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Carotid Cavernous Fistulas: Anatomy, Classification, and Treatment

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The cavernous sinus is defined as a dural envelope through which the intracranial extradural segment of the internal carotid artery (ICA) and its branches traverse from the petrous and lacerum segments to the interdural and intradural supraclinoid segments. An abnormal communication between the ICA and external carotid artery (ECA) or any of their branches and the cavernous sinus is termed a *carotid cavernous fistula* (CCF). Patients with CCFs usually present with Dandy's triad of pulsatile exophthalmos, chemosis, and bruit.

These lesions are usually classified as direct or indirect. Direct fistulas have an abnormal communication between the ICA and the cavernous sinus. Indirect fistulas have an abnormal communication between the meningeal branches of the ICA and ECA and the cavernous sinus [1,2]. Direct CCFs are usually caused by trauma, fibromuscular dysplasia, ruptured intracavernous artery aneurysm, collagen deficiency, arterial dissection, or iatrogenic causes (eg, surgical trauma) [3–8]. The causes of indirect CCFs are unknown, but indirect CCFs have been associated with pregnancy, sinusitis, trauma, cavernous sinus thrombosis, and surgery [9–13].

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Anatomy

Essential to the understanding of the causes and treatment of CCFs is a detailed knowledge of the cavernous sinus, intracavernous ICA, and meningeal branches of the ICA and ECA. Parkinson [14] first described the detailed anatomy of the cavernous sinus, which constitutes an unexpansible extradural venous plexus. The cavernous sinus communicates with adjacent regions through emissary veins in the following ways: anteriorly through the superior orbital fissure with the orbit, anteroinferiorly through the foramen rotundum with the superior portion of the pterygopalatine fossa, laterally through the foramen ovale and the foramen of Vesalius with the pterygoid region, and posteriorly through the superior petrosal sinus and inferior petrosal sinus (IPS) with the jugular vein of the upper neck (Fig. 1) [15].

The nerves in the lateral wall of the sinus (from superior to inferior) are the oculomotor, trochlear, and the first trigeminal division or ophthalmic nerves (Fig. 2). The abducens nerve courses medial to the ophthalmic nerve and lateral to the ICA. Sympathetic fibers course on the surface of the artery as it courses over the foramen lacerum. The fibers join the abducens nerve within the sinus before being distributed to the first trigeminal division, which sends sympathetic fibers that reach the pupillodilator through the long ciliary nerves by passing through the ciliary ganglion [14]. Some sympathetic fibers pass directly from the carotid plexus to the ciliary ganglion, and

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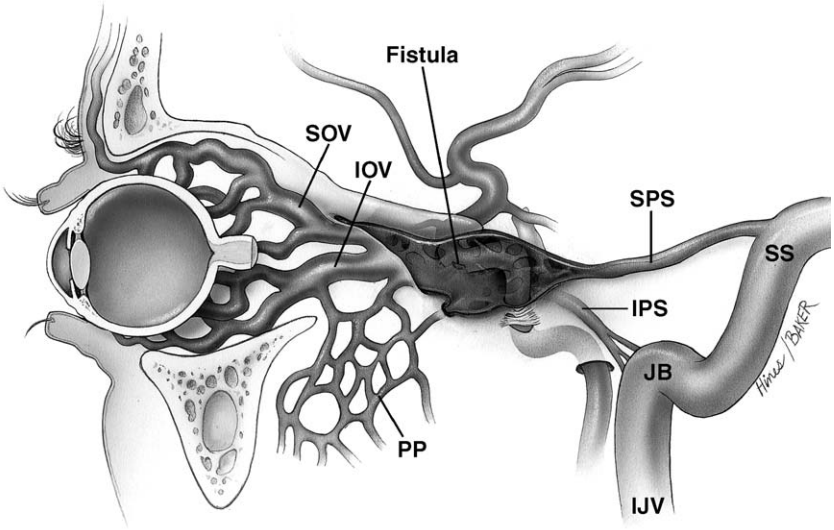

Fig. 1. Lateral view of a carotid cavernous fistula (CCF) causing enlargement of the cavernous sinus venous system and exophthalmos. IOV, inferior ophthalmic vein; IJV, internal jugular vein; IPS, inferior petrosal sinus; JB, jugular bulb; PP, pterygoid plexus; SS, sigmoid sinus; SOV, superior ophthalmic vein; SPS, superior petrosal sinus. (*Courtesy of* the Mayfield Clinic; with permission.)

other fibers may travel along the ophthalmic artery to the globe [16].

The ICA may be divided into seven anatomic segments (Fig. 3A). The cervical (C1) segment extends from the bifurcation to the skull base. The petrous (C2) segment courses through the petrous canal and terminates in the lacerum (C3) segment, where the ICA courses over the foramen lacerum. As the ICA enters the cavernous sinus, it is known as the C4 segment. The shortest segment, the clinoidal (C5) segment, extends from the distal dural ring to the ophthalmic artery. The ophthalmic (C6) segment extends to the posterior communicating artery origin, which marks the beginning of the communicating (C7) segment [17]. Debrun [4] further classified the intracavernous portion into five segments from the anterior clinoid process to the petrous canal as follows: anterior ascending segment, junction of the anterior ascending and horizontal segment, horizontal segment, and junction of the horizontal and posterior ascending segment. The clinoid or anterior ascending segment is surrounded by the anterior clinoid process laterally, the optic strut anteriorly, and the carotid sulcus medially to form a narrow space between the bone and artery. The intracavernous ICA is relatively fixed by this bony ring.

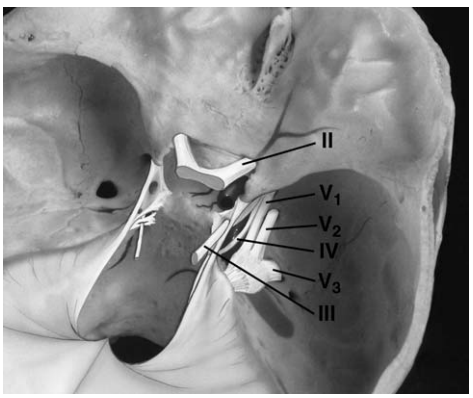

Fig. 2. The cranial nerves in and around the cavernous sinus. Optic nerve (II), oculomotor nerve (III), trochlear nerve (IV), and three divisions of the trigeminal nerve: ophthalmic (V_1), maxillary (V_2), and mandibular (V_3). (*Courtesy of* the Mayfield Clinic; with permission.)

The cavernous ICA gives off the following branches: the meningohypophyseal trunk, the inferolateral trunk or artery of the inferior cavernous sinus, McConnell's capsular artery, and, less frequently, the ophthalmic artery (Fig. 3B).

