Neurosurg Focus 32 (5):E9, 2012

Carotid-cavernous fistulas

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Carotid-cavernous fistulas (CCFs) are vascular shunts allowing blood to flow from the carotid artery into the cavernous sinus. The characteristic clinical features seen in patients with CCFs are the sequelae of hemodynamic dysfunction within the cavernous sinus. Once routinely treated with open surgical procedures, including carotid ligation or trapping and cavernous sinus exploration, endovascular therapy is now the treatment modality of choice in many cases. The authors provide a review of CCFs, detailing the current classification and clinical management of these lesions. Therapeutic options including conservative management, open surgery, endovascular intervention, and radiosurgical therapy are presented. The complications and treatment results as reported in the contemporary literature are also reviewed.
(http://thejns.org/doi/abs/10.3171/2012.2.FOCUS1223)

Key Words • **angiography** • **carotid artery** • **carotid-cavernous fistula** • **cavernous sinus** • **embolization** • **shunt**

Located just lateral to the sella turcica, the cavernous sinus is a trabeculated venous cavity invested by the dura mater and contains several neural and vascular structures. Named by Jacobus Winslow in 1734,[85] the term "cavernous sinus" is a misnomer, as it is neither cavernous nor is it truly a sinus. Dwight Parkinson[54,57,58] is credited for his extensive efforts in the 1960s and 1970s to further characterize the anatomy of the cavernous sinus and to define CCFs. Parkinson,[55,56] along with Hashimoto and colleagues,[24] emphasized the inappropriateness of the term "cavernous sinus," preferring the more anatomically correct term "lateral sellar compartment." In any event, the term "cavernous sinus" remains common in medical parlance today.

On average, the cavernous sinus measures 2 cm anteroposteriorly, 1 cm in lateral width, and 1.3 cm in vertical dimension.[55] The major neural and vascular structures traversing the cavernous sinus include the ICA and CNs III, IV, V1, V2, and VI (Fig. 1). The oculomotor nerve (CN III), trochlear nerve (CN IV), and the first and second divisions of the trigeminal nerve (V1 and V2) course between the 2 dural layers of the lateral wall of the cavernous sinus. The abducent nerve (CN VI) runs inside the cavernous sinus itself, lateral to the ICA.[6]

The cavernous sinus has been described as both a trabeculated venous channel and as a venous plexus. Harris and Rhoton[23] divided the cavernous sinus into the following 4 compartments: medial, lateral, anteroinferior, and posterosuperior with respect to the ICA. Normally, the cavernous sinus receives drainage from the sphenoparietal sinus, the superior ophthalmic vein, and the superficial sylvian vein. The middle meningeal vein empties into the cavernous sinus laterally, while the superior and inferior petrosal sinuses drain the cavernous sinus posteriorly. Various venous anastomoses connect the 2 cavernous sinuses, including an intercavernous sinus and a basilar plexus of veins. These anastomoses are clinically relevant, both in terms of the spread of extracranial infections and neoplasms and as alternative routes for venous drainage in patients with obstruction of the cavernous sinus.[6]

Abbreviations used in this paper: CCA = common carotid artery; CCF = carotid-cavernous fistula; CN = cranial nerve; ECA = external carotid artery; ICA = internal carotid artery.

Fistula Classification

Carotid-cavernous fistulas are abnormal vascular shunts, allowing blood to flow either directly or indirectly from the carotid artery into the cavernous sinus. Carotid-cavernous fistulas have been classified according to the hemodynamic properties, etiology, or anatomy of the fistula (Table 1). Hemodynamic classification separates CCFs into high-flow and low-flow fistulas. Etiological classification distinguishes spontaneous lesions from those occurring due to trauma. Anatomical classification defines direct CCFs as those arising directly from the carotid artery, while indirect CCFs are those originating from carotid artery branch vessels.

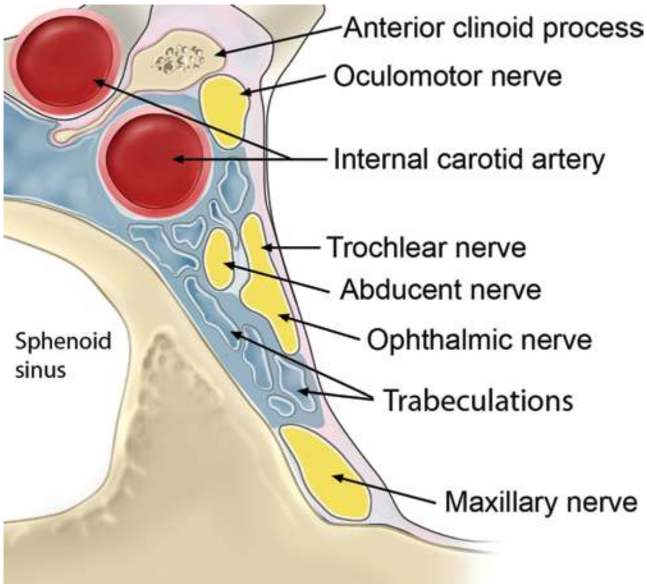

Fig. 1. Schematic organization of the cavernous sinus in coronal section. The major neurovascular structures including the ICA and CNs III–VI within the cavernous sinus are shown. Printed with permission from Jason A. Ellis.

Based on the nomenclature of Peeters and Kröger,[60] Barrow et al.[4] defined 4 types (Types A–D) of CCFs (Fig. 2). Type A CCFs are direct, high-flow lesions connecting the ICA directly to the cavernous sinus. Type A CCFs often result from a single tear in the carotid artery wall, caused either by trauma or aneurysm rupture. These are by far the most common type of CCFs, accounting for approximately 75%–80% of CCFs overall.[4,12] Type A CCFs are now less commonly seen in part due to increased motor vehicle safety measures such as the standardization of airbags and widespread usage of seat belts. Type B, C, and D CCFs are all indirect, low-flow lesions that arise from meningeal branches of either the ICA or ECA. Type B CCFs arise from meningeal branches of the ICA, Type C CCFs arise from meningeal branches of the ECA, and Type D CCFs arise from meningeal branches of the ICA and ECA.[4] In a study of 132 patients presenting with CCFs, 100 CCFs (75.8%) were classified as Type A, none were classified as Type B, 4 (3%) were classified as Type C, and 28 (21.6%) were classified as Type D.[12] Meyers et al.[48] characterized the arterial supply of 135 dural (Types B–D) CCFs and showed that the internal maxillary, middle meningeal, and meningohypophysial trunks and capsular arteries each supplied more than 50% of the fistulas.

