Endovascular Neurosurgery

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Abstract: There are two pathways for the surgical management of neurovascular diseases: one is microvascular neurosurgery and the other is endovascular neurosurgery. Nearly three decades have passed since the introduction of endovascular neurosurgery, and now it has taken its shape and definitive role in the management of neurovascular diseases such as intracranial aneurysms, arteriovenous fistulas of the brain and spinal cord, arterial stenosis, and intracranial and spinal AVMs. It can be used for curative or palliative purposes. It can also be used in combination with microsurgery and radiosurgery. This chapter will discuss the endovascular management of brain aneurysms, carotid and vertebral system arterial stenosis, intracranial and spinal AVMs, and dural AV fistulas (head and spine).

Abbreviations

ADC	afferent diffusion coefficient	ASA	anterior spinal artery
AVF	arteriovenous fistula	AVM	arteriovenous malformation
BAC	balloon-assisted coiling	BMT	best medical therapy
CARAT	cerebral aneurysm re-rupture after treatment	CAS	carotid artery stenting
CCA	common carotid artery	CCF	caroticocavernous fistula
CEA	carotid endarterectomy	CPA	cerebello-pontine angle
CT	computed tomography	CTA	computed tomographic arteriogram
CVR	cortical venous reflux	DAVF	dural arteriovenous fistula
DCCF	direct CCF	DSA	digital subtraction angiography
DW	diffusion-weighted	ECA	external carotid artery
EVT	endovascular therapy	FD	flow diverter
fMRI	functional magnetic resonance imaging	GRAS	gradient echo sequences
IA	intracranial aneurysm	ICA	internal carotid artery
ICCF	indirect CCF	ICG	indocyanine green
ICH	intracranial hematoma	ICP	intracranial pressure
MMA	middle meningeal artery	MRA	magnetic resonance angiography
MRI	magnetic resonance imaging	*n*-BCA	*n*-butyl 2-cyanoacrylate
PSA	posterior spinal artery	PVA	polyvinyl alcohol
PW	perfusion-weighted	SAC	stent-assisted coiling
SAH	subarachnoid hemorrhage	SDH	subdural hematoma
SVM	spinal vascular malformation	TIA	transient ischemic attack
TOF	time of flight	VA	vertebral artery

1. History of Endovascular Neurosurgery

Morgagni of Padua recorded dilatation of the posterior division of right and left carotid arteries in 1761. An aneurysm with a rupture was first noticed by Biumi of Milan in 1765. Blackall reported the case report of a subarachnoid hemorrhage (SAH) linked to a cerebral aneurysm in 1814. Earlier than the invention of angiography, only few aneurysms could be identified before an SAH. Sometimes, they may present as a space-occupying lesion that might be noticed on a pneumoencephalogram. Keen published Victor Horsley's surgery on a case with a big pulsatile blood cyst in 1890. Harvey Cushing discovered a brain aneurysm in managing a lesion that he believed to be a pituitary tumor. In his assertation of the pituitary body and its disorders, he compiled the case of a patient with a bitemporal visual deficit, hypopituitarism and a possible interpeduncular space aneurysm. In 1917, Cushing ligated an internal carotid artery (ICA) intracranially following an aneurysm rupture during operation, and the patient died. In 1926, Cushing wrapped an ICA aneurysm with muscle where hemiplegia had developed; the patient died later. However, post-mortem, the aneurysm was found to be thrombosed. The clinical diagnosis was an intracranial cyst (Smith et al. 1994).

Norman Dott conducted the first direct surgery on a cerebral aneurysm in 1933, wrapping a burst aneurysm, while Walter Dandy executed the first clipping of an aneurysm in 1938. When microsurgery was established in the 1960s, the surgical outcomes improved considerably (Maurice-Williams and Lafuente 2003).

Injecting iodinated contrast media into the carotid artery (direct puncture) and then utilizing Roentgen rays was the first experience with a cerebral angiogram, which was invented by Portuguese physician Egas Moniz at the University of Lisbon in 1927 (Lowis and Minagar 2003).

Per Amudsen, a Norwegian radiologist, was the first to conduct total cerebral angiography using a transfemoral technique in 1964. In 1964, Charles Dotter, the pioneer of angioplasty and interventional radiology, was the first to perform an endovascular operation, conducting a therapeutic angioplasty of the femoral artery in a female of 82 years with an ischemic lower limb who rejected amputation (Payne 2001).

Fedor Serbinenko devised a technique for the treatment of aneurysms in the ICA by occluding the light with balloons deployed into the vessel. The first treatment was performed in Moscow in 1970, when an internal carotid artery was occluded to manage a carotid–cavernous fistula (CCF). As such, he can be called the first interventional neuroradiologist, interventionist and endovascular neurosurgeon (Teitelbaum et al. 2000). Neuroradiologists all across the world perfected this technique, with Jacques Moret in Paris, Grant Hieshima in San Francisco and Gerard Debrun in Canada being the first and most outstanding. Image technology in radiology and neuroradiology units improved dramatically in tandem with the development of catheters. The technique of digital subtraction angiography (DSA) was pioneered by Charles Mistretta in 1979 (Celesia et al. 1983).

The work of two Italian physicians, Guido Guglielmi and Cesare Gianturco, revolutionized endovascular surgery by the end of the 1980s and the beginning of the 1990s. The first one had a strong understanding of diagnostic radiology, as well as a strong capacity to tackle technical and manual issues. He devised Gianturco's coils, which he utilized to embolize arteries, including aneurysms, for the first time. Gianturco also designed the first endovascular stent recognized by the American Food and Drug Administration, a device that has a long history. Hilal was the first at Columbia University to utilize coils to manage cerebral aneurysms in the late 1980s, but his technique was ineffective and risky since the coils were deployed with minimal control, putting the parent vessel at the risk of an occlusion. Guido Guglielmi's work at UCLA redefined coil embolization when he learned that electricity could be used to control the release of coils; in 1991, he described the embolization of cerebral aneurysms using detachable platinum coils (Guglielmi's coils). Aneurysm therapy has thus become more affordable and safe (Vaidya et al. 2008).

For decades, clipping a burst aneurysm was thought to be the only option; however, the invention of the GDC coil in 1990 provided an option that avoided the open surgery (Maurice-Williams and Lafuente 2003).

2. SAH and Intracranial Aneurysms

An intracranial aneurysm (IA) rupture is a dangerous and often fatal clinical emergency that necessitates prompt surgery. Approximately 12% of patients die before entering the hospital, 33% within 48 h and 50% within 30 days of the rupture, with 50% of survivors suffering from chronic disability and reliance (Taheri et al. 2015; Sehba et al. 2012). Therefore, it is advised that patients with a burst intracranial aneurysm should receive surgical treatment before dawn or sunset, whichever occurs first.

The approximate incidence of unruptured IAs is nearly 3.2% (Pierot and Wakhloo 2013; Vlak et al. 2011). Most IAs are usually symptomless until rupture, leading to an SAH. The mortality of IA rupture is very high (from 27% to 44%) (Nieuwkamp et al. 2009). Even though the management of a ruptured IA is direly an emergency, indication for the management of incidental IAs is still controversial. Management of the patients with IAs depends on aneurysm shape (saccular versus fusiform) and location (geometry), size (large/small/giant), neck size (large/small), location (posterior versus anterior circulation) and other factors. The heterogeneity suggests that EVTs must use diverse ways to treat all types of IAs. Various therapy strategies for an EVT of IAs have been developed over the previous three decades (Pierot et al. 2012a; Pierot and Wakhloo 2013).

2.1. Endovascular Coiling

The evolution of coils with a controlled deployable system (Figures 1–3) was definitely the first key step for the generalized utilization of endovascular therapy (EVT) (Guglielmi et al. 1991a, part 1; Guglielmi et al. 1991b, part 2).

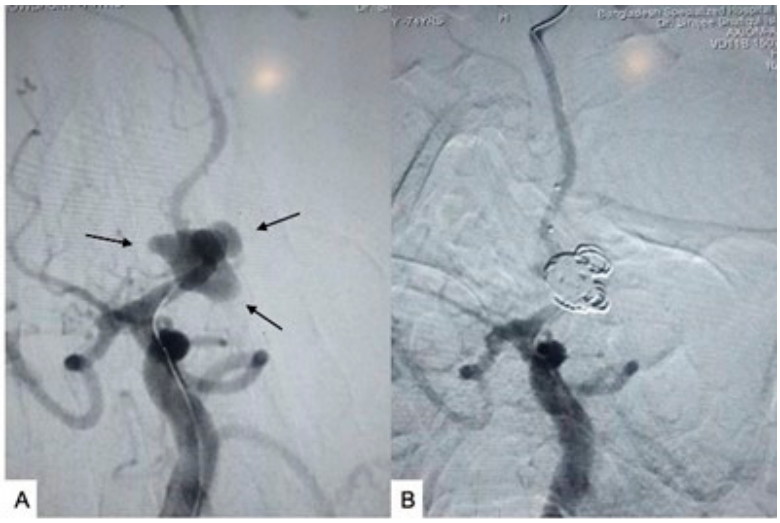

Figure 1. (**A**) DSA showing the pre-embolization state of an ACOM aneurysm; (**B**) DSA after coil embolization of an ACOM aneurysm. Source: Figure by authors.

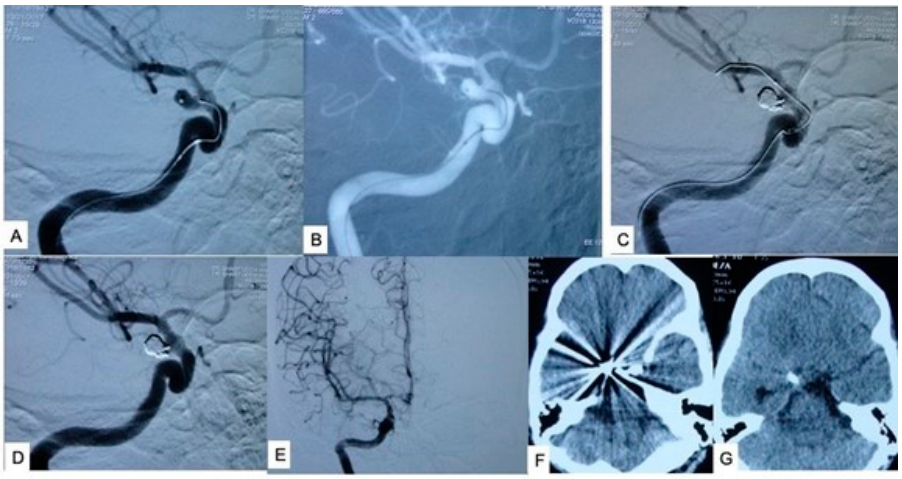

Figure 2. Preoperative pictures of a coil of a right PCOM aneurysm. (**A**) Microcatheter at the neck of the aneurysm; (**B**) road map; (**C**,**D**) coils inside the aneurysm; (**E**) post-coiling DSA; (**F**,**G**) postoperative CT scan after coiling. Source: Figure by authors.

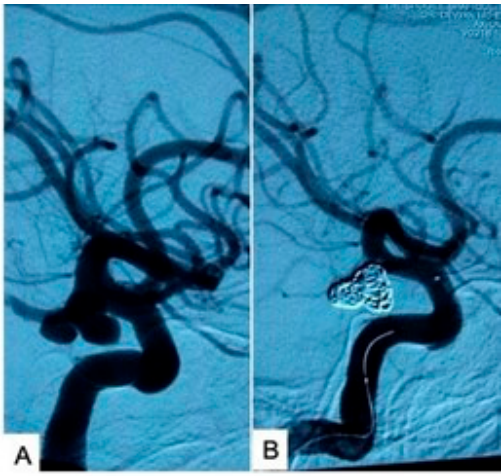

Figure 3. Bilobed PCOM aneurysm. (**A**) Before coiling; (**B**) after coiling. Source: Figure by authors.

The two most common hazards of coiling are thromboembolic hazards and intraprocedural rupture (Pierot et al. 2008a; Cognard et al. 2011). The incidence of thromboembolic complications and intraoperative ruptures linked

with coiling was found to be 7.3% and 2.0% in unruptured aneurysms, respectively. An intraoperative rupture was associated with a greater death rate (16.7%) than thromboembolic complications (4.1%). An intraprocedural ruptured aneurysm and thromboembolism were more common in ruptured aneurysms, with 13.3% and 3.7%, respectively (Cognard et al. 2011). Thus, the perioperative use of intravenous heparin and aspirin for incidental, and in few cases, for ruptured aneurysms as well, is adopted (Ries et al. 2006). The two main hurdles for coiling are as follows (Pierot and Wakhloo 2013):

(1) Difficult coiling due to their shape (giant and large aneurysms, fusiform types of aneurysms, wide neck aneurysms, aneurysms with an unaffordable size, relationship between aneurysm neck, dome and parent artery)—This resulted in the introduction of new technologies and techniques, incorporating balloon-assisted aneurysm coiling, stent abetting coiling and flow diversion (FD)/flow disruption.

(2) The decreased durability of coiling (Cognard et al. 1999; Raymond et al. 2003)— Reopening of an aneurysm occurs in 20.8% of cases, necessitating retreatment in 10.3%, according to a systematic study (Ferns et al. 2009). Recent burst, hypertension, smoking, neck size and aneurysm diameter, as well as the quality of early postoperative aneurysm occlusion are all related to a greater risk of recanalization and recurrence (coil packing density) (Hope et al. 1999; Vallée et al. 2004; Ortiz et al. 2008; Choi et al. 2010; Songsaeng et al. 2011; Gallas et al. 2005; Wakhloo et al. 2007; Willinsky et al. 2009; Pierot et al. 2012c). Surface-modified coils, such as polyglycolic–lactic acid coils, as well as hydrocoils, were created to address the recanalization rate, but they are no more effective than bare platinum coils (Pierot et al. 2008b; White et al. 2011). Aneurysm recanalization's clinical importance isn't well understood. The Cerebral Aneurysm Re-rupture After Treatment (CARAT) study found that the extent of aneurysm occlusion following initial therapy was a robust predictor of the probability of future rupture in SAH cases (Johnston et al. 2008). Because aneurysm recanalization is a probability, a DSA and MRA follow-up is required (Johnston et al. 2008; Pierot et al. 2006, 2012d, 2012f).

2.2. Balloon-Assisted Coiling

Moret et al. were the first to introduce balloon-assisted coiling (BAC, remodeling approach) for expanding EVT to broad-neck IAs (Moret et al. 1997). During each coil installation, a nondetachable balloon is momentarily inflated anterior to the aneurysm's neck (Figure 4) (Pierot et al. 2012b). The balloon is basically kept in the main anterior to the aneurysm neck in sidewall aneurysms. The procedure for bifurcation aneurysms is more difficult. There are several options available, including using two balloons, a round-shaped balloon, a hyper-compliant balloon or a double-lumen balloon. The balloon is deflated and withdrawn at the end of the intervention, and so no device is kept in situ unless a stenting is to be kept in place later.

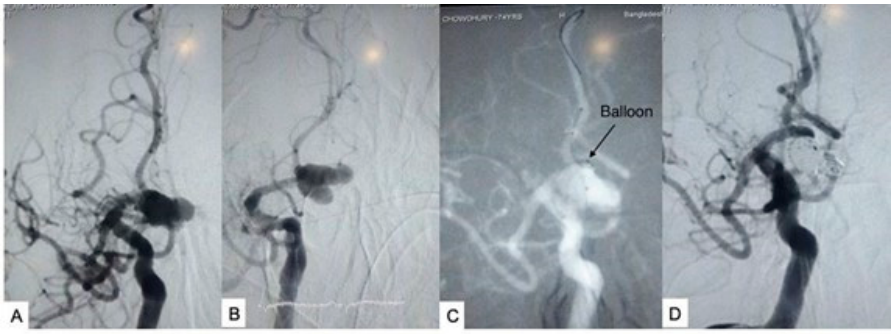

Figure 4. Balloon-assisted coiling of an ACOM aneurysm. (**A**,**B**) DAS before coiling showing a complex ACOM aneurysm and ACOM complex; (**C**) road map after the placement of a balloon, (**D**) after coil embolization. Source: Figure by authors.

When compared to standard coiling, BAC has more procedural complications (Sluzewski et al. 2006). This group had greater rates of thromboembolic events, including intraoperative rupture, 9.8% and 4.0%, respectively, than the coiling alone subgroup, which had 2.2% and 0.8%, respectively (Pierot et al. 2009; Pierot et al. 2011). Treatment morbidity in the coiling group was 3.9% compared to 2.5% in the BAC group, while treatment mortality was 1.2% in the coiling group and 1.3% in the BAC group. The impact of BAC on anatomic outcomes is still unknown (Pierot and Wakhloo 2013).

BAC has a higher incidence of incomplete aneurysm occlusion (27.7%) than normal coiling (16.9%); retreatment is also more common in BAC (16.9% versus 9.0% for standard coiling) (Sluzewski et al. 2006). In one series, 73% of cases in the BAC group and 49% of those managed with coiling alone had complete blockage (Pierot and Wakhloo 2013; Shapiro et al. 2008). Balloon-assisted coil embolization was originally designed to treat wide-neck aneurysms; however, it can also be employed in cases of an intraoperative burst, where the balloon aid may be linked to a higher likelihood of a similar or better clinical result than traditional coiling (Santillan et al. 2012). A balloon aid should be utilized not only to facilitate coiling, but also as a sentinel in the event of a preoperative rupture. The balloon remains deflated over the aneurysm's neck; it is solely inflated in the event of an intraprocedural rupture (Pierot and Wakhloo 2013).

2.3. Stent-Assisted Coiling

Some difficult aneurysms, such as broad-neck aneurysms, massive and giant aneurysms, as well as fusiform aneurysms, are managed by stent-assisted coiling (SAC) (Figure 5) (Wakhloo et al. 1998, 2008; Mericle et al. 1998; Lanzino et al. 1998).

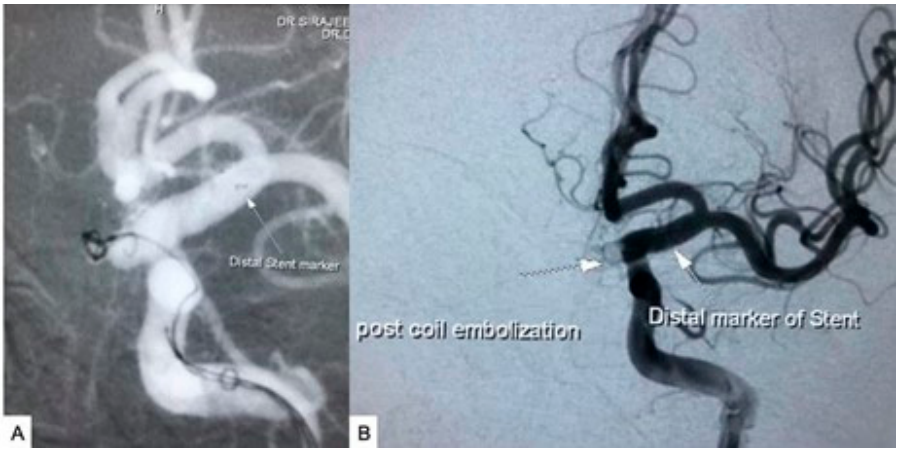

Figure 5. Stent-assisted coiling of an ICA aneurysm. (**A**) Stent-assisted placement of the coil in an aneurysm; (**B**) after coil embolization. Source: Figure by authors.