The meningohypophyseal trunk arises just before the apex of the first curve of the intracavernous ICA. This trunk is the most proximal branch of the intracavernous ICA, is fairly constant, and divides near the roof of the cavernous sinus. The meningohypophyseal trunk gives

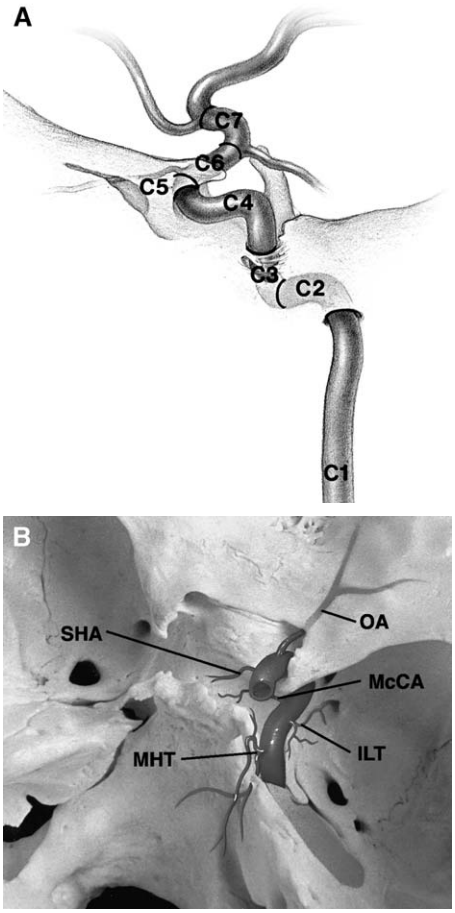

Fig. 3. (*A*) Classification of the internal carotid artery (ICA) segments includes cervical (C1), petrous (C2), lacerum (C3), cavernous (C4), clinoid (C5), ophthalmic (C6), and communicating (C7). (*From* Bouthillier A, van Loveren HR, Keller JT. Segments of the internal carotid artery: a new classification. Neurosurgery 1996;39:425–33; with permission.) (*B*) Branches of the cavernous (C4) carotid artery. ILT, inferolateral trunk; McCA, McConnell's capsular artery; MHT, meningohypophyseal trunk. The ophthalmic artery (OA) and superior hypophyseal artery (SHA) arise from the ICA just distal to the distal dural ring. (*Courtesy of* the Mayfield Clinic; with permission.)

off three branches. First, the tentorial branch or artery of Bernasconi-Cassinari, which runs along the medial edge of the tentorium, supplies the dura of the tentorium and proximal portion of cranial nerves III and IV. It is the most constant branch of the meningohypophyseal trunk and anastomoses with the meningeal branches of the ophthalmic artery. Second, the inferior hypophyseal artery courses medially to supply the posterior pituitary capsule and lobe and anastomoses with the inferior hypophyseal artery on the opposite side. Third, the dorsal meningeal artery, which passes posteriorly through the cavernous sinus to supply the dura over the clivus, sends a branch to cranial nerve VI and anastomoses with the dorsal clival artery on the opposite side.

The inferolateral trunk arises from the lateral side of the horizontal intracavernous ICA approximately 5 to 8 mm distal to the meningohypophyseal trunk [18]. The inferolateral trunk gives off four branches. First, a superior or tentorial branch supplies the roof of the cavernous sinus. Second, an anteromedial branch passes through the superior orbital fissure to supply cranial nerves V_1, III, IV, and VI, which may anastomose with the ophthalmic artery. Third, an anterolateral branch into the foramen rotundum may anastomose with the distal internal maxillary artery via the artery of the foramen rotundum. Fourth, a posterior branch passes medial and under the trigeminal ganglion. The inferolateral trunk also forms anastomoses with the proximal dural branches of the middle meningeal artery and is the branch that contributes most to cavernous sinus dural fistulas [19,20].

McConnell's capsular arteries arise from the medial side of the ICA distal to the origin of the inferolateral trunk. They supply the walls of the hypophyseal fossa and anastomose with branches of the inferior hypophyseal artery and its opposite mate [21].

The ECA contributes to the vascular network of the cavernous sinus. The accessory meningeal artery, which is a branch of the middle meningeal artery or maxillary artery, reaches the cavernous sinus through the foramen ovale or the foramen of Vesalius to form anastomoses with dural branches of the inferolateral trunk of the ICA [22]. The hypoglossal branch of the ascending pharyngeal artery gives off an ascending branch that anastomoses with the medial clival artery at the sella turcica. This anastomotic system makes it possible to see the posterior lobe of the hypophysis during injection of the ascending pharyngeal artery (Fig. 4).

The right and left cavernous sinuses communicate via a venous network localized to the clivus (Fig. 5). The cavernous sinus normally receives drainage from the superior and inferior ophthalmic veins as well as superiorly from the sphenoparietal sinus, sylvian veins, and cortical veins. The cavernous sinus drains posteriorly through the IPS and a superior petrosal sinus to the

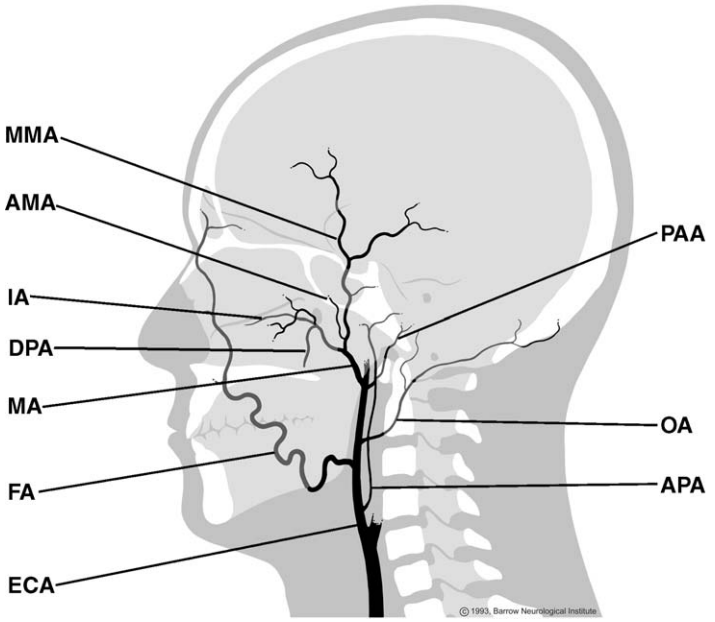

Fig. 4. Branches of the external carotid artery (ECA). AMA, accessory meningeal artery; APA, ascending pharyngeal artery; DPA, descending palatine artery; FA, facial artery; IA, infraorbital artery; MA, maxillary artery; MMA, middle meningeal artery; OA, occipital artery; PAA, posterior auricular artery. (*Courtesy of* the Barrow Neurological Institute; with permission.)

jugular bulb, inferiorly through the pterygoid plexus via emissary veins, and contralaterally through the contralateral cavernous sinus. With venous hypertension caused by an arteriovenous fistula, flow patterns may be revised.

Thus, the branches of the intracavernous ICA and ECA provide a circuit for the cavernous sinus. When one portion is occluded, this system usually provides a collateral pathway. Many of these intracavernous branches are enlarged in patients with CCFs. Thus, a detailed review of the anatomy and hemodynamics of CCFs is necessary for treatment.

Classification of carotid cavernous fistulas

CCFs have been classified by their cause (spontaneous or traumatic), hemodynamic properties (high or low flow), and anatomic variability. The etiologic classification does not consider the hemodynamic and anatomic features of the lesion in relation to prognosis and therapy. With this classification, we might not distinguish a spontaneous dural high-flow fistula from a fistula caused by a ruptured intracavernous artery aneurysm clinically. Although hemodynamic classification is important to explain symptoms and plan treatment, this classification is subjective. The determination of flow on clinical and radiologic grounds is difficult and depends significantly on the physician performing the procedure. The anatomic classification provides the clinician with the definite angioarchitecture of the lesion on which a therapeutic strategy can be based.

