16 Neurointervention and the Otolaryngologist: Head and Neck Surgeon

Randall C. Edgell and Jastin Antisdel

Introduction

The intersection of head and neck surgery and neurointervention dates to the turn of the twentieth century [1] when the endovascular devascularization of a tumor was first attempted. As neurointervention evolved toward greater safety with the introduction of the Seldinger technique [2] and the transfemoral approach [3], its application to disorders of the head and neck also increased. Preoperative tumor embolization is now widely applied. Endovascular treatment of epistaxis lessened the need for open vascular ligation [4] and is now used alongside endoscopic vessel ligation to treat refractory cases. In addition to these applications, neurointerventional techniques have been successfully applied to temporary and permanent vessel occlusion, treatment of carotid blowout syndrome, and treatment of iatrogenic vessel injuries to the head and neck.

R. C. Edgell
Department of Neurology and Psychiatry, Saint Louis University, St. Louis, MO, USA
e-mail: randall.edgell@health.slu.edu

J. Antisdel (✉)
Department of Otolaryngology – Head and Neck Surgery, Saint Louis University, St. Louis, MO, USA
e-mail: jastin.antisdel@health.slu.edu

Tools Review (See Chap. 1)

Coils

The coil consists of two components: a fine, soft, metal helix often made of platinum and a steel pusher wire. For intracranial application, these components are typically connected and must be mechanically or electrolytically detached. This connection allows for repositioning or removal of coils when the initial deployment is unsafe. For extracranial applications in which repositioning is rarely necessary, "pushable" coils are sometimes used. These coils are not attached to the pusher wire, and once deployed may not be retrieved. "Feathered" coils are not generally utilized intracranially, due to the perceived increased risk of thromboembolic complications. These coils are more often employed extracranially as the risk of cerebral ischemia is minimal, and their feathering may promote the desired vascular occlusion more rapidly.

Coils may be utilized in the treatment of a variety of head and neck disorders. They are perhaps most frequently used to sacrifice large cranial vessels in a controlled manner. They are less effective in devascularizing the nasal cavity or a tumor bed as they do not penetrate the microvasculature that is the target in these circumstances.

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Particulate Embolics

Polyvinyl alcohol particles (PVA; Boston Scientific, Boston, MA) and tris-acryl microspheres (microspheres) are the most frequently used particulate embolics in the modern treatment of head and neck pathology. They are available in a range of diameters, allowing control over the depth of vascular penetration. Gelfoam pledgets have also been utilized but are not as effective in microvascular penetration and therefore provide less complete devascularization of tumor beds and the nasal cavity.

Liquid Embolics

Liquid *n*-butyl cyanoacrylate (*n*-BCA) (Trufill, Codman Neurovascular, Raynham, NJ) and ethylene-vinyl alcohol copolymer (EVOH) (Onyx, Covidien, Irvine, California) have both been utilized to treat a variety head and neck disorders. They are effective in treating extracranial vascular malformations, with EVOH gaining favor in recent years due to its ability to occlude large portions of these malformations. They have also been successfully utilized in the embolization of epistaxis and tumors.

Stents: The use of stent technology in extracranial head and neck disease is relatively uncommon. Radiation-induced carotid stenosis is one condition for which stenting is indicated. The stents utilized in this setting are self-expanding nickel-titanium alloy (Nitinol). For iatrogenic vascular injuries or carotid blowout, balloon-mounted stents with a microfilament layer (covered stents) may be utilized.

Balloon Test Occlusion

Indications

Giant aneurysms (see Chap. 10): These ≥25 mm aneurysms often involve the intracranial carotid artery. The open surgical approach to these lesions, while increasingly rare, may involve a skull base approach, requiring collaboration between the head and neck surgeon and vascular neurosurgeon. Direct clip ligation of the aneurysm is often prohibitively risky due to adjacent cranial nerves, bony structures, and involvement of large portions of the parent vessel wall. Under these circumstances, vessel sacrifice may be contemplated through either open or endovascular means (discussed under section "Carotid Blowout Syndrome" and "Case Study 1: Carotid Blowout"). Surgical occlusion involves trapping of the aneurysm through clip application to the cervical carotid artery proximal to the aneurysm and clip application distal to the aneurysm to prevent retrograde filling.

Prior to undertaking these technically challenging and high-risk procedures, it is important to test the competence of the circle of Willis through balloon test occlusion (BTO). Patients who fail the BTO may require extracranial to intracranial bypass surgery prior to vessel sacrifice.

Skull base tumors: Examples of these lesions include pituitary macroadenomas, craniopharyngiomas, meningiomas, chordomas, and esthesioneuroblastomas. They often are adjacent to, and at times surrounding, arterial structures, making vessel sacrifice intraoperatively possible or, at times, unavoidable. Again, it is essential to test the collateral circulation via BTO prior to surgical resection.

Cervical tumors: Vascular encroachment or encasement of internal carotid artery is most commonly associated with metastatic lymphadenopathy from head and neck squamous cell carcinoma or the direct extension of the primary tumor. A variety of less common tumors may also present in this manner. These tumors may be malignant with rare histologies such as chondrosarcoma, papillary carcinoma (thyroid), adenocarcinoma, or undifferentiated carcinoma. Benign neoplasms of the neck including carotid body tumor, vagal paragangliomas, and peripheral nerve neoplasms (e.g., schwannomas or neurofibromas) are other lesions which can cause carotid artery encroachment. It is imperative to include angiography and BTO in the preoperative

workup of patients with strong clinical or radiological suspicion of vascular encasement from cervical tumors. The information from these two interventions helps to assess safety of carotid ligation and resection, which may be required for complete tumor removal if encroachment of the artery by malignant disease is 270° or more.

