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Functional angiography of the head and neck.

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Functional Angiography of the Head and Neck

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The basic concepts of the territorial approach to angiography and intravascular embolization of vasular lesions of the head and neck are reviewed. Superselective arteriographic injections are performed in sequences and projections tailored to the suspected location of the vascular lesion. The resulting studies are then reviewed as a whole, so that a complete angiographic map of the tissue territory involved is considered. From this map the interventionalist selects the best and safest route for embolization, taking into account developmental and acquired constraints to blood flow and the presence of any potentially dangerous collateral flow toward normal nervous tissue. Case studies are presented to illustrate the most frequently encountered anatomic variants that may, by their presence within the circulation to be embolized, increase the risk of complication. Attention is directed toward the probability of intraprocedural changes in hemodynamic balance and flow direction, which also may increase risk and which may be modified by catheter techniques and minimized by meticulous attention to detail. This article should support the contention that functional angiography and careful attention to the resulting angiographic map are essential for efficacious and safe head and neck embolization.

The radiologic evaluation of the influence of collateral flow and local developmental and acquired vascular constraints on regional hemodynamics may be termed *functional angiography*. Underlying this concept is consideration of the vascular lesion as it lies within a normal tissue territory. Both the lesion and the territory containing it must be angiographically evaluated before intravascular occlusive therapy is performed. This is accomplished by the development of a functional angiographic map drawn from study of individual superselective injections of all arterial pedicles supplying the territory in question. This article reviews the variable functional anatomy [1, 2] found within the head and neck and the techniques and angiographic protocols used to rapidly and completely evaluate a patient with a vascular lesion in this region. The potentially dangerous collateral networks that connect adjacent vascular territories are illustrated by case studies. It is the purpose of this article to review the basic tenets of functional anatomy and hemodynamic balance so that a clear understanding will promote the use of these principles, thereby resulting in improved safety and efficacy of interventional procedures in the head and neck.

Basic Concepts

While the histologic makeup of a vascular lesion has obvious prognostic and therapeutic importance, the territorial location of the lesion, aside from determining resectability, is a key factor in treatment. The regional arterial pattern and possible collateral routes determine potential risk and efficacy of embolization. As these vary greatly with lesion location, it is essential to consider each region of the head and neck separately for the purposes of functional angiographic evaluation [3]. Each tissue territory is nourished by a constant terminal arterial pedicle (Fig. 1A).
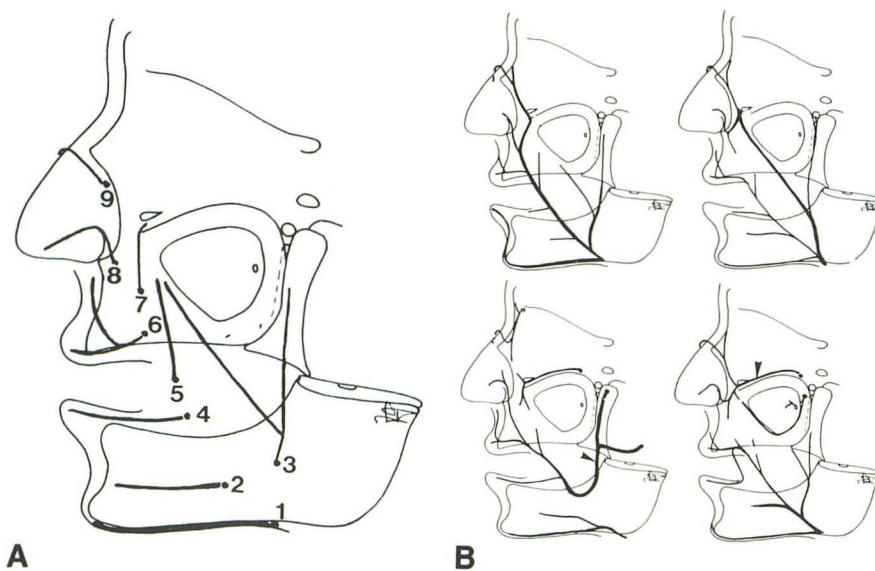
Fig. 1.—**A**, Arterial supply to face, lateral view. Various arterial pedicles that could be annexed by facial arterial trunk are submental artery (1), midmental artery (2), jugal trunk (3), inferior labial trunk (4), midjugal artery (5), superior labial artery (6), anterior jugal artery (7), alar artery (8), and nasal arch (9). **B**, Anatomic variations of facial artery supply. Upper right: long facial variant, its course that of posterior jugal branch. Upper left: middle facial artery variant, its course that of anterior jugal branch. Lower right: hypoplastic facial artery variant. Infraorbital trunk (of IMA) (*arrowhead*) supplies anterior jugal, alar, and nasal arch territories. Lower left: hypoplastic facial artery variant. Facial trunk has annexed only submental pedicle. Rest of face is supplied by masseteric and buccal systems (*arrowhead*). (Reprinted from [3].)

Each pedicle is supplied by a proximal arterial trunk, the course and size of which is determined by the variable elements of vascular regression and recruitment that occur during embryologic development. The structure of the final (adult) main arterial trunk depends on the number of distal pedicles developmentally recruited by that trunk. For example, a dominant facial artery recruits all distal pedicles supplying the facial tissue territories, which may have otherwise been supplied by other main trunks, usually the internal maxillary artery (IMA). Facial artery hypoplasia, on the other hand, signifies recruitment of many of these same territorial pedicles by an alternative branch, again usually the ipsilateral IMA (Fig. 1B). Main trunk patterns vary considerably from person to person and from one side of the head and neck to the other in the same individual. While it is important to be aware of truncal variations, it is perhaps more useful to consistently recognize the constant distal pedicles that supply any given territory. A full understanding of trunks, pedicles, and interconnecting anastomotic channels allows the angiographer to predict which of several arterial injections will demonstrate the territory harboring a lesion when an initial injection fails to supply and thereby define that area.

