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Intracranial Arterial Collateralization: Relevance in Neuro-Endovascular Procedures

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1. Introduction

Endovascular strategies for addressing intracranial and extracranial diseases continue to gain momentum. These techniques are limited principally by technology and imagination. As newer devices and implements are introduced to the endovascular surgeon, more diseases previously construed to be the realm of open surgery or untreatable are becoming amenable to endovascular interventions. Because of the nature of endovascular procedures, with liquid agents, flow-directed therapies, and embolic materials, it is critical for a neuro-interventionalist to be aware of the collaterals that exist between the vessels being embolized and other critical collaterally connected vessels, occlusion of which may result in undesirable outcomes. Similarly, for other occasions, such collaterals may provide unique conduits that may afford access in novel ways to the intracranial or extracranial circulation. The understanding of these collaterals is best undertaken with an initial understanding of the development of the cranial vasculature. The rich anastomotic connections and interlinked development shed great light and provide a firm basis for understanding the cranial collaterals. Secondly, the collateral circulation may be divided by collaterals between extracranial and intracranial systems on one hand and collaterals between the internal carotid and vertebrobasilar (VB) systems on the other. In this chapter, we endeavor to provide a brief overview of cranial vascular development, followed by specific clinically relevant examples of extracranial and intracranial anastomoses and the internal carotid artery (ICA) and vertebrobasilar (VB) anastomoses, intracranial and intracranial anastomoses.

2. Cranial vascular embryology

The cranial vasculature begins with the development of a vascular supply to the paired pharyngeal arches. This supply develops as vascular arches that emanate from the ventral aortic sac connect with the paired dorsal aortae. Each pharyngeal arch gets its own vascular arch. These vascular arches then develop and regress in rostrocaudal fashion. The pharyngeal arches become apparent at approximately 3 to 4 weeks’ gestation. The pharyngeal arches develop plexiform vascular channels that ultimately connect the ventral aortic sac with the paired dorsal aortae, forming the vascular arch. The first arch gives rise to
the primitive stapedial artery, whereas the second gives rise to the hyoid artery. These arches then regress and coalesce to form the primitive hyoidostapedial artery. These vessels are critical to the vascular development of the skull base. This primitive branch follows the three divisions of the trigeminal nerve such that one trunk that develops along the mandibular division becomes the adult internal maxillary artery; the superior trunk becomes the middle meningeal artery and contributes to the ophthalmic artery. The primitive maxillary artery develops as the meningohypophyseal trunk, whereas the third division becomes the corticotympanic branch, which communicates with the ICA in the petrous canal (Figure 1). An embryologic dorsal ophthalmic artery regresses to become the inferolateral trunk, rarely staying on as a cavernous origin to the adult ophthalmic artery. The third vascular arch on either side becomes the cervical ICA, eventually incorporating parts of the dorsal aortae bilaterally to form more cranial sections up the posterior communicating artery (PCoA) segment. From the third arch sprouts the external carotid artery (ECA) trunk, which anastomoses with the primitive hyoidostapedial artery branches to complete the ECA circuitry. Additionally, a pair of plexiform networks, called the ventral pharyngeal arteries, develop early on and connect to the hyoidostapedial trunk. These ventral–pharyngeal networks form prior to the development of the ECA trunk, eventually mostly regressing, but retaining parts that allow for anastomoses between the ascending pharyngeal and caroticotympanic arteries.

![Diagram of vascular arches](https://www.intechopen.com)

