

# Direct Microsurgery of Dural Arteriovenous Malformation Type Carotid-Cavernous Sinus Fistulas: Indications, Technique, and Results

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**OBJECTIVE:** There is a subgroup of patients with Barrow Type D carotid-cavernous sinus fistulas (CCFs) who have progressive neurological deficits despite endovascular attempts at obliteration. To effectively arrest the progression of neurological deficits, especially visual loss, these patients require direct operative intervention. We have used a direct approach to such lesions, which comprehensively occludes all fistulous connections of the CCF.

**METHODS:** We present a series of nine patients with Type D CCFs for which attempts at endovascular embolization failed and that, because of persistent symptoms, required surgical intervention. These lesions characteristically had extensive multiple external carotid artery feeders, often bilateral, in addition to the internal carotid artery feeders. The operative approach used was a combined extra- and intradural full exposure of the cavernous sinus and its contents, with identification and direct obliteration of all arterial input and selective ablation of the venous outflow from the cavernous sinus.

**RESULTS:** All nine patients experienced resolution of their symptoms, and complete ablation of the lesions, as demonstrated by postoperative angiography, was achieved. Transient diplopia and trigeminal hypesthesia was observed in all nine patients, which resolved by 6 months postoperatively. One patient suffered from a temporary hemiparesis and another from permanent hemiparesis. There were no deaths related to surgery in this series.

**CONCLUSIONS:** Patients with Type D CCFs who have persistent, progressive neurological deficits after failed endovascular attempts at obliteration may be treated by a direct surgical approach to ablate the fistulas. The pertinent anatomic concepts, indications for surgery, and operative techniques that are different from previously described methods are discussed. (Neurosurgery 41:1119-1126, 1997)

Key words: Anatomy, Carotid-Cavernous sinus fistula, Cerebrovascular, Surgery

Dural arteriovenous malformation type carotid cavernous sinus fistulas (CCFs) fed by both internal and external carotid artery feeders (Type D according to Barrow [1]) are particularly challenging to successfully obliterate. Endovascular methods are well established as the first line of therapy (4, 15) and are typically used in our practice. However, a subgroup has been identified that is comprised of lesions that are not successfully treated by endovascular techniques alone and that require direct surgical obliteration (1, 2, 15). Primarily, failure of an endovascular approach to halt rapid and progressive visual loss has been our indication to perform a direct surgical attack as a means of preserving vision.

Direct surgery of these lesions has been performed by several groups, all of whom used various methods of embolizing the fistulous connections (2, 6, 8-13). The surgical strategy used for our patients differs from previous methods of direct

surgery in that it directly exposes and obliterates all arterial and venous connections of the fistula, versus compression and thrombosis, by packing foreign material into the cavernous sinus. We have determined this method to be successful in completely obliterating the fistulous connections in the cavernous sinus and resulting in the resolution of symptoms.

## PATIENTS AND METHODS

Since 1985, we have treated nine patients with Barrow Type D (1) CCFs. The patients ranged in age from 11 to 71 years. There were 7 female and 2 male patients in the series. All patients presented with progressive visual loss that occurred during a period of weeks to several months as a component of their symptom complex. A diagnosis of Type D CCF was made using angiography in each case. Patients were evaluated by ophthalmological examination, which included acuity



and visual field testing. All nine patients were initially treated by endovascular methods using various types of embolic materials. Failure of endovascular therapy was recognized in all nine patients, both angiographically and by persistence of visual compromise. The time interval between endovascular intervention and surgery varied from 12 hours to 1 week. All nine patients underwent a combined intra- and extradural frontotemporal approach to the cavernous sinus for direct obliteration of the fistulous connections. The patient data are summarized in *Table 1*.

### Operative technique

The general details of the surgical approach have been described previously (3, 5, 6). We briefly describe the technique as applied in these cases.