In the beginning, due to the unavailability of stents definably devised for the endovascular therapy, tough coronary stents were utilized. Eventually, numerous stents devoted to the management of IAs became accessible, making treatment much more effective. Another intriguing advance is the creation of low-profile stents, which allow for BAC and SAC to be combined (Kadziolka et al. 2013).

SACs were also employed as a last resort in the event of coil herniation or coil migration into the main vascular lumen. As stents are used to bridge the aneurysm neck in the main artery, the chance of a stent thrombosis is more than with coiling alone. Antiplatelet therapy is therefore required both before and after surgery. SACs were first limited to aneurysms that had not ruptured. Despite this, stenting has been performed in burst aneurysms according to experience (Pierot and Wakhloo 2013).

An option for preventing aneurysm recanalization was stenting. This, in turn, contributed to an increase in the usage of SACs. The safety and effectiveness of SACs in comparison to normal coiling remain unknown. SACs were linked to a greater prevalence of irreversible neurological problems (7.4%) when compared to normal coiling (3.8%) in a study ($p = 0.644$). The procedure-related death rate was 4.6% in the stenting group versus 1.2% in the non-stenting group ($p = 0.006$). In 50% of the patients, follow-up was available, and angiographic reappearance was substantially higher in the non-stenting group (33.5%) than in the stenting group (14.9%; $p = 0.0001$) (Piotin et al. 2010).

According to a review by Shapiro et al. (2012), the total complication rate was 19%, with a fatality rate of 2.1%. In 10% and 2.2% of cases, respectively, thromboembolic and hemorrhagic consequences were found. In 9% of the instances, there were technical problems related to stenting. Of the aneurysms, 45% were quickly and totally occluded after stenting. The occlusion rate climbed to 61% on follow-up angiograms, with stent stenosis or stent occlusion occurring in 3.5% and 0.6% of cases, respectively. In spite of the increasing risk of thromboembolic events and hemorrhage, the literature analysis found that stents could be utilized in conjunction with coiling

to enhance the speed of a full occlusion in a subset of more complicated aneurysms (Pierot and Wakhloo 2013). Complications associated with SACs are pre-procedural complications, death, recanalization, in-stent stenosis, stent migration and a delayed infarction (Pierot and Wakhloo 2013; Lee et al. 2013).

SACs are commonly used to treat ruptured aneurysms (Wakhloo et al. 2012). A literature review on SACs for ruptured IAs found that they had a greater technical success rate (93%) but also had a higher incidence of clinically significant cerebral bleeding (up to 11%) and thrombosis (6%). A total of 14% of patients had bad outcomes, with up to 19% of them dying (Pierot and Wakhloo 2013; Bodily et al. 2011).

SACs have made more difficult aneurysms more treatable, with a decreased rate of recanalization and retreatment. However, the risk of bleeding and thromboembolism during the procedure, particularly in ruptured aneurysms, is higher than with normal coiling (Pierot and Wakhloo 2013).

2.4. Flow Diversion

A new armamentarium for aneurysm treatment flow diverters (FDs) was introduced in 2007 (Pierot and Wakhloo 2013). FDs (Figure 6) are low-porosity tubular stent-like endovascular devices which have two principal mechanisms of function (Pierot and Wakhloo 2013):

1. Flow redirection: It covers the aneurysm neck that decreases flow into the aneurysm sac by enhancing the resistance caused by the implant's mesh, while still enabling blood to flow through nearby perforators as well as the side branches. As a result, the flow of blood is diverted away from the aneurysm sac and toward the distal parent artery. Aneurysmal thrombosis is caused by a decrease in blood flow within the aneurysm sac, which causes circulatory stagnation.
2. Tissue overgrowth: The FD acts as a scaffold or framework for the neo-endothelialization of the aneurysm neck.

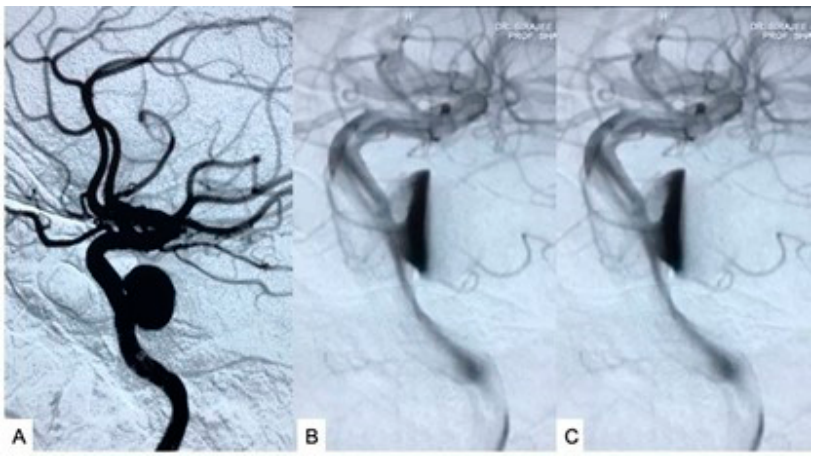

Figure 6. Flow diversion in a case of a cavernous segment ICA aneurysm. (**A**) Before the deployment of a flow diverter; (**B**,**C**) after the deployment of a flow diverter in a cavernous segment of the ICA. Source: Figure by authors.

FDs have been shown to be safe and efficacious in the management of aneurysms in preclinical investigations (Kallmes et al. 2007; Sadasivan et al. 2009).

The majority of preliminary clinical studies with FDs were described in small single-center or multicenter retrospective groups (Lylyk et al. 2009; Szikora et al. 2010; Byrne et al. 2010; Lubicz et al. 2010; Berge et al. 2012). The findings showed that treating a brain aneurysm is quite feasible, with tolerable periprocedural complications, low morbidity and fatality rates, and pleasing efficacy. These findings have recently been bolstered by substantial prospective and retrospective single-center and multicenter investigations (Kan et al. 2012; Piano et al. 2013; O'Kelly et al. 2013). Complex aneurysms, such as fusiform aneurysms, massive and giant aneurysms, broad-neck aneurysms and recurrent aneurysms after earlier coiling, are treated by flow diversion.

A recent prospective study employed in the management of complicated aneurysms found that FD has high effectiveness and a good safety profile. In 108 patients, this study treated massive and enormous, broad-necked aneurysms in the intracranial ICA. The procedure was technically possible in 99.1% of cases, with satisfactory safety, as well as high effectivity (73.6% of aneurysms fulfilled the study's combined primary efficacy end objective

of full closure at day 180 sans severe constriction of the parent channel; no supplementary coils were utilized) (5.6% of cases had a major same-sided infarct or death, which was the primary safety limit point) (Pierot and Wakhloo 2013; Becske et al. 2013).

Although the indications for FDs are still being worked out, it appears that FDs are most commonly utilized in big and giant aneurysms (along with fusiform aneurysms), multiple aneurysms within a segmental sick artery, wide-neck aneurysms and recurring aneurysms. As double antiplatelet therapy is required, the majority of aneurysms managed are unruptured. Flow diversion treatment, on the other hand, is effective in aneurysms that are too small to be treated with traditional coiling, such as blister-like aneurysms (Kulcsár et al. 2010).

More patient information on potential problems is becoming accessible as the use of FDs becomes more popular. Thromboembolic events and intraprocedural bursts are possible with any EVT of aneurysms. Although there is a lower chance of intraprocedural rupture due to the absence of endosaccular manipulations, the chance of thromboembolism is higher than with normal coiling or BAC. Preoperative and postoperative dual (usually) antiplatelet therapy is recommended to prevent thromboembolic events (Pierot and Wakhloo 2013).

It is worth noting that the majority of FD-related problems have occurred in giant and large aneurysms with a high natural risk of hemorrhage, that are otherwise endovascularly untreatable. Aneurysms rupture close after FD deployment in 1.0% of patients (Kulcsár and Szikora 2012). To prevent late rupture in FDs, especially in giant and large aneurysms, a few coils can be placed in the aneurysm sac before deployment of an FD, and steroids can be used after aneurysm treatment. Delayed ipsilateral parenchymal hemorrhage is another potentially lethal complication. The incidence is not exactly known, but Cruz et al. reported an 8.5% incidence (Cruz et al. 2012). Another important concerning issue of FDs is the patency of the perforators and side arterial branches covered by the implant. The migration of FDs is another rare concern. After the application of FDs, perforators can be occluded, resulting in deep infraction contributing to mortality and morbidity (Kulcsár et al. 2010). Late thrombosis of FDs can occur during the post-treatment period, and so, long-term follow-up is necessary (Fiorella et al. 2010; Kulcsár and Szikora 2012). The long-term results of FDs are yet to be known.

2.5. Flow Disruption

Intrasaccular flow disruption is an endovascular implant similar to intraluminal flow disruption, except that the mesh of the flow disruptor is positioned within the aneurysm pouch, causing blood flow stagnation and thrombosis.

Preclinical trials demonstrated the technique's practicality, as well as its efficacy and safety (Ding et al. 2011). The technical success of the treatment was high (100.0%) in a retrospective, preliminary, multicenter small series managed with the flow disruption device, with no death and low morbidity (4.8%) (Pierot et al. 2012e). This can be utilized to treat the basilar artery, middle cerebral artery, anterior communicating artery and ICA bifurcation aneurysms with a wide neck (Pierot et al. 2013). Antiplatelet therapy is not required because the flow disruptor implant is put entirely within the aneurysm, although the risk of a intraoperative burst is increased (Pierot and Wakhloo 2013). There is still a lot to learn about flow disruption.

2.6. Embolization with Liquid Embolic Agents

Liquid embolic agents have been investigated as a therapy for IAs. Onyx (Covidien/EV3, Irvine, CA) was the product with the most significant development, as well as the most extensive clinical evaluation (Molyneux et al. 2004). The product is gradually administered into the aneurysm sac under the supervision of a remodeling balloon, filling the aneurysm from the fundus to the neck. The preliminary results with uncoilable IAs were satisfactory, with good effectiveness and safety (Molyneux et al. 2004). However, rising safety issues (mass effect of giant and big IAs grew following filling with Onyx, stenosis of the parent vessel due to Onyx leaks) stunted the technique's expansion (Carlson et al. 2013).

In summary, various endovascular alternatives for the management of IAs are now available, including normal coiling, SAC, BAC and flow diversion. To preclude re-hemorrhage and subsequent complications linked to an SAH-related hydrocephalus and vasospasm, ruptured aneurysms must be treated on an emergency basis. Standard coiling or BAC is still used to treat ruptured aneurysms. Currently, flow disruption has not been adequately tested for emergency use. These procedures should not be utilized in burst aneurysms, since antiplatelet is required following SAC and flow diversion. Unruptured aneurysm treatment indications should be explored and reviewed on a case-by-case approach, taking into account clinical presentation, patient age and

comorbidities, as well as aneurysm size and location. Treatment with normal coiling or BAC is usually appropriate for minor aneurysms with small necks, while SAC and FDs are rarely used unless aneurysms are threatening to recur. Due to the possibly high rate of recanalization in giant and large, wide-neck and fusiform aneurysms, more comprehensive treatment, comprising SAC, flow diversion and flow disruption, should be used. More sophisticated devices and procedures have been developed as a result of scientific advancements in neuroimaging and device manufacturing, allowing for EVT to treat cerebral aneurysms that were previously believed to be untreatable by EVT. To assess the efficacy and safety of numerous upcoming new technologies, randomized trials will be required for the treatment of aneurysms in the brain (Pierot et al. 2012a; Pierot and Wakhloo 2013).

3. Carotid Atherosclerotic Stenosis and Stenting

The details of carotid artery stenosis are discussed in Chapter 10. Carotid artery stenting (CAS) (Figures 7–9) is an endovascular treatment that involves placing a stent within the artery's lumen to alleviate stenosis and minimize the risk of an ischemic stroke. When carotid endarterectomy (CEA) is deemed too dangerous, CAS is utilized to address the carotid artery stenosis in high-risk cases.

Carotid stenting is used to reduce the risk of an ischemic stroke caused by stenosis of the carotid artery. Carotid stenosis can be painless or cause symptoms (TIAs or strokes).

While CEA has traditionally been the treatment of choice for carotid stenosis, stenting provides an option for people who are not surgical candidates. When there are a lot of CEA risk factors, stenting is recommended instead. Medical comorbidities (severe lung disease, severe cardiac disease, cardiac failure) and anatomic attributes (opposite carotid occlusion, neck radiation therapy, prior same-sided carotid artery surgery, intracranial or intra-thoracic carotid disease) are all risk factors that can make CEA more difficult and dangerous (Gurm et al. 2008).

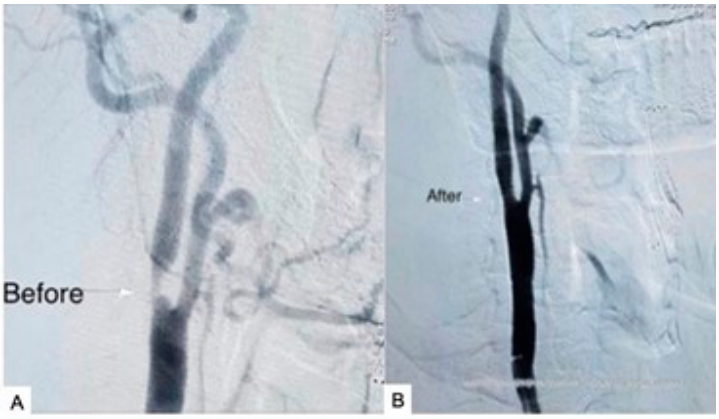

Figure 7. Initial segment of ICA stenting in severe carotid stenosis. (**A**) Before stenting; (**B**) after stenting. Source: Figure by authors.

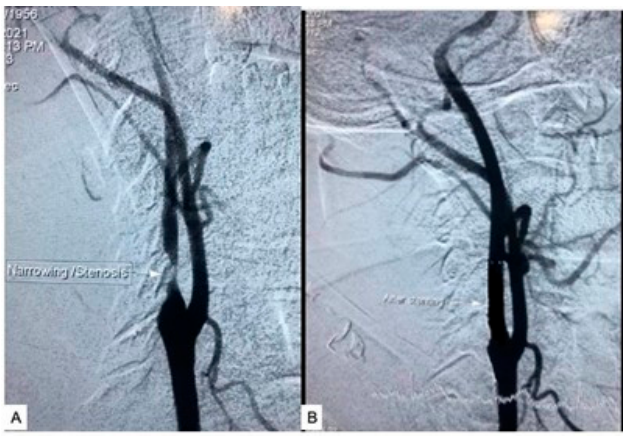

Figure 8. Initial segment of ICA stenting in severe carotid stenosis. (**A**) Before stenting; (**B**) after stenting. Source: Figure by authors.

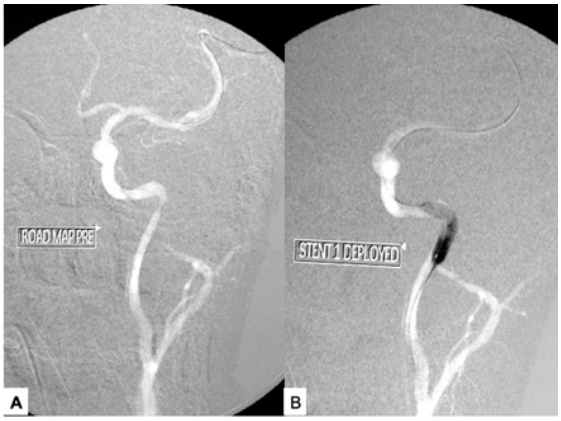

Figure 9. ICA stenting for stenosis at the junction of the cervical and petrous part of the ICA. (**A**) Road map of stent placement across the stenosis; (**B**) stent deployment with dilatation of stenosis. Source: Figure by authors.

Precaution: Although death and stroke following both CEA and CAS are low, the rates of death and stroke after a CAS may be higher than after a CEA, especially for a transfemoral route CAS in persons over 70 years of age (Bonati et al. 2012).

3.1. Endovascular Techniques

CAS is consistent with the placement of an intraarterial stent (https://en.wikipedia.org/wiki/Stent (accessed on 12 July 2021)) across arterial stenosis under general or local anesthesia.

CAS is commonly performed through the percutaneous femoral artery route. Critical steps include arterial access, passing of the guidewire across the stenosis, placing a stent across the stenosis and withdrawal of all the vascular access. The guide wire and sheath are progressed to the side to be treated through the femoral artery, external iliac artery, common iliac artery and the aorta. Other procedures, such as the utilization of a cerebral protection device (embolism protection device), pre/post-stent balloon angioplasty and cerebral DSA, may or may not be undertaken.

3.2. Post-Procedural Outcomes

Recovery after CAS is relatively simple provided there are no events. Patients typically stay in hospital for 0–1 day. The systolic blood pressure is kept under 140 mm of mercury. Reperfusion/hyperperfusion syndrome can be caused by high blood pressure in the early days after surgery.

The most concerning short-term consequence of any carotid artery stroke prevention operation is the occurrence of a stroke. Patients should be selected for CAS so that the procedure's "long-term risk prevention" is greater than the "short-term risk" of producing a stroke at the time of the surgery. Bleeding, infection and cardiac problems are among the other risks.

Recurrent stenosis/pseudoaneurysm development is a possible late consequence. It is necessary to follow up with a duplex ultrasonogram, CTA or MRA. When a patient has symptoms of carotid occlusive disease/stenosis (CEA/CAS), the risk reduction intervention is most effective—typically stroke or TIA (Paraskevas et al. 2009). For symptomatic individuals, there is inadequate evidence to say whether stenting or CEA is preferable. Asymptomatic carotid stenosis should only be treated in the background of randomized clinical trials (stenosis > 70%) (Derdeyn Colin 2007).

4. Intracranial Arterial Stenosis and Stenting

Intracranial arterial stenosis is a frequent etiology of ischemic strokes (middle cerebral artery, intradural ICA, anterior cerebral artery or intradural vertebrobasilar artery stenosis). At the end of 30 days, the stenting group had a 14.7% risk of an ischemic stroke or death compared to 5.8% in the medically managed group, and 23% in the stenting group compared to 15% in the medical group at a median follow-up of 32.4 months in the randomized multicenter study called Stenting and Aggressive Medical Management for the Prevention of Recurrent Stroke in Intracranial Stenosis (SAMMPRIS). The findings demonstrated that medical care is superior to endovascular stunting, and that intracranial arterial stenting has almost been phased out in recent years. In this series, 16 cases

in the stenting group (7.1%) experienced a severe debilitating or fatal stroke within 30 days, primarily due to perioperative events, analogous to 4 cases in the medical group (1.8%). On the other hand, 5 cases (2.2%) in the stenting group, as well as 14 cases (6.2%) in the medical group experienced a debilitating or fatal stroke after 30 days, demonstrating that stenting has a significant advantage if periprocedural problems can be controlled or avoided. It is necessary to assess the role and result of intracranial stenting in the preclusion of debilitating or fatal stroke (Chimowitz et al. 2011; Yu and Jiang 2018).