From second to second, blood flow shifts from trunk to trunk through anastomotic vessels, according to regional tissue demands. The hemodynamic balance between adjacent territories shifts when superimposed vascular constraints (e.g., surgical ligation) alter patterns of flow. Redirection of flow through such collateral channels may be induced by changes in angiographic technique (such as flow arrest) [4], so that reversal of flow toward the lesion may be induced within those channels. Such redirection may permit angiographic visualization of a lesion and its territory by injection of a trunk primarily supplying an adjacent territory, particularly when a constraint reduces flow from the usual route of supply. Reliable demonstration of all contributions to the lesion requires selective angiography. A protocol-guided selective study yields an angiographic map that clearly outlines the abnormality and adjacent normal tissue. Superselective injections are needed so that assessment of supply is not hindered by vascular superimposition, which may confuse the treatment plan. Arterial flow patterns may be studied one by one to confirm which of all possible alternate routes for embolization is best and safest. The map must also be examined for the presence of potentially dangerous flow toward the central nervous system, cranial nerves, or orbit. Such flow may arise from the same arterial trunk supplying the territory containing the lesion [5]. If such supply exists, it must be avoided during the embolization by adjusting the choice of catheters and materials, by intraprocedural catheter flow control, and by more selective catheterization techniques.

Sequences of angiographic injection have been developed for each head and neck tissue territory [6, 7]. Following these protocols can result in significant savings in procedural costs and radiation dosage to patient and physician. Generally the arterial trunks to be injected are those in hemodynamic balance in the territory containing the lesion. Table 1 provides a brief synopsis of common territories and the main trunks contributing to hemodynamic balance. Lateral filming projections are usually sufficient for forming an angiographic map, and subtractions are invaluable for the detection of small vascular stains and dangerous collateral flow.

Hemodynamic Balances

The normal hemodynamic balance between two adjacent arterial trunks is illustrated by example (Fig. 2). Selective injection of the facial artery (Fig. 2A) results in opacification of the IMA via anastomoses that may also function in reverse during maxillary injection (Fig. 2B). The direction of flow through anastomoses depends on many factors, including the selectivity of injection [4], force and rate of injection, local tissue oxygenation, acquired vascular constraints (ligation, embolization), and underlying developmental arterial pattern. Arteries within territories common to two or more adjacent

arterial systems serve as potential collateral pathways that may function only in response to hemodynamic compromise. The potential effects of local constraint are illustrated in a case of external carotid artery (ECA) ligation (Fig. 3). Here as elsewhere in the head and neck ipsilateral collateral flow occurs before contralateral flow. The first or second cervical space collaterals may (in reverse) become dangerous vessels during embolization of lesions supplied by the occipital artery (Fig. 4). (There are eight cervical spinal roots and seven vertebrae. Each root and its corresponding segmental arterial anastomotic branch courses from the spinal canal through a corresponding soft tissue intervertebral "space." The first space lies between occiput and atlas and contains the C1

TABLE 1: **Hemodynamic Balances**

Common Territory	Extracranial Arteries	Intracranial Arteries
Orbit	IMA	Ophthalmic artery
Cavernous sinus	IMA	ICA
First cervical space	OA	VA
Second cervical space	OA	VA
Third cervical space	APh	VA
Suboccipital neck	APh/OA/costocervical and thyrocervical trunks	VA
Palate	APh/IMA/FA	. . .
Cheek	IMA/FA	. . .

Note.—IMA = internal maxillary artery; ICA = internal carotid artery; VA = vertebral artery; APh = ascending pharyngeal artery; FA = facial artery; OA = occipital artery. For detailed analysis, see Lasjaunias [3, 7].
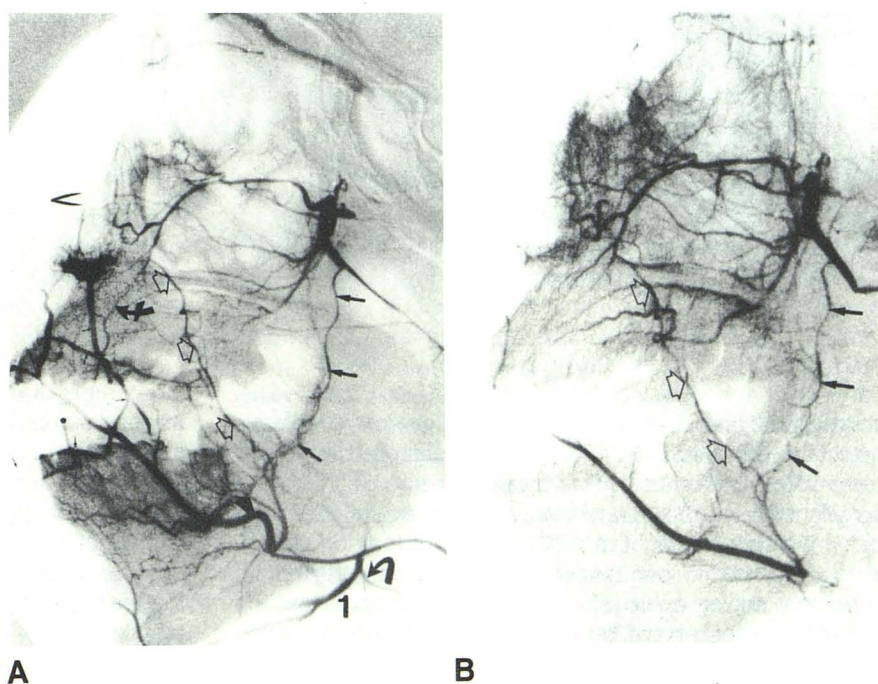
root. Lower spaces are numbered accordingly.)