After satisfactory induction of general anesthesia, the head is placed in three-pin fixation and rotated approximately 45 degrees. A routine pterional type scalp incision is made. The temporalis muscle and fascia are incised and elevated with the scalp as a single layer flap. The musculocutaneous flap is elevated to the margin of the orbital rim and held anteriorly. A frontotemporal craniotomy is cut centered one-third above and two-thirds below the superior temporal line. The dura is elevated away from the frontal and temporal cranial base, and the sphenoid ridge is then flattened using a high-speed drill. Complete skeletonization of the cranial nerves in their respective canals is next performed. The optic canal is first unroofed, and then the anterior clinoid process is removed. The superior orbital fissure, foramen rotundum, and foramen ovale are next unroofed to skeletonize 5 to 8 mm of their contents. This extradural bone removal is critical to exposing several of the triangular entry spaces into the cavernous sinus. Arterial feeders from the external carotid circulation are also exposed and occluded via this bone removal, both at the neural foramina and traversing bone in between. The middle meningeal artery is exposed at the foramen spinosum and divided. In some cases, the posterolateral and posteromedial cavernous trian-

gles are opened via extradural removal of the petrous apex (5, 6). This dissection provides access to the posterior cavernous sinus.

The dura propria over the temporal lobe is next separated from the outer cavernous membrane. As this layer is elevated, bleeding comes from small openings in the outer cavernous membrane. These bleeding points are controlled by packing with small pieces of Surgicel. The dura propria is elevated to near the incisural edge and petrous ridge. The anteromedial, anterolateral, and far lateral cavernous triangles are opened and packed with an appropriate amount of Surgicel.

Medially, the dura is opened in an L-shaped incision that begins over the sylvian fissure and extends to the optic nerve sheath. The second limb of the incision is directed along the tuberculum sellae. The fibrous dural ring surrounding the carotid artery is next opened. The medial, superior, and lateral cavernous triangles are opened and packed with an appropriate amount of Surgicel. The procedure of sequential packing of the cavernous triangles results in complete obliteration of the arteriovenous fistula in the cavernous sinus.

### Illustrative Case 1

A 60-year-old woman presented with a 3-month history of worsening chemosis, abducens palsy, and visual loss in the left eye. Her visual loss was characterized by an enlarging central scotoma and worsening acuity. The patient underwent a transarterial endovascular approach with embolization of the external carotid arterial feeding vessels with polyvinyl alcohol particles. It was not possible to embolize the internal carotid arterial supply to the fistula (*Fig. 1A*). There was no change in the patient's symptoms after embolization, and the patient was transferred to Mitsui Hospital in Tokyo for definitive therapy.

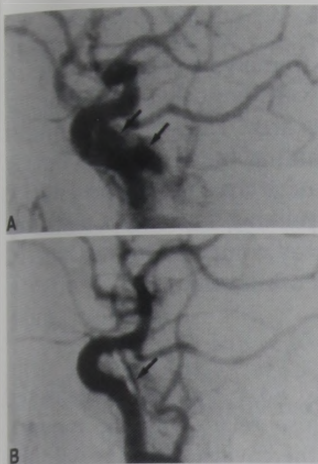
The patient underwent a frontotemporal craniotomy with intradural exposure of the cavernous sinus. The superior and lateral cavernous triangles were opened and the cavernous sinus packed with Surgicel. The meningo-hypophyseal trunk was exposed in the superior triangle and ligated with a small aneurysm clip. Postoperative angiography demonstrated complete obliteration of the fistula (*Fig. 1B*). The patient improved postoperatively, regaining normal