Epidemiology and Etiology

Traumatic CCFs

Traumatic CCFs are the most common type, accounting for up to 75% of all CCFs.[87] They have been reported to occur in 0.2% of patients with craniocerebral trauma and in up to 4% of patients who sustain a basilar skull fracture.[25,43] Consistent with the demographics associated with traumatic injuries, traumatic CCFs are most commonly seen in young male patients. They typically occur as a result of a closed head injury–associated basilar skull fracture. There are various theories regarding the mechanism of CCF formation after head trauma. One theory holds that the carotid artery is directly torn either by a bony fracture or by shear forces during the traumatic incident. Finding no skull fractures in a series of 42 patients with posttraumatic direct CCFs, Helmke et al.[25] proposed the following alternative theory: that there is a sudden increase in intraluminal pressure of the ICA with concurrent distal artery compression, which forces rupture of the vessel wall and results in a CCF. While the vast majority of CCFs caused by trauma are direct CCFs, posttraumatic indirect CCFs have been reported as well.[10,46]

TABLE 1: Classification of CCFs

Type	Classification
hemodynamic	high vs low flow
etiological	spontaneous vs traumatic
anatomical	direct vs indirect

Traumatic CCFs may also result from projectile or slash injuries that result in laceration of the cavernous carotid artery, allowing high-flow communication with the cavernous sinus. Although rare, there have also been reports of CCFs caused by iatrogenic injury during craniotomy, carotid endarterectomy, transsphenoidal exploration, endovascular procedures, and sinus surgery.[40,50,59,63,70,74] Bilateral CCFs are seen in 1%–2% of patients with posttraumatic CCFs.[42,43]

Spontaneous CCFs

Spontaneous CCFs, which account for approximately 30% of all CCFs, are typically found in older, female patients.[10,12,42,81] Ruptured cavernous ICA aneurysms are an often-cited cause of spontaneous, direct CCFs. Cavernous-carotid fistulas are reported in anywhere from 3% to 24% of patients with cavernous carotid aneurysms.[38,41,45,80] In addition to cavernous aneurysms, genetic conditions such as fibromuscular dysplasia,[29,39] Ehlers-Danlos syndrome,[8,15,69] and pseudoxanthoma elasticum[67] are known to predispose patients to spontaneous CCFs. It is thought that arterial wall defects in these patients predispose them to CCF formation after minor stress, such as coughing or Valsalva maneuver. In individuals without cavernous aneurysms or predisposition syndromes, it has been theorized that microscopic venous thrombosis or increases in venous sinus pressure may facilitate fistula formation by causing microscopic breaks in dural vessels to the cavernous sinus.[4,51,66,87] Factors thought to contribute to the breaks in these vessels include arterial hypertension, atherosclerotic vascular disease, pregnancy, minor trauma, straining, diabetic vascular disease, and collagen vascular disease.[44,51,65,72,77]

Clinical Presentation

Direct CCFs

Consistent with their etiology either from traumatic

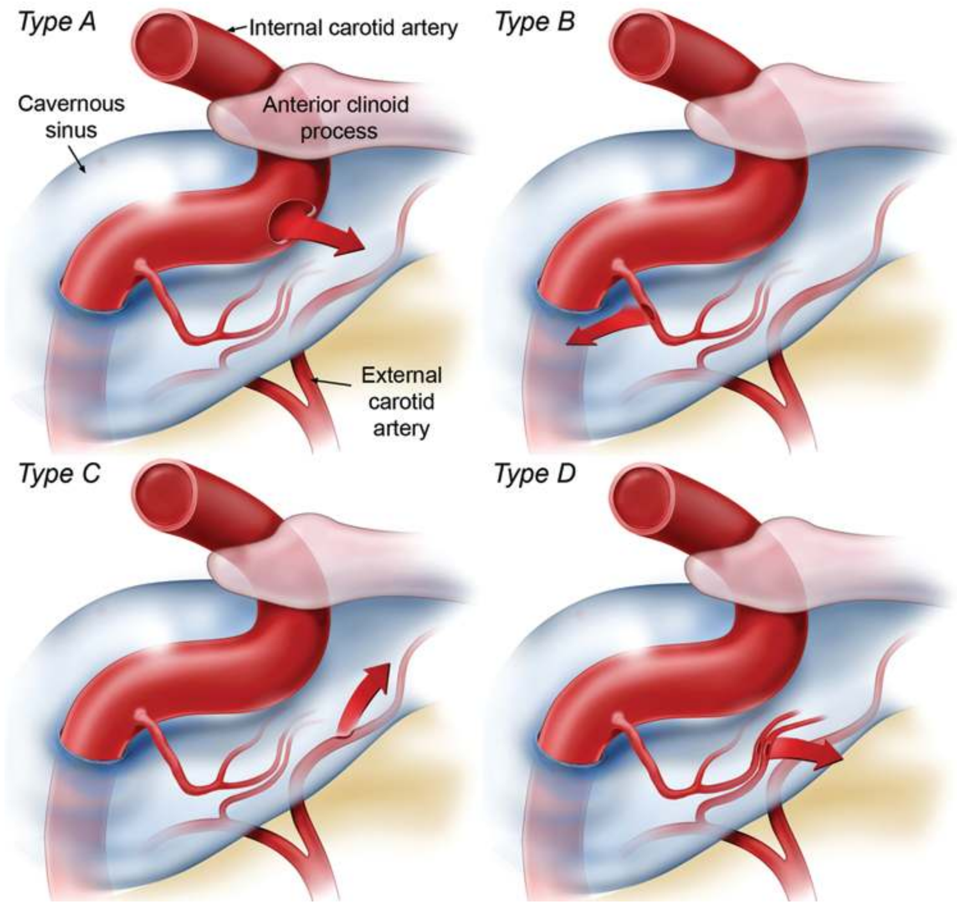

Fig. 2. Barrow classification of CCFs. Type A fistulas are characterized by direct shunting of blood flow from the ICA into the cavernous sinus. Type B and C fistulas are shunts to the cavernous sinus from branches of the ICA and ECA, respectively. Type D fistulas have shunts from both the ICA and ECA simultaneously. Printed with permission from Jason A. Ellis.

or aneurysmal rupture, high-flow, direct CCFs often present acutely. They typically progress rapidly, necessitating urgent treatment. The most common presenting signs and symptoms include proptosis in 72%–98%,[10,19,42,83] chemosis in 55%–100%,[19,42,83] orbital bruits in 71%–80%,[10,42,83] and headache in 25%–84% of patients.[19,42] Additionally, the majority of patients complain of visual disturbances, including diplopia reported in 88% of patients, blurry vision, and orbital pain.[10,12,19,40,42,83] These visual complaints may be due to retinal ischemia and may indicate the need for urgent intervention. Ophthalmoplegia has been reported in 23%–63% of patients,[42,83] while other cranial nerve deficits have been reported in 17%–44% of patients.[19,42] Less common presentations include intracerebral or subarachnoid hemorrhage in 5% of patients.[19,30]

Indirect CCFs

In contrast to high-flow CCFs, so-called low-flow, indirect CCFs tend to be more insidious in onset. Conjunctival injection is often the most prominent feature, and patients are commonly treated for other conditions, such as conjunctivitis, before a correct diagnosis is made. The disease course may be chronic or relapsing and remitting in nature, leading to a delay diagnosis and treatment of a year or more in more than 50% of patients.[51] In a series of 135 patients with indirect CCFs, common signs and symptoms included arterialization of conjunctival veins in 93%, chemosis in 87%, proptosis in 81%, diplopia with ophthalmoparesis in 68%, cranial bruit in 49%, retroorbital headache in 34%, elevated intraocular pressure in 34%, and decreased visual acuity in 31%.[48]

The precise pattern of symptoms is dependent on the rate of flow, the location of venous drainage of the CCF, inflammation, and pressure within the venous sinus. Additionally, the pattern of venous drainage may change with the development and resolution of a thrombosis, resulting in an inconsistent pattern of symptoms. Anterior draining CCFs, the most common type of indirect CCF, usually present with ocular and orbital symptoms such as chemosis, conjunctival injection, and proptosis. Diplopia may be caused by CN III, IV, or VI palsy, and loss of vision may be due to increased intraocular pressure due to orbital venous congestion with resultant glaucoma, venous retinopathy, and ischemic optic neuropathy.[73] Cavernous-carotid fistulas associated with retrograde cortical venous flow often present with neurological symptoms referable to the area of venous congestion or infarction. This may occur in the supratentorial or infratentorial compartments.