Ipsilateral collateral flow between the ECA and the internal carotid artery (ICA) may occur between small vessels that pass through foramina at the skull base and anastomose the cavernous ICA with the IMA [8–13]. Enlargement of these channels is inhibited by their restricted passage through these foramina (Figs. 5–9). This is quite different from the situation where collateral routes are not restricted by bone (Fig. 3) [3]. Transosseous ECA–ICA collaterals tend to be functionally important if other pathways have been occluded (Fig. 5) or are insufficient, or the demand by the lesion (sump effect) is marked. Potential collaterals connecting the ECA (IMA branch) and the cavernous ICA include the artery of the foramen rotundum (Fig. 5), the accessory meningeal artery (through the foramen spinosum), and arteries coursing through the foramen lacerum. All of these may anastomose with small branches of the inferolateral trunk of the ICA [8], formerly known as the artery of the inferior cavernous sinus [11], a vessel first described angiographically during investigation of intracranial meningioma [9]. Angiography of the IMA can be performed to demonstrate a lesion supplied primarily by the cavernous ICA because of such anastomoses [8].

When demand for flow exceeds that which can be accommodated by ipsilateral anastomotic flow, particularly if ipsilateral collateral routes are constrained, collateral routes develop contralaterally. Frontal plane arteriographic filming is then required to demonstrate such cross flow (Fig. 7).

In planning interventional therapy, this potential collateral flow must be taken into account. For example, the infraorbital and superior labial regions are territories through which a balance exists between the IMA and the facial artery. Proximal ligation of the facial artery will have little effect on a lesion within these territories, as immediate recollateralization oc-

Fig. 2.—Lateral subtraction arteriography. **A**, Selective facial artery injection fills dominant facial system, and via transfacial anatomoses fills IMA in retrograde fashion. Major collateral routes: buccal (*long arrows*), posterior jugal (*open arrows*), and anterior jugal (*crosshatched arrow*) arteries. Other facial artery pedicles: middle jugal (*short solid arrow*), submenta (1), submaxillary (*curved arrow*), and labial (*small arrows*) arterial pedicles. **B**, IMA injection fills facial trunk through same major anastomotic pathways. (Reprinted from [1].)

