases managed surgically might be amenable to newer endovascular stent options if encountered today. On review of our cases, at least 4 of the patients if evaluated today (those with saccular cavernous, paraclinoid, and supraclinoid lesions) would have been considered for flow-diverting stents, potentially further limiting the patient population who may require similar microsurgical techniques. Other centers adopting an even more aggressive endovascular approach might consider stent options in some of our other cases. Nevertheless, the experience remains important when addressing those lesions that cannot be treated endovascularly or that fail other attempted therapies with 16 of 18 treated aneurysms (89%) responding well to distal occlusion.

CONCLUSION

We describe our experience with the use of distal outflow occlusion in the management of a very select group of complex intracranial aneurysms. This option may be particularly useful when vascular sacrifice is planned, but simple proximal occlusion is difficult due to anatomic considerations. Although occasional reports have described distal occlusion in this setting, there is very little formal discussion of this management algorithm in the literature. We have found distal occlusion to be safe in our experience and effective in the majority of cases. Cases with no length of normal parent artery proximal to the aneurysm may not "respond" to distal occlusion. Similarly, giant dissecting aneurysms may continue to grow despite distal outflow occlusion treatment. Despite initial trepidation regarding the safety of distal occlusion, particularly in the setting of recent SAH, this technique has been safe and largely effective in our series.

Disclosure

The author has no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

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COMMENTS

This report describes the author's experience with the technique of "distal outflow occlusion," which has not been widely used nor reported on previously. The technique has been reported previously for the treatment of complex dissecting aneurysms of the posterior inferior cerebellar artery (PICA) with good results.[1] The author describes the outcomes in 18 aneurysms treated with occlusion of the parent artery distal to the aneurysm. The author retrospectively reviews the records of these patients and delineates the technique in a very well-illustrated article. The authors experienced good outcomes with this technique and encountered 2 patients whose aneurysms could not be controlled using distal outflow occlusion and were treated endovascularly.

As the author observes, some of the cases managed using distal outflow occlusion might now be amenable to newer endovascular techniques. Nevertheless, the microsurgical technique of distal outflow occlusion is elegantly illustrated described in this article and may aid vascular neurosurgeons in their decision making and adds to our surgical armamentarium.

Sam Safavi-Abbasi
Robert F. Spetzler
Phoenix, Arizona

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1. Nussbaum ES, Madison MT, Goddard JK, Lassig JP, Janjua TM, Nussbaum LA. Remote distal outflow occlusion: a novel treatment option for complex dissecting aneurysms of the posterior inferior cerebellar artery. Report of 3 cases. *J Neurosurg.* 2009;111(1):78-83.

The author presents a series of aneurysms not amenable to other surgical or endovascular treatment that were treated with surgical distal occlusion. He demonstrates excellent outcomes with this approach with surprisingly no rebleeding after treatment. As newer endovascular therapies emerge, some of these aneurysms may now be treated endovascularly. Nevertheless, it is an important, albeit rarely used, technique that should be considered and added to the armamentarium when faced with a complex aneurysm with no other available treatment.

Rose Du
Boston, Massachusetts