General Technique

The BTO is performed with no sedation in order to ensure that the patient's neurological function is not pharmacologically impaired. Local anesthesia is applied followed by puncture of the femoral artery. Some neurointerventionalists prefer to utilize the femoral artery ipsilateral to the artery to be occluded. This eliminates the possibility that vascular injury or insufficiency associated with the femoral access site will be confused with a neurological deficit induced by balloon inflation. A diagnostic catheter is then used to perform a four-vessel angiogram. The patency of the circle of Willis is carefully evaluated. When no visible filling across the anterior communicating artery is present, manual carotid compression with firm dye injection may make this collateral evident. Next, systemic heparin is administered to achieve an activated clotting time >250 s. A 6 French guide catheter is then navigated into the target vessel. Through this catheter, a compliant nondetachable balloon-tipped catheter is navigated into the petro-cervical carotid. The L-shaped curve of the artery at this location reduces the tendency for distal migration of the balloon caused by the arterial pressure within the cervical carotid artery. Among modern balloon types, the Hyperform (ev3) 7 × 7 mm and Scepter (Codman) balloon catheters are frequently employed. The Scepter has the advantage of being a dual-lumen catheter. This allows for removal of the microwire and administration of continuous heparinized flush distal to the balloon. Next the balloon is inflated, and angiography is performed to ensure the balloon is occlusive. At this point, it is useful to visualize the external carotid artery to look for external to internal collaterals and to assess the caliber of the superficial temporal artery (often utilized for external to internal bypass). Balloon inflation should be maintained for a minimum of 20 min. The patient's neurological function should be assessed in a standardized manner and at regular intervals of 2–5 min during balloon inflation. This examination often involves questioning of the patient for subjective symptoms, level of alertness, orientation, visual fields, language function, facial expression, and motor examination (drift and grip strength in upper extremities; movement of toes in lower extremities).

Predictive Value

When a circle of Willis collateral to the occluded carotid (posterior or anterior communicating artery) is angiographically visible, no neurological symptoms are noted during the period of occlusion, and cortical venous drainage is visible with less than a 1-s delay between hemispheres (venous phase assessment), the patient is said to have "passed" the balloon test occlusion. In one study, only 2% of patients who passed their BTO went on to develop a neurological deficit postoperatively [5].

A variety of attempts have been made to improve upon the positive predictive value of the findings above, including stump pressure measurement [6, 7], induced hypotension [8, 9], single-photon emission computed tomography (SPECT) imaging, CT perfusion imaging with acetazolamide challenge [10], xenon CT perfusion imaging [11–13], MR perfusion imaging [14, 15], transcranial Doppler [16], and neurophysiological monitoring [17, 18]. While some of these methods increase the sensitivity of the test, they have not been shown to improve surgical outcome.

Risks

The primary risk associated with BTO is thromboembolism. There is stasis of blood flow

proximal to the balloon within the carotid artery and decreased/turbulent flow distal to the balloon. This risk may be reduced through the use of systemic heparin as described above. In patients in whom surgery is not imminent or in whom endovascular vessel sacrifice is anticipated, aspirin may be considered. Additionally, balloon inflation may be associated with vessel dissection. If balloon inflation is performed at the carotid bulb or within the common carotid artery, it may trigger reflex bradycardia, hypotension, and in extreme cases, asystole. While this should not occur if the balloon is inflated in the location described above, it is advisable to be prepared for the possibility.

Technical Variations

Variations on the general approach to BTO include positioning of the balloon across the ophthalmic artery rather than at the petro-cervical junction. The purpose of this maneuver is to eliminate the possibility of external to internal carotid collateral recruitment that may cause a falsely normal test. Rarely, proximal basilar artery occlusion may be contemplated to treat large or giant basilar artery aneurysms or posterior fossa tumors. In these cases, the BTO is performed with the balloon in the proximal basilar artery. Some institutions have utilized the balloon guide catheter (Stryker Neurovascular) designed for use with the Solitaire thrombus retrieval system. The advantage of this catheter is that it allows robust delivery of heparinized saline distal to the balloon. The disadvantage is its large diameter and associated risk of vascular injury or baroreceptor stimulation.