A   B

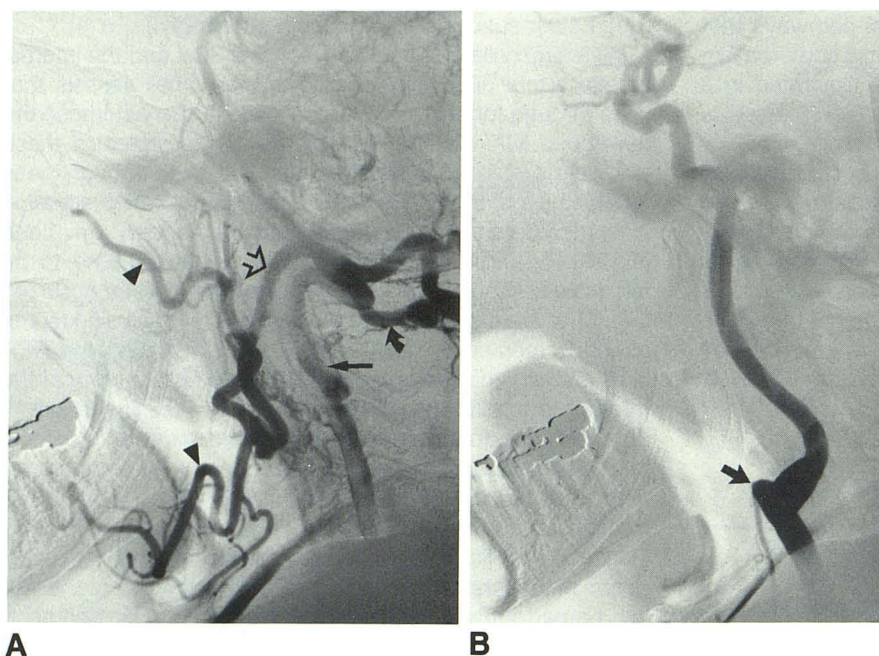**A**   **B**

Fig. 3.—ECA occlusion with reconstitution of flow from vertebral artery via C1 space collateral (suboccipital territory). **A,** Subtracted lateral view of left common carotid arteriogram. Complete occlusion of ECA just beyond superior thyroidal branch (*arrow*). **B,** Left vertebral artery (*solid straight arrow*), late phase. Large C1 space anastomotic vessel (*curved arrow*) provides flow that resupplies occipital artery (*open arrow*). Retrograde flow reconstitutes external artery distal to superior thyroidal, including maxillary and facial branches (*arrowheads*).
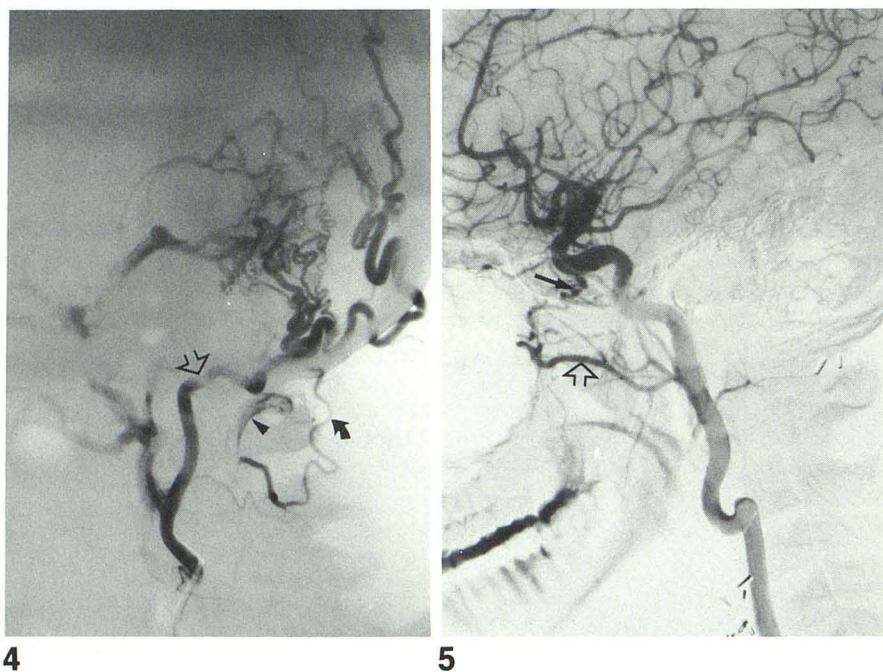

4   **5**

Fig. 4.—C2 collateral from occipital artery to vertebral artery in 38-year-old man with hemicalvarial fibrous dysplasia and dural arteriovenous malformation. Occipital artery (*open arrow*) supplies dural arteriovenous malformation. Opacification of vertebral artery (*arrowhead*) through C2 space collateral (*curved arrow*).

Fig. 5.—Subtracted lateral view of right common carotid angiogram. External carotid and vertebral arteries have been surgically clipped for control of cervical hemangioma. Reconstitution of IMA (*open arrow*) by retrograde flow through inferolateral trunk of ICA (*solid arrow*) via artery of foramen rotundum and accessory and middle meningeal arteries.

curs beyond the point of ligature, via flow from collaterals originating from the IMA. This is why distal embolization is more effective at reducing flow toward a lesion than proximal surgical ligation. Small particulate emboli can penetrate the lesion distal to the point at which a ligature may be placed, and beyond which collateral flow may resupply the lesion. Functional angiographic principles also allow us to test the results of embolization. The lack of opacification of the lesion

after postembolization injection of an occluded trunk is not sure evidence that the lesion itself has been occluded. Lack of such opacification indicates either a good distal occlusion or too proximal a blockage. One must investigate other potential collateral routes to the embolized territory to prove obliteration conclusively. If embolization is then found to be too proximal, and if another vessel is then found to contribute to supply, that vessel may then be used to embolize the
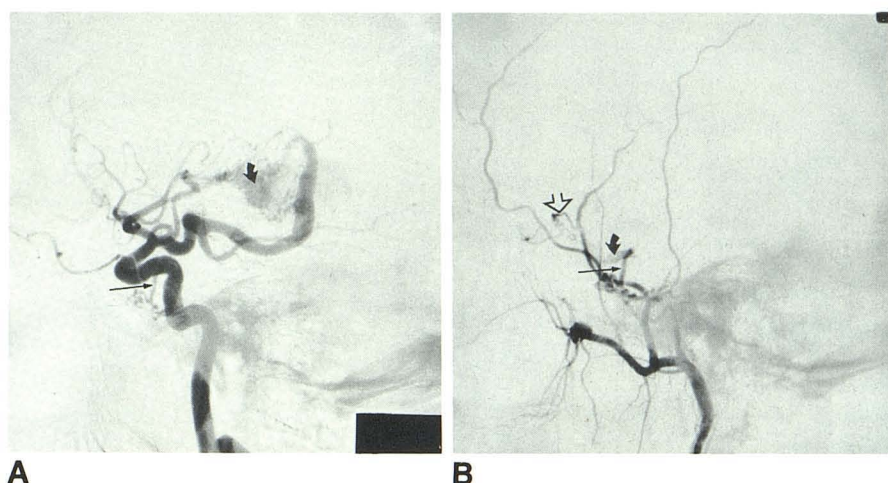
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B

Fig. 6.—21-year-old man with temporal lobe arteriovenous malformation (**A**, *curved arrow*). Bidirectional flow through dilated inferolateral trunk (*solid straight arrows*) in both internal and external carotid arteriograms. **A**, Flow toward external carotid (internal maxillary) system occurs as result of hemodynamic conditions present at time of pressure injection. **B**, Anastomotic branch of MMA courses through foramen spinosum, filling same inferolateral trunk shown in **A**, with blood now flowing *toward* intracranial circulation, as indicated by flash filling of cavernous carotid (*curved arrow*). Also, meningolacrimal variant branch of MMA (*open arrow*) is present. Direction of flow may vary from minute to minute according to cardiac cycle, regional demand, and arterial pressures, which may change during selective angiographic injections.