**TABLE 1. Clinical Summary of Patients Undergoing Direct Microsurgical Embolization<sup>a</sup>**

Age (yr)/Sex	Preoperative Symptoms	Persistent Deficits	Endovascular Approach	Embolic Agents	Residual Feeders	Time to Surgery
60/F	Chemosis, visual loss	None	Arterial	PVA	MHT	2 d
11/M	Chemosis, orbital pain, visual loss	None	Arterial	PVA	MHT and vertebral arterial branches	1 wk
71/F	Chemosis, visual loss	None	Arterial	PVA and gel foam	MHT	2 d
66/M	Chemosis, visual loss	Diplopia	Arterial	PVA	MHT	4 d
58/F	Chemosis, visual loss	Hemiparesis	Arterial	PVA	MHT	1 wk
65/F	Diplopia, chemosis, visual loss	None	Arterial and venous	GDC	MHT	1 d
69/F	Chemosis, visual loss, diplopia	None	Arterial and venous	GDC	MHT	2 d
54/F	Headache, diplopia, visual loss	None	Arterial and venous	GDC	MHT	2 d
45/F	Headache, visual loss	None	Arterial and venous	GDC and particles	MHT	12 h

<sup>a</sup> PVA, polyvinyl alcohol; GDC, Guglielmi detachable coils; MHT, meningo-hypophyseal trunk.





**FIGURE 1.** *A*, angiogram of the left ICA, obtained after embolization of external carotid arterial feeding vessels, demonstrating the persistent fistula (arrows), which is fed by the meningohypophyseal trunk. *B*, postoperative angiogram demonstrating occlusion of the fistula. An aneurysm clip (arrow) was placed across the meningohypophyseal trunk.

vision. Chemosis and the abducens palsy resolved during several days (Fig. 2).

### Illustrative Case 2

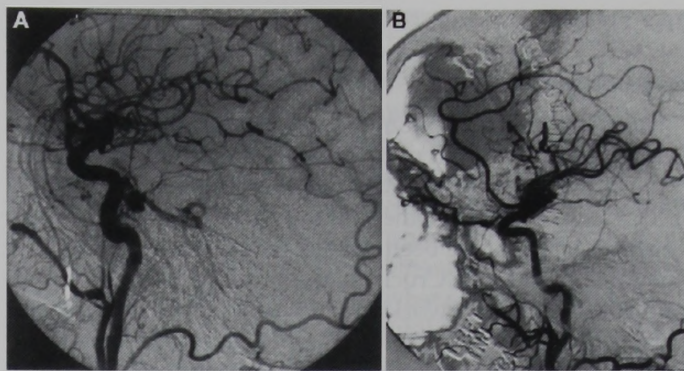
A 45-year-old woman presented with a history of several months' duration of headache and gradual visual loss in the left eye. She was examined by a neurologist who referred her for ophthalmological evaluation, including magnetic resonance imaging and magnetic resonance angiography. The ophthalmological evaluation was significant for an enlarged central scotoma and intraocular pressure of 26 mm Hg. The results of the magnetic resonance angiography were suspicious for a CCF. The patient was then referred for angiography and endovascular obliteration. Angiography revealed a Type D CCF with bilateral external carotid circulation feeding vessels in addition to feeding vessels from the meningohypophyseal trunk.

The patient underwent a combined transarterial and transvenous endovascular procedure with placement of Guglielmi detachable coils and polyvinyl alcohol microspheres, which successfully occluded the external carotid feeding vessels to the malformations. However, satisfactory embolization of the fistula supply from the internal carotid artery (ICA) was not achieved (Fig. 3A). The next morning, 12 hours after embolization, the intraocular pressure in the left eye was unchanged (24 mm Hg) and the patient reported deterioration in her vision. At that time, the decision was made to perform direct surgical obliteration of the fistula in an attempt to preserve the patient's vision.

A left frontotemporal craniotomy was performed with extradural exposure of the cavernous sinus. The anterolateral, lateral, superior, and medial cavernous triangles were opened and embolized with careful packing of Surgicel, as described above. Postoperatively, the patient recovered well. On the morning after surgery, the intraocular



**FIGURE 2.** *A*, photograph, obtained on postoperative Day 1, demonstrating severe chemosis of the left eye. *B*, photograph, obtained 3 months postoperatively, demonstrating resolution of chemosis.