**Fig. 1.** Anatomic diagram turned from anteroposterior to a left anterior oblique (LAO) position depicts the definitive left common carotid artery (CCA) as well as the external carotid artery (ECA) and internal carotid artery (ICA). The embryonic origin of these vessels is also shown. CTA, caroticotympanic artery; small single arrow, stapes; (arrowhead), foramen spinosum; (double arrows), optic canal; (open arrows), carotid canal. Distal ramifications from the internal maxillary artery (IMA), middle meningeal artery (MMA), and orbital branches of the ophthalmic artery (OA) are indicated by the dotted lines. Stapedial artery (STA). Permission requested from Osborn AG: Diagnostic Cerebral Angiography (2nd ed). Philadelphia: Lippincott Williams & Wilkins, Wolters Kluwer, 1999, figure 2-5, page 35.
The posterior circulation develops in parallel, first appearing towards the beginning of the fifth gestational week as paired dorsal longitudinal neural arteries. These eventually form the intracranial VA and BA. Further caudally, a plexiform network of the cervical intersegmental arteries anastomoses to form the paired vertebral arteries (VAs). As these channels continue to develop, they reliably develop anastomotic connections with the ICAs, forming the trigeminal, otic, hypoglossal, and proatlantal intersegmental connections (Figure 2). The seventh cervical intersegmental artery coalesces with the right fourth cervical intersegmental artery to form the proximal vertebral arteries (VAs, solid black arrows). These VAs form as longitudinal anastomoses between the seven cervical intersegmental arteries. The proximal connections between the C1-6 arteries and the dorsal aorta (DA) are regressing. For simplification, only one set of longitudinal neural and cervical intersegmental arteries is shown. These vessels are the precursors of the VB circulation. Initially, the longitudinal neural arteries are supplied from below via the intersegmental arteries. At this stage, several temporary connections between the developing VB circulation and the carotid arteries also form. From cephalad to caudal, these arteries are the trigeminal (T), otic (O), hypoglossal (H), and proatlantal intersegmental arteries (P) (this vessel forms slightly later). These transient anastomoses regress as the caudal divisions of the primitive internal carotid arteries (ICAs) anastomose with the cranial ends of the longitudinal neural arteries and form the future posterior communicating arteries (dotted lines with curved arrows). Persistence of the transient embryonic interconnections is abnormal and results in a so-called primitive carotid–basilar anastomosis. Sprouting of the external carotid arteries (ECAs) from the proximal common carotid arteries (CCAs) is also depicted. These vessels will annex first and second arch remnants (solid black areas). VA, ventral aorta. Permission requested from Osborn AG: Diagnostic Cerebral Angiography (2nd ed). Philadelphia: Lippincott Williams & Wilkins, Wolters Kluwer, 1999, figure 1-2A, page 8.
vascular arch (the right aortic arch) to form the proximal part of the subclavian artery and from it originates the VA. On the left side, the seventh cervical intersegmental artery coalesces with the left aortic arch (distal fourth arch–true adult aortic arch) to form the proximal left subclavian artery and from it originates the left VA (Figure 3). By 6 weeks' gestation, the cranial-most end of the ICAs have divided into rostral and caudal divisions. Although the rostral division will form the anterior, middle and anterior choroidal arteries, the caudal division fuses with the dorsal longitudinal neural arteries to form the PCoA. This results in eventual disappearance of the primitive carotid-basilar anastomoses.

Fig. 3. Diagrammatic sketch of the craniocerebral vasculature at 7 weeks of development. The arch and great vessels are approaching their definitive form. Origins of these vessels from their embryonic precursors are depicted schematically. The fourth and sixth aortic arches are undergoing asymmetric remodeling to supply blood to the upper extremities, dorsal aorta, and lungs. The right sixth arch has involuted (leaving only part of the right pulmonary artery). The right dorsal aorta distal to the origin of the subclavian artery (SCA) is regressing (dotted lines) but remains connected to the right fourth arch. The right third and fourth arches are forming the brachiocephalic trunk; the left fourth arch becomes the definitive aortic arch. The left dorsal aorta becomes the proximal descending aorta. The first six cervical intersegmental arteries have become the definitive vertebral arteries (VAs); the C7 arteries have enlarged to become part of the developing subclavian arteries (SCAs). The longitudinal neural arteries are fusing across the midline to form the definitive basilar artery (BA).