Radiographic Diagnosis

While cerebral angiography is the gold-standard imaging modality in the diagnosis of CCFs, patients typically undergo noninvasive cerebral imaging with CT scanning, MRI, or CT/MR angiography first. Evidence of cavernous sinus enlargement, proptosis, extraocular muscle enlargement, superior ophthalmic vein dilation, or dilation of cortical or leptomeningeal vessels, as well as associated skull fractures, may be seen on CT or MRI and are suggestive of CCF.[1,10] However, the absence of abnormalities on noninvasive imaging studies does not exclude the diagnosis of CCF.

If there is a high degree of clinical suspicion and/or imaging studies are consistent with the presence of CCF, the patient should be referred for diagnostic catheter cerebral angiography. Typically, this will be performed using transfemoral arterial catheterization with imaging of the bilateral CCAs, ICAs, and ECAs, as well as the vertebral arteries.[11,48] Ipsilateral carotid artery compression during vertebral artery injection, known as the Huber maneuver, may be helpful to demonstrate shunting to the distal aspect of the fistula from the posterior circulation vessels. Similarly, ipsilateral common carotid artery compression during ICA injection, known as the Mehringer maneuver, may help visualize very–high flow CCFs by limiting the concurrent amount of unopacified blood shunting through the fistula.

Treatment

The goal of CCF treatment is to completely occlude the fistula while preserving the normal flow of blood through the ICA. Historically, ligation of the CCA was the surgical intervention of choice for the treatment of patients with CCFs. Benjamin Travers[78] is credited with performing the first successful surgical CCA ligation in 1809 for a patient with pulsatile exophthalmos, attributed to an "aneurism by anastomosis." A century later, in 1908, DeSchweinitz and Holloway[13] reported the results of 114 patients treated with CCA ligation for pulsatile exophthalmos and found a 56% success rate and an 11.7% mortality rate. In 1934, Dorrance and Loudenslager[14] reported the results of 151 patients treated with either CCA ligation (82 patients) or cervical ICA ligation (69 patients) and found that 52% of the patients had a successful outcome, 12.6% of the patients developed hemiplegia, and 5.3% died. The high morbidity and mortality rates seen with these procedures clearly necessitated further treatment refinements.

Hamby[21] and Hamby and Gardner[22] were the first to propose the technique of intracranial ICA ligation, followed by insertion of a muscle embolus in the cervical ICA as a way to close the fistula. Brooks is credited as being the first to use intravascular flow-directed embolization alone, without carotid artery ligation, to treat a CCF in 1931.[82] Although Brooks reported a good result, the patient lost vision in the ipsilateral eye. In 1937, Browder[7] was perhaps the first to perform direct surgery on the cavernous sinus, packing it with muscle to treat a cavernous carotid aneurysm. In the 1970s, Fedor Serbinenko paved the way for the modern era of CCF treatment with the development of balloon catheters that could be navigated endovascularly to the fistula site.[75]

Conservative Management

Conservative management, consisting of external manual compression of the ipsilateral cervical carotid artery several times a day for 4–6 weeks, may be effective in the treatment of indirect, low-flow CCFs.[27,33] However, this is ineffective in the treatment of direct, high-flow fistulas. Higashida et al.[27] reported complete CCF occlusion without clinical or angiographic evidence of recurrence at the 1-year follow-up in 30% of patients with indirect CCFs but in only 17% of patients with direct CCFs. Similar results for manual compression in the treatment of indirect CCFs have been reported by other groups.[20,34] Interestingly, it has been reported that 20%–60% of patients with indirect CCFs have spontaneous fistula closure.[10,17,18,62]

When conservative management is used, it is important to have close ophthalmological follow-up, with serial vision tests, intraocular pressure measurements, and funduscopic examinations.[49] Progressive visual decline, papilledema, and refractory elevation of intraocular pressure are all indications for emergency endovascular intervention. Additionally, cortical venous drainage on diagnostic angiography, neurological symptoms, or intractable headache or eye pain are indications of increased risk of intracranial hemorrhage, and should prompt urgent endovascular intervention. Patients presenting with a failure of CCF closure after compression therapy should be considered for alternative treatment strategies.[12,30] It is worth noting that in our experience, conservative management of CCFs has generally been ineffective.

Endovascular Intervention

Transarterial or transvenous embolization is the first-line treatment modality for the treatment of most CCFs (Table 2).[19,61,71,83] Metallic coils and/or liquid embolic agents are now most commonly used for this purpose after the withdrawal of detachable balloons from the US market in 2003.[46,83] Transarterial access is often used when the CCF originates from branches of the ECA, as well as in select cases of direct fistulas (Fig. 3). When the CCF originates from branches of the ICA, transarterial embolization is significantly more difficult and carries an increased risk of stroke due to embolic reflux into the ICA. In these cases, a transvenous approach is used, and the fistula is occluded using either a coil or liquid embolization of the cavernous sinus.[36,37,48,49]

Transvenous access to the cavernous sinus can be achieved using standard transfemoral vein cannulation techniques with navigation through the inferior petrosal sinus or the facial and superior ophthalmic vein in most cases. Even an occluded inferior petrosal sinus can often be catheterized with careful microcatheter and microguidewire manipulation.[48] The superior petrosal sinus, the basilar plexus, and the pterygoid plexus are alternative transvenous access routes if the inferior petrosal sinus or superior ophthalmic vein cannot be accessed. In rare circumstances, direct cannulation of the superior ophthal-