**FIGURE 3.** *A*, preoperative lateral angiogram of the patient in the first illustrative case, demonstrating persistent flow via the ICA circulation. The external carotid artery feeders were obliterated using an endovascular approach. *B*, angiogram, obtained on postoperative Day 3, demonstrating complete obliteration of fistulous flow.

pressure in the left eye was 19 mm Hg and the patient's vision was improved. The intraocular pressure in the left eye subsequently dropped to 14 mm Hg by 1 month after surgery, and the patient's vision had returned to normal. Postoperative angiography confirmed the occlusion of the fistula on postoperative Day 3 (Fig. 3B). The patient suffered a transient partial left oculomotor nerve paresis, which resolved completely by the 2nd week after surgery. At follow-up 1 year after surgery, the patient had experienced no recurrence of symptoms.

### RESULTS

Complete resolution of the CCFs was achieved in all nine patients, as documented by postoperative angiography. None of the patients have suffered a recurrence of the fistulas to date. The follow-up period ranges from 1 to 14 years. No late follow-up angiograms have been obtained because none of the patients have experienced a return of symptoms. There were no mortalities in the series.

Vision was preserved at the preoperative level or improved postoperatively in all patients. The minimum goal of the procedure was to halt the progression of the visual deficit, which was realized in all patients. Three patients had diplopia as part of their presenting symptom complex. All three experienced reversal of their ocular motor disturbance by 3 months after surgery. One patient suffered from a new abducens nerve deficit, presumably secondary to compression by overpacking in Parkinson's triangle. The remaining five patients had temporary diplopia postoperatively, all of which resolved by 3 months. Chemosis and headache were also relieved postoperatively.

One patient suffered a permanent hemiparesis postoperatively. This was one of the initial patients treated in the series. Postoperative angiography demonstrated occlusion of the ICA; otherwise, the CCF was occluded. A second patient suffered temporary contralateral hemiparesis, which resolved within 1 week. A slight narrowing of the cavernous ICA was detected using postoperative angiography for this patient. Initial postoperative computed tomography demonstrated mild cerebral hemispheric edema.



## DISCUSSION

## Surgical anatomy

The microsurgical anatomy of the cavernous sinus has been well described in recent publications (7). The pertinent details of the surgical anatomy of the cavernous sinus for treatment of CCFs are reliant on the features of the triangular entry corridors, which have been detailed elsewhere (5, 6) and are illustrated in Figure 4. Crucial in diminishing surgical complications is an intimate working knowledge of these triangular entry corridors. The success of the procedure relies on packing a sufficient amount of oxidized cellulose (Surgicel) in the correct position within each triangular corridor. Overpacking in any one direction, in any particular corridor, can lead to carotid occlusion and/or compression of cranial nerves. Therefore, the following descriptions form the basis for successful obliteration of Type D CCFs using our approach.

## Anteromedial triangle

The anteromedial triangle contains the subclinoidal segment of the carotid artery. This segment of the vessel frequently is surrounded by an extension of the cavernous venous plexus and in cases of CCFs must therefore be packed. The subclinoidal segment of the carotid artery is almost invariably exposed in these procedures as a means of exposing and controlling the distal carotid artery. Heavy packing of oxidized cellulose in any direction has the potential to occlude the carotid artery.