Fig. 7.—Multiple contraints promote collateral flow from contralateral ECA in 58-year-old woman with large right traumatic arteriovenous fistula just distal to right external carotid ligation. Selective injection (anteroposterior subtracted view) of left IMA shows filling of contralateral right IMA (*open arrow*) by collateral flow from right palatine branches (*solid arrows*).

lesion. *Demonstration of proximal occlusion of a branch just embolized is not proof of how distal, and therefore how effective, the embolization is.*

Intraprocedural Changes in Hemodynamic Balances

The degree of angiographically demonstrable collateral flow may change with angiographic injection pressure and volume, contrast viscosity [4], and the presence of unopacified blood flow from a competing collateral branch. Significantly, from the point of view of safety, hemodynamic balance may change rapidly during embolization. As distal vessel occlusion progresses and territory and lesion are occluded, proximal intraluminal pressure increases. This increases the risk of reflux of embolic material from orifice of the branch and may result in occlusion of normal vessels. This may be prevented by careful fluoroscopic monitoring and staging of the embolization [15]. The increase in intraluminal pressure and local changes in Pco_2 that occur during embolization may also cause opening of and flow reversal in anastomotic vessels that did not opacify at the time of baseline angiography [16] (Fig. 4). If the late-opening anastomotic channel supplies blood to nervous tissue, and the opening is not appreciated fluoroscopically, a neurologic complication may result. It is imperative that the interventionalist be aware of this and refrain from further embolization in such a situation. Furthermore, if the territory to be occluded is known to commonly contain such a dangerous collateral, even if the vessel is not opacified one should consider the probability of its opening during distal occlusion. Techniques of embolization should be adjusted to minimize such risk. *Failure to demonstrate an anastomosis*

angiographically does not rule out its existence or its potential for function under ever-changing hemodynamic conditions encountered during vessel occlusion [7].

Hemodynamics may be controlled to the advantage of the angiographer. The use of a double-lumen balloon catheter or catheter wedging will allow embolization during flow arrest. Flow reversal within the distal territory may be advantageous for safety and efficacy of embolization [7]. In a similar fashion, intracranial flow may be redirected by manual compression of the cervical carotid, a useful technique to demonstrate vascular filling not observed at baseline angiography.

Dangerous Vessels—Neurologic Risks

Stroke

ECA branches may directly or indirectly provide blood flow to the central nervous system, retina, and cranial nerves. Particulate or fluid embolization may result in stroke, blindness, or cranial nerve palsy if such branches fill during occlusive therapy. Frequently occurring intracranial anastomoses and neural pedicles are listed in Table 2. The risk of stroke from the intracranial passage of embolic materials initially introduced extracranially is significant in two territories that serve as interfaces between cranial and cervicofacial regions. The suboccipital and high posterior cervical regions contain anastomotic channels that connect the ascending pharyngeal and occipital arteries with the vertebrobasilar system [5, 16–20] (Figs. 4 and 9). The first and second cervical space communications are almost always present, and should be considered when planning embolization of the occipital artery and the cervical arteries originating from the subclavian sys-
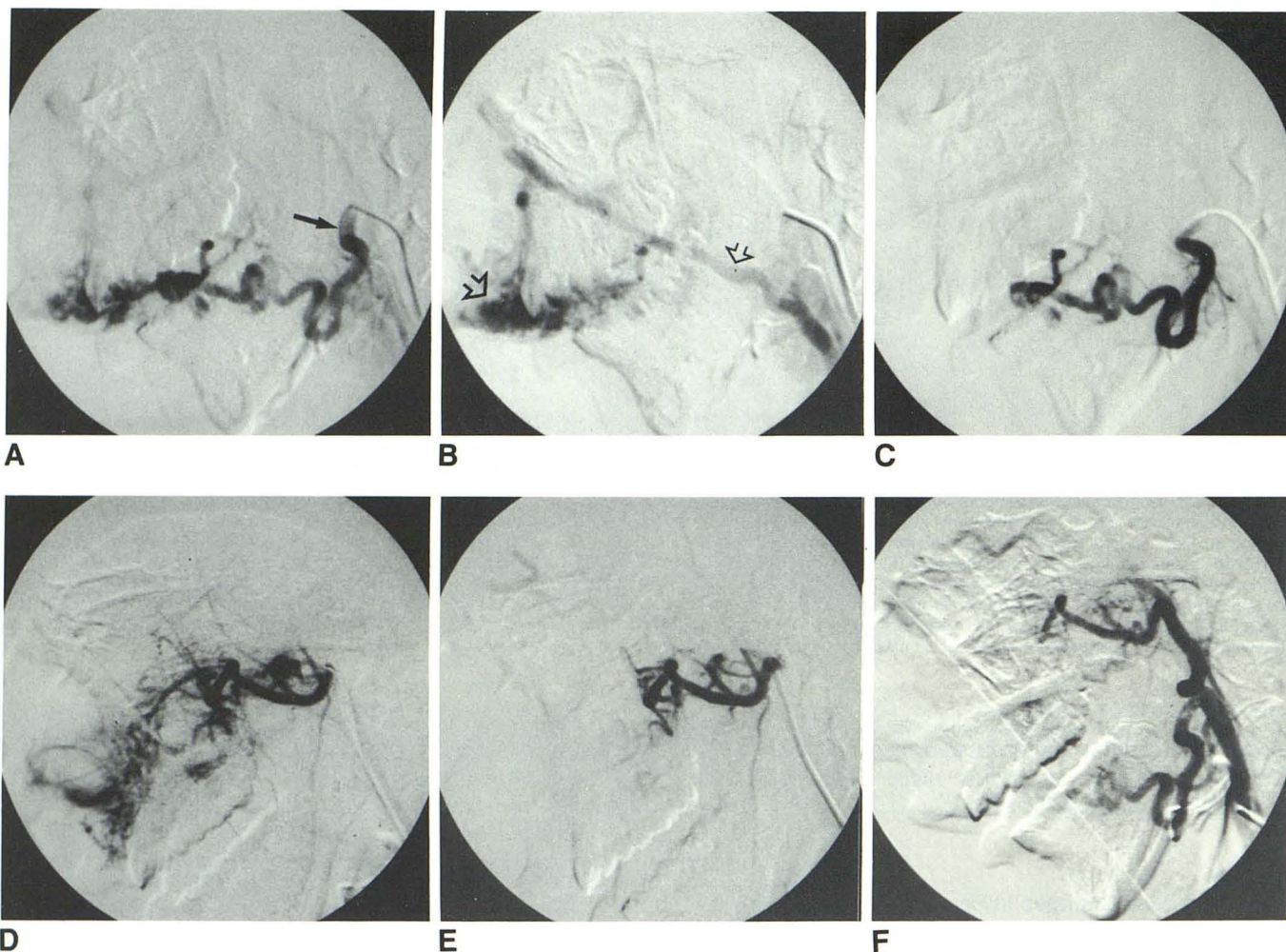
A