5. Vertebral Artery (VA) Stenosis and Stenting

Patients with a recent symptomatic vertebrobasilar insufficiency/stenosis, similar to carotid artery stenosis, have a substantial risk of a recurrent ischemic stroke, with the risk being the highest in the first month (Payne 2001). Angioplasty and/or stenting can be used to treat stenosis of the vertebral artery (VA) (Figure 10). Stenting may be a good therapeutic approach according to one case series (Oshizumi et al. 2004; Eberhardt et al. 2006; Stayman et al. 2011). Stenting for extracranial VA stenosis has been linked with very few complication rates (1–1.5%) (Stayman et al. 2011), whereas intracranial stenosis is accompanied with higher hazards rates (7–10%) (Eberhardt et al. 2006). However, new randomized trial data have dampened the enthusiasm. In patients with an intracranial arteries stenosis, the Stenting and Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis (SAMMPRIS) trial found that stenting was worse than the best medical therapy (BMT) in preventing recurrent stroke (Chimowitz et al. 2011); there were, however, only a few patients with a VA stenosis (Lutsep et al. 2015).

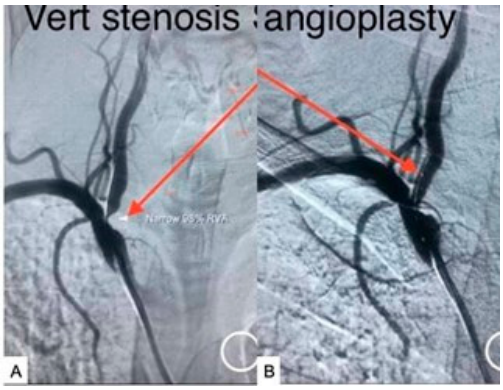

Figure 10. VA stenting at its origin. (**A**) Pre-stenting DSA; (**B**) after stent deployment. Source: Figure by authors.

6. Carotid–Cavernous Fistula (CCF)

The CCF (caroticocavernous fistula) (Figure 11) occurs due to a tear in the ICA, which permits it to develop a low-resistance, high-flow fistula with the cavernous sinus's venous system. A CCF can be direct or indirect (Barrow et al. 1985; Cohen and Rad 2004). Blood is diverted from the ICA into the cavernous sinus in a direct CCF (DCCF); in an indirect CCF (ICCF), there is a dural arteriovenous connection and a reduced flow rate. The details of a CCF are described in Chapter 15.

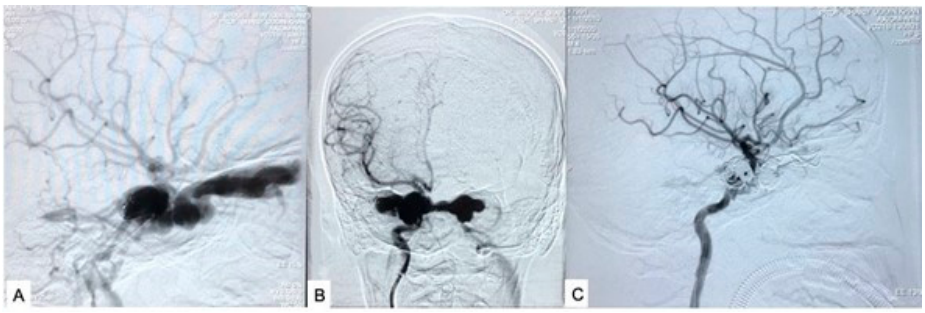

Figure 11. Direct CCF repair with endovascular coiling. (**A**,**B**) Pre-embolization; (**C**) after repair of the fistula with coils. Source: Figure by authors.

6.1. Classification

A CCF is divided into direct (Type A) or indirect (Types B–D) categories (Barrow et al. 1985; Cohen and Rad 2004; Henderson and Miller 2017):

Type A/direct: DCCF is a high-flow fistula between the ICA and cavernous sinus (CS):

(a) Traumatic (including iatrogenic): occurs in 0.2% of patients with traumatic head injury. Iatrogenic DCCF occurs following percutaneous trigeminal rhizotomy, an endovascular procedure.
(b) Spontaneous: ruptured cavernous sinus ICA aneurysm with connective tissue disorders.

Type B-D/Indirect: most ICCFs are shunts from dural arteries that are branches of the ICA/ECA:

Type B: from the dural branches of the ICA;
Type C: from the dural branches of the ECA;
Type D: from the dural branches of both the ICA and ECA.

6.2. Etiopathology with Mode of Presentation

The pressure difference in a DCCF causes flow reversal into the superior ophthalmic vein, as well as superficial middle cerebral vein and rapid diverting to the inferior petrosal sinus and pterygoid vein, resulting in a pulsating exophthalmos and orbital bruit, as well as visual changes, orbital pain and proptosis.

The majority of symptoms are unmistakably caused by arterialization of the CS with draining orbital veins. Venous congestion and hemorrhage coupled with headache, chemosis, vertigo, tinnitus and cranial nerve paralysis are some of the most prevalent complications.

In patients with a CCF, having arterial steal leads to cerebral hypoperfusion that causes focal neurological impairments.

6.3. Investigations: Evaluation

A CT–CTA or MRI–MRA of the head usually demonstrates proptosis, which is characterized by engorged and serpiginous intraorbital vessels, including the superior ophthalmic vein (best observed on T2WI coronals) and convexity of the lateral wall of the cavernous sinus.

A DSA shows diversion of blood from the ICA into the CS. Quick opacification of the petrosal sinus and/or ophthalmic vein may be seen.

Mehringer–Hieshima maneuver: injection of contrast at a rate of 2–3 mL/s into diseased carotid while compressing the cervical carotid artery (down to the catheter tip) to control flow to help demonstrate the fistula.

Huber maneuver: lateral view, inject VA and compress the affected carotid artery. It aids in detecting the upper limit of the fistula, many fistulous openings and the total transection of the ICA (Henderson and Miller 2017; Morris 2007).

6.4. Treatment

The patient's stability, the anatomy of the fistula and the hemodynamics of the system all play a role in CCF management. Management should ideally work on repairing or obliterating the tear or connection while maintaining flow through the ICA (Fiorella et al. 2008). Total blockage of the artery may be required in some cases. Because 20–50% of low-flow CCFs thrombose spontaneously, they can be observed as long as visual acuity is constant and intraocular pressure is less than 25. High-flow CCFs that are symptomatic (e.g., gradual vision degradation) seldom thrombose spontaneously, and therapy is generally required. Parkinson reported a straightforward surgical repair of a traumatic CCF while keeping the ICA intact in 1973. While any treatment in this anatomic area is difficult, open surgical repair in the acute environment amid probable polytrauma in patients carries a high risk of morbidity; hence, endovascular closure is the preferred option if the patient can tolerate it (Henderson and Miller 2017; Geibprasert et al. 2009).

6.4.1. Endovascular Treatment

The goal of treatment is eliminating the fistula.

A cerebral DSA is used to detect the precise size and location, as well as its venous drainage of the fistula. Angiography with 7.5 frames per second should be considered instead of the normal 2–4 frames per second to handle the excessive flow. Any vascular injuries/anomalies in addition to the CCF should be looked for. Both

ECAs and ICAs are catheterized selectively to measure their role in the CCF. To better measure the cross-flow from the contralateral side, angiography is conducted after physical compression of the CCA on the side of the fistula. The excessive blood circulation to the fistula will be reduced by digital compression, allowing for viewing of the fistula.

Rotational angiography with 3D reformatting may be performed to study the fistula and select proper working views for the interventional procedure. The CS, inferior and superior ophthalmic veins, sphenoparietal sinus, superior and inferior petrosal sinuses, and pterygoid plexus are all key venous involvements to be aware of.

The following routes may be utilized for managing CCFs: transarterial, transvenous and superior ophthalmic vein.

Coils: The ideal route is the transarterial coil embolization of CCFs. Utilizing road mapping, the microcatheter is moved over the microware into the CS via the fistula. Coils are then deployed and detached. Periodic angiography is performed and further coils placed. Complete occlusion is indicated by no further contrast entering the CS.

Onyx: In case of a high-flow fistula, prior to Onyx deposition, it may be recommended to deposit coils into the CS initially to slow down the blood flow. A balloon can be inflated within the parent ICA to protect it.

NBCA: To prevent undesired deposits in the venous sinuses, an NBCA should be used with extreme caution, especially after slowing the flow through the CCF. A balloon may be placed in the parent artery to safeguard it.

Stents: Stents provide the required parent vessel protection by acting as a non-occlusive scaffold along the ostium of the fistula. A self-expanding, flexible stent is utilized in the pipeline embolization procedure. Covered stents can be utilized instead of coils and balloon-assisted or stent insertion. The possible tortuosity of the vasculature plus the stiffness of the stent occludes any small arteries in the vicinity, which is one of the key obstacles in the proper delivery as well as deployment of covered stents.

Balloons: Type A CCFs can be repaired using a removable balloon occlusion. Low cost, easy navigation to the fistula, and the capability to intermittently inflate and deflate the balloon permit for the constant appraisal of the fistula anatomy as advantages of the balloon.

Choice of Technique

With indirect fistulas, it is mandatory to place coils on the side of venous (otherwise new feeders will be recruited). Coils or clips may be used to occlude direct fistulas (Henderson and Miller 2017; Fiorella et al. 2008; Geibprasert et al. 2009; Chalouhi et al. 2012).

7. Cranial Dural Arteriovenous Fistulas (DAVFs)

7.1. Introduction

A DAVF (Figure 12) is a vascular pathology in which an arterio-venous shunt is within the two layers of the dura mater. Multiple branches of the ICA/ECA or vertebral arteries create direct communication with the venous sinus and/ or cerebral veins. They are considered acquired rather than congenital lesions. Lesions leading the arterialization of intradural veins (leptomeningeal cortical vein) are linked with an ICH. Multiple fistulas may be found in up to 8% of cases. They are usually found adjacent to the dural venous sinuses. The details, including the Borden and Cognard classification of cranial AV fistula is discussed in Chapter 14.

7.2. Common Locations

- Transverse/sigmoid: the most common (63% of cases) with a slight left-sided predominance (Baharvahdat et al. 2020), with the epicenter of these almost invariably at the junctional site of the transverse and sigmoid sinuses (Figure 12);
- Tentorial/petrosal;
- Anterior fossa/ethmoidal;
- Middle fossa/Sylvian;
- Cavernous sinus (carotid–cavernous fistula—CCF);
- Superior sagittal sinus;
- Foramen magnum (Intracranial Dural Arteriovenous Fistula 2021).

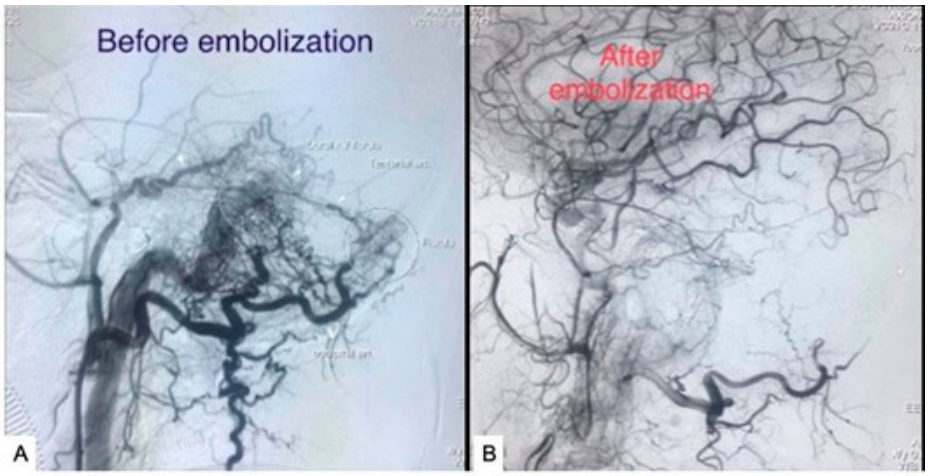

Figure 12. Sigmoid sinus dural AVF. (**A**) Before embolization; (**B**) after embolization. Source: Figure by authors.

7.3. Epidemiology

DAVFs constitute 10–15% of all intracranial AVMs. Of the patients, 61–66% occur in females, and patients are usually in their 40s or 50s. They can occur seldom in children. When they occur, they tend to be more complex, bilateral dural sinus malformations (Baharvahdat et al. 2020; Graeb and Dolman 1986; Arnautovic and Krisht 2000; Ashour et al. 2012).

7.4. Clinical Features

Pulsatile tinnitus is the most common presenting symptom of a DAVF. Cortical venous drainage with possible venous hypertension can produce IC-HTN, and this is the most common cause of mortality and morbidity, and thus the robust indication for DAVF management. DAVFs may also cause global cerebral edema or hydrocephalus due to impaired cerebral venous drainage or by disturbed function of the arachnoid granulations, respectively. Other DAVF symptoms/signs include headaches, seizures, cranial nerve palsies and orbital venous congestion (Intracranial Dural Arteriovenous Fistula 2021; Baharvahdat et al. 2020).

7.5. Natural History and Risk of Hemorrhage

The concept of a benign vs. aggressive DAVF behavior depends on the presence or absence of cortical venous drainage. Data reported by the University of Toronto group over a 3-year period show that 98% of benign lesions (no cortical venous drainage) remained benign (Intracranial Dural Arteriovenous Fistula 2021; Davies et al. 1997). On the other hand, over a 4-year period, the annual hemorrhage rates, non-hemorrhagic neuro-deficit and mortality were 8.1%, 6.9% and 10.4% for aggressive fistulas (with cortical venous drainage) (Intracranial Dural Arteriovenous Fistula 2021; van Dijk et al. 2002). In a meta-analysis of 377 cases (Ashour et al. 2012), three DAVF locations were associated with particularly aggressive behavior (aggressive: benign ratio): tentorial (31:1), middle fossa/Sylvian (2.5:1), anterior fossa/ethmoidal (2.1:1). Most Borden Type I lesions or Cognard Type I and IIa are benign lesion, whereas higher-grade fistulas are dangerous (Intracranial Dural Arteriovenous Fistula 2021).

7.6. Investigation: Evaluation

A brain CT or MRI without contrast is often normal.

A CTA may reveal dilated and tortuous vessels corresponding to enlarged arterial supplier or ectatic draining veins.

An MRA may show dilated and engorged pial vessels, early distinctive venous sinus filling, sinus engorgement or occlusion and white matter edema due to venous hypertension.

DSA: Full six-vessel cerebral angiography (bilateral ICAs, bilateral ECAs and bilateral vertebral arteries) is required to reach the diagnosis and plan the treatment.

DAVFs with angiographic findings include the following:

A selective DSA shows prolong cerebral circulation time. This occurs in venous congestive encephalopathy.

Pseudo phlebitis pattern: The cerebral surface shows the tortuous and dilated collateral veins in the venous phase of the DSA. This finding is connected with a higher risk of hemorrhage or neuro-deficits of non-hemorrhagic origin.

Cortical venous reflux (CVR): To confirm that this is not omitted, a selective (rather than non-selective, global) DSA should always be performed when investigating a DAVF. A venous obstruction or stenosis is frequently seen in patients with CVR.

Several classification systems have been published to characterize DAVFs.

The Borden and Cognard systems are the most commonly utilized grading systems. Cortical venous drainage is the definite angiographic feature that distinguishes benign (low-grade) from aggressive (high-grade) fistulas (Intracranial Dural Arteriovenous Fistula 2021; Baharvahdat et al. 2020). (Borden I, Cognard I and Cognard IIa are low-grade; all others are high-grade.)

7.7. Treatments

Management options:

Conservative;
Endovascular;
Microsurgery;
Radiosurgery;
Any combination of surgery, endovascular and radiosurgery.

An AVF with cortical venous drainage should usually be treated. Lesions without cortical venous drainage should be followed radiographically and clinically (2% may evolve to develop cortical venous drainage). An alteration in a bruit (either disappearance or worsening) should be reinvestigated. Here, endovascular options are discussed.

Indications for intervention:

1. Presence of cortical venous drainage;
2. Neurologic dysfunction;
3. Hemorrhage;
4. Orbital venous congestion;
5. Refractory symptoms (headache, pulsatile tinnitus) (Intracranial Dural Arteriovenous Fistula 2021; Baharvahdat et al. 2020; Arnautovic and Krisht 2000; Ashour et al. 2012; Davies et al. 1997).

7.7.1. DAVF Embolization

The approach (Figure 12) may be transvenous, transarterial or a combination. Whenever possible, a transvenous route is preferred, as the rate of fistula closures is higher through the transvenous route.

7.7.2. Transarterial Embolization

Transarterial embolization is classically utilized for (a) high-grade DAVFs, (b) direct cortical venous drainage, (c) situations where venous access is difficult or limited, (d) de novo DAVFs may occur at a secondary location after transvenous embolization, probably due to hypertension and (e) hazards related to the transvenous approach can be precluded (sixth nerve palsy from superior petrosal sinus catheterization).

7.7.3. Transvenous Embolization

The transvenous route is preferred when (a) the prime arterial supplier of a DAVF arises from the ICA or the vertebral artery, (b) possible extracranial-to-intracranial anastomoses sites are involved and (c) arterial supply to the cranial nerves is in danger.

Alternatively, in lieu of performing a total occlusion, partial treatment can be considered only such that CVR is ruled out, turning the fistula into the Borden type I (benign).

When utilizing the venous route, it should be ensured that the venous path is not tenuous (e.g., acute DAVF), making it vulnerable to rupture during catheter handling.

Coils: The right-sized coils according to the maximum diameter of the fistulous spot to be occluded should be selected. As many coils as necessary to close the fistula should be installed. A “combination” method may be performed, in which coils are deposited first to decrease the rapid blood circulation via the fistula, then a liquid

embolic agent is used to close the fistula. A microcatheter compliant with the liquid embolic agent if this method is used should be used.

Onyx: During transarterial embolization with liquid embolic agents, the microcatheter should be kept as near as possible to the fistula. It is important to disrupt the fistulous connection to achieve cure. Therefore, Onyx must penetrate into the venous side of the DAVF.

nBCA: It is used less commonly after the availability of Onyx.

The other modalities of treatments are manual carotid self-compression, surgery and stereotactic radiosurgery.

The therapy of choice for DAVFs is endovascular management. A transarterial or transvenous technique is one of the many alternatives available. Coils, Onyx and n-BCA are just a few of the embolizing agents that can be used.

8. Cranial Arterio-Venous Malformation (AVM)

8.1. Introduction

An irregular grouping of blood vessels occurs when arterial blood travels straight into the draining veins passing through the typical capillary beds in between. No parenchyma remains within the nidus. AVMs are generally congenital lesions that tend to increase in size somewhat with age and frequently turn from low-flow juvenile lesions at birth to medium-to-high-flow high-pressure AVMs in adulthood (Frosting 2003). Cerebral AVMs are also discussed in Chapter 13.

8.2. Anatomy

They are made up of a tangled web of afferent feeding arteries, as well as draining veins connected by an irregular intervening capillary bed called the nidus that may or may not have straight arteriovenous shunts. A compact nidus, which forms a tumor-like well-circumscribed system, and diffuse nidus, which has sparse, aberrant AV channels scattered across normal brain tissue, can be more or less distinguished.