Carotid Blowout Syndrome

Causes

Carotid blowout syndrome denotes rupture of the extracranial carotid artery or its major branches in the neck. It is mostly associated with local spread, treatment, or recurrence of squamous cell cancers of the head and neck. These tumors may arise from the nasopharynx, oral cavity, oropharynx, larynx, or the hypopharynx. Advanced, fungating tumors may directly encroach and at times invade the carotid artery, leading to imminent rupture. Though uncommon, carotid blowout is one of the most dreaded complications after surgery for advanced head and neck carcinoma. Surgical resection may involve radical (en bloc removal of the lymph node-bearing tissues on one side of the neck, as well as the removal of the spinal accessory nerve, internal jugular vein (IJV), and sternocleidomastoid muscle) or modified radical neck dissection (dissection with the goal of preserving the IJV, the sternocleidomastoid muscle, or the spinal accessory nerve). Impaired healing and wound breakdown are the main postoperative complications that precede carotid blowout. Predisposing factors for these postoperative complications are from infection, tumor recurrence, the use of vertical limb and three-point junction incision for radical neck dissection, rough handling of the vessel adventitia during surgery, flap necrosis, and systemic factors such as malnourishment, anemia, and hypoproteinemia [19]. Pharyngocutaneous fistula is another known postoperative complication with a high risk for carotid rupture due to exposure of the carotid wall to direct salivary contamination and damage from salivary enzymes. Finally, treatment protocols including radiotherapy play an etiological role. Radiation has been associated with a sevenfold increased risk of carotid rupture in head and neck cancer [20, 21]. Thrombosis of the vasa vasorum, adventitial fibrosis, and fragmentation of tunica media elastic fibers following irradiation of carotid sheath lead to weakening of the vessel wall and subsequent rupture [22]. Carotid blowout will be accelerated in this setting if there is chronic carotid exposure or can occur if there is delayed wound healing since radiation also increases postoperative healing complications and pharyngocutaneous fistulae, thus further predisposing to the risk of carotid blowout. Blunt or penetrating trauma to the neck is also reported in the literature as a rare cause of carotid blowout [23].

Epidemiology

Over 550,000 patients are diagnosed with head and neck cancers annually worldwide [24]. Carotid blowout has been reported to occur in 3–5% of patients with major head and neck resections [25]. Among patients with head and neck squamous cell carcinoma, up to 20% require radical neck dissection, and this procedure is associated with a 4% incidence of carotid blowout [26]. The reported rates vary across series, but the average mortality estimate from carotid blowout is 40% (9–64%), and severe neurological deficit occurs in about 60% (9–84%) [25, 27].

Clinical Presentation

Chaloupka et al. [27] have described three entities that fall within the carotid blowout syndrome:

- Exposed carotid: In which there is wound breakdown and direct exposure of the carotid artery postsurgical resection of tumor with or without irradiation. There may also be evidence of tumoral invasion of the carotid sheath or asymptomatic pseudoaneurysm of the carotid artery.
- Impending blowout: In which there is a "sentinel" hemorrhage presenting with nasal, oral, or transcervical hemorrhage that is self-limited. This may be due to an angiographically evident pseudoaneurysm or tumoral erosion into the vessel. Highly variable duration of sentinel bleeding from moments to months prior to hemorrhage has been described. Patients at high risk for potential blowout should be counseled to report such occurrences. Precautionary measures such as protection of the airway with a cuffed tracheostomy tube and cross-matching in anticipation of blood transfusion are recommended.
- Acute carotid blowout: Presenting with uncontrolled nasal, oral, or transcervical hemorrhage. There is often massive blood loss with the need for manual compression of the carotid and hemodynamic resuscitation.

Indications for Invasive Treatment

An *exposed carotid* requires adequate coverage of the weakened vessel wall with mobilization of a tissue flap and attempted wound closure to prevent both early and delayed rupture. Skin or fascial grafts are inadequate. Local flaps consisting of skin and subcutaneous tissue are required to provide sufficient healthy tissue for carotid coverage, particularly in post-irradiated patients. The most reliable protection is provided by pedicled muscular flaps composed of well-vascularized muscle. If there is a skin defect overlying the carotid, a pedicled myocutaneous flap is recommended which comprises the overlying skin, subcutaneous fat, and muscle. The most commonly utilized flap to prevent carotid rupture flaps is a pectoralis major myocutaneous flap which is mobilized from the chest and rotated to the neck. Less commonly used are latissimus dorsi and deltopectoral flaps. The muscle paddle of the flap is sutured to the tissue around the carotid, while the skin overlying the muscle is sutured to the edges of the cutaneous defect in the neck, thus providing complete carotid protection. In patients with pharyngocutaneous fistula with carotid exposure, reconstruction with the myocutaneous flap helps seal the salivary leak and promotes revascularization of the vessel wall, thus preventing rupture of the carotid.

When tumor invasion of the carotid or pseudoaneurysm formation is present, it is reasonable to proactively plan for possible carotid sacrifice. This would involve an elective cerebral angiogram to localize the arterial defect if present (common carotid, external carotid, or internal carotid) and assess collateral circulation. A BTO would also be helpful to assess the ability to safely sacrifice the affected vessel. The decision to prophylactically sacrifice the involved vessel could then be based on a thorough understanding of the clinical condition of the patient, native collateral circulation, and extent of tumoral involvement of the artery.

For patients with an *impending blowout* or *acute blowout*, the need for invasive treatment is more clear-cut. The difference in approach to these two entities is related to the speed with

which treatment must occur. In cases of impending blowout, the steps of workup may be more measured with reliance on more technically elegant vessel-sparing strategies such as stent-assisted pseudoaneurysm coiling and covered stent placement. For acute blowout syndromes, where control of bleeding must be obtained within minutes rather than hours, vessel sacrifice may be the only feasible option.

Risks

The most pressing risk associated with acute carotid blowout syndrome is the failure to control bleeding in time to prevent exsanguination and death due to hypovolemic shock. Vessel-preserving strategies may paradoxically increase this risk in the short term as they often involve the use of stent technology and the associated need for antiplatelet agents. Vessel occlusion and the use of stents without antiplatelet agents carry a risk of thromboembolism and ischemic stroke.