B

C

D

E

F

Fig. 8.—15-year-old girl with chronic epistaxis and facial (upper lip) asymmetry due to hemangioma. **A** and **B**, Lateral subtraction right facial artery (*solid arrow*) injection. Vascular malformation of upper lip (superior labial pedicle). Nidus (*large open arrow*) drains via facial vein (*smaller open arrow*). **C**, Repeat of **A** after flow-guided facial artery embolization with PVA microparticles. Proximal facial artery is preserved despite occlusion of labial nidus. **D**, Selective right internal maxillary angiogram after facial artery embolization. Vessel fills portion of labial nidus not previously occluded by facial artery injection and fills deep and posterior portion of lesion. **E** and **F**, Lateral subtraction view of internal maxillary angiogram after microparticle embolization (**E**). Complete occlusion of labial nidus confirmed by ipsilateral external carotid study (**F**) and by final contralateral external injection (not shown). Facial asymmetry had regressed and epistaxis had not recurred at 1 year after therapy. (Reprinted from [14].)
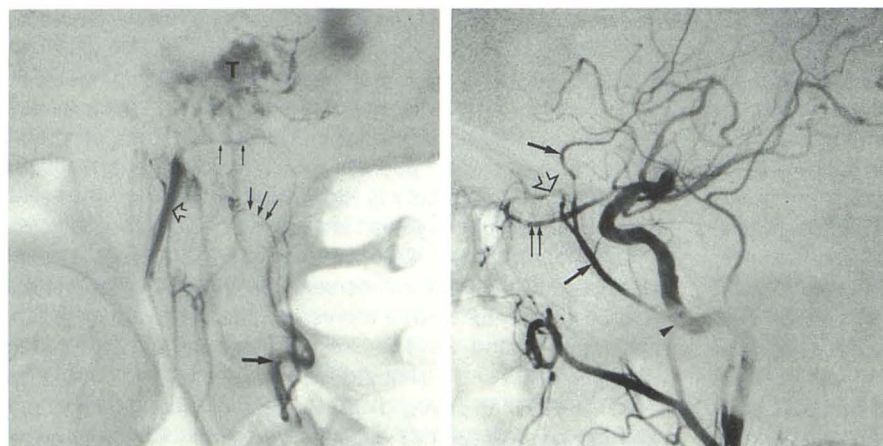

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Fig. 9.—Selective injection of posterior neuromeningeal branch of ascending pharyngeal artery (*open arrow*), lateral view. Hypoglossal branch (*double arrows*) fills tumor (T), and then sends medial descending branch to anastomose with vertebral artery at C3 level (*single arrow*). Note associated odontoid arterial arcade (*triple arrows*). Embolization of ascending pharyngeal supply to tumor was safely accomplished by reversing flow within dangerous hypoglossal-vertebral anastomosis by catheter flow arrest and use of larger microparticles (590–1000 μm size polyvinyl alcohol foam). (Reprinted from [14].)

Fig. 10.—Meningiolacrimal variant. Note origin of ophthalmic artery (*double arrows*) from C2 (supraclinoid) segment of ICA and a lacrimal branch (*open arrow*) of anterior division of MMA (*single arrows*). Note characteristic 90°–100° angle of MMA as it courses beyond foramen spinosum (*arrowhead*). Usual segment of origin of ophthalmic artery is C3 (preclinoid) portion of ICA. Ophthalmic artery may also arise from C4 (horizontal cavernous) portion or C5 (ascending precavernous) segment of ICA.

TABLE 2: **External Carotid Injections: Neurologic Risk**

Branch/Artery	Anastomosis/Neural Supply	Major Risk
Internal maxillary artery:		
Anterior deep temporal artery	Ophthalmic artery	Blindness
Artery of foramen rotundum	ICA (ILT)	Stroke
Accessory meningeal artery	ICA/CNs III–VII	Stroke
Middle meningeal artery:		
Petrous branch	ICA/CN VII	CN VII palsy
Orbital branch	Ophthalmic artery/CN IV, V	Blindness
Ascending pharyngeal artery:		
Carotid branch	ICA	Stroke
Eustachian branch	Mandibular artery	Stroke
Neuromeningeal trunk:		
Jugular artery	CNs VI, IX–XI	CNs IX–XI palsy
Hypoglossal artery	VA (C3 branches)/CN XII	Stroke
Occipital/posterior auricular arteries:		
C1 and C2 branches	VA (C1 and C2 branches)	Stroke
Stylomastoid artery	CN VII	CN VII palsy

Note.—ICA = internal carotid artery; ILT = inferolateral trunk of ICA; VA = vertebral artery; CN = cranial nerve. (Modified from [6].)

tem, as these anastomose. Fluid embolic agents must be used with caution in this territory [5].