Most of these changes are complete in the adult configuration by 8 weeks of gestation. For a review and further details, please see Osborn’s detailed descriptions. Further details have been described by Larsen and Lasjaunias et al.
### 3. Extracranial-to-intracranial anastomoses

For the purpose of grouping these anastomoses, Lasjaunias et al.\textsuperscript{15,16} and Berenstein et al.\textsuperscript{3} described regions within the cranial circulation that could be divided on the basis of primitive vascular connections. They divided the regions as follows:

1. Anterior or ophthalmic artery connections with facial and internal maxillary arteries
2. Middle or petrovenous ICA branches with internal maxillary and ascending pharyngeal branches
3. Posterior or VB connections with ascending pharyngeal, occipital, and subclavian artery branches, especially the ascending and deep cervical arteries.

This division, although overlapping, serves to identify vascular territories of concern; that is, functional areas in which anastomotic dysfunction may become most apparent, thereby tailoring angiographic and neurologic examination during intraprocedural monitoring. Even when not apparent on routine angiography, these connections exist as byproducts of a unified vascular development for the entire head and neck region.\textsuperscript{9} Under situations of increased flow, such as with arteriovenous fistulas (AVFs) or arteriovenous malformations (AVMs), there may be arterio-arterial embolization, resulting in deficits. In other cases, shared venous outflow may complicate options or results. Similarly, simply injecting materials, whether contrast or embolic materials, particularly liquid agents, may result in their crossing the anastomosis to occlude functional vessels supplying neural tissues, thereby creating unintended deficits. In addition, as embolization proceeds and the desired target vessel occludes, putative collateral anastomoses may be at increased risk. This may occur because of their presence as a relative lower-pressure sump (in the face of an occluded principal target vessel), resulting in the preferential shunting of embolic materials into these collaterals.\textsuperscript{5} Although these collaterals may increase the risk of embolization procedures in the skull base, they serve to provide critical collaterals in the face of acute or subacute carotid or VB occlusion. They provide an innate bypass that grows under an increased demand from a hyperperfused intracranial territory. A recent review by Geibprasert et al.\textsuperscript{9} describes these extracranial–intracranial anastomoses in considerable detail. These anastomotic connections are summarized in Table 1.

#### Table 1. Extracranial-to-Intracranial Anastomoses

<table>
<thead>
<tr>
<th>Location</th>
<th>External Carotid Artery Branch</th>
<th>Internal Carotid Artery Branch</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orbit, Superior Orbital Fossa</td>
<td>Internal maxillary artery-Middle meningeal artery</td>
<td>Ophthalmic artery-Orbital branch</td>
</tr>
<tr>
<td>Maxillary Sinus</td>
<td>Internal maxillary artery-Middle meningeal artery</td>
<td>Ophthalmic artery-Anterior ethmoid artery</td>
</tr>
<tr>
<td>Orbit</td>
<td>Internal maxillary artery-Deep temporal artery, inferior orbital artery</td>
<td>Ophthalmic artery-Orbital branch (Lateral branches)</td>
</tr>
<tr>
<td>Frontal Sinus</td>
<td>Internal maxillary artery-Sphenopalatine, Greater palatine artery</td>
<td>Ophthalmic artery-Anterior and Posterior ethmoid arteries</td>
</tr>
<tr>
<td>Scalp</td>
<td>Superficial temporal artery</td>
<td>Ophthalmic artery-Supero orbital artery</td>
</tr>
<tr>
<td>Nose</td>
<td>Facial artery-external branch, internal maxillary artery-internal orbital artery</td>
<td>Ophthalmic artery-Dentinal nasal artery</td>
</tr>
<tr>
<td>Cavernous Sinus</td>
<td>Internal maxillary artery-Middle meningeal artery</td>
<td>Internal carotid artery-Inferior lateral trunk</td>
</tr>
<tr>
<td>Sphenopalatine Fossa</td>
<td>Internal maxillary artery-Artery of the vidian canal</td>
<td>Internal Carotid artery-Branch of the foramen lacerum (Primitive stagnational artery)</td>
</tr>
<tr>
<td>Infraorbital Fossa</td>
<td>Internal maxillary artery-Accessory meningeal artery</td>
<td>Internal carotid artery-Artery of the foramen ovale (Artery of the foramen ovale)</td>
</tr>
<tr>
<td>Superior Nasopharynx</td>
<td>Ascending pharyngeal artery-Pharyngeal branches</td>
<td>Internal carotid artery-Artery of the foramen lacerum (Primordial pharyngeal artery)</td>
</tr>
<tr>
<td>Temporal Bone-Middle Ear</td>
<td>Ascending pharyngeal branches-Neuroradiculomeningeal branches</td>
<td>Internal carotid artery-Neuroroticynic branch</td>
</tr>
<tr>
<td>Hypoglossal Canal and Jugular Foramen</td>
<td>Ascending pharyngeal artery-Neuroradiculomeningeal branches</td>
<td>Internal carotid artery-Neurohypophyseal trunk</td>
</tr>
<tr>
<td>Occluded Process</td>
<td>Ascending pharyngeal artery-Neuroradiculomeningeal branches</td>
<td>Vertical artery-Anterior arch</td>
</tr>
<tr>
<td>Styloid foramen</td>
<td>Posterior Auricular, Occipital artery-Styloidomastoid branch</td>
<td>Internal carotid artery-Cereolotympanic branch (Facial artery supply)</td>
</tr>
<tr>
<td>Temporal process of C1</td>
<td>Occipital artery-Muscular branches</td>
<td>Vertical artery-Muscular branches</td>
</tr>
<tr>
<td>Posterior cervical muscle fossa</td>
<td>Deep and Ascending Cervical arteries-Muscular branches</td>
<td>Vertical artery-Muscular branches</td>
</tr>
</tbody>
</table>