TABLE 2: Endovascular and surgical intervention*

Authors & Year	Pt Population	Signs & Symptoms	Tx	Outcome	Complications
Wang et al., 2011	54 posttraumatic direct CCFs in 51 pts	pulsating exophthalmos (82%), chemosis (78%), retroorbital bruit (71%), ophthalmoplegia (63%), decreased visual acuity (57%), ICH (20%), incidental SAH (10%), spinal fracture (6%)	all pts treated endovascularly via transarterial approach: 40 w/ DBs alone, 8 w/ DBs & stents, 2 w/ stents alone, 2 w/ DBs & coils, 1 w/ coils alone, 1 w/ DBs, stents, & coils	overall, 98% cure w/ ICA preservation, 85% cure w/ DBs alone	22% required 2nd & 3rd endovascular interventions; 18% of pts treated w/ DBs alone developed pseudoaneurysms, 2 of which were symptomatic & 3 progressively enlarging (these 5 were subsequently treated w/ stents); 5 pts had persistent moderate decreased vision or CN deficits; no procedural complications or delayed neurological/vascular complications
Yoshida et al., 2010	44 indirect CCFs	chemosis (100%), ophthalmoparesis (36%), proptosis (14%), decreased visual acuity (11%)	initial Tx w/ transvenous endovascular embol using detachable coils, 4 pts required add'l transarterial embol, 2 pts required add'l radiosurgery	82% cure, 14% minor residual shunt, 4% significant residual shunt	9% recurrence, 7% permanent morbidity (2 brainstem infarcts, 1 ICH), 14% transient CN palsy
Gupta et al., 2006	91 direct CCFs in 89 pts (85 traumatic, 6 ruptured aneurysms)	chemosis (100%), proptosis (98%), diplopia (88%), HA (84%), tinnitus (72%), neurological deficits including CN deficit (44%), visual deficit (26%), seizure (6%), abnormalities of mentation (6%), IC bleed (5%)	79 treated endovascularly w/ transarterial DBs, 12 treated w/ transarterial coil occlusion, as a balloon could not be negotiated through the fistula	of the 79 CCFs treated w/ DBs, 91% cure; of the 12 treated w/ coils, 75% cure; overall at 1 mo, 89% cure, 10% significant improvement, 1% remained static	minimal complications in 3.4%, w/ 1 pt developing multiple CN palsies, w/ partial recovery w/ steroid Tx; 1 pt had asymptomatic ICA occlusion; of pts who did not achieve complete CCF occlusion, 9 had persistent minor HA & diplopia on extreme lateral gaze, & 2 had persistent mild proptosis & diplopia; of the 23 pts w/ visual deficits at presentation, 4 had fixed deficits
Kirsch et al., 2006	141 indirect CCFs	chemosis (94%), increased IOP (60%), exophthalmos (87%), CN palsy (54%), diplopia (51%), decreased vision (28%), bruit (19%), ptosis (13%)	initial Tx w/ transvenous coil placement, 23% required add'l partial arterial embol	81% cure, 13% minor residual shunt, 4% significant residual shunt, 2% Tx failed	14% required >1 transvenous procedure to achieve cure; 11% of pts had residual CN palsy or diplopia
Luo et al., 2006	176 traumatic direct CCFs	chemosis (89%), bruit (89%), decreased visual acuity (78%), proptosis (67%), impairment of CN function (33%), epistaxis (6%)†	initial Tx w/ endovascular transarterial embol w/ a DB or a coil, 22 required add'l transarterial balloon-assisted NBCA embol	80% cure w/ ICA preservation following initial procedure, 82% of pts who underwent transarterial balloon-assisted NBCA embol achieved CCF occlusion w/ ICA preservation on immediate postembol angiography	of the 18 pts w/ successful occlusion following transarterial balloon-assisted NBCA embol, 1 died of SAH on Day 2 postembol; 1 had a recurrent fistula on Day 2 postembol & presented w/ massive epistaxis requiring balloon occlusion of ICA & fistula; 4 had asymptomatic migration of the NBCA mixture distally to the superior ophthalmic vein (n=3) or the inferior petrous sinus (n=1); 5 had asymptomatic small false sac or pseudoaneurysms in the cavernous ICA

(continued)

TABLE 2: Endovascular and surgical intervention* *(continued)*

Authors & Year	Pt Population	Signs & Symptoms	Tx	Outcome	Complications
Meyers et al., 2002	135 indirect CCFs	arterialization of conjunctival veins (93%), chemosis (87%), proptosis (81%), diplopia w/ ophthalmoparesis (68%), cranial bruit (49%), retroorbital HA (34%), elevated IOP (34%), diminished visual acuity (31%)	133 treated endovascularly, 76% via a transvenous approach; embol achieved using a combination of metallic coils (87%), silk suture fragments (13%), or liquid adhesive (3%)	90% cure, 97% good clinical recovery, 1% moderate disability, 2% severe disability	29% of pts required ≥2 endovascular procedures to achieve cure; 2.3% had procedure-related permanent morbidity; 6% of pts had symptomatic complications (1 cerebral infarction, 2 pts w/ decreased visual acuity, 1 pt w/ DI, 1 w/ orbital ecchymosis, 1 w/ retroperitoneal hematoma, 2 w/ deep femoral vein thrombosis w/o PE)
Tu et al., 1997	78 CCFs (66 direct, 12 indirect)	bruit (95%), chemosis (89%), proptosis (79%), exophthalmos (58%), CN VI palsy (47%), ophthalmoplegia (37%), decreased visual acuity (32%), facial hypesthesia (11%)‡	initial Tx w/ endovascular transarterial DBs; 5 pts required subsequent transvenous intervention; 18 pts in whom endovascular Tx failed & 1 who presented w/ acute bleeding underwent direct surgical repair (9 treated w/ sinus packing, 2 treated by seal w/ fascia & glue, 2 w/ IC-IC bypass, 1 w/ clipping, w/ suture, 4 w/ multiple tech niques)	of the 77 pts who had endovascular Tx, 77% cure; of the 19 pts who underwent direct surgical repair, 100% cure, w/ 73% ICA patency; overall, 100% cure w/ 94% ICA patency	of the 19 pts who underwent direct surgical repair, 42% experienced transient CN III palsy & 1 experienced postop CN VI palsy w/ only partial recovery; 1 pt had a wound infection requiring prolonged antibiotic administration
Lewis et al., 1995	100 direct CCFs in 98 pts (76 traumatic, 22 ruptured aneurysms, 2 iatrogenic)	orbital bruit (80%), proptosis (72%), chemosis (55%), CN VI palsy (49%), conjunctival injection (44%)	initial Tx w/ transarterial embol w/ DBs	88% cure, w/ 75% ICA preservation; 4 pts had eventual spontaneous closure following unsuccessful balloon occlusion; 5 pts required direct op to achieve CCF occlusion; 2 pts had CCF closure w/ non-DBs	1 pt died of trauma-related injuries; 4% of pts experienced permanent neurological complications (1 pt w/ cerebral infarction, 1 w/ ICH, 1 w/ vision loss); 1 death related to balloon shift causing cerebral infarction; 3 pts experienced transient ischemia
Higashida et al., 1989	213 posttraumatic CCFs (206 direct, 7 indirect)	pulsating exophthalmos, chemosis, retroorbital bruit, ophthalmoplegia, decreased visual activity	Direct CCFs initially treated endovascularly w/ transarterial DBs; add'l endovascular interventions included use of liquid tissue adhesives, microcoils, & silk sutures; 3% required transvenous approaches. Indirect CCFs treated w/ embol using Ivalon particulate emboli &/or liquid tissue adhesives; half required add'l transvenous intervention	of the 206 direct CCFs, 88% cure w/ ICA preservation; of the 7 indirect CCFs, 100% cure	6 pts experienced transient cerebral ischemia, 5 had pseudoaneurysm formation, 5 experienced strokes, 1 had a peripheral nerve injury after Tx

(continued)

TABLE 2: Endovascular and surgical intervention* ***(continued)***

Authors & Year	Pt Population	Signs & Symptoms	Tx	Outcome	Complications
Debrun et al., 1988	132 CCFs: 100 direct (95 traumatic, 3 ruptured cavernous aneurysms, 2 spontaneous); 32 indirect (all spontaneous: 0 Type B, 4 Type C, 28 Type D)		traumatic: 92 treated endovascularly w/ DBs, 3 treated w/ direct surgical exposure; Type C: all treated endovascularly (2 w/ isobutyl cyanoacrylate & 2 w/ particles of polyvinyl alcohol foam); Type D: pts were initially watched for a minimum of 6 mos w/ subsequent Tx if symptoms persisted or earlier if there was visual deterioration. All endovas cular Tx.	traumatic: 100% cure w/ 70% ICA preservation; Type C: 100% cure; Type D: 3 healed spontaneously, 48% of those treated endovascularly were cured, w/ an add'l 16% cured after surgical exposure	traumatic: 3 pts required reop, 5 have minor residual limitations of motility of the ocular globe, & 3 died (1 of subdural empyema & 2 of severe brain damage associated w/ the initial trauma); Type D: in 2 pts, the ICA feeders increased in size postembol of ECA feeders

* add'l = additional; cure = complete CCF occlusion; DB = detachable balloon; DI = diabetes insipidus; embol = embolization; HA = headache; IC = intracranial; ICH = intracranial hemorrhage; IOP = intraocular pressure; NBCA = *N*-butyl 2-cyanoacrylate; PE = pulmonary embolism; pt = patient; SAH = subarachnoid hemorrhage; Tx = treatment.
† Percentages are based on 18 patients treated with transarterial balloon-assisted NBCA embolization.
‡ Percentages are based on 19 patients who underwent direct surgical repair.