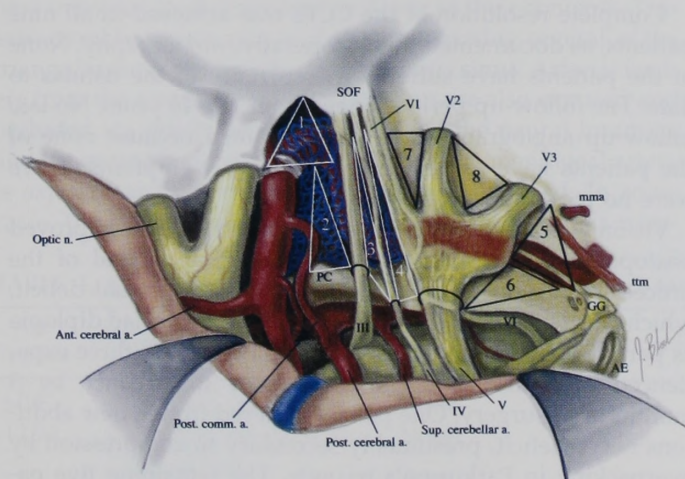


FIGURE 4. Illustration of the critical anatomic triangular entry corridors to the cavernous sinus used in direct obliteration of CCFs. 1, anteromedial triangle; 2, medial triangle; 3, superior triangle; 4, lateral triangle; 5, posterolateral triangle; 6, posteromedial triangle; 7, anterolateral triangle; 8, far lateral triangle; AE, arcuate eminence; GG, geniculate ganglion; mma, middle meningeal artery; PC, posterior clinoid process; SOF, superior orbital fissure; ttm, tensor tympani muscle; III, oculomotor nerve; IV, trochlear nerve; V, trigeminal nerve; VI, abducens nerve; n., nerve; a., artery; Ant., anterior; Post. comm., posterior communicating; Sup., superior.

## Anterolateral triangle

The anterolateral triangle is extremely important in cases of CCFs. The superior ophthalmic vein typically communicates with the cavernous sinus via this area. Therefore, it is invariably opened as a means of occluding this important venous connection. Packing toward the orbit may be performed with impunity. However, posterior packing has the potential to occlude the carotid artery and may compress the sixth nerve near its entrance to the superior orbital fissure.

## Far lateral triangle

This space, bounded by V2 and V3, commonly contains the sphenoidal emissary vein in close relation to the foramen ovale. This vein communicates the cavernous sinus with the pterygoid venous plexus and may be substantial in cases of CCFs. As in the case of the anterolateral triangle, packing toward the base, occluding the vein, is performed with impunity. However, posterior packing has the potential to occlude the carotid artery as it enters the cavernous sinus space via the foramen lacerum.

## Medial triangle

Packing the medial triangle in the lateral and anterior directions carries risk to the IIIrd cranial nerve and the ascending portion of the cavernous carotid artery. The posterior and inferior directions lead to the cavernous sinus communications with the basilar venous plexus of the clivus and the inferior petrosal sinus. Therefore, a large amount of embolic material may be directed in this direction without consequence.

## Superior triangle

This space provides the route to the most typical origin site of the meningohypophyseal trunk. Occlusion of this vessel may be achieved via this space. Packing of embolic material may be performed in the posterior and medial directions, which augments occlusion of the basilar venous plexus and inferior petrosal sinus via the larger medial triangle. Packing anteriorly and laterally will risk occlusion of the cavernous carotid artery and may result in compression of the Vth cranial nerve.

## Lateral triangle

Parkinson's triangle is relatively hazardous in terms of generous placement of embolic material. Packing in the lateral and anterior directions poses a high risk to the sixth nerve, VI, and the carotid artery. Posteriorly directed packing also risks compression of the sixth nerve. The medial direction is generally safe as a means of occluding the communication to the inferior petrosal sinus.

## Posterolateral triangle

Known as Glasscock's triangle, this space houses the horizontal intrapetrous carotid artery. This triangle may need to be opened for proximal control of the intracavernous carotid artery and to occlude fistulous connections through bone to



the pterygoid venous plexus. Care must be taken during packing to preserve patency of the carotid artery.

### Posteromedial triangle

This triangle roughly describes the petrous apex and is known as Kawase's triangle. Removing the bone in this area provides access to the posterior cavernous sinus region. This may be important in occluding fistulous connections to the superior petrosal sinus, inferior petrosal sinus, and petrosal veins.

In principle, all of the cavernous triangles are opened and packed in these cases. These Type D CCFs are diffuse lesions; therefore, they generally require full exposure for successful obliteration. However, the surgical approach may be tailored in individual cases, based on the results of postembolization angiography. For example, if after embolization the sole remaining feeding vessel is the meningohypophyseal trunk, selective opening of the superior or lateral triangle with ligation of the vessel may suffice. This was the situation in our first illustrative case.