Barrow et al [23] distinguished four types of CCFs based on arterial supply (Fig. 6). Type A is a direct fistula between the intracavernous ICA and cavernous sinus. Type A fistulas usually present with high-flow rates. Type B fistulas have dural ICA branches to the cavernous sinus, which are relatively uncommon. Type C fistulas have dural ECA branches to the cavernous sinus. Type D fistulas have dural ICA and ECA branches to the cavernous sinus. Tomsick [24–26] subclassified type D CCFs into type D1 or D2 depending on the presence of a unilateral or bilateral supply. Peeters and Kroger [1] discussed the first three fistula types, excluding type D fistulas of Barrow's classification. Larsen et al [27] described four types of CCFs as follows: type 1 are traumatically acquired direct arteriovenous fistulas; type 2 fistulas are caused by rupture of an intracavernous aneurysm into the cavernous

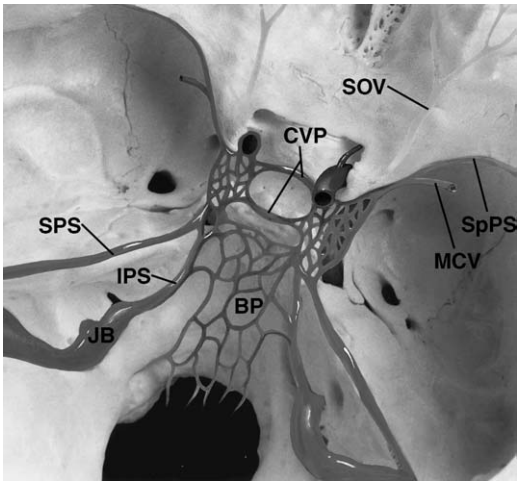

Fig. 5. Superior view of the right and left cavernous sinus communication across the sellae and clivus. BP, basilar plexus; CVP, circular venous plexus; IPS, inferior petrosal sinus; JB, jugular bulb; MCV, superficial middle cerebral vein; SOV, superior ophthalmic vein; SpPS, sphenoparietal sinus; SPS, superior petrosal sinus. (*Courtesy of* the Mayfield Clinic; with permission.)

sinus; type 3 fistulas have ECA or ICA branches to the cavernous sinus; and, finally, type 4 CCFs are a combination of direct and indirect fistula characteristics.

We discuss the classification of direct and indirect fistulas and the variations within these types.

Direct fistulas: causes and pathologic findings

Direct fistulas, type A, are acquired arteriovenous shunts between the ICA and the cavernous sinus. They are thought to be caused by a lesion in the wall of the cavernous ICA or in the wall of one of its branches, typically a result of head trauma or iatrogenic injury. Most of the lesions occur in young adults, and 20% may occur spontaneously as a result of a cavernous ICA aneurysm rupture or weakened ICA vessel walls [28]. Parkinson [28] postulated that direct CCFs are caused by tears of the ICA and meningeal vessels from the cavernous ICA. He theorized that the vessels can easily be torn by bony fractures. During trauma, movement of the artery might stretch and tear the ICA and its branches because it is fixed at the proximal petrosphenoid ligament just beyond the lacerum segment.

However, Helmke et al [29] refuted Parkinson's theories of skull fracture, tearing off, and ICA laceration. In their study of 42 cases, they found no history of skull fractures. Friedmann et al [30] showed that direct CCFs occurred in less than 1% of skull injuries and that this incidence did not parallel that of head trauma. The tearing-off hypothesis did not explain why Debrun's segment C3 is rarely involved in the genesis of direct CCFs and why most direct CCFs are localized in the C4 segment, which is always free of branches in adults. The trabeculae, which span out between the outer surface of the ICA and the outer wall of the cavernous sinus, are shown to insert tangentially into the adventitia of the ICA, not reaching the muscular layer. Thus, it seems unlikely that tugging of the trabeculae could destroy the wall of the ICA [29].

Helmke et al [29] concluded that direct CCFs develop from direct rupture of the vessel wall and that the ruptures are caused by distention of the vessel wall induced by increased intraluminal pressures. This increased tension may be caused by an intense axial acceleration of the body or by sudden compression of the carotid arteries, for example, by extreme sudden extending or bending of the neck.

Debrun et al [31] showed most direct CCFs occur in the proximal horizontal intracavernous segment. The next most common sites are the junction of the horizontal and intracavernous ascending segments, posterior ascending segment, junction of the anterior ascending and horizontal intracavernous segments, and the anterior ascending segment.

Most direct type A CCFs are high-flow shunts. Total steal, which is a complete absence of filling of the ICA above the fistula, occurs in 5% of patients at diagnosis. Type A fistulas typically range from 1 to 5 mm in size (average = 3 mm) [30]. Bilateral traumatic CCFs occur in approximately 1% to 2% of patients with traumatic CCFs [32].

Spontaneous direct CCFs usually arise from any condition that predisposes the ICA wall to weaken [33]. Predisposing factors to the development of spontaneous type A CCFs are collagen deficiency syndrome, which causes a defect in the arterial wall media, (eg, aneurysms of the cavernous ICA); Ehlers-Danlos syndrome [34]; fibromuscular dysplasia [35]; and pseudoxanthoma elasticum [36].

Clinical features of direct fistulas

Patients with direct fistulas initially may present with intracranial bruit. Most patients present

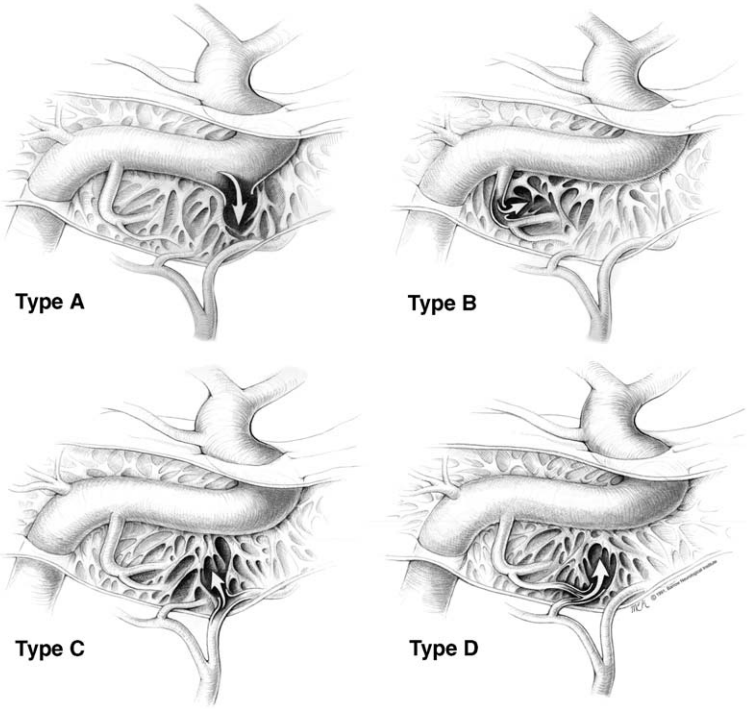

Fig. 6. Classification of carotid cavernous fistulas. (*A*) Type A fistulas involve a direct communication of the internal carotid artery (ICA) and the cavernous sinus. (*B*) Type B fistulas arise from a torn intracavernous branch of the ICA. Type C fistulas arise entirely from external carotid dural branches (*C*), whereas type D fistulas include small dural branches of the ICA and external carotid artery (*D*). Type D2 fistulas involve bilateral internal and external carotid supply. (*Courtesy of* the Barrow Neurological Institute; with permission.)

with proptosis (90%), chemosis (90%), diplopia (50%), pain (25%), trigeminal nerve dysfunction, elevated intraocular pressure, and visual loss (up to 50%) [25].