One or more feeding arteries are possible. They might be somewhat enlarged or have a nearly normal lumen. High flow can cause the following:

(a) Saccular aneurysms at the plane of the circle of Willis, the feeding arteries or the nidus.
(b) High-flow (angiopathy) with increasing stenosis and ultimate occlusion of the supplying arteries. Draining veins might be single or many, deep or cortical. The dilation and tortuosity of the affected veins are caused by direct shunting of blood at high pressure. High flow can also cause localized stenosis and secondary venous aneurysmal dilatation, especially when the veins pass through the dura to enter the sinus (Frosting 2003).

They may be divided as follows (Chaloupka and Huddle 1998):

1. Parenchymal AVMs (mentioned below). Sub-classified as:
   (a) Paraventricular;
   (b) Subcortical;
   (c) Pial;
   (d) Combined.
2. Pure dural AVM.
3. Mixed parenchymal and dural (rare).

AVM-related syndromes include Sturge–Weber, Rendu–Osler–Weber, Klippel–Trenaunay, Wyburn–Mason, Parks–Weber etc.

8.3. Etiopathogenesis

Congenital vascular abnormalities characterized by improper direct connections between venous and arterial systems are hypothesized to be the cause of cerebral AVMs (Mullan et al. 1996; van Beijnum et al. 2007).

Although the precise embryological genesis is uncertain, both the maintenance of a basic arteriovenous link and its development before or after delivery have been hypothesized (Fleetwood and Steinberg 2002).

8.4. Epidemiology

There is hardly any information in the published literature about the incidence of AVMs, or the percentage of a population with an AVM diagnosis at any given period. Because of the disease's rarity and the presence of

symptomless patients, determining a real prevalence estimate is hard and unlikely. A retrospective investigation in a Scottish region reported an incidence of AVMs of 15 per 100,000 live persons over the age of 16 years in unselected populations (Al-Shahi et al. 2002). Because asymptomatic AVMs are not included in this study, the prevalence is clearly underestimated.

8.5. Pathology

Cerebral AVMs consist of (a) aggregations and unusually muscularized feeders that may also have modifications such as doubling or damage of the elasticity, segmental thinning of the wall and fibrosis of the media; (b) arterialized veins of various sizes and wall thicknesses; (c) anatomically ambiguous vessels consisting of fibrous tissue only or showing both venous and arterial criteria; and (d) intervening gliotic tissue. They connect to a regular cerebral vessel by anastomosis (Frosting 2003).

8.6. Physiopathology and Biology

The pathophysiology of cerebral AVMs is uncertain; however, new research suggests that abnormal vasculogenesis or angiogenesis may have a role in their genesis and progression. Protein ligands bind and modulate the actions of transmembrane receptor tyrosine kinases, form and remodel blood vessels in both processes (Frosting 2003).

8.7. Natural History

Cerebral AVMs are diseases that are unaffected by significant anatomic changes over time. But, because AVMs are dynamic, they are subject to ongoing anatomic and hemodynamic changes. When the patient's ability to properly adapt has attained its limit, an AVM becomes symptomatic. They are most commonly clinically obvious in young individuals, especially those under the age of 40 years. The evolutionary history of cerebral AVMs rarely includes expansion, reduction or regression anatomically. The spontaneous obliteration of AVMs in the brain is extremely unusual. The following factors aid in AVM thrombosis regression:

- The AVM's anatomy;
- Surgical treatment of the AVM;
- Squeezing of the AVM by a neighboring mass (such as a hematoma).

In most cases, an AVM nidus thrombosis is caused by an intracerebral or SAH. In this situation, the blood clot's mass effect may change the dynamic of the AVM and reduce blood circulation, most likely by compressing draining veins to the point of thrombosis. Surgical intervention, such as the removal of a blood clot or the implantation of a shunt, has been connected to the regression of AVMs, which is understandable given the compression of the veins caused by hemorrhage or edema. It is also possible that spontaneous regression occurs.

The presence of a sole draining vein (84% of instances of spontaneous occlusion), a single arterial feeder (30%) and a small AVM nidus (less than 3 cm in 50% of cases) are all linked to a spontaneous occlusion of brain AVMs (Frosting 2003).

8.8. Clinical Features

8.8.1. General Information of Presentation

Hemorrhage (most common);

Seizures;

The mass effect: for example, trigeminal pain caused by a CPA AVM;

Stealth ischemia;

H/A: extremely unusual. AVMs have been linked to migraines in the past. Visual impairment (usually hemianopsia/quadrantanopsia) and H/A that are identical from migraine can be symptoms of occipital AVMs;

Bruit: this is especially true for dural AVMs;

A higher ICP;

Almost exclusively found in children, frequently with big midline AVMs draining into an expanded vein of Galen:

(a) Hydrocephalus and macrocephaly: As a result of constriction of the Sylvian aqueduct by an enlarged Galen vein or elevated venous pressure;
(b) Cardiomegaly with congestive cardiac failure;
(c) Prominence of the frontal veins (due to raised venous pressure) (Drake 1979; Kupersmith et al. 1996).

8.8.2. Hemorrhage

The peak age for rupture of a brain AVM is between 15 and 20 years. The reported morbidity and mortality from an AVM hemorrhage varies extensively. Approximately, it is 10% mortality and 30–50% morbidity (neuro-deficit) from each rupture.

Hemorrhage Location with AVMs

ICH (intraparenchymal): 82% (the most common site of hemorrhage).
Intraventricular hemorrhage:

(a) Usually happens in combination with an ICH due to rupture of the ICH into the ventricle;
(b) An intraventricular AVM may be indicated by a pure IVH (without an ICH).

Subarachnoid: SAH can also occur by the burst of a feeding artery aneurysm, which is prevalent with AVMs.
Subdural: this is a rare occurrence. It is possible that this is the genesis of a spontaneous SDH (Perret and Nishioka 1966; Hartmann et al. 1998).

Hemorrhage Rate Related to AVM Size

Small AVMs have the tendency to bleed more than large ones.
Larger AVMs are associated with seizure more frequently as they are more likely to engage the cerebral cortex because of their size. Small AVMs, on the other hand, have substantially higher pressure in the supplying arteries. As a result, smaller AVMs are more deadly than larger ones (Crawford et al. 1986; Spetzler et al. 1992).

Hemorrhage Rate in Relation to Spetzler–Martin Grade (Controversial)

Some studies have demonstrate a higher risk with Spetzler–Martin (S-M) grade 4–5 AVMs, while others show the opposite:
S-M grade 1–3: annual risk of bleeding is 3.5%;
S-M grade 4–5: hemorrhage is a 2.5% annual risk in S-M grade 4–5 (Jayaraman et al. 2007).

Yearly and Lifetime Risk of Hemorrhage and Re-Hemorrhage

An AVM has a 2–4% chance of causing bleeding on average. The risk of rebleeding in the first year after a hemorrhage was 6–14%, which declines to 2% per year after 10 years (Kondziolka et al. 1995).
Factors increasing the risk of bleeding (Frosting 2003; Kondziolka et al. 1995):

Anatomic Factors

(a) Feeding Vessels:
Arterial aneurysms;
Feeders from the ECA;
Feeders by perforators and the vertebra-basilar system.
(b) Nidus:
Size;
Location;
Angiogenesis.
(c) Venous Drainage:
Venous stenosis.
Deep venous drainage.
Venous reflux;
Solitary draining vein;
Venous ectasia.

Hemodynamic Factors

Feeding artery pressures;
Draining vein pressures.

Factors decreasing the risk of bleeding (Frosting 2003):

1. Nidus—reduction in the pressure into the nidus:

(a) Arterial stenosis;
(b) Arterial angioectasia.

2. Arteriovenous fistulas.

Severity of the Hemorrhage

The rupture of cerebral AVMs (Frosting 2003; Hartmann et al. 1998) is less serious in comparison with that of aneurysms, with fatality rates of 10 to 15%, as well as overall morbidity rates of less than 50%. Subarachnoidal (30%), intraventricular (16%), parenchymal (23%) and mixed sites (31%) are the most common locations for cerebral AVM hemorrhages. A neurological deficiency is generally accompanied by parenchymal hemorrhages (52%). In general, 47% of cases had a positive outcome following the hemorrhage, and another 37% of cases were self-sufficient in their daily lives.

In truth, an AVM rupture is just as destructive as an aneurysm rupture. While an aneurysm rupture is more fatal than an AVM rupture (21% versus 9%), AVM ruptures have a lower success rate (49% versus 56%) as a result of the increased risk of a parenchymal hematoma.

8.8.3. Seizures

The younger the patient, greater the chance of experiencing seizures at the time of detection. Patients who arrive with bleeding have a 22% chance of having epilepsy in the next 20 years. No AVM discovered by chance or presenting with a neurological impairment develops seizures. Seizures are usually partial or partially complex in nature. Between 27% and 35% of patients experience generalized seizures. Seizures are more commonly related to cortical AVMs. Antiepileptic medicines are effective in controlling seizures in a large majority of instances (Ding et al. 2015; Lv et al. 2010).

8.8.4. Headache

In 7–48% (mean: 31%) of cases, the first symptom is a chronic headache (Mast et al. 1995). There is no recognized link between headache, migraine and AVMs. There are no characteristics that point to the diagnosis of an AVM, such as incidence, duration or severity.

8.8.5. Focal Neuro-Deficits

In 1–40% of cases, focal neurological impairments without bleeding are the first symptom. Focal neuro-deficiencies can be gradual, persistent or reversible in nature. The cause of reversible focal neurologic impairments is unclear, as a post-ictal origin may not be ruled out. Different hypotheses for the evolution of neurologic deficits exist: the mass effect, venous hypertension or steal phenomena are all terms for the same thing (Frosting 2003; Mast et al. 1995).

8.9. AVMs and Aneurysms

About 7% of cases with cerebral AVMs have associated aneurysms (Frosting 2003). Seventy five percent of them are situated on the major supplying artery.

These AVM-associated aneurysms may be classified as the following five types:

Type	Aneurysm location
I	Proximal to the ipsilateral main artery that feeds the AVM;
IA	Proximal to a large artery but on the opposite side of an AVM;
II	The superficial feeding artery is situated at the distal end of the artery;
III	On the deep feeding artery, proximal or distal (“bizarre”);
IV	On a non-AVM artery; on draining veins or within the nidus, aneurysms can occur.

Symptomatic AVMs or aneurysms are usually addressed first when treating AVMs and aneurysms (whenever feasible, both should be treated simultaneously). If it is not evident which one bled, the aneurysm is most likely to be accountable, though the majority of AVM-related aneurysms will regress after the AVM is removed (66%).

8.10. Investigations and Evaluation

8.10.1. Aims of Neuro-Imaging

1. To reach the diagnosis of cerebral AVMs;
2. For pre-treatment assessment of brain AVMs (to abet in decision-making);
3. To manage brain AVMs as a only therapy or in combination with radiosurgery or surgery;
4. For post-treatment follow-up and evaluation.

8.10.2. CT and CTA of the Head

A CT scan of the head is the best neuroimaging to exclude acute hemorrhage and its types, i.e., parenchymal, subarachnoid and intraventricular. It can also show the calcifications within the pathology. When the patient is young, the parenchymal hematoma is lobar. The calcifications or spontaneously highly dense serpiginous formations (Vessels voids) are present and should be discussed.

The changed appearances of the ventricular system can be visualized:

- Effacement of the ventricular system due to the mass effect generated by the AVM;
- Focal dilatation in the case of concomitant parenchymal atrophy;
- Hydrocephalus can occur as a result of a prior hemorrhage or when the ventricular cavity is squeezed by the AVM's engorged drainage veins;
- A contrast CT scan may reveal enhancement within the arteries, as well as a sharpening of the nidus (Frosting 2003; Chaloupka and Huddle 1998; Spetzler and Martin 1986; Peschillo et al. 2014).

8.10.3. MRI and MRA of the Head

In unruptured AVMs or lobar hematomas, an MRI is frequently used days or weeks following the hemorrhage to look for the underlying pathology. Three levels of evaluation of the AVM can be obtained using a separate sequence of MRI images:

- Analytical sequences for anatomical analysis;
- MR angiography for vascular analysis and fMRI for functional analysis.

8.10.4. Anatomic Analysis of AVMs

T1, T2 and T1 with a gadolinium MRI can completely define the anatomic location and the size of the nidus. An MRI always defines anatomic localization better than angiography. As a result of the flow void phenomenon, the circulating vessels have no signal on T1W and T2W imaging. With traditional imaging sequences, portrayal of arterial feeders as well as draining veins is frequently unclear. An MRI is also a superior tool for showing the specific location of parenchymal lesions produced by an AVM. A current and earler hematoma can be seen using magnetic resonance imaging. A recent hematoma, on the other hand, may obscure a minor AVM, resulting in a false negative MR. Perinidal signal anomalies, notably hypersignal on T2W imaging, can be a marker of perinidal ischemia in the absence of blood. An MR can detect AVM-induced morphological alterations, as well as their parenchymal and ventricular consequences:

- Ventricular system dilatation due to parenchymal atrophy;
- Hydrocephalus in the case of an earlier hemorrhage or if the ventricular cavity is squeezed by engorged draining veins;
- Hydrocephalus in the case of a previous hemorrhage or whether the ventricular system is squeezed by engorged draining veins.

8.10.5. Vascular Analysis of Cerebral AVMs

Three-dimensional depictions of AVM architecture can be obtained by phase contrast and time-of-flight (TOF) techniques. Anatomic coverage and preciseness are the limitations of these techniques. The accurate size of the nidus cannot be calculated, intranidal aneurysms are not commonly visible, portrayal of the draining veins is not consistent, and small-caliber vessels and areas of sluggish blood circulation cannot be reliably seen. In addition, dynamic information cannot be obtained by these sequences. New gadolinium-contrasted MRAs are better than TOF MRAs, but still less demonstrating than DSA images for picturesque components of AVMs, as both temporal and spatial resolution are not available.

8.10.6. Functional Analysis of Brain AVMs

A perfusion PWI and diffusion DWI, apparent diffusion coefficient (ADC) imaging and bold sequences are all part of a functional MRI (fMRI). The nidus normally has a weak signal with a big and uniform increase in ADC. In AVMs, DWI and PWI have a small impact. The functional sections of the brain in an eloquent area AVM, especially the visual, sensorimotor and language areas, can be visualized with an fMRI. The use of bold sequences helps identify hemodynamic alteration in the cortex while performing a certain activity. A change in the activated cortex with common interhemispheric transmission is noted in most where AVMs are situated in the eloquent cortex.

8.10.7. Characteristics of a Cerebral AVM on MRI

- Increased intensity on partial flip-angle T1WI or T2WI within the AVM;
- Flow void on T1WI or T2WI within the AVM;
- Increased intensity on partial flip-angle T1WI or T2WI within the AVM;
- Flow void on T1WI or T2WI within the AVM;
- A significant amount of edema around the lesion could indicate a bleeding tumor rather than an AVM;
- A complete ring of low density (as a result hemosiderin) around the lesion alludes to an AVM over neoplasm on gradient echo sequences (GRASs);
- An entire ring of low density (as a result of hemosiderin) around the lesion alludes to an AVM over neoplasm on gradient echo sequences (GRASs) (Frosting 2003; Chaloupka and Huddle 1998; Spetzler and Martin 1986; Peschillo et al. 2014).

8.10.8. CT Angiography (CTA)/MR Angiography (MRA) Findings in a Brain AVM

On angiography, AVMs have the following characteristics:

- Large feeding artery;
- Angle of vessels;
- Draining veins are shown in the same pictures as the arteries (arterial phase);
- Large draining veins.

Angiography can detect most AVMs, but not all (Frosting 2003; Chaloupka and Huddle 1998; Spetzler and Martin 1986; Peschillo et al. 2014).

8.10.9. Selective and Super-Selective DSA

CTA and MRA are not adequate for an accurate description of the anatomic and hemodynamic features of an AVM. Functional and anatomical information that is therapeutically important must still be obtained through angiography. Injecting into the ICA, ECA and vertebral arteries is required for selective angiography. Multiple projections are used to analyze and gather data on arterial feeders, nidus and venous drainage (anteroposterior, lateral and oblique). A 3D DSA (three-dimensional DSA) could be useful. However, even a perfect DSA is frequently insufficient for making precise therapy judgments. Large feeding arteries may have a hazy architecture, whereas small feeding arteries may not be apparent on a selective DSA. Intranidal aneurysms and straight intranidal AV fistulas are frequently ignored, despite the fact that the extent of the nidus is usually well demonstrated by selective angiography. The venous drainage of the brain AVM is usually well received by a selective DSA, but as the AVM is injected as a whole, the compartments of the AVM, as well as their venous drainage are frequently not well understood. Because of these factors, super-selective angiography is frequently required for a more complete investigation of AVMs and may become increasingly relevant in decision-making. The manual injection of each particular artery feeder is used in super-selective angiography. It is usually the first step in the therapeutic embolization procedure (Frosting 2003; Chaloupka and Huddle 1998; Spetzler and Martin 1986; Peschillo et al. 2014).

8.11. Grading of Cranial AVM

8.11.1. Spetzler–Martin Grade of AVMs

Grade = sum of points from Table 1 ranges from 1 to 5. Untreatable AVMs (by any means: surgery, SRS, etc.) are assigned a distinct grade 6, since excision would almost always result in a debilitating deficiency or death. This scale has been shown to have good prognostic predictability. It may not be applicable to pediatrics

(AVMs are immature and change with time; AVMs mature at ≈age 18 years and tend to become more compact). Spetzler–Martin Grading of AVM with Supplementary grading is shown in Table 1.

Table 1. Grading of AVMs.

Spetzler–Martin Grading	Points	Supplementary Grading
Size		**Age, years**
<3 cm	1	Less than 20
3–6 cm	2	20–40
>6 cm	3	More than 40
Venous drainage		**Hemorrhage**
Superficial	0	Yes
Deep	1	No
Eloquent brain		**Compactness**
Non-eloquent	0	Yes
Eloquent	1	No
Total Grade	5	

Note: Sensorimotor, language, visual cortex, hypothalamus, thalamus, internal capsule, brain stem, cerebellar nuclei, cerebellar peduncles or regions directly adjacent to these structures are eloquent brain. Source: Authors' compilation based on data from Spetzler et al. (2015) and Spetzler and Martin (1986).

Size: On non-magnified angiography, the largest nidus diameter measured is related to (and therefore implicitly includes other factors relating to) the hardship of AVM excision, e.g., number of feeders, severity of steal, etc.

Eloquence (eloquent brain): This includes the language, sensorimotor and visual areas; thalamus and hypothalamus; internal capsule; brainstem; deep cerebellar nuclei; cerebellar peduncles.

Venous Drainage: If all venous drainage occurs via the cortical venous system, it is called superficial; if any or all drainage is through deep veins, it is considered deep (e.g., internal cerebral vein (great cerebral vein), basal vein of Rosenthal or pre-central cerebellar vein).

8.11.2. Outcome Based on Spetzler–Martin Grade

Spetzler published a three-tiered management recommendation scheme as follows:

Class A (S–M Grade I & II): surgical excision;

Class B (S–M Grade III): multimodality management;

Class C (S–M Grades IV and V): follow clinically and repeat angiogram every 5 years.