Surgical Approach

Emergent treatment of carotid blowout requires expeditious transfer of the patient to the operating room for definitive control. Traditional surgical intervention has been exploration of the neck and ligation of the bleeding vessel. Pressure is maintained at the site of hemorrhage until the vessel is exposed. Skin incision is planned to achieve the best exposure of the carotid artery along its entire length in the neck. In previously operated patients, the incision is made along the scar and extended as required. Skin flaps are elevated followed by identification of the site of bleeding. Sternocleidomastoid muscle (in patients with no history of radical neck dissection) is retracted laterally to expose the carotid sheath. Dissection in postoperative cases is complicated due to distortion of the anatomy by edema, granulation, or necrotic tissue. Once identified, the common carotid artery is carefully dissected to obtain vessel control both proximal and distal to the bleeding site. If the site of rupture is in the internal or common carotid artery, vertical sutures are preferred instead of horizontal sutures to prevent compromise of the cerebral blood flow [25]. Such ligatures may not always be sufficient, and there may be a need to ligate or to resect the carotid artery. Reconstruction of the artery with interpositional grafts with Gore-Tex or saphenous vein may be undertaken. Reconstruction is avoided in the presence of infected or irradiated tissue due to increased risk of postoperative disruption. Hemorrhage may also occur from blowout of the external carotid artery or one of its major branches in the neck; these are usually safe to ligate unilaterally. Pharyngocutaneous fistula, if present, should be repaired, and salivary diversion be created as required. It is, however, rare that the fistula can be repaired directly. The safest option is repair of the pharyngeal defect with a myocutaneous flap. This reconstruction will also bring vascularized muscle into the wound to cover the ligated vessels. Pharyngeal diversion may then be established with wound packing and a salivary bypass tube. All necrotic or infected tissue is thoroughly debrided, and swabs are sent for microbiology. The wound is copiously irrigated after ensuring complete control of the hemorrhage. Vascularized muscular or myocutaneous flaps are mobilized to cover the wound especially in irradiated necks and in patients with pharyngocutaneous fistula.

Ligation of the internal or common carotid artery is a rapid technique to secure bleeding in carotid blowout, but the rate of neurological sequelae and death is high in patients with insufficient collateral circulation at the circle of Willis. Lower cerebral ischemic complication rates are reported in patients who are hemodynamically stable prior to the surgery [28]. Therefore, vigilant monitoring of vitals and maintenance of adequate blood pressure in the perioperative period is paramount. Surgical ligation should be ideally preceded with BTO and cerebral angiography, but the unstable disposition and the emergent need to prevent death from exsanguination usually do not allow planning for such interventions. Newer endovascular techniques, however,

have been developed for management of carotid blowouts with lesser complications versus open surgery and are discussed below.

Endovascular Approach

- *Vessel sacrifice*: While at first blush, vessel sacrifice may appear to be the least sophisticated treatment for carotid blowout syndrome, there are in fact several challenges that must be overcome. Although time is short in most cases, it is important to gain a rapid assessment of the circle of Willis. This will allow assessment of the risk of hemispheric stroke after vessel sacrifice. The next goal of treatment is to significantly slow the loss of blood in acute blowout. To this end, it may be useful to position a nondetachable balloon proximal to the point of vessel rupture, or across the rupture site itself. At this point, detachable coils are used to permanently occlude the vessel as they are the most controllable tool currently available in the neurointerventional armamentarium. Despite this relative control, coil migration is a risk in the early stages of vessel sacrifice, especially in large-caliber vessels such as the common carotid artery. Proximal balloon inflation will serve to reduce or eliminate anterograde flow, while the first coils are positioned and deployed. Using oversized, longer coils will further reduce the risk of coil migration intracranially, as will anchoring of the first coils in the rupture point of the vessel. Also, key in this endeavor is occluding the vessel distal to the rupture point and working backward to a position proximal to that point. This strategy avoids the risk of retrograde filling of the occluded vessel and continued blood loss. While coils alone may achieve complete vessel occlusion, the matrix they create is not occlusive in all cases, especially in large-caliber vessels. In these cases, the high-density version of EVOH (Onyx 34) may be injected once significant flow reduction has been achieved to completely eliminate flow.
- *Stent-assisted coiling* (see Chaps. 10 and 11): In cases of exposed carotid with pseudoaneurysm formation or impending rupture with pseudoaneurysm formation, stent-assisted coiling is an effective treatment option. The safe use of stent technology requires dual antiplatelet therapy, and in controlled circumstances, this can be achieved via oral administration of aspirin (81 or 325 mg) and clopidogrel (300 mg loading dose followed by 75 mg daily), as long as there is no active bleeding and treatment is planned in close proximity to antiplatelet administration. An alternative strategy is to utilize IV antiplatelet agents (e.g., eptifibatide or abciximab) immediately after stent deployment followed by administration of oral agents at the conclusion of the procedure. The latter approach reduces the interval of time between platelet inhibition and pseudoaneurysm occlusion.
- *Covered stent placement*: The covered stent offers the potential for quick control of blood loss while preserving vessel patency. It requires the ability to navigate a large-diameter guide catheter or sheath (8F or larger) proximal to the point of rupture. These devices tend to be more stiff and difficult to navigate than uncovered stents, and therefore the target vessel must be free of significant tortuosity. The larger surface area created by the stent membrane also increases the risk of thromboembolism, making the use of antiplatelet agents more pressing. It is essential to oversize the stent diameter and allow for a substantial landing zone on either side of the rupture point to ensure complete occlusion of the lesion.