Retrograde venous drainage from the cavernous sinus into the orbit produces venous hypertension and increased orbital venous volume. This increase in orbital vascular volume also causes enlargement of the extraocular muscles, which subsequently leads to proptosis. Increased exposure of the cornea may cause corneal damage. Diplopia may also occur because of limited mobility of the extraocular muscles. Chemosis is another clinical presentation of ocular venous hypertension. The conjunctival vasculature becomes incompetent to hold fluid and then leaks into the conjunctiva, causing it to swell [19].

Normal intraocular pressure is maintained by a pressure gradient between the anterior chamber and its aqueous fluid and the episcleral vein; the former has a higher pressure than the latter. Increases in episcleral venous pressure cause a reversal of flow. Subsequently, the aqueous fluid is not reabsorbed appropriately, and intraocular pressure elevates. Visual loss may also result from decreased ocular or retinal perfusion because of orbital venous stasis. The pain in direct CCFs is presumably caused by involvement of the dural walls of the cavernous sinus [37].

Intracranial hemorrhage develops in 5% of patients, probably as venous drainage reverses into the sphenoparietal sinus with occlusion of other drainage pathways, with resultant cerebral cortical venous hypertension. One percent to 2% of these cases manifest life-threatening epistaxis caused by rupture of a pseudoaneurysmal cavernous sinus varix [25].

Radiographic evaluation

Noninvasive imaging, such as CT and MRI, is useful in the diagnostic workup of traumatic CCFs. A CT scan of the orbit usually demonstrates proptosis of the affected globe, dilatation of the cavernous sinus and superior ophthalmic vein, and enlargement of the extraocular muscles

[37]. It is also useful to identify skull fractures that may compromise the carotid artery lumen. Orbital ultrasound is another noninvasive means to image these changes. The "gold standard" for diagnosis is selective cerebral angiography, however. The initial angiographic evaluation should be tailored to obtain the following information: size and location of the fistula, presence of any associated cavernous carotid aneurysm, identification of high-risk features (eg, cortical venous drainage, pseudoaneurysm, cavernous sinus varix), venous drainage patterns, differentiation of direct from indirect lesions, and associated vascular injuries.

The angiographic workup must include the five following parts. First, assessment of the morphology of the carotid bifurcation and the origin of the ICA is performed by injection of both common carotid arteries (CCAs). Visualization of the contralateral CCA is needed to rule out a carotid dissection or stenosis, pseudoaneurysm, or contralateral CCF. Second, ipsilateral ICA and ECA angiograms can identify the location of the fistula to assess whether the CCF is of high or low flow and to determine the presence of a complete or partial steal. Complete steal is of enormous interest because it confirms that the CCF is of high flow, that the fistula tear is large, and that the patient has an excellent circle of Willis if there are no contralateral deficits [24]. In patients with high-flow fistulas, regular ICA angiography at three frames per second cannot possibly detect the morphology of the fistula. Therefore, specific maneuvers can slow down the flow through the fistula. The Mehringer-Hieshima maneuver consists of a gentle ipsilateral ICA injection and manual compression of the ipsilateral carotid artery while filming at a slower rate [2]. Use of a double-lumen balloon catheter in the ipsilateral ICA is another way to demonstrate the fistula. With the balloon in place, slow injection of contrast at 1 mL/s at one or two frames per second allows opacification of the fistula [38]. Third, assessment of the patency of the anterior communicating artery is performed by ipsilateral carotid compression of the contralateral ICA. Fourth, ipsilateral carotid compression of the vertebral artery, called the Heuber maneuver, opacifies the fistula through a patent posterior communicating artery. Fifth, assessment of venous drainage is extremely important for treatment by correlation with patient's symptoms. Patients with cranial nerve deficits usually have petrosal venous drainage. Patients with ocular symptoms usually have superior ophthalmic venous drainage. Patients with severe headaches may have cortical venous drainage and should be treated aggressively because of their predisposition to bleed. The venous route also provides excellent access for treatment.

Indirect fistulas (types B, C, and D): causes and pathologic findings

Indirect CCFs (types B, C, and D) are typically low-flow fistulas and are also called dural fistulas. They may occur spontaneously in women older than 50 years of age. The cause of these lesions is still unclear, but some evidence points to a congenital origin [39]. Congenital dural fistulas have been reported in infants as young as 5 weeks of age. Taniguchi et al [40] postulated that indirect CCFs may represent a collateral response to thrombosis of the cavernous sinus and may be a manifestation of a thrombotic tendency, which may lead to multiple dural fistulas. Barrow favors the theory of Newton and Hoyt [41], who postulated that spontaneous low-flow CCFs form after the rupture of one of the thin-walled dural arteries that normally traverse the cavernous sinus. Different factors that may predispose patients to this rupture include hypertension [42], pregnancy [11], trauma and straining [40], atherosclerotic disease [40], and collagen vascular disease.

Clinical features of indirect fistulas

The onset of symptoms of indirect CCFs is more insidious compared with direct CCFs. The symptoms and signs of the former are often fewer and less severe than those of the latter. Among 260 cases reviewed at diagnosis, signs and symptoms included red eye (90%), proptosis (89%), increased intraocular pressure (83%), double vision or cranial nerve paresis (68%), pain (40%), and bruit (39%) [37]. Diplopia is usually horizontal because of the involvement of the abducens nerve, which occurs in 50% of cases; in contrast, vertical diplopia of a trochlear nerve palsy or upward gaze occurs because of oculomotor nerve involvement. The possible causes of cranial nerve paresis are venous congestion, venous compression, or, less often, ischemia caused by arterial steal from the meningohypophyseal and inferior cavernous arterial supply to the nerves.

Spontaneous resolution of dural fistulas can occur independent of treatment. This resolution may be attributed to further thrombosis of the involved segment of the cavernous sinus. Its

incidence ranges from 10% to 60% in the literature [21]. Hamby noted cases of spontaneous resolution after diagnostic angiography alone. In fact, the rate of spontaneous cure after diagnostic angiography has been reported to be as high as 43% [43]. Sasaki et al [44] reported that 18 of 19 patients with dural CCFs had complete regression of the signs and symptoms during an observation period ranging from 6 months to 8 years.