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3.1 Ophthalmic artery anastomoses
The ophthalmic artery is the principal vascular supply to contents in the orbit.\textsuperscript{13} It is the principal supply to the central retinal artery, which in turn, supplies the retina and choroid. Occlusion of this vessel will result in monocular blindness. Therefore, visualization of a choroid blush with an ECA injection should raise alarms about potential anatomic variations and dangerous collaterals.\textsuperscript{25} The ophthalmic artery originates from the ICA; however, during its development, it necessarily develops connections with other sources of supply to the contents of the orbit. The primitive stapedial artery contributes to the middle meningeal artery, and this develops some connections to the ophthalmic artery through the superior orbital fissure.\textsuperscript{26} The magnitude of this connection may vary; rarely, the middle meningeal and internal maxillary arteries completely assume the principal source of supply to the ophthalmic artery. In cases in which the ophthalmic artery is not visualized, such variation should be suspected.

![An ECA angiogram in a patient with ICA occlusion, with reconstitution of the ICA through collaterals via the ophthalmic artery and the cavernous ICA. The vidian artery (A) is supplying collateral flow from the internal maxillary artery (B) to the petrous ICA (C). The frontal branch of the superficial temporal artery (D) is providing transosseous collaterals to the frontal meningo-pial collaterals, whereas the parietal branch (E) is not involved. The PCoA (F) is involved in the extensive neovascularization through its perforators, typical of Moya Moya disease. The ophthalmic artery (G) is the largest source of retrograde collateral flow to the ICA. It is receiving its supply principally through its ethmoidal arteries (H) via connections with the internal maxillary artery. Another source of supply to the ophthalmic artery is the recurrent meningeal branch (I) of the middle meningeal artery.](https://www.intechopen.com)

The anastomotic connections of the ECA branches with the ophthalmic artery may be divided according to the segments of the ophthalmic artery.\textsuperscript{13} The first segment of the ophthalmic artery arises as the first supraclinoidal branch of the ICA and travels on the
underside of the optic nerve in the optic canal. Upon entering the orbit, it maintains its close relationship with the optic nerve traveling towards the posterior globe. The recurrent meningeal artery is a reliable branch often noted by microvascular surgeons along the lateral aspect of the superior orbital fissure and is a branch of the middle meningeal artery. This supplies the contents of the superior orbital fissure and then anastomoses with the second segment of the ophthalmic artery in the orbit. This anastomosis is of particular importance when embolizing the middle meningeal artery branches, particularly for convexity meningiomas. The recurrent meningeal artery can often be visualized as the middle meningeal artery crosses the sphenoid wing. In cases in which embolization is desirable, it is best to obtain distal access close to or in the tumor proper and be vigilant to reflux to this more proximal branch point. In other cases where a branch is not noted, an intraarterial injection of sodium amytal and lidocaine may allow Wada testing of the anastomosis, which may not be apparent (Figures 4 and 5).