Case Study 1: Carotid Blowout

A 62-year-old man with a history of squamous cell carcinoma of the larynx presented with hypovolemic shock and bleeding from tracheostomy, nose, and mouth. He was found to have a recurrent mass with erosion into the esopha-

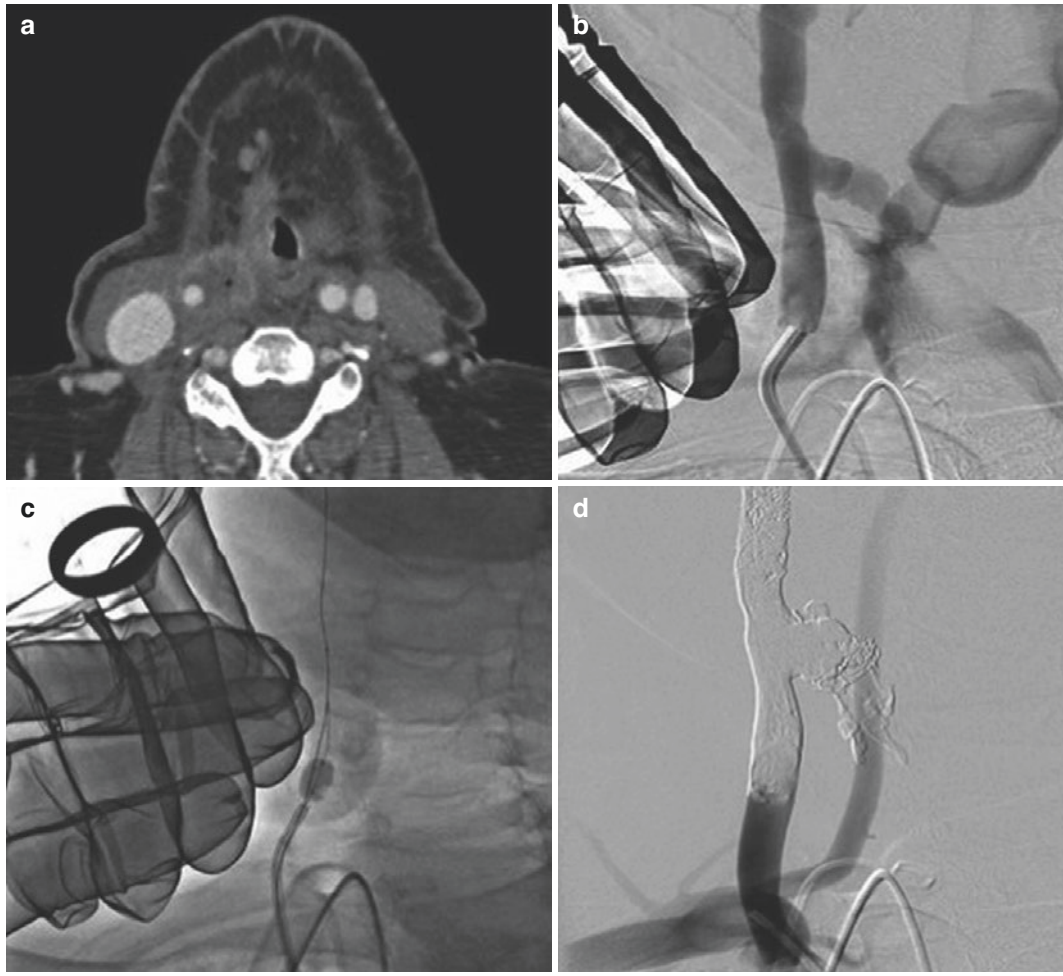

Fig. 16.1 Carotid blowout. (**a**) Contrast cervical axial CT scan showing recurrent laryngeal cancer encasing the right carotid artery (*arrow*). (**b**) Pretreatment angiogram showing a carotid-esophageal fistula with manual compression being applied. (**c**) Inflation of a balloon in the common carotid artery proximal to the fistula to slow flow and reduce blood loss. (**d**) Carotid occlusion with coil deployment distal, within the fistula, and proximal to the fistula with EVOH deposition to complete the occlusion

gus and common carotid, creating a fistulous connection. While undergoing emergent hemodynamic resuscitation with manual compression being applied to the common carotid artery, the patient was brought to the interventional suit. Diagnostic angiography revealed a patent anterior communicating artery. A proximal balloon catheter was inflated to reduce bleeding and allow coiling placement within the vessel. Onyx 34 was used to seal the coil matrix. The patient was successfully resuscitated and stabilized (Fig. 16.1).

Epistaxis

Epidemiology

Idiopathic epistaxis affects at least 60% of the adult population during a lifetime; however, only 6% of epistaxis cases require medical attention. Males and females are equally affected with an increase in frequency over the age of 40. Most cases arise from the anterior septal area; however, 5% of cases arise more posteriorly and are difficult to control [29].