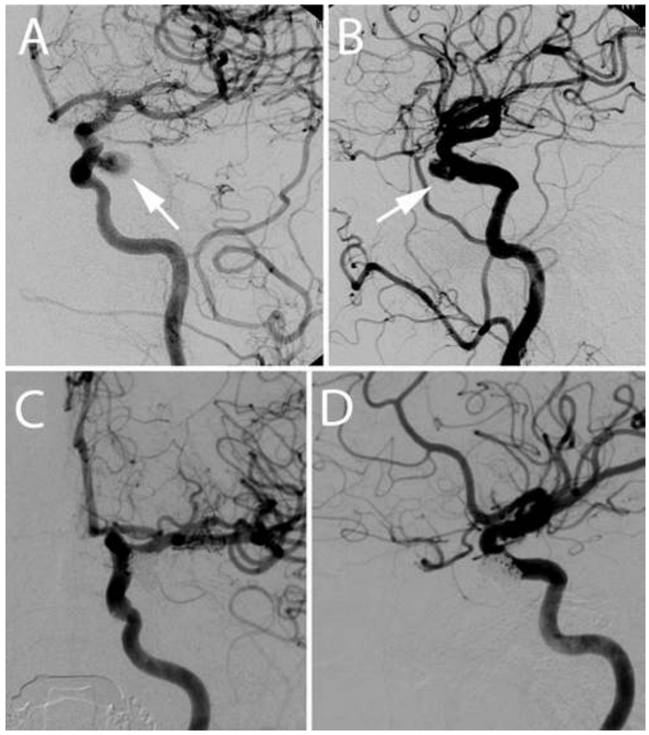

Fig. 3. Transarterial coil embolization of a CCF. Catheter arteriography demonstrating a 2-cm pseudoaneurysm of the left cavernous ICA in anteroposterior **(A)** and lateral **(B)** projections. The resulting high flow fistula was completely cured after the placement of platinum coils within the aneurysm **(C and D)**.

mic vein after surgical exposure may be necessary to gain access to the cavernous sinus.[86]

Endovascular placement of covered or flow-diverting stents is being investigated as an alternative to embolization for the treatment of CCFs. Flow-diverting stents are deployed within the cavernous ICA, over the site of the fistula, redirecting blood flow away from the cavernous sinus.[76,84] While these devices demonstrate dramatic results initially, delayed thrombosis remains a problem with the use of currently available covered stents in the cerebral circulation. The use of flow diverters alone or in combination with other embolic materials has been used anecdotally.

Reported complications of endovascular treatment include cerebral infarction, decreased visual acuity, diabetes insipidus, retroperitoneal hematoma, femoral vein thrombosis, and ophthalmoplegia in 2%–5% of patients.[16,48] Other complications include subarachnoid or intracerebral hemorrhage, sinus rupture, extradural extravasation of contrast, and CN palsies. It is notable that up to 42% of patients will experience a transient worsening of symptoms after cavernous sinus embolization that generally resolves over time.[68] Resolution of preexisting symptoms is related to their duration and severity.

More than 80% of patients who undergo endovascular treatment for direct and indirect CCFs will experience a complete cure.[5,17,42,52,88] This is demonstrated both clinically by reversal of the signs and symptoms of the CCF and angiographically by fistula obliteration with reversal of retrograde cortical venous flow and vascular steal. In a series of more than 200 patients presenting with posttraumatic direct CCFs, Higashida et al.[26] reported complete fistula occlusion with preservation of the ICA in 88% of patients. Similarly, in a series of 135 patients presenting with indirect CCFs, Meyers et al.[48] reported complete fistula occlusion in 90% of patients.

Surgical Intervention

In cases in which endovascular treatment is not possible or is unsuccessful, open surgical intervention may be warranted (Table 2). Surgical intervention may involve suturing, clipping, or trapping the fistula, packing the cavernous sinus to occlude the fistula, sealing the fistula with fascia and glue, ligating the ICA, or a combination of these procedures. Isamat and colleagues[31–33] are proponents of direct intracavernous obliteration by muscle packing combined with transmural injection of fibrin glue, citing good results with less complexity, less time, and a higher probability of preserving the patency of the ICA compared with other surgical procedures.[81]

Overall success rates using surgical intervention in the treatment of CCFs have been reported at between 31% and 79%. Some practitioners have reported 100% obliteration rates in small series.[9,17,66,79] Tu et al.[79] reported a series of 19 patients in whom direct surgical intervention was used after endovascular treatment failed. Closure of the CCF was achieved in all 19 patients, with a 73% ICA patency rate. Day and Fukushima[9] reported similar success using direct surgical intervention after endovascular failure in the treatment of 9 patients with Type D CCFs. In this series, there was a 100% cure rate with no deaths. Transient diplopia and trigeminal hypesthesia were noted in all 9 patients, 1 patient suffered from temporary hemiparesis, and another patient had a permanent hemiparesis.

Radiosurgical Intervention

First performed by Barcia-Salorio and colleagues[2] in 1977, radiosurgical intervention may play a role in the treatment of patients with indirect, low-flow CCFs (Table 3). Barcia-Salorio et al.[3] reported complete or near-complete reversal of the neuroophthalmological symptoms associated with indirect CCFs in 91.6% of patients with spontaneous, low-flow lesions treated with radiosurgery. However, in patients presenting with previously treated direct CCFs, radiosurgery was effective in only 2 of 5 patients. Other small series of patients with indirect CCFs have shown long-term obliteration of the fistula after treatment with radiosurgery in 75%–100% of patients.[3,17,28,53,64] Radiosurgery should not be used in emergency cases, as there is a latency of several months to years before complete obliteration of the CCF is accomplished.[3]

Prognosis

After successful intervention with complete closure of a CCF, symptoms such as chemosis and proptosis generally resolve within hours to days.[36,48] Cranial nerve palsies typically resolve over the course of several weeks.[47,83] The degree of vision recovery, if vision loss was experienced prior to intervention, is largely dependent on the