Treatment is only for progressive neurologic deficit, steal-related symptoms or aneurysms identified on surveillance angiograms.

8.12. Treatments

8.12.1. Microsurgical Treatment

Whenever possible, the gold standard in cerebral AVM treatment is microsurgery. When microsurgery is accomplished by skilled vascular neurosurgeons, an angiographic recovery with lower rates of morbidity and mortality (from 1% to 10%) can be accomplished for smaller (nidus 3 cm) AVMs in 94–100% of patients. For larger AVMs located in crucial or eloquent brain regions, these percentages vary greatly. Only 22% and 17% of AVMs of the IV and V grades of the Spetzler and Martin classifications can be cured by angiography, respectively. In cases of microsurgery, permanent neurological impairments or mortality occur in 7.4% of cases (range: 0–40%). In this study, an effective AVM occlusion was accomplished in 96% of instances (range: 0–100%) (Spetzler and Martin 1986). Microsurgery may be used as part of a multimodal management plan that includes endovascular surgery to decrease the nidus volume and treatment or mitigation of future vascular abnormalities. In most cases, vascular neurosurgeons desire selective embolization of deep arterial feeders on the flip side of the surgical field from their neuro-interventional colleagues, ideally using Onyx instead of glue to enhance surgical removal (Peschillo et al. 2014).

In circumstances where an AVM appeared to be inoperable earlier, a combination of surgery plus radiosurgery (Sanchez-Mejia et al. 2009) may be used. First, radiosurgery is used to shrink the AVM. Radiosurgery can also be utilized as a follow-up treatment following microsurgery.

Unprecedented advancements in the armamentarium of vascular neurosurgeons have significantly broadened the therapeutic choices available. The utilization of indocyanine green (ICG) during microsurgery allows for the visualization of residual AVM sections, resulting in better surgical outcomes (Killory et al. 2009). Furthermore, the advent of non-stick bipolar forceps has resulted in a significant technological advancement. The recent groundbreaking 3D implementations in microsurgery permit the real-time merging of MRA or MRI images with the microsurgical operation field, resulting in an additive role with other imaging devices such as navigation, DTI and tractography, as well as fMRI to further accelerate progress toward an upgraded understanding of the brain AVM anatomy and, as a result, superior microsurgical results (Peschillo et al. 2014).

8.12.2. Endovascular Therapy

Although the ultimate target of cerebral AVM treatment is the total occlusion of its nidus, this is not achievable or feasible all the time. An endovascular treatment (Figure 13) may be utilized in the following scenarios:

1. Embolization may be accomplished either before to surgery or radiosurgery;
2. To manage vascular anomalies with an AVM;
3. As a curative treatment;
4. In a palliation target (i.e., mitigation of blood circulation in steal symptoms) (Fiorella et al. 2006).

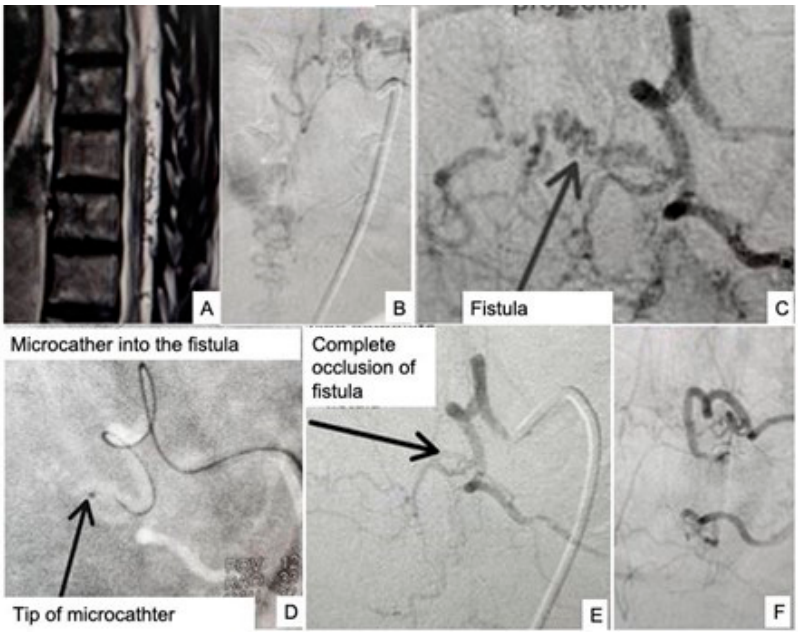

Figure 13. Endovascular closure of a dorsal spinal AVF. (**A**) MRI T2W image in sagittal view showing a suspected dorsal AVM. (**B**,**C**) Spinal DSA showing an AVF supplied by the left 8th intercostal artery; (**D**) road map microcatheter tip at the site of the fistula; (**E**) occlusion of the fistula; (**F**) DSA after occlusion of an AVF. Source: Figure by authors.

Embolic agents come in a variety of forms. n-Butyl cyanoacrylate (n-BCA), PVA/ Embospheres, ETOH, coils and, recently, Onyx (a biocompatible polymer of ethyl vinyl alcohol copolymer dissolved in an organic solvent (dimethyl sulfoxide) that promotes substantial AVM volume shrinkage and, in some cases, anatomic and angiographic cure) are all commonly used agents (Peschillo and Delfini 2012). The invention of microcatheters with detachable tips completely changed the techniques employed to inject embolic agents, since these microcatheters are significantly less prone to risks (due to microcatheter entrapment), resulting in improved endovascular therapy efficacy (Peschillo et al. 2014).

Despite the fact that the intra-arterial route is the most common, a transvenous technique has been developed in recent years with promising results (Consoli et al. 2013; Kessler et al. 2011). Due to the potential hemorrhagic consequences from any manipulation of the venous portion of a brain AVM while its core and nidus are still patent, endovascular neurosurgeons are frequently hesitant to employ the venous route. Only in

exceptional circumstances should the transvenous technique be used (such as when it is impossible to negotiate the microcatheter through tortuous and small arteries to reach in the AVM nidus, in the case of high-flow venous-side aneurysm obliteration or when surgery/radiosurgery is not practical). The progressive and well-controlled deposition of Onyx within the draining vein, as well as the transvenous fast nidal closure with the backward filling of all its feeders can prevent hemorrhage (Massoud 2013).

In general, satisfactory Onyx injection results are achieved by first forming a plug in the vascular lumen just prior to the microcatheter tip and thereafter injecting. Chapot's "pressure-cooker" approach involves trapping the detachable component of an Onyx-compatible microcatheter with glue and coils to achieve wedge-flow situations, allowing for a better knowledge of macrofistulous cerebral AVMs and more thorough, forceful and well-controlled Onyx embolization (Chapot et al. 2014). In the same way, double-lumen balloons could help with endovascular therapy of brain AVMs (inflating a balloon at the Onyx injection site could eliminate the necessity for a plug and its hazards). However, the main issue with this technique is the difficulty in negotiating the dual-lumen balloon into distal arterial feeders, particularly small ones, as these vessels can often only be accessed with small microcatheters, or flow-directed microcatheters and overpenetration of the nidus, which has negative hemodynamic implications, particularly with venous penetration (Jagadeesan et al. 2013).

After the embolization of an AVM, 6.6% (range, 0–28%) experience permanent neurologic impairments or death. While embolization is successful, cerebral AVM obliteration is only achieved in 13% of cases (range: 0–94%) (Spetzler and Martin 1986).

8.12.3. Radiosurgery

In some situations of high-grade AVMs that are considered inoperable or have a significant risk of severe or even fatal consequences if managed with microsurgery or endovascular surgery, radiosurgery may be successful. The delay of postoperative effects and iatrogenic morbidity are two major drawbacks of radiosurgery. The average wait period for the results is two years (can be up to four years). Individuals with an AVM are regrettably exposed to hemorrhage dangers analogous to non-operated patients during the latency period. The second constraint is that radiation may harm structures close to the radiosurgical target volume, resulting in iatrogenic morbidity.

In 50–90% of cases, total obliteration is documented, and the incidence is inversely proportionate to the size of the AVM nidus in the brain. Even if a patient is thought to be cured after radiosurgery, a hemorrhagic episode can occur in less than 1% of cases (Peschillo et al. 2014). In 5.1% of instances (range, 0–21%), permanent neurological impairments or death occur (Spetzler and Martin 1986).

For achieving the greatest outcomes for AVMs, a multidisciplinary, case-by-case approaching technique should be used. Anatomical and biological individual-case unique aspects and natural history with a concentration on clinical symptoms should be addressed and kept in mind while developing treatment for brain AVMs on a case-by-case approach. A team of neuro-endovascular interventionists, vascular neurosurgeons and radiosurgery specialists should review all patients.

9. Spinal Vascular Malformation (SVM)

Spinal arteriovenous malformations (SVMs) are rarer pathologies to come across, with devastating consequences if left untreated. The improvement in and invention of endovascular embolization for these lesions has resulted from a growing understanding of the angioarchitecture and pathophysiology of SVMs. In the context of a multimodal management strategy, additional advancements in imaging, interventional and surgical procedures enable neurosurgeons to address these lesions more successfully and efficiently (Patsalides et al. 2011).

9.1. Classification

Early and initial classifications were absolutely descriptive and founded on histology, neglecting the essential realization of the pathophysiology of these different pathologies. There are two types of AVMs depending on hemodynamic attributes: 1) spinal AVFs with a direct shunt between the artery and vein, and 2) AVMs with a nidus (a network of aberrant arteries) between the artery and vein (Krings et al. 2005). Capillary telangiectasias and cavernous hemangiomas are vascular abnormalities that require surgery and cannot be treated with endovascular techniques (Barnwell et al. 1990).

9.1.1. Topographic Classification of SVMs

A. AVM

1. Intramedullary (also known as type II or glomus-type AVM);
2. Pial;
3. Epidural;
4. Intra- and extramedullary (also called type III, juvenile AVM, intradural– extradural or metameric AVM).

B. AVF

1. Pial AVF (also called type IV, ventral intradural AVF, spinal cord AVF or perimedullary AVF):

   a. Giant;
   b. Large;
   c. Small.

2. Dural AVF (also called dorsal intradural or type I AVF);
3. Epidural AVF (also called extradural AVF) (Patsalides et al. 2011).

9.2. Pathophysiology

Myelopathy (motor and sensory deficits, bowel and bladder dysfunction), back pain, radicular discomfort or deficit, or spinal column deformities can all be caused by spinal arteriovenous lesions. Bleeding, arterial steal, venous hypertension and mass effect are all potential causes of spinal cord injury, and their significance varies depending on the pathology. Acute neurologic impairments can result from a spinal cord parenchymal hemorrhage and/or spinal subarachnoid hemorrhage. In spinal AVMs, the risk of hemorrhage is higher. Hemorrhage can occur in giant and big spinal AVFs, cervical DAVFs and intracranial DAVFs with perimedullary venous drainage, although thoracic and lumbar DAVFs and small spinal cord AVFs are linked to less hemorrhage (Rosenblum et al. 1987; Mourier et al. 1993). Spinal artery aneurysms and intranidal aneurysms have a higher risk of rupture (Biondi et al. 1992).

Seldom, spinal AVMs along with intracranial venous drainage may have posterior fossa bleeding (Di Chiro and Doppman 1970). With arteriovenous lesions and perimedullary venous outflow, venous hypertension is typically severe.

A spinal DAVF is the most common etiology of venous hypertension, although it can also be caused by any lesion with perimedullary venous drainage like pial spinal AVFs or numerous intracranial dural AVFs. As a result of a lack of valves, the pressure in the perimedullary veins is unusually high, which is transferred to the intrinsic veins of the spinal cord, resulting in "arterialization" with tortuous and thickened walls. The reduced intramedullary arteriovenous pressure difference leads to reduced tissue perfusion, as well as spinal cord hypoxia (Hurst et al. 1995). The disruption and malfunction of the blood–cord barrier come from the lack of autoregulation of the intrinsic cord vessels, resulting in cord edema (Jellema et al. 2006). In the upright position, the conus is the lowest section of the spinal cord; therefore, venous hypertension predominates here, abated by a valveless venous system. Because the pressure in the draining veins differs from arterial pressure, exercise causes increased discomfort. Venous hypertension can be established with a DSA of the Adamkiewicz artery, which shows the prolonged severe venous phase (Merland et al. 1980). Pathologies involving high-flow arteriovenous shunts might cause arterial blood to be stolen from a normal spinal cord section nearby (Djindjian et al. 1978). Due to the poor possibility for collateral arterial supply to normal spinal cord tissue, lesions in the dorsal side of the spinal cord fed by the ASA are also vulnerable to arterial steal. Myelopathy caused by mass impact is a fairly uncommon occurrence. Large aneurysms (el Mahdi et al. 1989) and dilated veins/varices, like those found in huge spinal cord AVFs, can apply pressure on the spinal cord and nerve roots.

9.3. Clinical Manifestation

9.3.1. Spinal Cord AVMs

AVMs in the spinal cord comprise 20–30% of all SVMs (Krings et al. 2005). They are high-flow lesions with a distinct nidus that are fed by one branch of the ASA and/or PSA. They have a localized pattern of arteriovenous shunts which drain into the spinal veins, similar to brain AVMs. Aneurysms of the supplying arteries and the nidus

are common complications (Biondi et al. 1992). AVMs in the spinal cord are evenly distributed along the long axis of the spinal cord and can possess a more complicated architecture, with both extramedullary and intramedullary components that disregard tissue planes. The conus medullaris AVM (Spetzler et al. 2002) is a separate kind that may extend along the filum terminale and is found on the cauda equina or conus medullaris. AVMs of the spinal cord usually appear in infancy or early adulthood, with clinical symptoms appearing suddenly due to bleeding or compression-related myelopathy. Patients may experience sensory and/or motor deficiencies, as well as bowel and bladder problems and pain. Following the initial incident, most patients have a partial improvement, but further occurrences are almost certain to occur, resulting in progressive cord function degradation. Venous hypertension and arterial steal are two possible sequences that might lead to progressive myelopathy; however, they are uncommon. AVMs of the conus medullaris commonly cause myelopathy and radiculopathy at the same time (Spetzler et al. 2002).

9.3.2. Pial AVFs

Pial AVFs (Figure 13) have a few or a single intradural direct arteriovenous shunts sans an intervening nidus as a hallmark. It is a condition of the cord's pial surface. The ASA or PSA supplies the feeder/s (one or more), and the shunt/s drain into the spinal cord veins. Based on the size and flow of the direct shunt, pial AVFs are categorized into three types: tiny (Type 1), large (Type 2) and giant (Type 3) (Gueguen et al. 1987).

AVFs of type 1 (small) have a low flow shunt between a branch of the ASA and a slightly dilated spinal vein. These are frequently seen on the filum terminale or the anterior surface of the conus medullaris. DAVFs and small AVFs on the conus medullaris are frequently mistaken. There may be a single or several shunts in Type 2 or large AVFs, with increased flow and ampullary dilatation of the draining vein. They are fed by one or more modestly dilated feeders from the PSA and normally lie on the posterolateral surface of the conus medullaris. Many arterial feeders converge on one or a few shunts in large AVFs. Type 3 AVFs have one or more high-flow shunts, as well as one or more dilated arterial supplies from the ASA and PSA. The arterial suppliers converge to form a single shunt that drains into dilated arterialized draining veins. Giant AVFs have a stronger affinity for the conus medullaris area and can be found in complex vascular malformation syndromes, although they may also be detected in other conditions (Nakstad et al. 1993).

AVFs of Type 2 and 3 are more common in childhood and adolescence. An SAH can cause symptoms to appear suddenly, although venous hypertension, vascular steal or pressure on the spinal cord and/or nerve roots are more likely to cause progressive sensory and motor degeneration, including sphincter dysfunction. SAHs are caused by venous rupture (Ricolfi et al. 1997).

Type 1 AVFs appear later in life, with progressive neuro-deficiencies caused by venous hypertension, whereas SAHs occur infrequently. Hematomyelia can occur when the anterior spinal vein, that is located subpially, ruptures (Rodesch et al. 2004). A spinal DSA is required for all of these illnesses in order to clinch the diagnosis, describe the shunt and choose the treatment strategy.

9.3.3. Epidural AVF

This kind of AVF is more uncommon and carries a higher risk of morbidity. The shunt is located between an artery and the epidural venous plexus. Presenting symptoms are mainly due to the mass effect and subsequent hematoma; venous hypertension or steal syndrome are uncommon unless the shunt drains into the spinal cord vein/s (Arnaud et al. 1994; Clarke et al. 2009; Weingrad et al. 1979; Kawabori et al. 2009; Willinsky et al. 1990). It is most common in the cervical region. Surgery or embolization are the two therapeutic choices, with the latter being preferred. Liquid embolic agents should be used to occlude the shunt and the proximal draining vein. It is also possible to use a transvenous route. When the spinal cord is not draining via the vein to be embolized, a transvenous route can be used to finish the embolization (Szajner et al. 1999; Willinsky et al. 1993).

9.3.4. Intramedullary–Extramedullary Spinal AVM

This is also called an intradural–intradural spinal AVM, and is the rarest form. This complex spinal AVM evolves along a discrete embryonic somatic level and involves more than one structure of spinal dura, cord, vertebra, paravertebral soft tissue or skin. Total somatic involvement occurs in Cobb syndrome. Incomplete somatic involvement is more common and may be associated with diffuse angiomatosis (e.g., Rendu–Osler–Weber

disease). They typically present in childhood or in young adults, like manifestations of other spinal AVMs. Numerous arterial supplies from several vertebral levels are prevalent (Patsalides et al. 2011).

9.3.5. Intracranial DAVF with Cervical Perimedullary Venous Drainage

In the Djindjian–Merland classification, this is categorized as Type V (Houdart et al. 1993), where it represents a special type of intracranial lesion that manifests with spinal cord manifestations. It receives blood flow from the meningeal branches of the ECA, ICA and VA, and drains into veins around the brainstem and upper cervical spinal cord. It generally presents between the third and seventh decades of life due to venous hypertension (Ricolfi et al. 1999). These lesions demand the need for catheter angiographic evaluation from the cranium to the sacrum.

9.4. Spinal DSA and ITS Techniques

Despite major advancements in non-invasive spine vascular imaging, the spinal DSA is the gold standard test for diagnosing and classifying SVMs. For patient comfort and needed apnea during thoracic spine examination, a general anesthetic is preferred. The 5F sheath is inserted through the femoral route into the common femoral artery. Power injection with a pig-tail high-flow catheter put in the descending thoracic aorta at the mid-dorsal level can be used to obtain an aortogram. Each dorsal and lumbar artery should be examined on both sides. For cervical spinal DSA, the vertebral, deep cervical arteries and ascending cervical arteries must be canulated and examined. For lumbo-sacral spinal locations, internal iliac and iliolumbar arteries should be investigated. The examination of venous drainage should be included in spinal angiography, especially following injection in the Adamkiewicz artery. Venous drainage is protracted or nonexistent in severe venous hypertension and myelopathy involving the dorso-lumbar spine. The cause (typically a DAVF) must be present if venous hypertension is discovered. After therapy, an elevated venous outflow is a positive predictive sign (Gobin et al. 1992).