As previously noted, the IMA is in hemodynamic balance with the cavernous ICA through anastomoses at the skull base. A common pathway toward the ICA is the inferolateral trunk, which may fill retrograde from several maxillary and meningeal arteries (Fig. 5). Danger of stroke is present during any procedure using fluid agents or microparticles in the IMA, even if the small communications are not appreciated at the time of angiographic mapping.

The superior laryngeal artery arises from the superior thyroidal branch of the ECA and nourishes the mucosa of the larynx and upper digestive tract. Tissue necrosis of these structures, therefore, is a risk of liquid or microparticulate embolization in this vessel.

While ECA anastomotic channels do not normally carry blood toward the ICA, under conditions of raised intraluminal pressure (occlusion angiography and progressive distal embolization) these channels will transmit small particles and liquids. The neurologic risk may be minimized if larger particles are used; however, this choice may lessen the effectiveness of the occlusion. If large communications are seen on review of the map or are observed during fluoroscopic monitoring of the procedure (Fig. 4), prior selective large-particle occlusion of the anastomoses themselves may allow more complete microparticle occlusion of the lesion without further risk. Catheter control of flow may allow redirection of collateral flow away from critical structures, thereby reducing neurologic risk (Fig. 9) [2]. In the unusual circumstance that no safe route exists, the increased risks of embolization must be weighed against the risk of not treating the lesion or of performing surgery without prior embolization.

Visual Loss

ECA embolization may result in central retinal artery occlusion and permanent blindness if anatomic variants of the origin of the ophthalmic artery are present but either not sought or not appreciated on review of the angiographic map. Embryologically, the development of the ophthalmic artery is intimately related to that of the IMA and its middle meningeal

(MMA) and anterior deep temporal branches [21–26]. Many adult variations exist, ranging from annexation of the MMA by the ophthalmic artery to a less common but potentially dangerous variant: ophthalmic artery origin from the MMA or the anterior deep temporal artery (Figs. 10 and 11). In the latter variant there is complete assimilation of the primitive ophthalmic artery by the supraorbital division of the stapedial artery [21]. The anastomosing vessel may be hidden by superimposition of the sphenoid bone, so that high-quality subtracted images may be needed for detection. Alternatively, the presence of a retinal blush on the ECA study may indicate the presence of such an anomaly. Even in competent hands, blindness may complicate embolization, and this risk must be fully considered in appropriate cases. Ophthalmic artery embolization should be avoided even in cases of long-standing blindness in the affected eye, as retinal necrosis may lead to contralateral autoimmune retinal injury in the nonaffected eye.

Cranial Nerve Palsy

The injection of embolic agents within arteries providing nutrient blood supply to cranial nerves may result in acute cranial nerve palsy [27]. Liquid embolic agents may progress more distally within the vasa nervosa and therefore carry a higher risk of lasting deficit than do larger particulate emboli. While most iatrogenically produced nerve deficits fully resolve with time, transient disturbance in function may also be clinically devastating (e.g., IX–XI nerve palsy leading to abnormal deglutition and aspiration) [27]. Therefore, knowledge of the arterial supply to the cranial nerves is essential for full angiographic assessment of the risks of embolization. This information is presented as part of Table 2.

Facial nerve palsy results in a cosmetically disfiguring deficit. Its occurrence is well documented during embolizations of the MMA using liquid embolic agents such as silicone fluid [27]. The MMA gives rise to the petrosal branch several millimeters beyond the foramen spinosum (Fig. 12) [28–30]. This branch is the only arterial supply to the seventh nerve ganglion in 25% of cases, explaining the high incidence of palsy. Other branches that anastomose with the petrosal branch of the MMA form a collateral loop. These include the
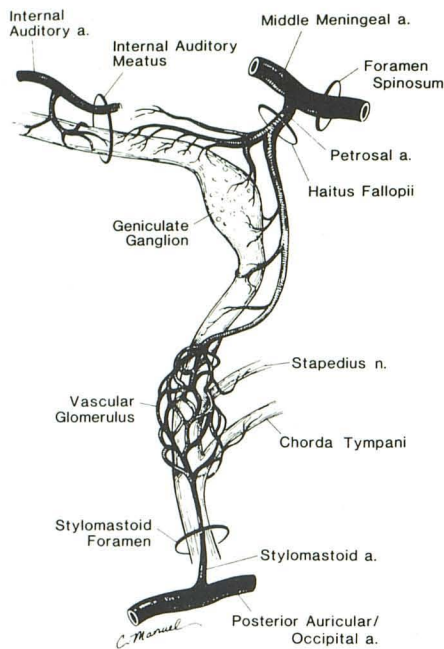
Fig. 11.—Neurologically significant variation in arterial supply to eye. **A,** Common variation, in which only functional supply to retina arises from ICA. Small, nonfunctional connections with MMA may be present, but are not angiographically demonstrated. **B,** Dangerous but rare variation, in which dominant supply to eye arises from meningeal artery. Connection between ICA and retinal artery is not functional. This variant results from ophthalmic artery annexation by embryologic stapedial artery and carries risk of blindness during embolization of maxillary/meningeal arterial system.
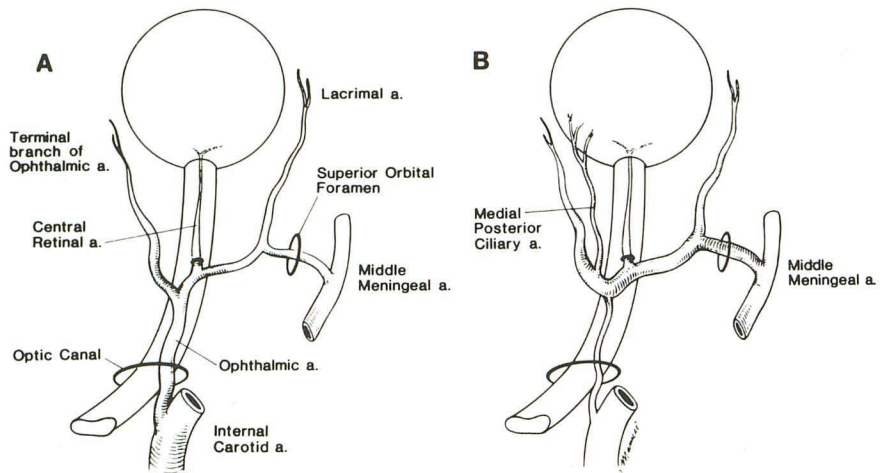
Fig. 12.—Arterial blood supply to seventh cranial nerve. Major anastomotic locus within stylomastoid foramen, between petrosal branch of MMA and stylomastoid artery, a branch of either occipital or posterior auricular artery. Petrosal branch supplies geniculate ganglion, and may represent its only signficant supply.