TABLE 3: Radiosurgical intervention*

Authors & Year	Year	Pts	Signs & Symptoms	Tx	Outcome	Complications
Pollock et al., 1999	1999	20 indirect CCFs	chemosis/proptosis (90%), diplopia (65%), decreased vision (40%), HA/eye pain (30%)	7 pts treated w/ radiosurgery alone; 13 pts treated w/ radiosurgery & transarterial embol	95% symptomatic improvement; of 15 pts who underwent FU angiography, 13 showed complete obliteration & 1 near-total obliteration of the fistula	2 pts developed new neurological deficits after embol, 2 pts required a 2nd endovascular procedure to achieve cure, 1 pt experienced recurrence postradiosurgery & required transvenous embol
Hirai et al., 1998	1998	26 indirect CCFs	diplopia, HA, ophthalmic pain, bruit, exophthalmos, chemosis, CN palsy, visual disturbance	12 pts treated w/ radiation alone; 14 pts treated initially w/ endovascular embol, w/ subsequent radiation in 6 in whom endovascular intervention failed	of the 12 pts treated w/ radiation alone, 75% complete cure & 8% w/ improvement, w/ a mean FU of 62 mos; of the 6 pts treated w/ radiation after endovascular embol, 67% (4/6) complete cure, w/ an additional 1 pt having improvement w/ a mean FU of 24 mos	of the 12 pts treated w/ radiation alone, no complications were seen; of the 14 pts 1st treated w/ endovascular embol, 1 experienced rt hemiparesis lasting 1 yr
Barcia-Salorio et al., 1994	1994	25 CCFs (22 indirect, 3 previously treated direct)		stereotactic radiosurgery	20/22 (91%) low-flow CCFs completely obliterated, 1/3 (33%) previously treated direct CCFs cured	none

* FU = follow-up.

pathogenesis, severity, and duration of the preintervention deficit.[35,43] Recurrence of CCFs due to recanalization postembolization is uncommon but can typically be treated by repeat embolization.

Conclusions

Catheter cerebral angiography is the gold standard imaging modality used in the diagnosis and classification of CCFs. Although historically difficult to treat, these lesions are now routinely managed with low rates of morbidity and mortality. Endovascular intervention with a goal of complete fistula occlusion while preserving normal blood flow through the internal carotid artery has emerged as the treatment of choice. In select cases, open surgery, radiosurgery, or conservative management are also treatment options. Symptom resolution with low rates of recurrence can be expected in most cases after appropriate therapy.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Ellis, Meyers. Acquisition of data: Ellis, Goldstein. Analysis and interpretation of data: all authors. Drafting the article: Ellis, Goldstein. Critically revising the article: Ellis, Connolly, Meyers. Reviewed submitted version of manuscript: Ellis, Connolly, Meyers. Approved the final version of the manuscript on behalf of all authors: Ellis.

References

1. Acierno MD, Trobe JD, Cornblath WT, Gebarski SS: Painful oculomotor palsy caused by posterior-draining dural carotid cavernous fistulas. **Arch Ophthalmol 113:**1045–1049, 1995
2. Barcia-Salorio JL, Broseta J, Hernandez G, Ballester B, Masbout G: Radiosurgical treatment of a carotid-cavernous fistula. Case report, in Szikla G (ed): **Stereotactic Cerebral Irradiations.** Amsterdam: Elsevier, 1979, pp 251–256
3. Barcia-Salorio JL, Soler F, Barcia JA, Hernández G: Stereotactic radiosurgery for the treatment of low-flow carotid-cavernous fistulae: results in a series of 25 cases. **Stereotact Funct Neurosurg 63:**266–270, 1994
4. Barrow DL, Spector RH, Braun IF, Landman JA, Tindall SC, Tindall GT: Classification and treatment of spontaneous carotid-cavernous sinus fistulas. **J Neurosurg 62:**248–256, 1985
5. Barry RC, Wilkinson M, Ahmed RM, Lim CS, Parker GD, McCluskey PJ, et al: Interventional treatment of carotid cavernous fistula. **J Clin Neurosci 18:**1072–1079, 2011
6. Borba LAB, Al-Mefty O: Normal anatomy of the cavernous sinus, in Eisenberg MB, Al-Mefty O (eds): **The Cavernous Sinus: A Comprehensive Text.** Philadelphia: Lippincott Williams & Wilkins, 2000, pp 21–34
7. Browder J: Treatment of carotid-cavernous sinus fistula. Report of a case. **Arch Ophthalmol 18:**95–102, 1937
8. Chuman H, Trobe JD, Petty EM, Schwarze U, Pepin M, Byers PH, et al: Spontaneous direct carotid-cavernous fistula in Ehlers-Danlos syndrome type IV: two case reports and a review of the literature. **J Neuroophthalmol 22:**75–81, 2002
9. Day JD, Fukushima T: Direct microsurgery of dural arteriovenous malformation type carotid-cavernous sinus fistulas: indications, technique, and results. **Neurosurgery 41:**1119–1126, 1997
10. de Keizer R: Carotid-cavernous and orbital arteriovenous fistulas: ocular features, diagnostic and hemodynamic considerations in relation to visual impairment and morbidity. **Orbit 22:**121–142, 2003
11. Debrun GM: Angiographic workup of a carotid cavernous sinus fistula (CCF) or what information does the interventionalist need for treatment? **Surg Neurol 44:**75–79, 1995
12. Debrun GM, Viñuela F, Fox AJ, Davis KR, Ahn HS: Indications for treatment and classification of 132 carotid-cavernous fistulas. **Neurosurgery 22:**285–289, 1988
13. DeSchweinitz G, Holloway T: **Pulsating Exophthalmos: its Etiology, Symptomatology, Pathogenesis and Treatment.** Philadelphia: WB Saunders, 1908
14. Dorrance GM, Loudenslager PE: Physiological considerations in treatment of pulsating exophthalmos. **Am J Ophthalmol 17:**1099–1111, 1934
15. Farley MK, Clark RD, Fallor MK, Geggel HS, Heckenlively JR: Spontaneous carotid-cavernous fistula and the Ehlers-Danlos syndromes. **Ophthalmology 90:**1337–1342, 1983
16. Fifi JT, Meyers PM, Lavine SD, Cox V, Silverberg L, Mangla S, et al: Complications of modern diagnostic cerebral angiography in an academic medical center. **J Vasc Interv Radiol 20:**442–447, 2009
17. Gemmete JJ, Chaudhary N, Pandey A, Ansari S: Treatment of carotid cavernous fistulas. **Curr Treat Options Neurol 12:** 43–53, 2010
18. Gobin Y, Duckwiler G, Gugliemi G: Endovascular techniques in the treatment of carotid cavernous fistulas, in Eisenberg MB, Al-Mefty O (eds): **The Cavernous Sinus: a Comprehensive Text.** Philadelphia: Lippincott Williams & Wilkins, 2000, pp 209–225
19. Gupta AK, Purkayastha S, Krishnamoorthy T, Bodhey NK, Kapilamoorthy TR, Kesavadas C, et al: Endovascular treatment of direct carotid cavernous fistulae: a pictorial review. **Neuroradiology 48:**831–839, 2006
20. Halbach VV, Higashida RT, Hieshima GB, Reicher M, Norman D, Newton TH: Dural fistulas involving the cavernous sinus: results of treatment in 30 patients. **Radiology 163:**437–442, 1987
21. Hamby WB: Carotid-cavernous fistula. Report of 32 surgically treated cases and suggestions for definitive operation. **J Neurosurg 21:**859–866, 1964
22. Hamby WB, Gardner WJ: Treatment of pulsating exophthalmos with report of two cases. **Arch Surg 27:**676–685, 1933
23. Harris FS, Rhoton AL: Anatomy of the cavernous sinus. A microsurgical study. **J Neurosurg 45:**169–180, 1976
24. Hashimoto M, Yokota A, Yamada H, Okudera T: Development of the cavernous sinus in the fetal period: a morphological study. **Neurol Med Chir (Tokyo) 40:**140–150, 2000
25. Helmke K, Krüger O, Laas R: The direct carotid cavernous fistula: a clinical, pathoanatomical, and physical study. **Acta Neurochir (Wien) 127:**1–5, 1994
26. Higashida RT, Halbach VV, Tsai FY, Norman D, Pribram HF, Mehringer CM, et al: Interventional neurovascular treatment of traumatic carotid and vertebral artery lesions: results in 234 cases. **AJR Am J Roentgenol 153:**577–582, 1989
27. Higashida RT, Hieshima GB, Halbach VV, Bentson JR, Goto K: Closure of carotid cavernous sinus fistulae by external compression of the carotid artery and jugular vein. **Acta Radiol Suppl 369:**580–583, 1986
28. Hirai T, Korogi Y, Baba Y, Nishimura R, Hamatake S, Kawanaka K, et al: Dural carotid cavernous fistulas: role of conventional radiation therapy—long-term results with irradiation, embolization, or both. **Radiology 207:**423–430, 1998
29. Hirai T, Korogi Y, Goto K, Ogata N, Sakamoto Y, Takahashi M: Carotid-cavernous sinus fistula and aneurysmal rupture associated with fibromuscular dysplasia. A case report. **Acta Radiol 37:**49–51, 1996
30. Huai RC, Yi CL, Ru LB, Chen GH, Guo HH, Luo L: Trau-