9.5. Endovascular Therapy for SVMs

The hemodynamics of SVMs, their position in the longitudinal and axial planes, and the angioarchitecture of the pathology all have a role in the management of spinal vascular lesions. For many arteriovenous malformations, endovascular treatment may be the preferred treatment. Nonetheless, microsurgery plays an important role, and a comprehensive therapeutic approach is required. One of the most crucial factors to examine before any neuro-intervention is the patient's preoperative neurologic condition. Because postoperative functional success is strongly linked to the preoperative neuro-status, maximum functional restoration can be attained by treating patients early before serious neuro-deterioration sets in. In patients with severe neuro-deficiencies, partial results may still be attainable (Patsalides et al. 2011).

9.5.1. General Principles for Embolization

To reduce the chance of a vascular attack on the spinal cord, the vascular anatomy must be clearly defined prior to operation. PSAs have many more arterial anastomoses than ASAs, implying that sufficient collateral flow is more likely following an occlusion of the PSA rather than the ASA. As a result, an occlusion of the posterior radiculomedullary artery supplying, the PSA may occur without any clinical effects. The obstruction of an anterior radiculomedullary artery that supplies the ASA, on the other hand, is linked to a substantial risk of spinal cord infarction/ischemia (Patsalides et al. 2011).

It refers to the levels below and above for anastomoses that would supply the ASA region prior to embolization. When embolizing a vascular abnormality with arterial steal, extra caution is required because partial obstruction of the feeding artery would result in diminished arterial steal, as well as the possible emergence of normal spinal arteries; unintentional embolization of these arteries should be avoided (Patsalides et al. 2011).

The rich pial perimedullary anastomoses joining the ASA and PSA are also a matter for worry, as they may result in the unintentional embolization of the ASA while embolizing a posterior radiculomedullary artery. This embolization in wedge flow can alter normal flow patterns, allowing for the embolic agent to reach arteries that were not visible on the pre-embolization DSA. In an AVM, obstruction of the venous drainage of a big arteriovenous shunt can cause nidus rupture and rupture, while in an AVF, it can produce increased venous hypertension. Last but not least, an occlusion of a feeding artery to an arteriovenous shunt too close to the shunt is usually ineffectual since alternative arterial connections may be recruited and expanded to supply the shunt;

however, because more blood is diverted to the shunt, there may be greater arterial theft. At the same time, access to the shunt for additional embolization is limited (Patsalides et al. 2011).

9.5.2. Malformation-Specific Therapy

Intramedullary AVM

Intramedullary AVMs should be managed to change the course of the disease and lower the risk of bleeding. The outcome of untreated spinal AVMs is bleak (Aminoff and Logue 1974; Hurth et al. 1978). Microsurgery, embolization or a combination of the two is currently a possible treatment. When a long endovascular approach or an unstable catheter location make catheter access problematic, surgery is the only choice. A lesion that is superficially placed in the back is more appealing for a safe resection. Surgical treatment of anteriorly placed lesions is still possible, particularly in the cervical area, where collateral circulation to the spinal cord might develop caudally. Filum terminale AVMs can also be treated surgically.

Embolization can be used as a main treatment or as a supplement for microsurgery in the treatment of intramedullary spinal AVMs (Djindjian et al. 1973; Doppman et al. 1968). Modern microcatheters allow for the selective catheterization of the anterior and posterior spinal arteries supplying the AVM; therefore, embolization with a liquid or particle embolic agent is possible. These AVMs do not use coils as they need relatively rigid microcatheters that are dangerous to maneuver. As a result, coils can only be utilized for proximal embolization, which might cause collateral circulation to the nidus to develop, preventing future safe interventional access.

The advantages of particle embolic agents include sequential embolization and the capability to monitor the result clinically as well as angiographically during the intervention. However, recanalization has a long-term negative impact. Particle embolization necessitates angiographic monitoring every year, as well as further embolization in the event of recanalization.

Multiple embolizations may be required as the AVM recanalization rate is as high as 80% (Biondi et al. 1990). Essentially, particle embolization is a palliative procedure that affects the natural history, and it may produce a positive clinical result rather than a definitive solution.

Liquid embolic agents have the advantage of providing a more lasting occlusion that is less likely to recanalize, but they also carry the danger of unintentional embolization of normal perforating arteries which are not shown on DSA. When possible, a liquid embolic agent should be utilized, particularly if embolization is the primary or exclusive treatment. The liquid embolic agent should be injected into the nidus or as close to it as possible. The use of n-BCA, (Rodesch et al. 2003) for spinal AVF embolizations had the satisfactory clinical outcome of up to 83%. Thirteen percent of individuals get permanent morbidity as a result of embolization. A severe deficit occurs in cases where embolization is performed through the ASA. Nowadays, Onyx (ev3, Irvine, California) is also utilized to manage spinal cord AVMs.

Embolization for Intramedullary AVMs

Embolization of an intramedullary AVM is generally accomplished while the patient is sedated and receiving systemic heparin. A microcatheter is inserted into the radiculomedullary branch that nourishes the AVM, and a 5F guide catheter is implanted at the ostium of the segmental artery. In most cases, flow-directed microcatheters are preferable to tougher braided microcatheters. The microcatheter should be placed inside or as near to the nidus as possible for lesions fed by the ASA, particularly in a sulcal artery beyond the ASA's longitudinal axis. This reduces the risk of embolization of a normal ASA branch by accident. Although the implications of unintentionally occluding typical PSA branches are not as severe, similar concepts apply to lesions fed by a feeder from the PSA. Because of the lower procedure-related risk, AVMs fed by both ASA and PSA feeders should be treated initially. Embolization is commonly carried out with a 1:2 to 1:3 mixture of n-BCA and ethiodized oil, with tantalum powder added to boost the embolic material's radiodensity. A larger concentration of n-BCA can be employed for high-flow shunts. To avoid recurrent hemorrhage, it is critical to embolize false and flow-related aneurysms (Konan et al. 1999). For the next 24–48 h, the patient should be observed in a neurologic critical care unit. To preclude thrombosis of the normal spinal arteries, systemic heparin is maintained for 24 h (low-dose regimen with a target-activated partial thromboplastin time of 50 to 60 s) (Patsalides et al. 2011).

Intramedullary–Extramedullary AVM and Complex Angiomatosis

Due to the complex architecture and the real fact of the intermingling of the cord parenchyma in an actual AVM, the treatment of these lesions is very difficult. Although there is no known optimal treatment for these lesions, microsurgery and embolization may be carried out alone or in combination. In reality, treatment should be palliative, with the goal of alleviating symptoms produced by a hematoma, arterial steal, venous hypertension or direct mass effect. The closure of a feeding artery (Mourier et al. 1993) and decompressive laminectomy are two simple procedures (Biondi et al. 1992; Di Chiro and Doppman 1970; Hurst et al. 1995). When utilized preoperatively, embolization can be utilized with particles.

Pial AVFs

Pial AVFs are a diverse set of vascular pathologies, with treatment options based mostly on the angioarchitecture of the lesion. Surgical or endovascular treatment should be used as soon as possible. Embolization can also be used in conjunction with microsurgery (Hida et al. 1999). The only approach to prevent long-term recurrence is to obliterate the pial fistula completely and permanently. Liquid embolic agents are the favored materials for embolization, while PVA particles should only be utilized for pre-microsurgical embolization. As the feeder is a distal branch of a thin ASA, super-selective catheterization and retaining the microcatheter in the fistula for efficient embolization is problematic in small pial fistulas (Riche et al. 1983). Because there are several feeding arteries, some of which are perimedullary or transmedullary branches that cannot be catheterized safely, embolization alone is rarely helpful for larger AVFs. Embolization has been documented in a few occurrences, with some cases being partially embolized and recurrence (Oran et al. 2005; Cho et al. 2005). Microsurgical management, on the other hand, is curative, particularly in lesions of the dorsal/dorsolateral cord. The purpose of the surgery is to cut off the junction between the venous and arterial systems while leaving the ASA branches intact. Because of their bigger diameter and rapid flow through the shunt, super-selective arterial feeder catheterization is more practicable in huge fistulas. The enlarged draining veins raise the chances of an intraoperative rupture, making surgical therapy more difficult. As a result, embolization is the preferred treatment for these kinds of lesions. The most difficult aspect of embolization is getting the embolization agent into the right spot without causing venous migration (Mourier et al. 1993; Ricolfi et al. 1997).

As the particles can pass past the shunt and into the venous circulation, causing thrombosis or pulmonary embolism, they should not be used alone. Instead, coils should be utilized alone or in combination with liquid embolic materials. Coils can be inserted in huge AVFs to act as a template for the liquid embolic agent, preventing it from passing through the shunt and onto the venous side. This approach necessitates the utilization of a microcatheter with a big enough inner diameter to facilitate coil deployment. For recurrence prevention, the fistula and proximal draining veins should be embolized. Transient worsening of symptoms may be due to progressive backward thrombosis of the draining veins. Systemic heparin can prevent this for 24–48 h after embolization (Patsalides et al. 2011).

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References

Al-Shahi, Rustam, J. S. Y. Fang, S. C. Lewis, and Charles P. Warlow. 2002. Prevalence of adults with brain arteriovenousmalformations: A community-based study in Scotlandusing capture-recapture analysis. *Journal of Neurology, Neurosurgery & Psychiatry* 73: 547–51.

Aminoff, M. J., and Valentine Logue. 1974. The prognosis of patients with spinal vascular malformations. *Brain* 97: 211–18. [CrossRef] [PubMed]

Arnaud, O., F. Bille, J. Pouget, G. Serratrice, and G. Salamon. 1994. Epidural arteriovenous fistula with perimedullary venous drainage: Case report. *Neuroradiology* 36: 490–91. [CrossRef]

Arnautovic, Kenan I., and Ali F. Krisht. 2000. Transverse-Sigmoid Sinus Dural Arteriovenous Malformations. *Contemporary Neurosurgery* 21: 1–6. [CrossRef]

Ashour, Ramsey, Mohammad Ali Aziz-Sultan, Maryam Soltanolkotabi, Samantha E. Schoeneman, Tord D. Alden, Michael C. Hurley, Arthur J. Dipatri, Tadanori Tomita, Mohamed Samy Elhammady, and Ali Shaibani. 2012. Safety an decacy of onyx embolization for pediatric cranial and spinal vascular lesions and tumors. *Neurosurgery* 71: 773–84. [CrossRef]

Baharvahdat, Humain, Yinn Cher Ooi, Wi Jin Kim, Ashkan Mowla, Alexander L. Coon, and Geoffrey P. Colby. 2020. Updates in the management of cranial dural arteriovenous fistula. *Stroke and Vascular Neurology* 5: 50–58. [CrossRef]

Barnwell, Stanley L., Christopher F. Dowd, Richard L. Davis, Michael S. B. Edwards, Philip H. Gutin, and Charles B. Wilson. 1990. Cryptic vascular malformations of the spinal cord: Diagnosis by magnetic resonance imaging and outcome of surgery. *Journal of Neurosurgery* 72: 403–7. [CrossRef]

Barrow, Daniel L., Robert H. Spector, Ira F. Braun, Jeffrey A. Landman, Suzie C. Tindall, and George T. Tindall. 1985. Classification and treatment of spontaneous carotid-cavernous sinus fistulas. *Journal of Neurosurgery* 62: 248–56. [CrossRef]

Becske, Tibor, David F. Kallmes, Isil Saatci, Cameron G. McDougall, István Szikora, Giuseppe Lanzino, Christopher J. Moran, Henry H.Woo, Demetrius K. Lopes, Aaron L. Berez, and et al. 2013. Pipeline for uncoilable or failed aneurysms: Results from a multicenter clinical trial. *Radiology* 267: 858–68. Available online: http://radiology.rsna.org/ (accessed on 26 February 2013). [CrossRef]

Berge, J., A. Biondi, P. Machi, H. Brunel, L. Pierot, J. Gabrillargues, K. Kadziolka, X. Barreau, V. Dousset, and A. Bonafé. 2012. Flow-diverter silk stent for the treatment of intracranial aneurysms: 1-year follow-up in a multicenter study. *American Journal of Neuroradiology* 33: 1150–55. [CrossRef]

Biondi, Alessandra, J. J. Merland, J. E. Hodes, J. P. Pruvo, and D. Reizine. 1992. Aneurysms of spinal arteries associated with intramedullary arteriovenous malformations. I. Angiographic and clinical aspects. *American Journal of Neuroradiology* 13: 913–22.

Biondi, Alessandra, Jean-Jacques Merland, Daniel Reizine, A. Aymard, J. E. Hodes, P. Lecoz, and A. Rey. 1990. Embolization with particles in thoracic intramedullary arteriovenous malformations: Long-term angiographic and clinical results. *Radiology* 177: 651–58. [CrossRef]

Bodily, K. D., H. J. Cloft, G. Lanzino, D. J. Fiorella, P. M. White, and David F. Kallmes. 2011. Stent-assisted coiling in acutely ruptured intracranial aneurysms: A qualitative, systematic review of the literature. *American Journal of Neuroradiology* 32: 1232–36. [CrossRef] [PubMed]

Bonati, Leo H., Philippe Lyrer, Joerg Ederle, Roland Featherstone, and Martin M. Brown. 2012. Percutaneous transluminal balloon angioplasty and stenting for carotid artery stenosis. *The Cochrane Database of Systematic Reviews* 9: CD000515. [CrossRef] [PubMed]

Byrne, James V., Radu Beltechi, Julia A. Yarnold, Jacqueline Birks, and Mudassar Kamran. 2010. Early experience in the treatment of intra-cranial aneurysms by endovascular flow diversion: A multicentre prospective study. *PLoS ONE* 5: e12492. [CrossRef] [PubMed]

Carlson, Andrew P., Ali Alaraj, Sepideh Amin-Hanjani, Fady T. Charbel, and Victor A. Aletich. 2013. Continued concern about parent vessel steno-occlusive progression with Onyx HD-500 and the utility of quantitative magnetic resonance imaging in serial assessment. *Neurosurgery* 72: 341–52. [CrossRef]

Celesia, Gastone G., Charles M. Strother, Patrick A. Turski, Michael F. Stieghorst, Joseph F. Sackett, and Charles A. Mistretta. 1983. Digital Subtraction Arteriography. A New Method for Evaluation of Extracranial Occlusive Disease. *Archives of Neurology* 40: 70–74. [CrossRef]

Chalouhi, Nohra, Aaron S. Dumont, Stavropoula Tjoumakaris, L. Fernando Gonzalez, Jurij R. Bilyk, Ciro Randazzo, David Hasan, Richard T. Dalyai, Robert Rosenwasser, and Pascal Jabbour. 2012. The superior ophthalmic vein approach for the treatment of carotid-cavernous fistulas: A novel technique using Onyx. *Neurosurgical Focus* 32: E13. [CrossRef] [PubMed]

Chaloupka, John C., and Daniel C. Huddle. 1998. Classification of vascular malformations of the central nervous system. *Neuroimaging clinics of North America* 8: 295–321.

Chapot, René, Paul Stracke, Aglaé Velasco, Hannes Nordmeyer, Markus Heddier, Michael Stauder, Petra Schooss, and Pascal J. Mosimann. 2014. The pressure cooker technique for the treatment of brain AVMs. *Journal of Neuroradiology* 41: 87–91. [CrossRef] [PubMed]

Chimowitz, Marc I., Michael J. Lynn, Colin P. Derdeyn, Tanya N. Turan, David Fiorella, Bethany F. Lane, L. Scott Janis, Helmi L. Lutsep, Stanley L. Barnwell, Michael F. Waters, and et al. 2011. Stenting versus aggressive medical therapy for intracranial arterial stenosis. *New England Journal of Medicine* 365: 993–1003. [CrossRef] [PubMed]

Cho, Keun Tae, Dong Yeob Lee, Chun Kee Chung, Moon Hee Han, and Hyun Jib Kim. 2005. Treatment of spinal cord perimedullary arteriovenous fistula: Embolization versus surgery. *Neurosurgery* 56: 232–41. [CrossRef] [PubMed]

Choi, Dae Seob, Mun Chul Kim, Seon Kyu Lee, Robert A. Willinsky, and Karel G. Terbrugge. 2010. Clinical and angiographic long-term follow-up of completely coiled aneurysms using endovascular technique. *Journal of Neurosurgery* 112: 575–81. [CrossRef] [PubMed]

Clarke, Michelle J., Todd A. Patrick, J. Bradley White, Harry J. Cloft, William E. Krauss, E. P. Lindell, and David G. Piepgras. 2009. Spinal extradural arteriovenous malformations with parenchymal drainage: Venous drainage variability and implications in clinical manifestations. *Neurosurgical Focus* 26: E5. [CrossRef]

Cognard, Christophe, Alain Weill, Laurent Spelle, Michel Piotin, Lina Castaings, Alain Rey, and Jacques Moret. 1999. Long-term angiographic follow-up of 169 intracranial berry aneurysms occluded with detachable coils. *Radiology* 212: 348–56. [CrossRef]

Cognard, Christophe, Laurent Pierot, René Anxionnat, Frédéric Ricolfi, and Clarity Study Group. 2011. Results of embolization used as the first treatment choice in a consecutive nonselected population of ruptured aneurysms: Clinical results of the Clarity GDC study. *Neurosurgery* 69: 837–41. [CrossRef]

Cohen, James, and Ionel Rad. 2004. Contemporary management of carotid blowout. *Current Opinion in Otolaryngology & Head and Neck Surgery* 12: 110–15.

Consoli, A., L. Renieri, S. Nappini, N. Limbucci, and S. Mangiafico. 2013. Endovascular treatment of deep hemorrhagic brain arteriovenous malformations with transvenous onyx embolization. *The American Journal of Neuroradiology* 34: 1805–11. [CrossRef]

Crawford, P. M., C. R. West, D. W. Chadwick, and M. D. Shaw. 1986. Arteriovenous Malformations of the Brain: Natural History in Unoperated Patients. *Journal of Neurology, Neurosurgery & Psychiatry* 49: 1–10.

Cruz, J. P., M. Chow, C. O'Kelly, B. Marotta, J. Spears, W. Montanera, D. Fiorella, and T. Marotta. 2012. Delayed ipsilateral parenchymal hemorrhage following flow diversion for the treatment of anterior circulation aneurysms. *American Journal of Neuroradiology* 33: 603–8. [CrossRef]

Davies, M. A., J. Saleh, K. Ter Brugge, R. Willinsky, and M. C. Wallace. 1997. The natural history and management of in tracranial dural arteriovenous fistulae. Part 1: Benign lesions. *Interventional Neuroradiology* 3: 295–302. [CrossRef] [PubMed]

Derdeyn Colin, P. 2007. Carotid Stenting for Asymptomatic carotid. *Stroke* 38: 715–20. [CrossRef] [PubMed]

Di Chiro, Giovanni, and John L. Doppman. 1970. Endocranial drainage of spinal cord veins. *Radiology* 95: 555–60. [CrossRef] [PubMed]

Ding, Dale, Robert M. Starke, Mark Quigg, Chun-Po Yen, Colin J. Przybylowski, Blair K. Dodson, and Jason P. Sheehan. 2015. Cerebral Arteriovenous Malformations and Epilepsy, Part 1: Predictors of Seizure Presentation. *World Neurosurgery* 84: 645–52. [CrossRef]

Ding, Yong H., Debra A. Lewis, Ram Kadirvel, D. Dai, and David F. Kallmes. 2011. The Wowen EndoBridge: A new occlusion device. *American Journal of Neuroradiology* 32: 607–11. [CrossRef]

Djindjian, M., R. Djindjian, M. Hurth, R. Houdart, and A. Rey. 1978. Steal phenomena in spinal arteriovenous malformations. *Journal of Neuroradiology* 5: 187–201.