Exacerbation and remission of signs and symptoms is the hallmark of the disease, possibly because of cavernous sinus thromboses and shunting of venous flow in different directions. Redirection of blood flow to the superior ophthalmic veins may cause ocular signs and symptoms, that to the retinal vein may cause blindness, that to the spinal pial venous drainage may cause myelopathy, and that to the brain via pial veins may cause intracerebral hemorrhage. Retrograde cortical venous drainage is a well-known cause of neurologic deficits and severe headaches. In Ernst and Tomsick's study [24] of dural CCFs, 20% of patients had known pial venous cortical drainage but no hemorrhage. Halbach et al [45] noted that the risk of intracerebral hemorrhage in patients with cortical venous drainage from direct fistulas may be fatal if untreated. The incidence of hemorrhage with direct or indirect fistulas is remarkably low.

Radiographic evaluation

Angiography is the diagnostic modality of choice. Its goals are to determine the location of the fistula, define the arterial supply to the fistula and pattern of venous drainage, identify any dangerous extracranial-to-intracranial or ophthalmic collaterals, and evaluate the carotid bifurcations before initiation of carotid compression therapy. Tolerance for ICA occlusion should also be evaluated using balloon test occlusion (BTO) to aid in identification of appropriate therapeutic choices.

The angiographic workup should include selective angiography of both ICAs, including visualization of both ascending pharyngeal arteries, both internal maxillary arteries, and ipsilateral vertebral arteries. During ECA injection, the following arteries should be noted: the middle meningeal artery, accessory meningeal artery, distal internal maxillary artery (including the artery of the foramen rotundum and vidian artery), and ascending pharyngeal artery. These arteries usually supply type C and D fistulas. During ICA injections, the following arteries should be noted: the meningohypophyseal artery, inferolateral artery, and McConnell's arteries, because they supply type B and D fistulas [24].

During angiography of CCFs, dangerous anastomoses should be noted. If arterial embolization is contemplated, the following are important: the ophthalmic, meningeal, and vidian arteries and the artery of the foramen rotundum to the ICA branch; ascending pharyngeal to cavernous and petrous carotid arteries; and occipital artery communication to the ICA or vertebral artery. If transvenous embolization is contemplated, the venous phase should be studied to identify venous drainage patterns and determine the route.

Identification of the exact point of fistula in direct CCFs may be difficult because of the high-flow state. In many cases, this point may be visualized by injection of the vertebral artery during manual compression of the ipsilateral carotid artery. The source of flow in indirect CCFs can typically be identified by selective injection of the external carotid branches as mentioned previously.

Determination of the patient's ability to tolerate ICA occlusion is important before embarking on a therapeutic intervention. BTO is the currently accepted technique for evaluation. Proper evaluation requires documentation of collateral flow during ipsilateral ICA occlusion if not already demonstrated on routine angiography. Evaluation of the contralateral carotid or vertebral artery may be performed during BTO using bilateral femoral access with a diagnostic catheter or before BTO by manual compression of the ipsilateral carotid artery. Occlusion of the ICA is performed after gaining access to the CCA with a 6-French guide catheter. Although we used the Endeavor nondetachable balloon (Target Therapeutics, Fremont, California) for occlusion because of its compliant nontraumatic characteristics, this product has recently become unavailable. We now use the Hyperglide balloon (Micro Therapeutics, Irvine, California) for the same purpose. This wire-valve balloon uses a 0.010-in wire to occlude the distal exit port during inflation and has a similar compliance profile to the Endeavor. Administration of intravenous heparin (50–70 U/kg) helps the patient to achieve an activated clotting time (ACT) of greater than 300 seconds before undergoing balloon inflation. Throughout the procedure, a heparinized saline flush (2.5 U/mL at 30 mL/h) is continued via the guide catheter and the groin

sheath. The ACT is checked hourly and maintained over 300 seconds. The ICA may be best occluded with the balloon positioned within the petrous (C2) segment of the ICA. The rigid carotid canal permits ICA occlusion at lower balloon volumes, thereby reducing the risk of dissection. The disadvantage of occlusion at this location is the risk of creating a steal phenomenon with retrograde filling of the fistula. This phenomenon can be recognized during evaluation of collateral flow as described previously. Passing the BTO despite a steal predicts clinical tolerance to ICA occlusion. If the patient develops neurologic signs with a steal above, it does not mean that the ICA cannot be occluded. Therefore, the occlusion test should be repeated with the ostium occluded or by trapping the ostium with two balloons.

After confirmation of ICA occlusion, the patient is evaluated clinically with detailed testing of mental status, speech, visual fields, facial animation, and motor power in all four extremities. If no deficits are noted, the patient is observed for 15 to 20 minutes and re-examined. If the patient tolerates occlusion at normal blood pressure, nitroprusside infusion is initiated and titrated to achieve a mean arterial pressure two thirds of the patient's baseline. The patient is examined again and observed for 15 to 20 minutes. Periodic visualization of the balloon ensures that it has not migrated or deflated. At our institution, standard evaluation includes single proton emission computed tomography (SPECT) to rule out significant asymmetry in perfusion during BTO. This is accomplished by intravenous injection of ^{99m}Tc-hexamethylpropyleneamine oxime during the period of ICA occlusion. A SPECT scan must be performed within 1 hour of injection because of the short half-life of the tracer. This can best be accomplished by calling for tracer injection after achieving target hypotension. The importance of SPECT evaluation, even in patients who seem to tolerate BTO during relative hypotensive challenge, was illustrated in the review by Larson et al [27], in which 2 of 58 patients apparently tolerating BTO with hypotensive challenge suffered a major stroke after permanent carotid occlusion.

Indications for emergency treatment

In a study of 155 patients with CCFs, Meyers et al [46] noted that the presence of the following angiographic features increases the risks of morbidity and mortality: pseudoaneurysm, large varix of the cavernous sinus, venous drainage to cortical veins, and thrombosis of venous outflow pathways distant from the fistula. Clinical signs and symptoms that should concern the interventionalist are increased intracranial pressure; progressive proptosis, which may signify spontaneous thrombosis of venous outflow pathways to the orbit; diminished visual acuity; hemorrhage; and transient ischemic attacks, which may signify impaired cerebral autoregulation secondary to chronic steal phenomenon. Recognition of these signs, symptoms, and radiographic findings should warrant immediate and definitive treatment to improve outcome [46].

Surgical management

Although surgical treatment of CCFs has been relegated to historical status for the last 30 years, it remains a consideration for salvage of failed endovascular attempts. The earliest technique, described in the *Lancet* in 1875, was ligation of the CCA, which was fraught with failure that resulted in symptom resolution for only one third of patients [47]. In 1935, Dandy described a conceptually more appropriate technique for trapping the fistula in which cervical ICA ligation was followed by craniotomy and ligation of the supraclinoid (C5) ICA. Although this proved more successful, a high failure rate, which was blamed on collateral supply from the ECA, remained. In 1942, Jaeger [48] addressed this problem by isolation and ligation of the supraclinoid carotid artery (C5 segment), which was followed by packing the muscle into the cervical ICA. Hamby [49] later modified this by surgically packing muscle into the entire cavernous (C4) ICA segment.