matic carotid cavernous fistula concomitant with pseudoaneurysm in the sphenoid sinus. **Interv Neuroradiol 14:**59–68, 2008
31. Isamat F: On posttraumatic intracavernous false aneurysms and arteriovenous fistulas. **Acta Neurochir (Wien) 85:**148–153, 1987
32. Isamat F, Ferrer E, Twose J: Direct intracavernous obliteration of high-flow carotid-cavernous fistulas. **J Neurosurg 65:** 770–775, 1986
33. Isamat F, Twose J, Conesa G: Surgical management of cavernous-carotid fistulas, in Eisenberg MB, Al-Mefty O (eds): **The Cavernous Sinus: a Comprehensive Text.** Philadelphia: Lippincott Williams & Wilkins, 2000, pp 201–208
34. Kai Y, Hamada J, Morioka M, Yano S, Kuratsu J: Treatment of cavernous sinus dural arteriovenous fistulae by external manual carotid compression. **Neurosurgery 60:**253–258, 2007
35. Kim MS, Han DH, Kwon OK, Oh CW, Han MH: Clinical characteristics of dural arteriovenous fistula. **J Clin Neurosci 9:**147–155, 2002
36. Kirsch M, Henkes H, Liebig T, Weber W, Esser J, Golik S, et al: Endovascular management of dural carotid-cavernous sinus fistulas in 141 patients. **Neuroradiology 48:**486–490, 2006
37. Klisch J, Huppertz HJ, Spetzger U, Hetzel A, Seeger W, Schumacher M: Transvenous treatment of carotid cavernous and dural arteriovenous fistulae: results for 31 patients and review of the literature. **Neurosurgery 53:**836–857, 2003
38. Kobayashi N, Miyachi S, Negoro M, Suzuki O, Hattori K, Kojima T, et al: Endovascular treatment strategy for direct carotid-cavernous fistulas resulting from rupture of intracavernous carotid aneurysms. **AJNR Am J Neuroradiol 24:**1789–1796, 2003
39. Kupersmith MJ, Berenstein A, Choi IS, Warren F, Flamm E: Management of nontraumatic vascular shunts involving the cavernous sinus. **Ophthalmology 95:**121–130, 1988
40. Kupersmith MJ, Berenstein A, Flamm E, Ransohoff J: Neuroophthalmologic abnormalities and intravascular therapy of traumatic carotid cavernous fistulas. **Ophthalmology 93:** 906–912, 1986
41. Kupersmith MJ, Stiebel-Kalish H, Huna-Baron R, Setton A, Niimi Y, Langer D, et al: Cavernous carotid aneurysms rarely cause subarachnoid hemorrhage or major neurologic morbidity. **J Stroke Cerebrovasc Dis 11:**9–14, 2002
42. Lewis AI, Tomsick TA, Tew JM Jr: Management of 100 consecutive direct carotid-cavernous fistulas: results of treatment with detachable balloons. **Neurosurgery 36:**239–245, 1995
43. Liang W, Xiaofeng Y, Weiguo L, Wusi Q, Gang S, Xuesheng Z: Traumatic carotid cavernous fistula accompanying basilar skull fracture: a study on the incidence of traumatic carotid cavernous fistula in the patients with basilar skull fracture and the prognostic analysis about traumatic carotid cavernous fistula. **J Trauma 63:**1014–1020, 2007
44. Lin TK, Chang CN, Wai YY: Spontaneous intracerebral hematoma from occult carotid-cavernous fistula during pregnancy and puerperium. Case report. **J Neurosurg 76:**714–717, 1992
45. Linskey ME, Sekhar LN, Hirsch W Jr, Yonas H, Horton JA: Aneurysms of the intracavernous carotid artery: clinical presentation, radiographic features, and pathogenesis. **Neurosurgery 26:**71–79, 1990
46. Luo CB, Teng MM, Chang FC, Chang CY: Transarterial balloon-assisted n-butyl-2-cyanoacrylate embolization of direct carotid cavernous fistulas. **AJNR Am J Neuroradiol 27:** 1535–1540, 2006
47. Marques MC, Caldas JG, Nalli DR, Fonseca JR, Nogueira RG, Abdala N: Follow-up of endovascular treatment of direct carotid-cavernous fistulas. **Neuroradiology 52:**1127–1133, 2010
48. Meyers PM, Halbach VV, Dowd CF, Lempert TE, Malek AM, Phatouros CC, et al: Dural carotid cavernous fistula: definitive endovascular management and long-term follow-up. **Am J Ophthalmol 134:**85–92, 2002
49. Miller NR: Diagnosis and management of dural carotid-cavernous sinus fistulas. **Neurosurg Focus 23(5):**E13, 2007
50. Motarjeme A, Keifer JW: Carotid-cavernous sinus fistula as a complication of carotid endarterectomy. A case report. **Radiology 108:**83–84, 1973
51. Newton TH, Hoyt FW: Dural arteriovenous shunts in the region of the cavernous sinus. **Neuroradiology 1:**71–81, 1970
52. Ng PP, Higashida RT, Cullen S, Malek R, Halbach VV, Dowd CF: Endovascular strategies for carotid cavernous and intracerebral dural arteriovenous fistulas. **Neurosurg Focus 15(4):**ECP1, 2003
53. Onizuka M, Mori K, Takahashi N, Kawahara I, Hiu T, Toda K, et al: Gamma knife surgery for the treatment of spontaneous dural carotid-cavernous fistulas. **Neurol Med Chir (Tokyo) 43:**477–483, 2003
54. Parkinson D: Carotid cavernous fistula: direct repair with preservation of the carotid artery. Technical note. **J Neurosurg 38:**99–106, 1973
55. Parkinson D: Lateral sellar compartment: history and anatomy. **J Craniofac Surg 6:**55–68, 1995
56. Parkinson D: Lateral sellar compartment O.T. (cavernous sinus): history, anatomy, terminology. **Anat Rec 251:**486–490, 1998
57. Parkinson D: A surgical approach to the cavernous portion of the carotid artery. Anatomical studies and case report. **J Neurosurg 23:**474–483, 1965
58. Parkinson D: Transcavernous repair of carotid cavernous fistula. Case report. **J Neurosurg 26:**420–424, 1967
59. Pedersen RA, Troost BT, Schramm VL: Carotid-cavernous sinus fistula after external ethmoid-sphenoid surgery. Clinical course and management. **Arch Otolaryngol 107:**307–309, 1981
60. Peeters FL, Kröger R: Dural and direct cavernous sinus fistulas. **AJR Am J Roentgenol 132:**599–606, 1979
61. Phatouros CC, Meyers PM, Dowd CF, Halbach VV, Malek AM, Higashida RT: Carotid artery cavernous fistulas. **Neurosurg Clin N Am 11:**67–84, viii, 2000
62. Phelps CD, Thompson HS, Ossoinig KC: The diagnosis and prognosis of atypical carotid-cavernous fistula (red-eyed shunt syndrome). **Am J Ophthalmol 93:**423–436, 1982
63. Pigott TJ, Holland IM, Punt JA: Carotico-cavernous fistula after trans-sphenoidal hypophysectomy. **Br J Neurosurg 3:** 613–616, 1989
64. Pollock BE, Nichols DA, Garrity JA, Gorman DA, Stafford SL: Stereotactic radiosurgery and particulate embolization for cavernous sinus dural arteriovenous fistulae. **Neurosurgery 45:** 459–467, 1999
65. Raskind R, Johnson N, Hance D: Carotid cavernous fistula in pregnancy. **Angiology 28:**671–676, 1977
66. Ringer AJ, Salud L, Tomsick TA: Carotid cavernous fistulas: anatomy, classification, and treatment. **Neurosurg Clin N Am 16:**279–295, viii, 2005
67. Rios-Montenegro EN, Behrens MM, Hoyt WF: Pseudoxanthoma elasticum. Association with bilateral carotid rete mirabile and unilateral carotid-cavernous sinus fistula. **Arch Neurol 26:**151–155, 1972
68. Roy D, Raymond J: The role of transvenous embolization in the treatment of intracranial dural arteriovenous fistulas. **Neurosurgery 40:**1133–1144, 1997
69. Schievink WI, Piepgras DG, Earnest F IV, Gordon H: Spontaneous carotid-cavernous fistulae in Ehlers-Danlos syndrome Type IV. Case report. **J Neurosurg 74:**991–998, 1991
70. Sekhar LN, Heros RC, Kerber CW: Carotid-cavernous fistula following percutaneous retrogasserian procedures. Report of two cases. **J Neurosurg 51:**700–706, 1979
71. Siniluoto T, Seppänen S, Kuurne T, Wikholm G, Leinonen S, Svendsen P: Transarterial embolization of a direct carotid cavernous fistula with Guglielmi detachable coils. **AJNR Am J Neuroradiol 18:**519–523, 1997
72. Slusher MM, Lennington BR, Weaver RG, Davis CH Jr: Oph-