Associated Conditions

- Hereditary Hemorrhagic Telangiectasia (Osler–Weber–Rendu syndrome): A rare autosomal dominant, systemic disease in which epistaxis is caused by rupture of telangiectasias and is often refractory to treatment. While embolization will control an acute episode, symptoms generally recur over time [30].
- Eroding cavernous carotid aneurysms (see Chaps. 10 and 11): Large and giant aneurysms of the cavernous carotid artery may erode into the sphenoid sinus, rupture, and present with epistaxis [31].
- Arteriovenous malformation or fistula (see Chaps. 12 and 13): A rare cause of epistaxis but is the subject of several case reports [32].
- Trauma: Traumatic maxillofacial injury is sometimes associated with laceration of branches of the external carotid artery and massive oronasal blood loss. More rarely, skull base fractures are associated with laceration of the cavernous carotid artery. Generally, this leads to symptoms of carotid cavernous fistulae (see Chap. 15). However, when there is rupture into the sphenoid sinus, oronasal bleeding may be seen [33].
- Sinonasal neoplasm: Juvenile nasopharyngeal angiofibroma (see below) is the neoplasm most commonly associated with epistaxis. Other tumors that may present in this fashion include hemangioma, hemangiopericytoma, acute myelogenous leukemia, pyogenic granuloma gravidarum, nasopharyngeal carcinoma, esthesioneuroblastoma, malignant fibrous histiocytoma, adenoid cystic carcinoma, and metastatic disease [34].

Stepwise Treatment Algorithm [29]

1. Nasal pressure.
2. Topical hemostatic and vasoconstricting agents.
3. Anterior packing.
4. Reversal of underlying factors (i.e., platelet inhibition, anticoagulation, or hypertension).
5. Endoscopic cauterization: chemical, e.g., silver nitrate or electrocautery.
6. Posterior packing with inpatient observation.
7. Endoscopic or endovascular ligation of arterial supply to posterior nasal fossa.

Angiographic Assessment

During the angiographic assessment of epistaxis, selective catheterization and angiography should be performed in bilateral internal and external carotid arteries. At least one vertebral artery injection may be useful to look for evidence of dural arteriovenous fistula or arteriovenous malformation.

Imaging of the internal carotid artery will rule out rare causes of epistaxis including aneurysms, AVMs, DAVF, or traumatic carotid cavernous fistula. In addition, the ophthalmic artery provides important vascular supply to the superior nasal cavity via the anterior and posterior ethmoidal arteries. These arteries are not considered targets for endovascular therapy due to the risk of inadvertent embolization of cerebral or ophthalmic vessels.

Assessment of the external carotid artery is particularly important as the target vessels for endovascular treatment arise from it. Moving from the anterior to posterior nasal cavity, these include the superior labial artery (a branch of the facial artery) and the greater palatine and sphenopalatine arteries (branches of the internal maxillary artery). It is also crucial to look for angiographically subtle external to internal anastomoses through the angular branch of the facial artery and branches of the internal maxillary artery (vidian, accessory meningeal, and middle meningeal). At times it may be feasible to remove nasal packing during angiography to look for extravasation of contrast and localize the side of bleeding more precisely.

Surgical/Endoscopic Treatment

Surgical treatment is indicated in patients with epistaxis who continue to bleed despite conservative measures including decongestants, application of cautery, hemostatic agents, and short-term nasal packing. The origin of such intractable epi-

staxis is difficult to visualize and usually arises from the vessels in the posterior and superior nasal cavity, most commonly the sphenopalatine artery. Surgical intervention provides prompt treatment in these situations. It not only prevents pressure necrosis and infections in the nasal cavity but also reduces the hospital stay from prolonged packing. The nature of surgical interventions has evolved both in the technique and the target of ligation from open ligation of the external carotid artery (ECA) to transantral ligation of internal maxillary artery (IMA) to the endoscopic ligation of the sphenopalatine artery (SPA).

Ligation of the ECA through neck exploration is associated with risk of inadvertent injury to the hypoglossal nerve. It can also cause ischemic complications in atherosclerotic patients whose cerebral circulation is dependent on the external to internal carotid system anastomoses. Open ligation of ECA has been replaced by transantral ligation of IMA that is performed through a Caldwell-Luc approach. In this approach, a window is created through the anterior surface of the maxillary sinus via a gingivobuccal sulcus incision. Through this transantral window, the IMA is ligated in the pterygopalatine fossa which lies posterior to the maxillary sinus. A complication rate of 25–30% is associated with this technique and mainly includes oroantral fistula, cheek and dental anesthesia, and injury to the nasolacrimal duct.

The most favored approach currently is endoscopic ligation of SPA at it has less postoperative complications compared to transantral ligation of IMA [35]. A detailed endoscopic examination of the nasal cavity is performed under general anesthesia after adequately preparing the nasal cavity with topical decongestants. Nasal mucosa and the greater palatine canal (optionally) are injected with 1% Xylocaine with 1:100,000 epinephrine for additional vasoconstriction. The middle turbinate is medialized and followed to its posteriormost aspect. The sphenopalatine foramen is situated just inferior to the posterior end of the middle turbinate and is accessed by a vertical mucoperiosteal incision on the lateral nasal wall. The SPA is clipped and/or cauterized as it exits the foramen and the mucosal flap is reapproximated, thus completing the procedure. The success rate with this procedure is reported to be over 85% [36]. The commonly reported complications include minor rebleeding, nasal crusting, palatal numbness, septal perforation, injury to the nasolacrimal duct, and acute sinusitis. Postoperatively, nasal saline irrigation is recommended to reduce crusting.

Rarely, bleeding from the anterior ethmoidal artery (AEA) can be a source of intractable posterior epistaxis. This is seen mainly in patients with a history of midfacial trauma or iatrogenic injury during sinus surgery and often fails to subside with conservative measures. Surgical intervention requires ligation of AEA via either a traditional approach through an external Lynch incision placed over the medial orbital wall or an endoscopic approach. External approach achieves better control of AEA and also avoids the complications that may occur from an endoscopic approach such as cerebrospinal fluid leak and orbital injury.