### Clinical considerations

The contemporary first-line therapy for any type of CCF that is symptomatic is an endovascular approach to embolize the fistula. Patients who present with rapid visual loss constitute urgent cases requiring immediate therapy to save vision. Clearly, it is the rare patient who does not respond to endovascular obliteration of the CCF (1, 4, 15). The initial five patients in this series were treated before the advent of transvenous approaches and Guglielmi detachable coils. The last four patients were treated using a combination of transarterial and transvenous approaches. Despite these contemporary techniques, these patients failed to respond to therapy. Those patients who fail endovascular procedures and continue to suffer visual loss and diplopia are candidates for a direct obliteration procedure. The optimal timing of surgery after a supposedly failed endovascular procedure is uncertain. In this series of patients, the interval varied from 12 hours to 1 week. This variance was secondary to several factors. Some patients were treated endovascularly outside the authors' institution and were transferred to us after failing to realize any improvement. Patients who were operated more proximate to the endovascular procedure had intervention because of persistence of the fistula on angiograms, the continued severity of symptoms, and the presumed imminent threat to their vision. The selection of the surgical approach should optimally ensure patency of the carotid artery while resulting in complete fistula occlusion.

Other techniques have been used and presented. In 1965, Parkinson (11) reported direct surgical obliteration of a CCF under conditions of hypothermic cardiac arrest. His surgical approach aimed to directly interrupt the fistulous connections in the cavernous sinus (11, 12). Although successful in cases of Type A CCFs, the complexity of Type D fistulas does not lend itself to this limited approach through the lateral triangle. Additionally, advances in surgical technique have now obviated the necessity for hypothermic cardiac arrest in such

cases. Mullan (10) is also recognized for his pioneering work on this complicated problem via direct venous occlusion of the cavernous sinus via the anterolateral triangle and other routes. The early series of 33 patients presented by Mullan (10) produced uniformly excellent results, with no case of carotid occlusion. As in our series, cranial nerve deficits were uniformly improved postoperatively. The method presented by Mullan (10) is essentially incorporated into a more comprehensive approach to surgical obliteration with our method. Isamat et al. (9) published a somewhat similar technique with muscle packing through a transdural approach used in four patients with high-flow, Type A CCFs. Although the results reported were good, this is an imprecise technique that does not guarantee carotid patency nor accurate obliteration of all venous spaces in the cavernous sinus. Such techniques rely on a sophisticated sensitivity to packing pressure and volume by an experienced surgeon to prevent overpacking and carotid compromise.

### Complication avoidance

The dominant concept that requires study and comprehension is the triangular entry corridor organization of cavernous sinus anatomy. Avoiding compression of the intracavernous carotid artery, the abducens nerve, and the nerves within the lateral wall relies on an intimate knowledge of this anatomic organization. A sense of how much embolic material can be placed in any particular direction is crucial. This is a "subjective" matter of packing pressure, which has been commented on by Mullan (10). One patient early in our series suffered from permanent hemiparesis, which was secondary to occlusion of the carotid artery from overpacking, as observed on the postoperative angiogram. A second patient suffered transient contralateral hemiparesis postoperatively, which resolved within several days. Postoperative computed tomographic scans showed some element of cerebral hemispheric edema, possibly as a result of retraction. This patient may have also had some element of vasospasm from manipulation of the carotid artery, which may have caused this problem. The method presented by Mullan (10) of transvenous obliteration of CCFs similarly relies on this technical point for success without complication from overpacking.

The pathological mechanism of visual loss involves venous hypertension leading to raised intraocular pressures. High intraocular pressure results in a partial ischemic deficit (14). This mechanism requires that intraoperatively, the patient's blood pressure is kept at least at a normotensive level. Intraoperative hypotension may lead to retinal hypoperfusion because of an inability to overcome high intraocular pressures from venous obstruction. Therefore, we maintain adequate perfusion pressures to prevent further ischemic retinal damage during the procedure.