Fig. 5. Selective ECA angiogram reveals a tumor blush of an anterior cranial fossa meningioma. The principal supply is the middle meningeal artery (A), which divides into the parietal branch (B) and an enlarged recurrent meningeal branch (C) that, along with the accessory meningeal artery (D), is the principal supply for this tumor (E, tumor blush).

The second group of anastomoses is a product of the shared supply to the ethmoidal sinuses from the anterior and posterior branches of the ophthalmic artery as well as branches from septal, sphenopalatine, and greater palatine branches of the internal maxillary artery. These occur along the second and third segments of the ophthalmic artery. In addition, anastomoses may also exist between the ophthalmic artery and deep temporal and infraorbital branches of the internal maxillary artery. These arise particularly because of shared ophthalmic and internal maxillary supply to the lacrimal apparatus (Figure 4). These anastomotic connections carry great significance during embolization for epistaxis. Most of the anastomotic connections through the lacrimal and ethmoidal arteries tend to be very
Fig. 11. A 40-year-old man presented with amaurosis fugax in the right eye. An imaging study confirmed a partially thrombosed giant aneurysm of the right ICA cavernous segment that likely had caused the embolic event. (A) T1-weighted magnetic resonance image reveals a partially thrombosed cavernous aneurysm (arrow). (B and C) Angiography shows the fusiform aneurysm with serpentine channel (arrow). (D) Right ICA TBO test with angiogram from left ICA reveals robust cross-filling of the contralateral circulation. The ACoA serves to opacify the right ACA and MCA without delay. (E) The patient tolerated TBO and was successfully treated with right ICA sacrifice.

revealed or opacified during angiography only after proximal vessel test occlusion. We typically perform a superselective micro-balloon test occlusion of the proximal vessel (as distal as possible remaining proximal to the aneurysm). Under micro-vessel occlusion we perform proximal angiograms as well as concurrent neurophysiologic testing to demonstrate angiographic and functional collateralization. If the patient tolerates the test occlusion, we then proceed immediately with endovascular embolization permanent occlusion of the tested vessel distal to the test site (Figure 13). If the patient does not tolerate test occlusion, one then has to weigh the risks of vessel-preserving strategies through microsurgical or endovascular means versus the expected neurologic deficit of vessel deconstruction. Obviously, microsurgical bypass anastomosis prior to artery sacrifice is one well-established treatment strategy. It should be noted that Hallacq et al. reported a series of 10 cases of P2 PCA aneurysms treated with occlusion of the aneurysm and parent vessel without balloon test occlusion in which no postocclusion occipital lobe ischemia occurred.
Similar patterns of distal arterial collateralization can often be expected in PICA, AICA and SCA circulations. In cases of PICA ruptured dissections or dissecting aneurysms, proximal PICA can be occluded without needs of microsurgical bypass anastomosis. (Figure 21).

Fig. 12. A 38-year-old man with a left PCA territory embolic infarct was found to have a traumatic giant left VA dissecting aneurysm and DAVF from a fall 2 years prior to treatment. (A and B) Three-dimensional and two-dimensional angiograms show a giant left VA dissecting aneurysm and a DAVF. (C) The right VA angiogram demonstrates competent flow in the right VA. (D and E) Due to extremely high flow in the giant aneurysm, the DAVF was unable to be catheterized until occlusion of the left VA at the origin of the aneurysm. The left traumatic vertebral DAVF was catheterized from the right VA through the VB junction to the left VA. The black arrow indicates the fistula. Open arrows indicate the microcatheter route. (F) The fistula and residual filling of the giant aneurysm were treated successfully with coil occlusion through the contralateral VA to the left vertebral DAVF and giant aneurysm.