Endovascular Treatment

Endovascular procedures to control epistaxis are most often performed under general anesthesia to reduce patient movement and protect the patient's airway from blood and saliva. Femoral or radial artery access is obtained in the standard manor and a 5 or 6 F guide catheter is navigated into the origin of the external carotid. This vessel is prone to catheter-induced spasm, and consideration should be given to topical application of 1 inch of nitroglycerin to the angle of the ipsilateral jaw prior to catheterization. Intra-arterial nitroglycerine should also be available to relieve spasm that occurs during the case. A large inner diameter microcatheter (e.g., a 0.021 Rapid Transit, Codman) is then navigated over a microwire and positioned within the facial artery distal to the submandibular artery. The authors rely on microparticles for embolization of the ECA branches. We utilize 250–355 μm PVA particles suspended

in a contrast slurry ipsilateral to the site of bleeding. Injection is performed under negative roadmap imaging. Brief puffs are administered with careful attention paid to anterograde penetration of the nasal cavity. Once reflux is noted, embolization of these vessels is complete. The particle-contrast slurry must be constantly agitated, and attention must be paid to accumulation of microparticles within the hub or the microcatheter to avoid occlusion. A new microcatheter is utilized to select the contralateral ECA, is navigated in the internal maxillary artery, and is positioned distal to the deep temporal vessels. The process above is then repeated. The authors will embolize the contralateral ECA to reduce collateral supply to the area of hemorrhage. However, larger-diameter PVA particles (500–710 μm) are utilized in this case to avoid excessive penetration and devascularization of the nasal cavity and skin overlying the nose.

As described above, some interventionalists rely on proximal vessel occlusion of the IMAX through deployment of detachable or pushable coils rather than microparticles. Coils have the advantage of speed and simplicity but do not penetrate the capillary bed and obstruct re-treatment should symptoms recur. Liquid embolic agents are enjoying increasing favor but are higher cost, may be more prone to travel through external to internal collaterals, and also inhibit re-treatment if needed. They are also rarely associated with skin "tattooing" when cutaneous branches are inadvertently embolized.

Some practitioners will have nasal packing removed in the angiography suite to assess the need for further treatment before concluding the procedure. Others will remove nasal packing the next day.

Efficacy

Embolization is associated with a 90–100% efficacy in idiopathic epistaxis; however, eventual recurrence is the rule in HHT patients. When early rebleeding (within 30 days) is taken into account, rates of effective treatment are between 70 and 90% [29].

Risks

While the overall risk of embolization for epistaxis is low, there is risk of serious adverse events such as blindness and stroke that may occur due to reflux of the embolic material into cerebral or ophthalmic collaterals. In addition, external to internal collateral may lead to inadvertent cerebral artery embolization, making the assessment of collaterals crucial. Finally, excessive devascularization of the nasal cavity may lead to erosion and ulceration of the nasal mucosa or skin overlying the nose.

Illustrative Case 2: Epistaxis

A 55-year-old woman was admitted with several days of epistaxis. Upon admission, she underwent posterior nasal packing but upon removal once again experienced severe bleeding, requiring transfusion of two units of packed red blood cells. She was then brought to the angiography suit where she underwent PVA particle embolization of both distal facial and internal maxillary arteries. She tolerated the procedure well and was discharged, free of further bleeding, on postoperative day number 2 (Fig. 16.2).

Preoperative Tumor Embolization

General Technique

See Chap. 14.

Risks

The risks associated with tumor embolization are similar to those seen in the treatment of epistaxis and include stroke due to reflux of the embolic agent into the cerebral vasculature or transit through external to internal or external to vertebral artery connections. There is also risk associated with unintentional devascularization of cranial nerves, skin, and mucosa. When large tumors are treated, perioperative