Although the aforementioned techniques are effective, sacrifice of the ICA is required. Transcavernous occlusion of a CCF may permit preservation of ICA flow as described by Parkinson [50] and Mullan [51] Parkinson exposed the C4 segment of the ICA between the trochlear nerve and the first division of the trigeminal nerve. Mullan exposed the ICA between the first and second divisions of the trigeminal nerve or packed the cavernous sinus through the superior petrosal sinus, IPS, or superior ophthalmic vein (Fig. 7). When performing surgical transvenous packing, as in transvenous endovascular repair, the surgeon must avoid occlusion of only the posterior drainage, which could result in pressurization of the superior ophthalmic vein with visual loss or

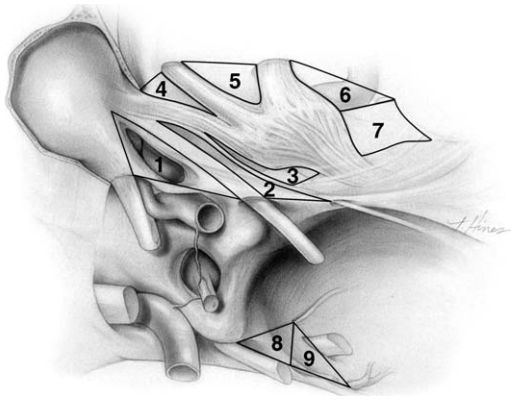

Fig. 7. Surgical view of the cavernous sinus after posterior orbitotomy and anterior clinoidectomy. Anatomic triangles of the cavernous sinus region are (1) anteromedial triangle (Dolenc), (2) paramedical triangle, (3) Parkinson's triangle, (4) anterolateral triangle (Mullan), (5) lateral triangle, (6) posterolateral triangle (Glasscock), (7) posteromedial quadrilateral (Kawase), (8) inferomedial triangle, and (9) inferolateral triangle. (*From* Tew JM, van Loveren HR, Keller JT. Atlas of operative microneurosurgery, vol. 2. Philadelphia: WB Saunders; 2001. p. 76–7; with permission.)

occlusion of the anterior drainage with cortical venous pressurization.

Indications for surgical repair include compromised proximal arterial access that prevents endovascular repair or failure of transarterial and transvenous endovascular repair. Preoperative evaluation should include complete angiographic definition of the fistula and BTO as described previously. The appearance and condition of the superficial temporal artery should also be noted when extracranial-to-intracranial bypass is needed. Although the details of surgical technique and management are beyond the scope of this article, the topic is expertly described by van Loveren et al [52].

Endovascular repair: type A carotid cavernous fistulas

Balloon occlusion

The large carotid defect commonly present in type A CCFs frequently permits transarterial balloon occlusion of the fistula with preservation of the ICA. The earliest procedures were performed with latex balloons hand-tied to the delivery catheter with latex strips. The development of the gold-valve balloon (GVB) permitted selection of a preformed detachable balloon fashioned for manual attachment to the delivery catheter. The balloon was mounted on a 2-French Teflon microcatheter that was passed through the delivery catheter, typically a 3-French catheter, and was detached by manual traction on the catheter. The Heishima detachable silicone balloon (DSB) made use of a miter valve that required a slightly higher detachment force than the GVB. When the US Food and Drug Administration (FDA) limited the use of implanted latex devices, the DSB became the preferred method for balloon occlusion of CCFs.

The technique for DSB occlusion of a CCF involves transfemoral access to the proximal CCA with a 7-French guide catheter or long 6-French sheath. If the ICA origin arises at an oblique angle from the CCA, an angled tip to the guide catheter may aid by directing the balloon during advancement. Once femoral access is gained, the patient undergoes anticoagulation with intravenous heparin (50–70 U/kg) and an ACT is confirmed over 300 seconds. The ACT is checked again hourly, and supplemental heparin is given as needed.

After the guide catheter is positioned, angiography is performed to document the anatomy of the lesion and define the site of the fistula. The specific DSB chosen depends on the size of the venous compartment that needs to be filled and the flow rate through the fistula. Selection of a balloon too small for the cavernous sinus compartment into which the fistula opens results in a persistent or recurrent fistula. For high-flow fistulas, selection of a balloon with a low detachment force may risk premature detachment, whereas a balloon with a high detachment force may not detach safely on reaching the target.

Next, the uninflated balloon is advanced to the distal end of the guide catheter; at this point, roadmap imaging is used for further balloon positioning. Partial inflation and deflation advance the balloon within the artery, permitting blood flow to carry the balloon distally. In many cases, the high flow of the fistula directs the balloon into the cavernous sinus. If the balloon is carried distal to the fistula within the ICA, a nondetachable balloon may be advanced first and inflated distal to the fistula, thus occluding distal flow. Subsequent attempts to advance the DSB should result in deflection into the cavernous sinus through the fistula. On entering the cavernous sinus, the DSB is inflated and drawn back against the fistula. Angiography is performed to document occlusion of the fistula. If flow persists through the fistula, the DSB is inflated further and

the process is repeated until the fistula is occluded (Fig. 8). When occlusion is documented, the balloon is detached by withdrawal of the delivery catheter from the balloon. The force required for detachment depends on the type of balloon chosen.

The advantage of balloon occlusion of a CCF is its ability to occlude the fistula rapidly with preservation of the ICA. Potential disadvantages include fistula recurrence and ICA compromise as the inflated balloon protrudes through the fistula site to narrow the adjacent ICA lumen. Although this compromise may require prolonged anticoagulation after completion of the procedure, it rarely restricts distal flow. The fistula may recur if the balloon migrates, deflates, or ruptures. The concern raised in the past with silicone balloons was that filling with a hypotonic solution could cause osmotic loss of fluid volume and partial deflation. For this reason, many interventionalists chose to inflate the DSB with mildly hypertonic saline or metrizamide solutions or with permanent solid material, such as silicone [53] or hydroxyethyl methacrylate [54]. Laboratory research implies that deflation rarely occurs by osmotic fluid loss and that saline-diluted contrast is safe for DSB inflation, however [55]. In cases of traumatic CCFs, we have seen balloons punctured by bony spicules and migration of balloons without deflation presumably because of dilation of the venous compartment by the balloon. The DSB was most recently available from Target Therapeutics and came in three sizes, with each available in low (20–30 g), medium (30–40 g), and high (40–55 g) force of detachment ranges. Like the nondetachable Endeavor balloon, the DSB has been discontinued, making detachable balloon treatment currently unavailable in the United States.

Coil occlusion

Coil occlusion for CCFs became popular after FDA approval of the Guglielmi detachable coil (GDC) system in 1995. The advantages of coil occlusion of CCFs include ease of access and availability of a variety of sizes of the embolic device when compared with balloon embolization. Potential disadvantages include slower gradual occlusion of the fistula, which increases procedure time, and the risk of incomplete fistula occlusion with loss of transarterial access; this loss would then require a second transvenous approach. As with intracranial aneurysms, coil compaction with recurrence remains a concern. This compaction may be minimized by oversizing the first coils used for framing and by keeping the microcatheter tip well into the cavernous sinus. These steps allow for some expansion of the venous compartment during embolization and for dense coil packing distal to the fistula site, thus preventing coil compaction or early loss of microcatheter access.