5. Alternative microcatheterization of target vessel via a collateral route

The circle of Willis provides a natural conduit to access contralateral or carotid to basilar (or vice-versa) circulations. In the vast majority of cases, adequate access may be obtained through direct ipsilateral routes; however, due either to proximal vessel occlusion or distal vessel entry angles, these alternate routes through the circle of Willis provide easier microcatheter entry angles.
Fig. 13. A 48-year-old woman with a subarachnoid hemorrhage due to a left P2-P3 junction ruptured aneurysm. (A and B) Lateral view and anteroposterior (AP) view of original VA angiogram (arrow in B). (C and D) Left ICA AP and lateral angiography. (E and F) Left PCA P2 TBO. The left ICA angiogram, while the left P2 was occluded with a balloon, revealed left ACA to PCA cortical collateralization (black arrow) with retrograde opacification during the late arterial phase. In F, open arrow indicates the balloon; arrowhead indicates the retrograde partially opacified P2-P3 aneurysm. (G and H) Ultimately, this aneurysm was coil embolized completely with balloon-remodeling technique; and the distal PCA branches were preserved anterogradely.

A sharp reversely angulated origin of a vessel makes superselective ipsilateral catheterization difficult. For instance, access to the PICA or anterior inferior cerebellar artery (AICA) from the VA can be occasionally difficult, or in cases in which the carotid or bilateral VAs are occluded, the PCoA offers a distinct advantage. In these situations, if the caliber of the PCoA is adequate, effective catheterization of the desired vessel can be achieved (Figures 14 and 15). Naturally, a microcatheter can also go from one VA to the other through the VB junction (Figure 12, 16). Similarly, during treatment of broad-based aneurysms at the basilar or ICA terminus, ipsilateral placement of a stent may be inadequate for complete coverage of the aneurysm neck, necessitating a Y-configuration with increased risk of thromboembolic complications. In these cases, the following strategies offer distinct advantages: access from the contralateral carotid artery to pass a stent across the AcoA so
Fig. 14. A 40-year-old man with a left intracranial VA dissection with a pseudoaneurysm just distal to the left PICA origin. (A and B) three-dimensional angiogram and lateral view of left VA two-dimensional angiogram. Arrows indicate the pseudoaneurysm at the dissection site. (C) Left ICA angiogram revealed robust PCoA connecting the left ICA system to the VB system. (D) Due to limited distal flow from the dissecting aneurysm to the normal PICA origin, complete occlusion of the left VA was not achieved by balloon-remodeling coiling through left VA catheterization. Therefore, superselective catheterization through a left ICA-PCoA-BA-left VA route allowed us to successfully occlude the diseased segment of the VA. Arrows in E indicate the microcatheter route. By comparison with the angiogram in C, the 3-month follow-up angiogram with right VA (E) and left VA (F, lateral view) runs shows the left VA supplying the left PICA without evidence of recurrent aneurysm.

that it ultimately sits across the entire neck of the aneurysm in the ICA terminus (from the ipsilateral ACA to the MCA) or from the carotid artery across the PCoA so that it sits from the ipsilateral PCA to contralateral PCA (Figure 17). In cases in which there is occlusion of one or more vessels to the circle of Willis, the circle provides an optimal opportunity to access diseased segments of vessels that are proximally occluded. These routes can be used to treat a variety of conditions, such as aneurysms (Figure 18), AVMs, or AVFs. Similarly, in cases of acute occlusion, even if the proximal vessel cannot be adequately revascularized, reestablishing flow across the circle may alleviate acute cerebral ischemia (Figure 19). In other situations, the circle may allow passage of endovascular implements that, despite patency, cannot be brought up through ipsilateral routes (Figure 22 -24). This is particularly relevant to situations in which the ipsilateral vessel may have spasm after subarachnoid hemorrhage (Figure 20) or be congenitally hypoplastic.
Fig. 15. A 70-year-old woman presented with multiple events of VB insufficiency. Evaluation revealed bilateral VA occlusions. A) Injection of the left ICA (C) revealed the PCoA (B) as the principal supply to the BA (A) and the PCA (D). (B) A road-map angiogram reveals a guide catheter in the left ICA (B) with a microwire through the PCoA (C) and through the proximal BA (A) across an obvious stenosis into the distal VA. (C) After retrograde BA angioplasty, an ICA (A) angiogram reveals robust filling through the PCoA (B) into the basilar artery with markedly improved flow across the stenotic lesion into the BA junction (C), bilateral PICA, and VA (E, F). With permission from Chiam PT, Mocco J, Samuelson RM, Siddiqui AH, Hopkins LN, Levy EI: Retrograde angioplasty for basilar artery stenosis: bypassing bilateral vertebral artery occlusions. J Neurosurg 110:427-430, 2009