Djindjian, R., J. Cophignon, A. Rey, J. Theron, J. J. Merland, and R. Houdart. 1973. Superselective arteriographic embolization by the femoral route in neuroradiology: Study of 50 cases. II. Embolization in vertebromedullary pathology. *Neuroradiology* 6: 132–42.

Doppman, JohnL, GiovanniDi Chiro, and Ayub Ommaya. 1968. Obliteration of spinal-cord arteriovenous malformation by percutaneous embolisation. *Lancet* 1: 477. [CrossRef]

Drake, Charles G. 1979. Cerebral AVMs: Considerations for and Experience with Surgical Treatment in 166 Cases. *Clinical Neurosurgery* 26: 145–208. [CrossRef]

Eberhardt, Olaf, Thomas Naegele, Sabine Raygrotzki, Michael Weller, and Ulrike Ernemann. 2006. Stenting of vertebrobasilar arteries in symptomatic atherosclerotic disease and acute occlusion: Case series and review of the literature. *Journal of Vascular Surgery* 43: 1145–54. [CrossRef] [PubMed]

el Mahdi, Mokhtar A., Mohamed A. Rudwan, Salah M. Khaffaji, and F. A. Jadallah. 1989. A giant spinal aneurysm with cord and root compression. *Journal of Neurology, Neurosurgery & Psychiatry* 52: 532–35.

Ferns, Sandra P., Marieke E. S. Sprengers, Willem Jan van Rooij, Gabriel J. E. Rinkel, Jeroen C. van Rijn, Shandra Bipat, Menno Sluzewski, and Charles B. L. M. Majoie. 2009. Coiling of intracranial aneurysms: A systematic review on initial occlusion and reopening and retreatment rates. *Stroke* 40: e523–29. [CrossRef] [PubMed]

Fiorella, David, Daniel Hsu, Henry H. Woo, Robert W. Tarr, and Peter Kim Nelson. 2010. Very late thrombosis of a pipeline embolization device construct: Case report. *Neurosurgery* 67: onsE313–14; discussion on E314. [CrossRef] [PubMed]

Fiorella, David, Felipe C. Albuquerque, Henry H. Woo, Cameron G. McDougall, and Peter A. Rasmussen. 2006. The role of neuroendovascular therapy for the treatment of brain arteriovenous malformations. *Neurosurgery* 59: S3-163–S3-177. [CrossRef] [PubMed]

Fiorella, David, Henry H. Woo, Felipe C. Albuquerque, and Peter K. Nelson. 2008. Definitive reconstruction of circumferential, fusiform intracranial aneurysms with the pipeline embolization device. *Neurosurgery* 62: 1115–20. [CrossRef] [PubMed]

Fleetwood, Ian G., and Gary K. Steinberg. 2002. Arteriovenous malformations. *Lancet* 359: 863–73. [CrossRef]

Frosting, M. 2003. *Intracranial Vascular Malformation and Aneurysm. From Diagnostic Work-Up to Endovascular Therapy*. Berlin/Heidelberg and New York: Springer.

Gallas, Sophie, Anne Pasco, Jean-Philippe Cottier, Jean Gabrillargues, Jacques Drouineau, Christophe Cognard, and Denis Herbreteau. 2005. A multicenter study of 705 ruptured intracranial aneurysms treated with Guglielmi detachable coils. *American Journal of Neuroradiology* 26: 1723–31.

Geibprasert, S., S. Pongpech, D. Armstrong, and T. Krings. 2009. Dangerous Extracranial– Intracranial Anastomoses and Supply to the Cranial Nerves: Vessels the Neuro- interventionalist Needs to Know. *American Journal of Neuroradiology* 30: 1459–68. [CrossRef]

Gobin, Y. Pierre, Andre Rogopoulos, Armand Aymard, Mazen Khayata, Daniel Reizine, Jacques Chiras, and Jean-Jacques Merland. 1992. Endovascular treatment of intracranial dural arteriovenous fistulas with spinal perimedullary venous drainage. *Journal of Neurosurgery* 77: 718–23.

Graeb, Douglas A., and Clarisse L. Dolman. 1986. Radiological and Pathological Aspects of Dural Arteriovenous Fistulas. *Journal of Neurosurgery* 64: 962–7. [CrossRef] [PubMed]

Gueguen, B., J. J. Merland, M. C. Riche, and A. Rey. 1987. Vascular malformations of the spinal cord: Intrathecal perimedullary arteriovenous fistulas fed by medullary arteries. *Neurology* 37: 969–79.

Guglielmi, Guido, Fernando Vinuela, Ivan Sepetka, and Velio Macellari. 1991a. Electrothrombosis of saccular aneurysms via endovascular approach. Part 1: Electrochemical basis, technique, and experimental results. *Journal of Neurosurgery* 75: 1–7. [CrossRef] [PubMed]

Guglielmi, Guido, Fernando Vinuela, Ivan Sepetka, and Velio Macellari. 1991b. Electrothrombosis of saccular aneurysms via endovascular approach. Part 2: Preliminary clinical experience. *Journal of Neurosurgery* 75: 8–14. [CrossRef]

Gurm, Hitinder S., Jay S. Yadav, Pierre Fayad, Barry T. Katzen, Gregory J. Mishkel, Tanvir K. Bajwa, Gary Ansel, Neil E. Strickman, Hong Wang, Sidney A. Cohen, and et al. 2008. Long-term results of carotid stenting versus endarterectomy in high-risk patients. *New England Journal of Medicine* 358: 1572–79. [CrossRef] [PubMed]

Hartmann, Andreas, Henning Mast, J. P. Mohr, Hans-Christian Koennecke, Andrei Osipov, John Pile-Spellman, D. Hoang Duong, and William L. Young. 1998. Morbidity of intracranial hemorrhage in patients with cerebral arteriovenous malformation. *Stroke* 29: 931–34. [CrossRef] [PubMed]

Henderson, A. D., and N. R. Miller. 2017. Carotid-cavernous fistula: Current concept of aetiology, investigation, management. *Eye* 32: 164–72. [CrossRef] [PubMed]

Hida, Kazutoshi, Yoshinobu Iwasaki, Katsuya Goto, Kazuo Miyasaka, and Hiroshi Abe. 1999. Results of the surgical treatment of perimedullary arteriovenous fistulas with special reference to embolization. *Journal of Neurosurgery* 90: 198–205.

Hope, J. K. Ayton, James V. Byrne, and Andrew J. Molyneux. 1999. Factors influencing successful angiographic occlusion of aneurysms treated by coil occlusion. *American Journal of Neuroradiology* 20: 391–99.

Houdart, E., Y. P. Gobin, A. Casasco, A. Aymard, D. Herbreteau, and J. J. Merland. 1993. A proposed angiographic classification of intracranial arteriovenous fistulae and malformations. *Neuroradiology* 35: 381–85. [CrossRef]

Hurst, R. W., L. C. Kenyon, E. Lavi, E. C. Raps, and P. Marcotte. 1995. Spinal dural arteriovenous fistula: The pathology of venous hypertensive myelopathy. *Neurology* 45: 1309–13. [CrossRef]

Hurth, M., R. Houdart, R. Djindjian, A. Rey, and M. Djindjian. 1978. Arteriovenous malformations of the spinal cord: Clinical, anatomical and therapeutic consideration: A series of 150 cases. *Progress in Neurological Surgery* 9: 238–66.

Intracranial Dural Arteriovenous Fistula. 2021. Available online: https://operativeneurosurgery.com/doku.php?id=intracranial_dural_arteriovenousfistula (accessed on 21 April 2021).

Jagadeesan, Bharathi D., Mikayel Grigoryan, Ameer E. Hassan, Andrew W. Grande, and Ramachandra P. Tummala. 2013. Endovascular balloon-assisted embolization of intracranial and cervical arteriovenous malformations using dual-lumen coaxial balloon microcatheters and onyx. *Neurosurgery* 73: 238–43. [CrossRef] [PubMed]

Jayaraman, M. V., M. L. Marcellus, H. M. Do, S. D. Chang, J. K. Rosen berg, G. K. Stein berg, and M. P. Marks. 2007. Hemorrhage rate in patients with Spetzler-Martin grades IV and V arteriovenous malformations: Is treatment justified? *Stroke* 38: 325–29. [CrossRef]

Jellema, K., C. C. Tijssen, and J. van Gijn. 2006. Spinal dural arteriovenous fistulas: A congestive myelopathy that initially mimics a peripheral nerve disorder. *Brain* 129: 3150–64. [CrossRef]

Johnston, S. Claiborne, Christopher F. Dowd, Randall T. Higashida, Michael T. Lawton, Gary R. Duckwiler, and Daryl R. Gress. 2008. Predictors of rehemorrhage after treatment of ruptured intracranial aneurysms: The Cerebral Aneurysm Rerupture after Treatment (CARAT) study. *Stroke* 39: 120–25. [CrossRef]

Kadziolka, Krzysztof, Catherine Tomas, Georges Robin, and Laurent Pierot. 2013. Combined use of a double-lumen remodeling balloon and a low-profile stent in the treatment of intracranial aneurysms ('remostent' technique): A technical note. *Journal of Neuroradiology* 40: 50–53. [CrossRef] [PubMed]

Kallmes, David F., Yong Hong Ding, Daying Dai, Ramanathan Kadirvel, Debra A. Lewis, and Harry J. Cloft. 2007. A new endoluminal, flow-disrupting device for treatment of saccular aneurysms. *Stroke* 38: 2346–52. [CrossRef]

Kan, Peter, Adnan H. Siddiqui, Erol Veznedaroglu, Kenneth M. Liebman, Mandy J. Binning, Travis M. Dumont, Christopher S. Ogilvy, J. R. Gaughen, Jr., J. Mocco, G. J. Velat, and et al. 2012. Early post-market results after treatment of intracranial aneurysms with the Pipeline Embolization device: A US multicenter experience. *Neurosurgery* 71: 1080–87. [CrossRef] [PubMed]

Kawabori, Masahito, Kazutoshi Hida, Shunsuke Yano, Takeshi Asano, and Yoshinobu Iwasaki. 2009. Cervical epidural arteriovenous fistula with radiculopathy mimicking cervical spondylosis. *Neurologia Medico-Chirurgica* 49: 108–13. [CrossRef]

Kessler, Iruena, Roberto Riva, Maria Ruggiero, Monica Manisor, Maher Al-Khawaldeh, and Charbel Mounayer. 2011. Successful transvenous embolization of brain arteriovenous malformations using onyx in five consecutive patients. *Neurosurgery* 69: 184–93. [CrossRef]

Killory, Brendan D., Peter Nakaji, L. Fernando Gonzales, Francisco A. Ponce, Scott D. Wait, and Robert F. Spetzler. 2009. Prospective evaluation of surgical microscope-integrated intraoperative near-infrared indocyanine green angiography during cerebral arteriovenous malformation surgery. *Neurosurgery* 65: 456–62. [CrossRef] [PubMed]

Konan, Alexis Victorien, Jean Raymond, and Daniel Roy. 1999. Transarterial embolization of aneurysms associated with spinal cord arteriovenous malformations: Report of four cases. *Journal of Neurosurgery* 90: 148–54. [CrossRef] [PubMed]

Kondziolka, Douglas, Mark R. McLaughlin, and John RW Kestle. 1995. Simple Risk Predictions for Arteriovenous Malformation Hemorrhage. *Neurosurgery* 37: 851–55. [CrossRef] [PubMed]

Krings, Timo, Michael Mull, Joachim M. Gilsbach, and Armin Thron. 2005. Spinal vascular malformations. *European Radiology* 15: 267–78. [CrossRef]

Kulcsár, Zsolt, and István Szikora. 2012. The ESMINT Retrospective Analysis of Delayed Aneurysm Ruptures after flow diversion (RADAR) study. *EJMINT* 2012: 1244000088. Available online: http://www.ejmint.org/ (accessed on 26 February 2010).

Kulcsár, Zsolt, Stephan G. Wetzel, Luca Augsburger, Andreas Gruber, Isabel Wanke, and Daniel Andre Rüfenacht. 2010. Effect of flow diversion treatment on very small ruptured aneurysms. *Neurosurgery* 67: 789–93. [CrossRef]

Kupersmith, M. J., M. E. Vargas, A. Yashar, M. Madrid, K. Nelson, A. Seton, and A. Berenstein. 1996. Occipital arteriovenous malformations: Visual disturbances and presentation. *Neurology* 46: 953–57. [CrossRef]

Lanzino, G., A. K. Wakhloo, R. D. Fessler, M. L. Hartney, L.R. Guterman, and L. N. Hopkins. 1998. Efficacy and current limitations of intravascular stents for intracranial internal carotid, vertebral, and basilar artery aneurysms. *Journal of neurosurgery* 91: 538–46. [CrossRef]

Lee, S. J., Y. D. Cho, H.-S. Kang, J. E. Kim, and M. H. Han. 2013. Coil embolization using the self-expandable closed-cell stent for intracranial saccular aneurysm: A single-center experience of 289 consecutive aneurysms. *Clinical Radiology* 68: 256–63. [CrossRef]

Lowis, George W., and Alireza Minagar. 2003. The neglected research of Egas Moniz of internal carotid artery (ICA) occlusion. *Journal of the History of the Neurosciences* 12: 286–91. [CrossRef]

Lubicz, Boris, Laurent Collignon, Gaï Raphaeli, Jean-Pierre Pruvo, Michael Bruneau, Olivier De Witte, and Xavier Leclerc. 2010. Flow-diverter stent for the endovascular treatment of intracranial aneurysms: A prospective study in 29 patients with 34 aneurysms. *Stroke* 41: 2247–53. [CrossRef]

Lutsep, Helmi L., Michael J. Lynn, George A. Cotsonis, Colin P. Derdeyn, Tanya N. Turan, David Fiorella, L. Scott Janis, Bethany F. Lane, Jean Montgomery, Marc I. Chimowitz, and et al. 2015. Does the Stenting Versus Aggressive Medical Therapy Trial Support Stenting for Subgroups With Intracranial Stenosis? *Stroke* 46: 3282–84. [CrossRef]

Lv, X., Y. Li, C. Jiang, X. Yang, and Z. Wu. 2010. Brain arteriovenous malformations and endovascular treatment: Effect on seizures. *Interventional Neuroradiology* 16: 39–45. [CrossRef] [PubMed]

Lylyk, Pedro, Carlos Miranda, Rosana Ceratto, Angel Ferrario, Esteban Scrivano, Hugh Ramirez Luna, Aaron L. Berez, Quang Tran, Peter K. Nelson, and David Fiorella. 2009. Curative endovascular reconstruction of cerebral aneurysms with the pipeline embolization device: The Buenos Aires experience. *Neurosurgery* 64: 632–42. [CrossRef] [PubMed]

Massoud, Tarik F. 2013. Transvenous retrograde nidus sclerotherapy under controlled hypotension (TRENSH): Hemodynamic analysis and concept validation in a pig arteriovenous malformation model. *Neurosurgery* 73: 332–42. [CrossRef] [PubMed]

Mast, Henning, J. P. Mohr, Andrei Osipov, John Pile-Spellman, Randolph S. Marshall, Ronald M. Lazar, Bennett M. Stein, and William L. Young. 1995. 'Steal' Is an Unestablished Mechanism for the Clinical Presentation of Cerebral Arteriovenous Malformations. *Stroke* 26: 1215–20. [CrossRef] [PubMed]

Maurice-Williams, R. S., and J. Lafuente. 2003. Intracranial aneurysm surgery and its future. *Journal of the Royal Society of Medicine* 96: 540–43. [CrossRef]

Mericle, Robert A., Giuseppe Lanzino, Ajay K. Wakhloo, Lee R. Guterman, and L. Nelson Hopkins. 1998. Stenting and secondary coiling of internal carotid artery aneurysm: Technical case report. *Neurosurgery* 43: 1229–34. [CrossRef]

Merland, J. J., M. C. Riche, and J. Chiras. 1980. Intraspinal extramedullary arteriovenous fistulae draining into the medullary veins. *Journal of Neuroradiology* 7: 271–320.

Molyneux, Andrew J., Saruhan Cekirge, Isil Saatci, and Gyula Gál. 2004. Cerebral Aneurysm Multicenter European Onyx (CAMEO) trial: Results of a prospective observational study in 20 European centers. *American Journal of Neuroradiology* 25: 39–51.

Moret, J., C. Cognard, A. Weill, L. Castaings, and A. Rey. 1997. The "remodelling technique" in the treatment of wide neck intracranial aneurysms. *Interventional Neuroradiology* 3: 21–35. [CrossRef] [PubMed]

Morris, Pearse. 2007. *Practical Neuroangiography*. Philadelphia: Lippincott, pp. 498–99.

Mourier, K. L., Y. P. Gobin, B. George, G. Lot, and J. J. Merland. 1993. Intradural perimedullary arteriovenous fistulae: Results of surgical and endovascular treatment in a series of 35 cases. *Neurosurgery* 32: 885–91. [CrossRef] [PubMed]

Mullan, Sean, Saeid Mojtahedi, Douglas L. Johnson, and R. Loch Macdonald. 1996. Embryological basis of some aspectsof cerebral vascular fistulas and malformations. *Journal of Neurosurgery* 85: 1–8. [CrossRef] [PubMed]

Nakstad, P. H., J. K. Hald, and S. J. Bakke. 1993. Multiple spinal arteriovenous fistulas in Klippel-Trenaunay-Weber syndrome treated with platinum fibre coils. *Neuroradiology* 35: 163–65. [CrossRef]

Nieuwkamp, Dennis J., Larissa E. Setz, Ale Algra, Francisca H. H. Linn, Nicolien K. de Rooij, and Gabriël J. E. Rinkel. 2009. Changes in case fatality of aneurysmal subarachnoid haemorrhage over time, according to age, sex, and region: A meta-analysis. *The Lancet Neurology* 8: 635–42. [CrossRef] [PubMed]

O'Kelly, C. J., J. Spears, M. Chow, J. Wong, M. Boulton, A. Weill, R. A. Willinsky, M. Kelly, and T. R. Marotta. 2013. Canadian experience with the pipeline embolization device for repair of unruptured intracranial aneurysms. *American Journal of Neuroradiology* 34: 381–87. [CrossRef] [PubMed]

Oran, Ismail, Mustafa Parildar, and Abdurrahim Derbent. 2005. Treatment of slow-flow (type I) perimedullary spinal arteriovenous fistulas with special reference to embolization. *American Journal of Neuroradiology* 26: 2582–86.