Our approach to coil embolization of CCFs is similar to balloon occlusion. Transfemoral access is gained before anticoagulation using the heparin dosing outlined previously. As with balloon occlusion, an ACT of 250 to 300 seconds is acceptable as long as parent vessel occlusion is not planned. The small caliber of the microcatheters used in coiling enables access to the ICA with a 6-French guide. If distal balloon occlusion is required to access the fistula as described previously, supplemental heparin may be required to

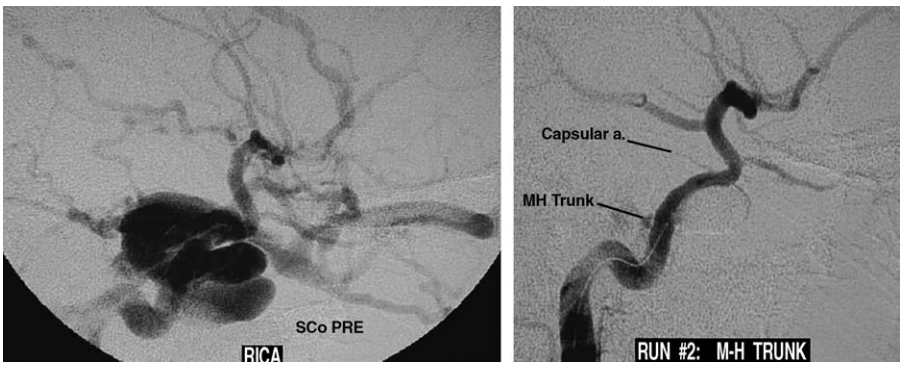

Fig. 8. Before and after angiograms of a direct (type A) carotid cavernous fistula treated with balloon occlusion. During an assault, this patient suffered fractures of the orbit, resulting in a direct fistula. Proptosis and chemosis at presentation later improved after balloon occlusion of the fistula. (*Courtesy of* the Mayfield Clinic; with permission.)

achieve an ACT greater than 300 seconds. The fistula is entered using roadmap guidance after full angiographic definition of the fistula and collateral flow. Dense coil packing is performed using the same principles of aneurysm coiling. We choose our first coil, often a three-dimensional coil, based on the size of the venous compartment opacified on angiography. As in aneurysm coiling, we begin by framing the compartment to be embolized with large coils to protect the fistula site, thus preventing coil prolapse or loss of microcatheter access before fistula occlusion. The fistula remains partially open on angiography in most cases until coil packing is quite dense. For this reason, it is usually advisable to choose coils with some resistance to stretching or unraveling as the coiling progresses.

We have used the GDC system in all our CCF coiling cases, taking advantage of the ''stretch-resistant'' coils in the latter stages of embolization (Fig. 9). After the fistula is closed by angiography, additional coils should only be passed if no resistance is met during advancement. If coil advancement becomes unsafe because of coil prolapse or loss of microcatheter access, the transarterial embolization may be aborted and the fistula may be occluded via a transvenous approach. Complications of transarterial coil embolization include thromboembolus, ICA compromise from protruding coil mass, and ICA dissection. When compared with balloon occlusion, coil occlusion is associated with lower rates of ICA compromise and similar rates of other complications.

Transvenous occlusion may be necessary in cases of inadequate transarterial access or incomplete occlusion after transarterial embolization (see Fig. 9D). The performance of transvenous occlusion requires arterial side angiography to allow complete visualization of the fistula. For this reason, we typically gain venous and arterial access in opposite groins to avoid confusion. The 5-French venous catheter is advanced under roadmap guidance through the

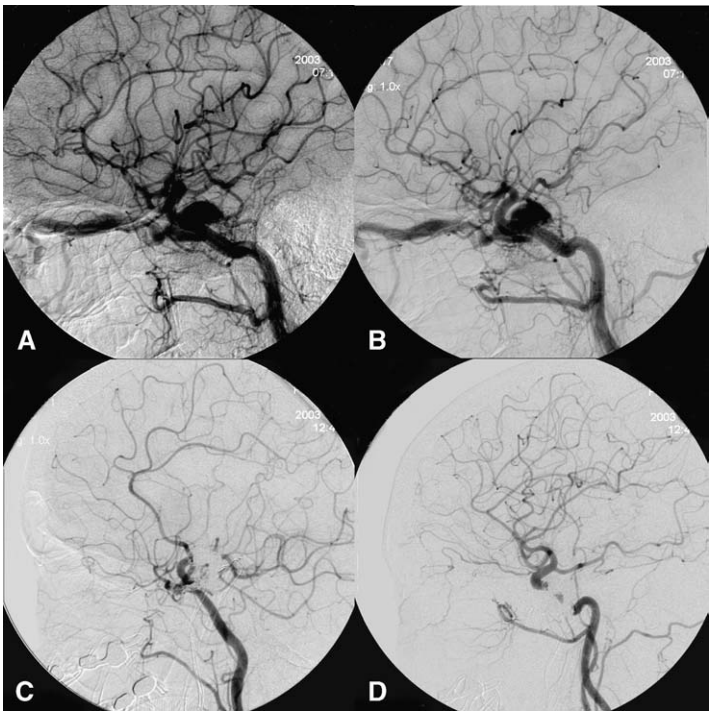

Fig. 9. Coil occlusion of direct (type A) carotid cavernous fistula. (*A*) During an automobile accident, this patient suffered multiple injuries, including a dissecting pseudoaneurysm of the right internal carotid artery. During observation with anticoagulant therapy, he acutely developed exophthalmos with an audible bruit. Angiography demonstrated the fistula (*B*), which was initially treated with transarterial coil occlusion with incomplete closure (*C*). (*D*) Subsequent transvenous coil occlusion cured the fistula. (*Courtesy of* the Mayfield Clinic; with permission.)

inferior vena cava to the superior vena cava and the internal jugular vein. On reaching the jugular bulb, we typically perform a venogram for road-map visualization of the IPS. A dual-tipped microcatheter is subsequently advanced through the IPS into the cavernous sinus to perform the coiling. The same principles of coiling or balloon occlusion apply as were described for transarterial embolization. Throughout the procedure, a 4-French diagnostic catheter is positioned in the ipsilateral CCA for control angiography. The procedure may be terminated when fistula flow is occluded. We prefer to maintain a heparinized saline flush through the 5-French venous guide catheter and the 4-French arterial diagnostic catheter. Alternatively, the arterial catheter may be withdrawn after accessing the cavernous sinus and reinserted for control angiograms. Potential complications include thromboembolus or coil embolus by pushing coils through the fistula into the ICA and thrombosis of the internal jugular vein. Both complications can be prevented by regular use of control angiography or road-mapping and with appropriate anticoagulation. A nondetachable balloon may be inflated in the ICA at the ostium to prevent coil prolapse into the ICA, the ''balloon-assist technique.'' Series for coil embolization describe good results using the transarterial or transvenous route [56,57]. Lewis et al [58] described great success using detachable balloons.

Parent artery occlusion

In general, occlusion of a direct CCF with preservation of the ICA is preferred. In some cases, however, attempts at fistula occlusion with the techniques described previously are unsuccessful or impossible. In other cases, ICA compromise from balloon or coil prolapse may pose risks for thromboembolism such that ICA occlusion is preferred. In these cases, assessment of the patient's ability to tolerate ICA occlusion is paramount. In cases of extremely high-flow fistulas, complete diversion of ipsilateral ICA flow may already be present, with collateral flow defined from other sources with simple angiography. If the patient has tolerated this condition without ischemia, test occlusion may be unnecessary. If the posterior communicating and anterior communicating artery collaterals are patent, the safety of parent occlusion is high. Otherwise, BTO is mandatory.