Because of the comprehensive nature of our surgical approach, most elements of the cavernous sinus region are exposed and manipulated. This results in at least temporary dysfunction of the affected nerves. In our series of nine patients, all had temporary diplopia, which resolved at varying times within 3 months postoperatively. This raises the impor-



tant point of tailoring the surgical approach for each specific case. The necessary elements of the surgical approach are determined by careful review of the postembolization angiograms. These films are critical to specifically exposing and embolizing the areas that were not treated by the endovascular approach. It is unnecessary and introduces added elements of risk to expose and pack segments of the anatomy that are already treated.

## CONCLUSIONS

We have treated nine patients with Type D CCFs via direct surgical obliteration of the fistula after endovascular attempts at treatment failed. This is a highly select group of patients who required a direct surgical approach. The mainstay of current therapy for CCFs of all types is endovascular embolization. However, some patients continue to have symptoms after endovascular therapy. When the patient's vision is threatened, a direct surgical procedure to occlude the fistula is indicated. Our method results in complete obliteration of the venous connections of the cavernous sinus, as well as any remaining arterial input to the fistula. Reliable use of this technique depends on the surgeon's having an intimate working knowledge of the triangular entry corridors into the cavernous sinus. With proper application, it has a high rate of success in obliterating CCFs with a minimum of morbidity.

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## COMMENTS

The authors report the results of direct surgical treatment for nine patients with Type D carotid-cavernous sinus fistulas (CCFs). Attempts at endovascular embolization failed in these patients. Surgery was accomplished using the "triangular entry corridors" to the cavernous sinus, selectively packing those areas of the cavernous sinus involved in the fistula.

Endovascular therapy failed in all patients in the study, and the authors report that Type D CCFs are challenging to successfully obliterate using the endovascular approach. This is true for transarterial approaches. However, since 1985 (the time frame of this study), microguidewires and microcatheter advances have made the transvenous endovascular approach the treatment of choice for Type D CCFs (1). These cases for which treatment by a combination of transarterial and transvenous embolization failed are difficult to manage, and these patients may be candidates for further endovascular treatment options. For example, in the illustrative case, a woman with deterioration in her vision 12 hours after incomplete embolization of a CCF was taken to surgery because of visual loss. The angiogram, however, shows no drainage into the superior ophthalmic vein (SOV) but instead potential pial venous drainage through the posterior aspect of the fistula. A transvenous approach to the fistula would likely have been successful. However, it is well known that visual deterioration may occur for a period of time despite effective obliteration of the fistula.

The indications for surgery were persistent or progressive neurological deficit related to the CCF, including "rapid and progressive visual loss." As part of the natural history, patients frequently experience worsening of vision and visual acuity as a fistula thromboses (3). The worsening is usually caused by petrosal vein thrombosis that causes redirection of flow to, or thrombosis of, the SOV. The cavernous sinus sinusoids may thrombose, become less compliant, and acutely increase pressure in the orbit. However, continued thrombosis usually leads to fistula thrombosis before severe visual loss becomes permanent. Anticoagulation may improve some of the acute vision deterioration. In our experience, there is no evidence that surgery preserves vision and that failure to



perform surgery would have led to vision loss. Therefore, the indications of direct surgery are infrequent. A possible indication for the illustrative case would be persistent CCF with pial venous drainage.

Of the nine patients treated using direct surgery, one (11%) patient had a permanent ischemic complication and one (11%) had a transient ischemic complication. The permanent morbidity in an early case may have been treated before fully understanding the volumes required for packing. In our experience, the CCF can be packed via triangular corridors and then unpacked to avoid excessive compression of the intracavernous internal carotid artery (ICA) or the cranial nerves (3). However, current endovascular techniques have recently invalidated the need for direct surgical treatment for Type D direct CCFs.