Fig. 16. A 65-year old man presented with neck-movement-related VB insufficiency. The left VA was noted to be occluded extracranially. (A) Right VA (A) angiogram revealed excellent flow into the BA with fenestration (B), a tight stenosis at the junction (C) of the left VA (D) with the BA, and a peak systolic angiographic jet opacifying the PICA (E). (B) A microballoon (A) was brought from the right VA (C) over a wire, which was placed into the left proximal VA (B). (C) Final angiogram revealing significant improvement (despite persistent stenosis) in distal left VA (A) flow noted into the proximal left VA (B) and left PICA (C) following a right VA (D) injection. (E) Basilar artery.
Fig. 17. A 44-year-old man with a Spetzler-Martin Grade IV AVM post-staged embolization and Gamma Knife radiosurgery presented with a residual, broad-based basilar terminus aneurysm. (A) Combined left carotid (E) and left VA (F) angiogram reveals a robust PCoA (C), BA (A), aneurysm (B), MCA (D). (B) Road-map image of an Enterprise stent (Codman Neurovascular) being deployed via the left ICA (E) through the left PCoA (F) from the ipsilateral P1 segment of the PCA (C) across the neck of the aneurysm into the contralateral PCA (B). Deployed stent markers (A). BA (D). (C) The patient was subsequently brought for a second coil embolization session with access to the BA aneurysm achieved from the left VA (C). Angiographically, complete aneurysm obliteration (A) was obtained. The left PCoA (B) is noted.