stylomastoid branch of either the occipital or posterior auricular arteries and the internal auditory branch of the anterior inferior cerebellar artery. The accessory meningeal artery may also supply the geniculate ganglion. The extrapetrous facial nerve (intraparotid) receives supply from the stylomastoid artery, proximal twigs of the occipital artery, and transverse facial branches of the superficial temporal artery. If the petrosal branch of the MMA cannot be avoided during catheterization for selective occlusion of the MMA, such as for devascularization of meningioma, liquid agents should be avoided. Although provocative testing for cranial nerve deficit during at-risk vessel occlusion has been advocated [31], it remains to be proven effective, and the possibility of deficit should be considered in all appropriate situations until such proof exists.

The posterior neuromeningeal branch of the ascending pharyngeal artery supplies blood to lower cranial nerves IX–XII [19]. The lateral jugular division enters the skull through the jugular foramen and supplies IX–XI. This branch may alternately arise from the occipital and anterior cervical arteries. The hypoglossal (medial) branch traverses the hypoglossal canal to supply cranial nerve XII and portions of the posterior fossa meninges. An ascending branch supplies clival dura and a medial branch descends to anastomose with the

vertebral artery, which, at the third cervical space, forms an easily recognizable extradural intraspinal arcade just behind the odontoid process and the body of C3 (Fig. 2). Apart from ischemic injury caused by occlusion of vasa nervosa, palsies of cranial nerves IX–XI (jugular foramen syndrome) may result from neural compression by acute edema within acutely embolized and infarcted tumor. This is more likely to occur with liquid embolization from distension of tumor with fluid agent, and such materials should probably be avoided in this region.

Tissue Necrosis

Tissue necrosis may result if arteries are inadvertently embolized that supply skin or mucosal surfaces. Necrosis of normal tissue is undesirable and may be avoided even during embolization of superficial lesions if certain facts are considered. Liquid embolic agents (bucrylate tissue adhesive and silicone fluids) penetrate distally into terminal arterioles and capillary beds and, therefore, more often cause necrosis than do larger particulate emboli. These materials should be used in superficial tissues only if superselective embolization can be performed to avoid normal circulation or if necrosis is an acceptable clinical consequence of the procedure. Particulate agents such as polyvinyl alcohol foam sponge may be precut to a desired size so that tailored embolization may be performed with particles larger than cutaneous vessels. Although these particles generally carry little risk of necrosis [32], the interventionalist should be aware that blending PVA [33] to increase the ease of injection through small catheters will produce smaller secondary particles that may enter cutaneous circulation and result in tissue necrosis (personal observation).

Compartmentalization [7, 34]

Vascular lesions are composed of one or many separate compartments. Lesions with a single vascular compartment

may be fed by one or by more than one arterial pedicle, and injection of any one pedicle may or may not result in complete opacification of the lesion. When a lesion fed by more than one pedicle fully opacifies after a single injection, it is considered to be unicompartmental, and embolization can at least in theory be performed by embolization through a single pedicle. Incomplete or too-proximal occlusion of one feeder, however, will result in recollateralization of the entire lesion, and the lesion must then be occluded via an alternate pedicle.

Multicompartmented lesions cannot be devascularized fully by embolization via one pedicle, and many individual arteries may need to be occluded for obliteration of the entire lesion. However, if a clinical syndrome or symptom can be attributed to one of the compartments, embolization can be directed to that compartment alone and a "partial" embolization may result in clinical success [14]. Such limited goals based upon careful clinical evaluation and correlation of the patient's problem with angiographic appearances may result in long-term benefits while limiting patient risks. In short, all regional arterial pedicles should be selectively injected before intervention for full evaluation of the nature of lesional compartmentalization.

Summary

In summary, developmental and acquired vascular constraints may affect the hemodynamics of territorial blood flow between adjacent major arterial trunks, through connection of distal arterial pedicles at points of collateral flow. Such changes may be induced during progressive embolic obliteration of a distal vascular bed, increasing the risk of the procedure. Familiarity with functional anatomy and the judicious use of functional angiography will allow selection of the best and safest route for embolization, and will also allow the circumvention of acquired constraints, improving procedural efficacy and decreasing risk.

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