It is clear from the authors' detailed discussion of their surgical experience with the "triangular corridor" anatomy that they have a superb knowledge and understanding of the spatial relationship of the cavernous sinus and its relationship to the intracavernous ICA and the anastomotic network of dural arteries. This anatomic expertise has led them to advance the pioneering methodology for CCF obliteration presented by Mullan (2). The authors document an anatomic understanding of the direct surgical treatment of dural CCFs, and the detailed methodology will be appreciated by neurosurgeons who treat this problem. Their sophisticated technique relies on precise angiographic identification of arterial feeders to the CCF for selection of the appropriate triangular corridors to be packed in a stepwise fashion to obliterate the CCF without compromising intracavernous ICA flow or the cranial nerves of the lateral wall.

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Day and Fukushima describe a direct microsurgical approach for a complex subgroup of CCFs supplied by both internal and external carotid artery feeders (Barrow Type D CCFs). They appropriately state that endovascular techniques have become the mainstay of treatment for CCFs, but there are situations in which endovascular approaches are inadequate. The authors present nine cases of Type D CCFs for which endovascular therapy had failed and that were subsequently treated using microsurgery to achieve direct obliteration. Their indications for direct microsurgery were clear and appropriate. Candidates for open surgery were patients who, in the surgeon's opinion, had continued rapid progressive

visual loss despite attempts at treatment with available endovascular techniques. The authors include an appropriate discussion of the microsurgical anatomy relevant to the approach. Results of their interventions are concisely discussed and summarized in table format.

It is interesting to note that all nine of their endovascular failures were at least partially secondary to inadequate occlusion of the meningohypophyseal trunk (MHT) feeders. Endovascular therapy failed in four of these cases despite the use of transvenous approaches. Catheterization of the MHT from the ICA is often difficult because of its size, and a transvenous approach with endovascular packing of the cavernous sinus is frequently required. If the standard transvenous approach via the inferior petrosal or other sinus fails, one should consider an SOV approach via an orbital lid cutdown (2). In our experience, this approach can be successful even when other endovascular procedures fail. The SOV approach is possible when the SOV is an enlarged prominent draining vein from the CCF. If this also fails or is not possible, the microsurgical approach is indicated. Direct ligation of the MHT feeder with an aneurysm clip or suture is possible through the superior triangle, as described in the authors' first illustrative case. In the few cases for which all endovascular approaches fail to obliterate the MHT feeders, this direct ligation may be the only method available to obliterate the CCFs.

Most of the unique risks of this microsurgical approach are related to the difficulty of packing the cavernous sinus triangles to an optimal pressure that occludes the fistula while only minimally disturbing the ICA and cranial nerves. The authors state that such techniques rely on a "sophisticated sensitivity to packing pressure and volume by an experienced surgeon," and they discuss the "subjective" nature of this crucial step to prevent overpacking. However, even with the excellent technical skills of these authors and their combined surgical experience with this type of lesion, they still encountered a 22% (2 of 9 patients) incidence of complications directly attributable to overpacking the triangles, with subsequent ICA compromise. However, if they performed significantly less packing, there could have been persistence of the CCF and failure of treatment.

There are several combination approaches available that could eliminate or reduce the risk of ICA compromise from overpacking during this microsurgical procedure. Endovascular temporary balloon test occlusion with hypotensive challenge during the initial transarterial procedure is a simple maneuver that might have prevented the two cases of hemiparesis secondary to ICA compromise (3). This test could ascertain which patients might not tolerate aggressive triangle packing before the open surgical procedure, perhaps indicating that increased precautionary measures are needed.

Intraoperative angiography is another simple procedure that can be used during the open microsurgical obliteration of a complex CCF. If a catheter remains in the carotid artery intraoperatively, each stage of packing can be evaluated with the "gold standard" to ensure patency of the ICA and obliteration of the fistula. This information would obviously be invaluable in preventing complications secondary to ICA occlusion or narrowing, such as the authors experienced. Also,