Fig. 18. A 40-year-old man presented with a history of a bullet injury to the head and neck and surgical ligation of the left ICA and now with acute development of a carotid-cavernous fistula. He was noted to have a ruptured, giant cavernous ICA pseudoaneurysm. (A) A left VA (A) angiogram reveals filling of the aneurysm (D) across the PCoA (C) to the supraclinoidal carotid artery (B) with early venous filling of the ophthalmic vein (F) and pterygoid venous plexus (E). (B) The aneurysm was accessed through the same route from the left VA (B) across the PCoA (C) to achieve complete obliteration of the aneurysm and carotid-cavernous fistula (A). MCA (D).
Fig. 19. A 65-year-old man presented with an acute stroke with occlusion of his left ICA. He was beyond the window for intravenous thrombolysis. His ancillary studies suggested potentially viable brain in the MCA territory. However, computed tomographic angiography suggested occlusion of the left MCA territory in addition to more proximal carotid occlusion. An emergent angiogram was planned. (A) A right carotid (D) angiogram performed to assess cross-flow reveals a left ICA terminus occlusion (A) with flow to the bilateral distal ACA circulation across the ACoA (C) but no flow to the left MCA (B). (B) Selective left ICA angiogram reveals complete cavernous occlusion (B) of the left ICA (A). (C) The occlusion (B) was carefully crossed, and a thrombectomy suction catheter (D) was advanced into the occluded left MCA. (D) Following suction aspiration, the clot was retrieved from the MCA (B), resulting in revascularization of the ICA terminus (A) and reestablishment of flow across the ACoA (C) from the right ICA (F). Both ACAs remained patent (E, D). Despite these efforts, the proximal occlusion remained recalcitrant to revascularization efforts and was therefore left occluded. The patient recovered from all deficits.
Fig. 20. A 42-year-old man presented with a ruptured ACoA aneurysm, which was treated via coil embolization. On Day 6, he developed severe symptomatic spasm, which remained refractory to maximal medical therapy. He had severe spasm of his left ACA, which resulted in the use of the ACoA artery as a conduit to angioplasty the left distal ACA. (A) Right carotid angiogram reveals a microcatheter and wire across the right ACA (A), AcoA, and aneurysm (C) into the left distal ACA (B). Right distal ACA (D). (B) Non-subtracted angiogram revealing the relationship of the aneurysm and the inflated balloon in the left distal ACA (A). AcoA (B), right distal ACA (C). Access microcatheter in the right ICA (D).
Fig. 21. A 24-year-old man presented with subarachnoid hemorrhage due to proximal right PICA dissection. After superselective micro-balloon test occlusion, it was clear that there are anastomoses from right SCA and AICA to right PICA distal branches. Therefore, the dissecting segment right PICA was successfully occluded with coils without the need of microsurgical bypass procedure, and the patient revealed no deficits following the procedure. The distal branches remains filling after the proximal PICA occlusion. (A) Right vertebral artery angiogram lateral view and (C) anterior-posterior view prior to the treatment. The black arrows point to the dissected proximal PICA. (B) Right vertebral artery angiogram lateral view and (D) anterior-posterior view after occlusion of proximal right PICA with coils. The solid white arrows indicate coil occlusion of the dissected proximal PICA. Open white arrows indicate the distal right PICA filling from collaterals.
Fig. 22. 64-year old woman presented with increasing headaches after a motor vehicle accident. She was neurologically intact upon presentation. Diagnostic workup revealed a giant cavernous aneurysm (Figure 22 A, B). She was enrolled in the PUFFs trial for utilization of a flow diversion Pipeline Device™ for treatment of large proximal carotid aneurysms. After deployment of the first device the distal access through the aneurysm was lost and despite multiple attempts to regain access through the proximal end of the stent, which had migrated laterally into the aneurysm, distal anterograde access could not be established (Figure 22 C, D).
Fig. 23. Review of the circle of Willis appeared to demonstrate both a sizeable ACoA and PCoA arteries therefore a second groin access was established with the guide catheter in the vertebral artery (Figure 23 A, B). A microcatheter was used to catheterize the PCoA artery from the vertebral artery and thereby gain access into the aneurysm using retrograde catheterization of the proximally dislocated but distally tethered Pipeline Device™ (Figure 23 C, D).
Fig. 24. Once the microwire was in the aneurysm a Snare was deployed to grab the microwire and the Marksman microcatheter was advanced using the snare over the microwire into the Pipeline device by pulling the microwire back out through the PCoA (Figure 24 A, B). Once distal access was reestablished additional Pipeline Devices were used to complete the endovascular reconstruction of the aneurysm. The aneurysm appeared almost completely obliterated at the end of the procedure (Figure 24 C) and remained obliterated at the 6-month follow-up (Figure 24 D).

6. Conclusion

An in-depth knowledge of intracranial and extracranial collateral anastomoses, overt or hidden, is crucial for a neurointerventionist to devise optimal endovascular strategies to manage a host of pathological conditions; to ascertain potential pitfalls; and ultimately, to
avoid complications that could have been prevented by a better understanding of underlying vascular anatomy. As the scope and extent of endovascular interventions for cerebrovascular and cranial disease continues to expand, the recognition of these putative anastomoses will continue to become a larger part of diagnostic evaluation and interventional design.

7. References


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Neuroimaging for clinicians sourced 19 chapters from some of the world's top brain-imaging researchers and clinicians to provide a timely review of the state of the art in neuroimaging, covering radiology, neurology, psychiatry, psychology, and geriatrics. Contributors from China, Brazil, France, Germany, Italy, Japan, Macedonia, Poland, Spain, South Africa, and the United States of America have collaborated enthusiastically and efficiently to create this reader-friendly but comprehensive work covering the diagnosis, pathophysiology, and effective treatment of several common health conditions, with many explanatory figures, tables and boxes to enhance legibility and make the book clinically useful. Countless hours have gone into writing these chapters, and our profound appreciation is in order for their consistent advice on the use of neuroimaging in diagnostic work-ups for conditions such as acute stroke, cell biology, ciliopathies, cognitive integration, dementia and other amnestic disorders, Post-Traumatic Stress Disorder, and many more.

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