Occlusion of the ICA, like direct CCF occlusion, may be performed with balloon or coil embolization. Balloon occlusion is performed using the identical access techniques as those used for CCF occlusion. In this case, however, balloon size is matched to the size of the ICA. This match is best predicted by noting the volume required to occlude the ICA during BTO. Medium- or high-detachment force balloons are preferred because of the risk of early detachment in the high-flow environment of the ICA. We prefer a multiple balloon approach, often using three balloons, to prevent distal migration of the distal balloon. In this approach, the first balloon is positioned distal to or across the fistula opening but is not detached until the proximal balloons are in position. On angiographic confirmation of fistula occlusion and safe detachment of the proximal balloon(s), the distal balloon is detached. The risk of distal balloon migration has been described in the acute and delayed settings [59]. The multiple balloon technique minimizes this risk. As mentioned in the section on balloon occlusion of CCFs, detachable balloons are currently unavailable in the United States; therefore, use of this technique with approved devices is impossible until a new product becomes available.

Fistula occlusion with coils depends somewhat more on ICA occlusion distal to the fistula than balloon embolization, because multiple coils are needed to eliminate flow. Positioning coils at the level of the fistula within the ICA may permit continued retrograde filling as the microcatheter migrates proximally. For this reason, moderately dense coil packing is needed distal and proximal to the fistula site. Coil occlusion of the ICA also risks distal migration during the procedure as well as partial fistula occlusion with loss of arterial access. Although this latter complication is less of a concern than with attempted fistula occlusion during ICA preservation, it remains possible if significant retrograde filling is present. Distal coil migration is a significant concern, however, particularly during detachment of the initial distal coils. Antegrade flow may be stopped to permit safe detachment of the coils within the ICA by temporary occlusion of the proximal ICA with a nondetachable silicone balloon. Proximal balloon occlusion may also limit the risk of thromboembolus that arises from the coils by limiting the flow through them while thrombus is developing. Accurate visualization of the distal ICA is necessary during placement of the distal coils. Therefore, road-mapping is performed before proximal balloon occlusion. After road-mapping, further control angiography is

unnecessary until dense coil packing is achieved proximal to the fistula. The proximal balloon is left inflated until this is accomplished. The ease of coil delivery and the ability to retrieve coils before detachment, if necessary, make coil occlusion of the ICA a technically uncomplicated and safe procedure. Even before DSBs became unavailable, we preferred the coil occlusion technique for these reasons.

Endovascular repair: indirect carotid cavernous fistulas (types B, C, and D)

Transarterial embolization

The complex anatomy of indirect CCFs makes cure by transarterial embolization unlikely. In addition, potential complications of the transarterial approach (eg, thromboembolic stroke, cranial nerve palsies) can be virtually eliminated by use of the transvenous approach. For these reasons, the transarterial approach is typically used only to reduce arterial inflow before transvenous occlusion for high-flow indirect CCFs and after failure of transvenous attempts. Some interventionalists think that flow reduction before venous side occlusion may reduce the risk of venous hypertension that may occur before complete venous occlusion. Selection of a site for transarterial embolization requires careful selective angiography of the branches of the ECA. Typically, a 5- or 6-French guide catheter placed in the ostium of the ECA allows selective catheterization of the meningeal branches with a microcatheter using roadmap guidance. When meningeal branches feeding the CCF are identified as a potential site of embolization, provocative testing is performed to evaluate the potential for postembolization cranial nerve palsy. This evaluation can be reliably performed by lidocaine injection (1% solution, 2.5–5 mL), followed by careful examination of cranial nerve function. Development of a new cranial nerve deficit indicates a risk for cranial nerve ischemia with embolization. If permanent embolization is deemed safe, catheter position is confirmed and the embolic agent is prepared.

In general, two embolic agents are considered for use. First, polyvinyl alcohol (PVA) particles (150–250 μm) may be mixed with a dilute contrast solution and slowly injected. If a blank roadmap image is created initially, visualization of the injectate becomes easier. Visualization of arteries not opacified in the microcatheter angiogram may indicate reflux of embolic agent. This reflux must be avoided to prevent embolization to intracranial arteries or the ophthalmic artery [60–62]. PVA embolization has the advantage of easy delivery to the fistula site but may flow through the fistula or result in proximal vessel occlusion because of the difficulty in precisely matching particle size to fistula size. This discrepancy frequently leads to early recanalization.

Second, liquid embolic agents, such as N-butyl cyanoacrylate (NBCA), have an advantage in transarterial embolization because of their ability to conform to the size of the fistula and feeding artery. The viscous opacified acrylic typically does not flow to the smallest involved branches, however, preferring to polymerize in larger arteries. Permanent occlusion may also be achieved if acrylic is deposited within the fistula and into the cavernous sinus. The risk of reflux or embolization through anastomoses to more dangerous locations still exists but may be avoided with similar road-mapping and injection techniques as described for PVA injection.

For either technique, selection of the intracavernous ICA branches has limited use. In most cases, microcatheter access cannot be gained distal enough to the ICA for safe embolization with PVA or NBCA. In some cases, enlargement of the meningohypophyseal trunk permits safe access. Detachable coils may permit safe embolization when only proximal access is available. Vinuela et al [62] described some success with this technique.

Transvenous occlusion

Most interventionalists prefer the transvenous approach because of the lower likelihood of immediate complete occlusion and the ischemic risk of transarterial occlusion of indirect CCFs. Success rates with the transvenous approach are significantly higher than with the transarterial approach for indirect CCFs, as reported by Halbach et al [63]. General approaches to the cavernous sinus access and imaging are identical to the technique described for transvenous occlusion of type A CCFs. Venous side occlusion may be achieved by balloon or coil occlusion (Fig. 10). Liquid embolics and PVA may occlude distal venous pathways, leading to increased pressure within the cavernous sinus and aggravated symptoms.

Summary

CCFs are a heterogeneous group of lesions with varying anatomy and causes. Despite the

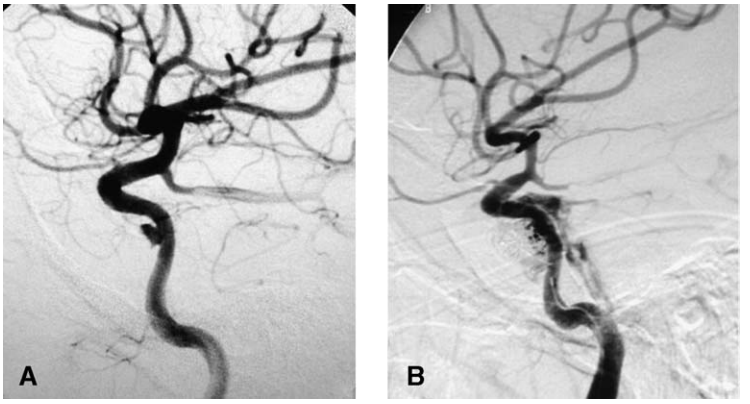

Fig. 10. Indirect (type D2) carotid cavernous fistula treated with transvenous coiling. Access to the posterior cavernous compartment was gained through the inferior petrosal sinus.

complexity of these lesions, a logical treatment plan can be derived with a good understanding of the different types of fistulas and knowledge of the relevant anatomy. Treatments tailored to the specific fistula type and anatomic considerations allow satisfactory treatment with good safety.

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