thalmic findings in dural arteriovenous shunts. **Ophthalmology 86:**720–731, 1979
73. Stiebel-Kalish H, Setton A, Nimii Y, Kalish Y, Hartman J, Huna Bar-On R, et al: Cavernous sinus dural arteriovenous malformations: patterns of venous drainage are related to clinical signs and symptoms. **Ophthalmology 109:**1685–1691, 2002
74. Takahashi M, Killeffer F, Wilson G: Iatrogenic carotid cavernous fistula. Case report. **J Neurosurg 30:**498–500, 1969
75. Teitelbaum GP, Larsen DW, Zelman V, Lysachev AG, Likhterman LB: A tribute to Dr. Fedor A. Serbinenko, founder of endovascular neurosurgery. **Neurosurgery 46:**462–470, 2000
76. Tiewei Q, Ali A, Shaolei G, Feng L, Zhongsong S, Xuesong L, et al: Carotid cavernous fistulas treated by endovascular covered stent grafts with follow-up results. **Br J Neurosurg 24:**435–440, 2010
77. Toya S, Shiobara R, Izumi J, Shinomiya Y, Shiga H, Kimura C: Spontaneous carotid-cavernous fistula during pregnancy or in the postpartum stage. Report of two cases. **J Neurosurg 54:**252–256, 1981
78. Travers B: A case of aneurism by anastomosis in the orbit, cured by the ligature of the common carotid artery. **Med Chir Trans 2:**1–16, 1811
79. Tu YK, Liu HM, Hu SC: Direct surgery of carotid cavernous fistulae and dural arteriovenous malformations of the cavernous sinus. **Neurosurgery 41:**798–806, 1997
80. van Rooij WJ, Sluzewski M, Beute GN: Ruptured cavernous sinus aneurysms causing carotid cavernous fistula: incidence, clinical presentation, treatment, and outcome. **AJNR Am J Neuroradiol 27:**185–189, 2006
81. Viñuela F, Fox AJ, Debrun GM, Peerless SJ, Drake CG: Spontaneous carotid-cavernous fistulas: clinical, radiological, and therapeutic considerations. Experience with 20 cases. **J Neurosurg 60:**976–984, 1984
82. Vitek JJ, Smith MJ: The myth of the Brooks method of embolization: a brief history of the endovascular treatment of carotid-cavernous sinus fistula. **J Neurointerv Surg 1:**108–111, 2009
83. Wang W, Li YD, Li MH, Tan HQ, Gu BX, Wang J, et al: Endovascular treatment of post-traumatic direct carotid-cavernous fistulas: A single-center experience. **J Clin Neurosci 18:**24–28, 2011
84. Weber W, Henkes H, Berg-Dammer E, Esser J, Kühne D: Cure of a direct carotid cavernous fistula by endovascular stent deployment. **Cerebrovasc Dis 12:**272–275, 2001
85. Winslow J: **Exposition Anatomique de la Structure du Corps Humain.** London: Prevast, Vol II, 1734
86. Wolfe SQ, Cumberbatch NM, Aziz-Sultan MA, Tummala R, Morcos JJ: Operative approach via the superior ophthalmic vein for the endovascular treatment of carotid cavernous fistulas that fail traditional endovascular access. **Neurosurgery 66 (6 Suppl Operative):**293–299, 2010
87. Yoo K, Krisht AF: Etiology and classification of cavernous-carotid fistulas, Eisenberg MB, Al-Mefty O (eds): **The Cavernous Sinus: a Comprehensive Text.** Philadelphia: Lippincott Williams & Wilkins, 2000, pp 191–200
88. Yoshida K, Melake M, Oishi H, Yamamoto M, Arai H: Transvenous embolization of dural carotid cavernous fistulas: a series of 44 consecutive patients. **AJNR Am J Neuroradiol 31:**651–655, 2010

Manuscript submitted January 15, 2012.
Accepted February 20, 2012.
Please include this information when citing this paper: DOI: 10.3171/2012.2.FOCUS1223.
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