Ortiz, Rafael, Michael Stefanski, Robert Rosenwasser, and Erol Veznedaroglu. 2008. Cigarette smoking as a risk factor for recurrence of aneurysms treated by endosaccular occlusion. *Journal of Neurosurgery* 108: 672–75. [CrossRef] [PubMed]

Oshizumi, H., H. Inoya, H. Masuzawa, K. Sato, and S. Nakamura. 2004. Stent-assisted percutaneous transluminal angioplasty for intracranial vertebral and basilar artery stenosis: Two case reports. *No Shinkei Geka* 32: 627–34. (In Japanese). [PubMed]

Paraskevas, Kosmas I., Dimitri P. Mikhailidis, and Frank J. Veith. 2009. Carotid artery stenting may be losing the battle against carotid endarterectomy for the management of symptomatic carotid artery stenosis, but the jury is still out. *Vascular* 17: 183–89. [CrossRef]

Patsalides, A., J. Knopman, A. Santillan, A. J. Tsiouris, H. Riina, and Y. P. Gobin. 2011. Endovascular Treatment of Spinal Arteriovenous Lesions: Beyond the Dural Fistula. *American Journal of Neuroradiology* 32: 798–808. [CrossRef]

Payne, Misty M. 2001. Charles Theodore Dotter: The Father of Intervention. *Texas Heart Institute Journal* 28: 28–38. [PubMed]

Perret, George, and Hiro Nishioka. 1966. Report on the Cooperative Study of Intracranial Aneurysms and Subarachnoid Hemorrhage: Arteriovenous Malformations. *Journal of Neurosurgery* 25: 467–90. [CrossRef] [PubMed]

Peschillo, Simone, Alessandro Caporlingua, Claudio Colonnese, and Giulio Guidetti. 2014. Brain AVMs: An Endovascular, Surgical, and Radiosurgical Update. *The Scientific World Journal* 2014: 834931. [CrossRef] [PubMed]

Peschillo, Simone, and Roberto Delfini. 2012. Endovascular neurosurgery in Europe and in Italy: What is in the future? *World Neurosurgery* 77: 248–51. [CrossRef]

Piano, Mariangela, Luca Valvassori, Luca Quilici, Guglielmo Pero, and Edoardo Boccardi. 2013. Midterm and long-term follow-up of cerebral aneurysms treated with flow diverter devices: A single-center experience. *Journal of Neurosurgery* 118: 408–16. [CrossRef]

Pierot, Laurent, and Ajay K. Wakhloo. 2013. Endovascular Treatment of Intracranial Aneurysms: Current Status. *Stroke* 44: 2046–54. [CrossRef]

Pierot, Laurent, C. Delcourt, F. Bouquigny, D. Breidt, B. Feuillet, O. Lanoix, and S. Gallas. 2006. Follow-up of intracranial aneurysms selectively treated with coils: Prospective evaluation of contrast-enhanced MR angiography. *American Journal of Neuroradiology* 27: 744–49.

Pierot, Laurent, X. Leclerc, A. Bonafé, S. Bracard, and French Matrix Registry Investigators. 2008a. Endovascular treatment of intracranial aneurysms with matrix detachable coils: Midterm anatomic follow-up from a prospective multicenter registry. *American Journal of Neuroradiology* 29: 57–61. [CrossRef]

Pierot, Laurent, Laurent Spelle, Fabien Vitry, and ATENA Investigators. 2008b. Clinical outcome of patients harbouring unruptured intracranial aneurysms treated by endovascular approach: Results of the ATENA trial. *Stroke* 39: 2497–504. [CrossRef]

Pierot, Laurent, Laurent Spelle, Xavier Leclerc, Christophe Cognard, Alain Bonafé, and Jacques Moret. 2009. Endovascular treatment of unruptured intracranial aneurysms: Comparison of safety of remodeling technique and standard treatment with coils. *Radiology* 251: 846–55. [CrossRef]

Pierot, Laurent, C. Cognard, L. Spelle, J. Moret, and CLARITY group. 2011. The remodelling technique for endovascular treatment of ruptured intracranial aneurysms is more efficacious than standard coiling with a similar safety. *Radiology* 258: 546–53. [CrossRef] [PubMed]

Pierot, Laurent, C. Cognard, F. Ricolfi, R. Anxionnat, and on behalf of CLARITY Group. 2012a. Mid-term anatomical results after endovascular treatment of ruptured intracranial aneurysms with GDC™ and Matrix™ coils: Analysis of the CLARITY series. *American Journal of Neuroradiology* 33: 469–73. [CrossRef] [PubMed]

Pierot, Laurent, C. Cognard, L. Spelle, and J. Moret. 2012b. Safety and efficacy of balloon remodeling technique during endovascular treatment of intracranial aneurysms: Critical review of the literature. *American Journal of Neuroradiology* 33: 12–15. [CrossRef] [PubMed]

Pierot, Laurent, C. Cognard, R. Anxionnat, F. Ricolfi, and CLARITY Investigators. 2012c. Endovascular treatment of ruptured intracranial aneurysms: Factors affecting midterm quality anatomic results: Analysis in a prospective, multicenter series of patients (CLARITY). *American Journal of Neuroradiology* 33: 1475–80. [CrossRef] [PubMed]

Pierot, Laurent, C. Portefaix, J.-Y. Gauvrit, and A. Boulin. 2012d. Follow-up of coiled intracranial aneurysms: Comparison of 3DTOF at 3T and 1.5T in a large, prospective series. *American Journal of Neuroradiology* 33: 2162–66. [CrossRef]

Pierot, Laurent, T. Liebig, V. Sychra, K. Kadziolka, F. Dorn, and C. Strasilla. 2012e. Intrasaccular flow disruption: A new endovascular approach for the treatment of intracranial aneurysms. Results of a preliminary clinical evaluation in a multicenter series. *American Journal of Neuroradiology* 33: 1232–38. [CrossRef]

Pierot, Laurent, Christophe Portefaix, Anne Boulin, and Jean-Yves Gauvrit. 2012f. Follow-up of coiled intracranial aneurysms: Comparison of 3D time-of-flight and contrast-enhanced magnetic resonance angiography at 3T in a large, prospective series. *European Radiology* 22: 2255–63. [CrossRef]

Pierot, Laurent, Joachim Klisch, Christophe Cognard, Istvan Szikora, Benjamin Mine, Krzysztof Kadziolka, Vojtech Sychra, Istvan Gubucz, Anne-Christine Januel, and Boris Lubicz. 2013. Endovascular WEB flow disruption in middle cerebral artery aneurysms: Preliminary feasibility, clinical, and anatomical results in a multicenter study. *Neurosurgery* 73: 27–34. [CrossRef]

Piotin, Michel, Raphaël Blanc, Laurent Spelle, Charbel Mounayer, Rhelen Piantino, Paul J. Schmidt, and Jacques Moret. 2010. Stent-assisted coiling of intracranial aneurysms: Clinical and angiographic results in 216 consecutive aneurysms. *Stroke* 41: 110–15. [CrossRef]

Raymond, Jean, François Guilbert, Alain Weill, Stavros A. Georganos, Louis Juravsky, Anick Lambert, Julie Lamoureux, Miguel Chagnon, and Daniel Roy. 2003. Long-term angiographic recurrences after selective endovascular treatment of aneurysms with detachable coils. *Stroke* 34: 1398–403. [CrossRef]

Riche, M. C., J. P. Melki, and J. J. Merland. 1983. Embolization of spinal cord vascular malformations via the anterior spinal artery. *American Journal of Neuroradiology* 4: 378–81. [PubMed]

Ricolfi, F., C. Manelfe, J. F. Meder, P. Arrué, P. Decq, P. Brugieres, C. Cognard, and A. Gaston. 1999. Intracranial dural arteriovenous fistulae with perimedullary venous drainage: Anatomical, clinical and therapeutic considerations. *Neuroradiology* 41: 803–12. [CrossRef] [PubMed]

Ricolfi, F., P. Y. Gobin, A. Aymard, F. Brunelle, A. Gaston, and J. J. Merland. 1997. Giant perimedullary arteriovenous fistulas of the spine: Clinical and radiologic features and endovascular treatment. *American Journal of Neuroradiology* 18: 677–87.

Ries, Thorsten, Jan-Hendrik Buhk, Thomas Kucinski, Einar Goebell, Ulrich Grzyska, Hermann Zeumer, and Jens Fiehler. 2006. Intravenous administration of acetylsalicylic acid during endovascular treatment of cerebral aneurysms reduces the rate of thromboembolic events. *Stroke* 37: 1816–21. [CrossRef] [PubMed]

Rodesch, G., M. Hurth, H. Alvarez, B. Ducot, M. Tadie, and P. Lasjaunias. 2004. Angio- architecture of spinal cord arteriovenous shunts at presentation: Clinical correlations in adults and children—The Bicetre experience on 155 consecutive patients seen between 1981–1999. *Acta Neurochirurgica* 146: 217–26. [CrossRef]

Rodesch, Georges, Michel Hurth, Beatrice Ducot, Hortensia Alvarez, Philippe David, Marc Tadie, and Pierre Lasjaunias. 2003. Embolization of spinal cord arteriovenous shunts: Morphological and clinical follow-up and results—Review of 69 consecutive cases. *Neurosurgery* 53: 40–49.

Rosenblum, Bruce, Edward H. Oldfield, John L. Doppman, and Giovanni Di Chiro. 1987. Spinal arteriovenous malformations: A comparison of dural arteriovenous fistulas and intradural AVM's in 81 patients. *Journal of Neurosurgery* 67: 795–802. [CrossRef]

Sadasivan, Chander, Liliana Cesar, Jaehoon Seong, Audrey Rakian, Qing Hao, Fermin O. Tio, Ajay K. Wakhloo, and Baruch B. Lieber. 2009. An original flow diversion device for the treatment of intracranial aneurysms: Evaluation in the rabbit elastase-induced model. *Stroke* 40: 952–8. [CrossRef]

Sanchez-Mejia, Rene O., Michael W. McDermott, Jeffery Tan, Helen Kim, William L. Young, and Michael T. Lawton. 2009. Radiosurgery facilitates resection of brain arteriovenous malformations and reduces surgical morbidity. *Neurosurgery* 64: 231–28. [CrossRef]

Santillan, A., Y. P. Gobin, E. D. Greenberg, L. Z. Leng, H. A. Riina, P. E. Stieg, and A. Patsalides. 2012. Intraprocedural aneurysmal rupture during coil embolization of brain aneurysms: Role of balloon-assisted coiling. *American Journal of Neuroradiology* 33: 2017–21. [CrossRef]

Sehba, Fatima A., Jack Hou, Ryszard M. Pluta, and John H. Zhang. 2012. The importance of early brain injury after subarachnoid hemorrhage. *Progress in Neurobiology* 97: 14–37. [CrossRef] [PubMed]

Shapiro, M., J. Babb, T. Becske, and P. K. Nelson. 2008. Safety and efficacy of adjunctive balloon remodeling during endovascular treatment of intracranial aneurysms: A literature review. *American Journal of Neuroradiology* 29: 1777–81. [CrossRef] [PubMed]

Shapiro, M., T. Becske, D. Sahlein, J. Babb, and Peter Kim Nelson. 2012. Stent-supported aneurysm coiling: A literature survey of treatment and follow-up. *American Journal of Neuroradiology* 33: 159–63. [CrossRef] [PubMed]

Sluzewski, Menno, Willem Jan Van Rooij, Guus N. Beute, and Peter C. Nijssen. 2006. Balloon-assisted coil embolization of intracranial aneurysms: Incidence, complications, and angiography results. *Journal of Neurosurgery* 105: 396–99. [CrossRef] [PubMed]

Smith, Robert R., Yuri N. Zubkov, Yahgoub Tarassoli, Robert R. Smith, Yuri N. Zubkov, and Yahgoub Tarassoli. 1994. The History of Aneurysm Surgery. In *Cerebral Aneurysms*. New York: Springer, pp. 1–9. [CrossRef]

Songsaeng, Dittapong, Sasikhan Geibprasert, Karel G. Ter Brugge, Robert Willinsky, Michael Tymianski, and Timo Krings. 2011. Impact of individual intracranial arterial aneurysm morphology on initial obliteration and recurrence rates of endovascular treatments: A multivariate analysis. *Journal of Neurosurgery* 114: 994–1002. [CrossRef]

Spetzler, Robert F., and Neil A. Martin. 1986. A proposed grading system for arteriovenous malformations. *Journal of Neurosurgery* 65: 476–83. [CrossRef]

Spetzler, Robert F., M. Yashar S. Kalani, and Peter Nakaji. 2015. *Neurovascular Surgery*. New York: Thieme.

Spetzler, Robert F., Paul W. Detwiler, Howard A. Riina, and Randall W. Porter. 2002. Modified classification of spinal cord vascular lesions. *Journal of Neurosurgery* 96: 145–56. [CrossRef]

Spetzler, Robert F., Ronald W. Hargraves, Patrick W. McCormick, Joseph M. Zabramski, Richard A. Flom, and Richard S. Zimmerman. 1992. Relationship of Perfusion Pressureand Size to Risk of Hemorrhage from Arteriovenous Malformations. *Journal of Neurosurgery* 76: 918–23. [CrossRef]

Stayman, Aaron N., Raul G. Nogueira, and Rishi Gupta. 2011. A systematic review of stenting and angioplasty of symptomatic extracranial vertebral artery stenosis. *Stroke* 42: 2212–26. [CrossRef]

Szajner, Maciej, Alain Weill, Michel Piotin, and Jacques Moret. 1999. Endovascular treatment of a cervical paraspinal arteriovenous malformation via arterial and venous approaches. *American Journal of Neuroradiology* 20: 1097–99.

Szikora, I., Z. Berentei, Z. Kulcsar, M. Marosfoi, Z. S. Vajda, W. Lee, A. Berez, and Peter Kim Nelson. 2010. Treatment of intracranial aneurysms by functional reconstruction of the parent artery: The Budapest experience with the pipeline embolization device. *American Journal of Neuroradiology* 31: 1139–47. [CrossRef] [PubMed]

Taheri, Zeinab, Mohammad Hosein Harirchian, Hosein Ghanaati, Alireza Khoshnevisan, Payman Salamati, Mojtaba Miri, Kavous Firouznia, Mina Saeednejad, Madjid Shakiba, and Vafa Rahimi-Movaghar. 2015. Comparison of endovascular coiling and surgical clipping for the treatment of intracranial aneurysms: A prospective study. *Iranian Journal of Neurology* 14: 22–28. [PubMed]

Teitelbaum, George P., Donald W. Larsen, Vladimir Zelman, Anatolii G. Lysachev, and Leonid B. Likhterman. 2000. A tribute to Dr. Fedor A. Serbinenko, founder of endovascular neurosurgery. *Neurosurgery* 46: 462–70. [CrossRef] [PubMed]

Vaidya, Sandeep, Kathleen R. Tozer, and Jarvis Chen. 2008. An Overview of Embolic Agents. *Seminars in Interventional Radiology* 25: 204–15. [CrossRef] [PubMed]

Vallée, Jean-Noël, Laurent Pierot, Alain Bonafé, Francis Turjman, Pierre Flandroy, Jérôme Berge, Georges Rodesch, and Serge Bracard. 2004. Endovascular treatment of intracranial wide-necked aneurysms using three-dimensional coils: Predictors of immediate anatomic and clinical results. *American Journal of Neuroradiology* 25: 298–306.

van Beijnum, J., H. B. Van der Worp, H. M. Schippers, O. Van Nieuwenhuizen, L. J. Kappelle, G. J. E. Rinkel, J. W. Berkelbach van der Sprenkel, and C. J. M. Klijn. 2007. Familial occurrence of brain arteriovenous malformations: A systematic review. *Journal of Neurology, Neurosurgery & Psychiatry* 78: 1213–17.

van Dijk, J. Marc C., Karel G. terBrugge, Robert A. Willinsky, and M. Christopher Wallace. 2002. Clinical course of cranial dural arteriovenous fistulas with long-term persistent cortical venous reflux. *Stroke* 33: 1233–36. [CrossRef]

Vlak, Monique H. M., Ale Algra, Raya Brandenburg, and Gabriël J. E. Rinkel. 2011. Prevalence of unruptured intracranial aneurysms, with emphasis on sex, age, comorbidity, country, and time period: A systematic review and meta-analysis. *The Lancet Neurology* 10: 626–36. [CrossRef]

Wakhloo, Ajay K., Giuseppe Lanzino, Baruch B. Lieber, and L. Nelson Hopkins. 1998. Stents for intracranial aneurysms: The beginning of a new endovascular era [Editorial comment]? *Neurosurgery* 43: 377–79. [CrossRef]

Wakhloo, Ajay K., I. Linfante, C. F. Silva, E. A. Samaniego, G. Dabus, V. Etezadi, Gabriela Spilberg, and Matthew J. Gounis. 2012. Closed-cell stent for coil embolization of intracranial aneurysms: Clinical and angiographic results. *American Journal of Neuroradiology* 33: 1651–56. [CrossRef]

Wakhloo, Ajay K., Jake Mandell, Matthew J. Gounis, Christopher Brooks, Italo Linfante, Jesse Winer, and John P. Weaver. 2008. Stent-assisted reconstructive endovascular repair of cranial fusiform atherosclerotic and dissecting aneurysms: Long-term clinical and angiographic follow-up. *Stroke* 39: 3288–96.

Wakhloo, AK, J Gounis, JS Sandhu, N Akkawi, A Schenck, and I Linfante. 2007. Complex- shaped platinum coils for intracranial aneurysms—Higher packing density and improved mid-term angiographic outcome. *American Journal of Neuroradiology* 28: 1395–1400. [CrossRef]

Weingrad, Daniel N., John L. Doppman, Paul B. Chretien, and Giovanni Di Chiro. 1979. Paraplegia due to posttraumatic pelvic arteriovenous fistula treated by surgery and embolization: Case report. *Journal of Neurosurgery* 50: 805–10. [CrossRef]

White, Philip M., Stephanie C. Lewis, Anil Gholkar, Robin J. Sellar, Hans Nahser, Christophe Cognard, Lynn Forrester, Joanna M. Wardlaw, and HELPS Trial Collaborators. 2011. Hydrogel-coated coils versus bare platinum coils for the endovascular treatment of intracranial aneurysms (HELPS): A randomised controlled trial. *The Lancet* 377: 1655–62. [CrossRef] [PubMed]

Willinsky, R. A., J. Peltz, L. Da Costa, R. Agid, and R. I. Farb. 2009. Clinical and angiographic follow-up of ruptured intracranial aneurysms treated with endovascular embolization. *American Journal of Neuroradiology* 30: 1035–40. [CrossRef] [PubMed]

Willinsky, R., W. Montanera, M. C. Wallace, and F. Gentili. 1993. Spinal epidural arteriovenous fistulas: Arterial and venous approaches to embolization. *American Journal of Neuroradiology* 14: 812–17. [PubMed]

Willinsky, Robert, Karel Terbrugge, Pierre Lasjaunias, and Walter Montanera. 1990. The variable presentations of craniocervical and cervical dural arteriovenous malformations. *Surgical Neurology* 34: 118–23. [CrossRef] [PubMed]

Yu, Wengui, and Wei-Jian Jiang. 2018. Stenting for intracranial stenosis: Potential future for the prevention of disabling or fatal stroke. *Stroke and Vascular Neurology* 3: e000158. [CrossRef]