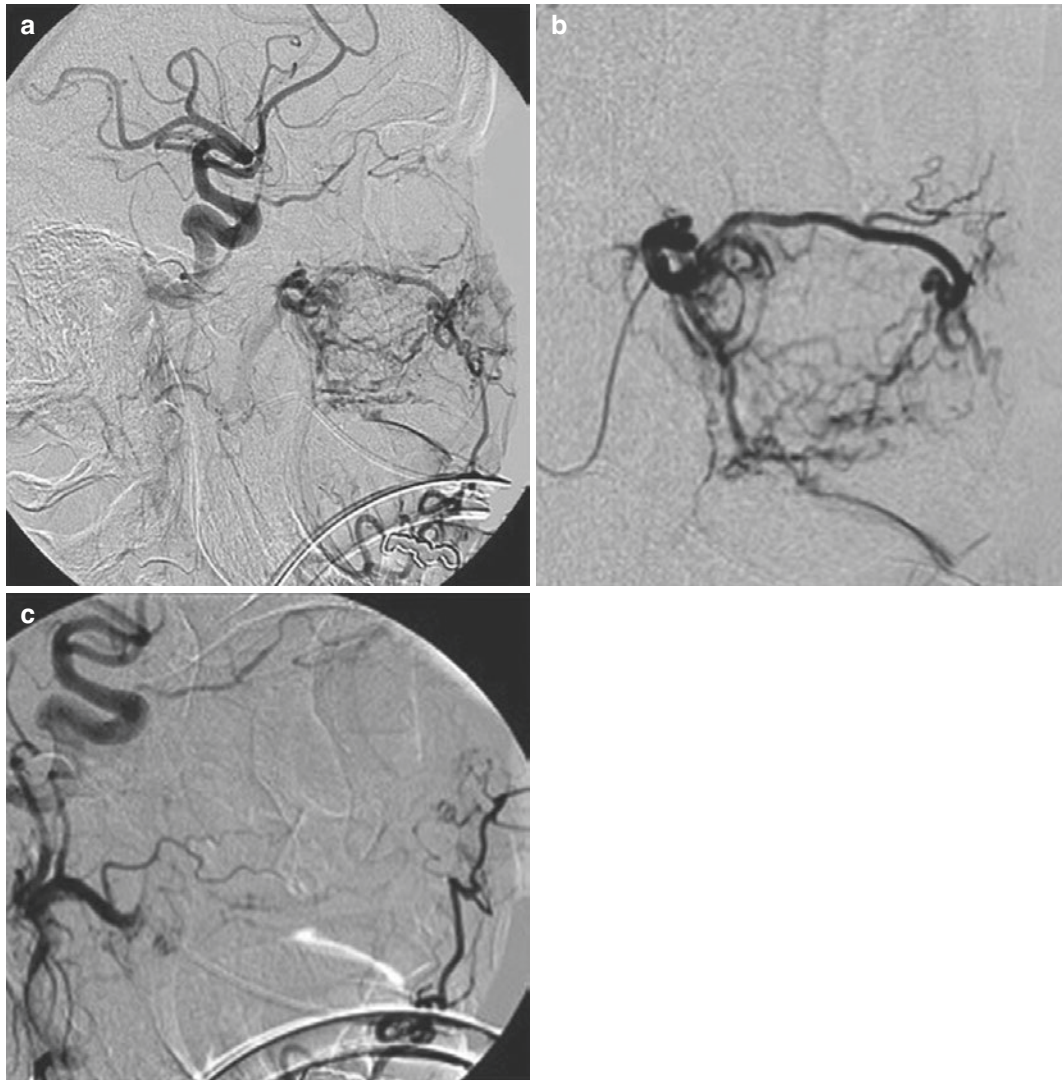

Fig. 16.2 Epistaxis. (**a**) Guide catheter angiogram showing vascular supply to the nasal cavity (sphenopalatine arteries, *bracket*; inferior orbital, *open arrow*; superior labial, *small arrow*). (**b**) Microcatheter angiogram of the distal internal maxillary artery (IMAX). (**c**) Post-PVA embolization showing occlusion of the distal IMAX

edema and mass effect may occur. This risk can be minimized through the judicious use of postoperative steroids and timing the embolization procedure in close proximity to surgical resection.

Vascular Tumors

- Meningiomas: Benign tumors arising from the dura mater occurring more frequently in females and in advanced age. They may present incidentally, with headache, focal neurological deficits, or seizures. At the skull base, they arise from the sphenoid wing, olfactory groove, and clivus. In these locations, they may be supplied by dilated branches of the ophthalmic artery, cavernous carotid artery (meningohypophyseal trunk or inferolateral trunk), or branches of the posterior circulation. Preoperative embolization of large meningiomas has been shown to reduce blood loss and operative time compared to case-matched controls [37].

- Juvenile nasopharyngeal angiofibromas: Represent 0.5% of all head and neck tumors. They generally affect adolescent boys, presenting as a painless, unilateral nasal obstruction or epistaxis. They originate in the sphenopalatine foramen or pterygopalatine fossa. 10–20% of JNAs have intracranial extension [38].
- Paragangliomas: Represent 0.6% of all head and neck neoplasms. They are vascular neoplasms arising from chemoreceptors of nerves. Women are more commonly affected with the peak incidence occurring between 30 and 60 years old. At times, they are multiple, familial, and/or secretory. Patients may present with a mass lesion in the neck, a cranial nerve deficit, or pulsatile tinnitus. Cranial nerve deficits are sometimes associated with the glomus jugulare subtype. These tumors are most often supplied by branches of the ascending pharyngeal artery [39]. The following locations have been described:
  - Tympanic (glomus tympanicum).
  - Jugular bulb (glomus jugulare).
  - Vagal (glomus vagale.
  - Carotid body tumor.
  - Aorta/larynx.

Surgical resection with or without preoperative embolization provides the best chance of cure. Preoperative embolization may cause inflammation of the dissection plane of the carotid adventitia and is not always preferred prior to resection. However, radiation or observation with serial imaging is recommended in highly advanced or multicentric, bilateral tumors where surgery may lead to significant functional impairment from cranial nerve deficits or vascular complications.

Radiation-Induced Carotid Stenosis

Association of Carotid Stenosis and Radiation

There is a known association between head and neck irradiation and carotid stenosis. In a series of 240 patients treated with head and neck radiation, 12% had >70% stenosis at 5 years with a tenfold higher relative risk of stroke at 10 years [40–43].

Pathophysiology

Most authors attribute the stenotic lesions seen post radiation to accelerated atherosclerosis; however, endothelial hyperplasia has also been hypothesized [40].

Indications for Treatment (See Chap. 4)

Patients with symptomatic stenosis (TIA or stroke) >50% should be considered for revascularization. Asymptomatic patients with 80% stenosis or greater may also be candidates. Due to the obscuration of tissue planes and thickening of the tissues seen post-neck irradiation, endovascular treatment is generally preferred over carotid endarterectomy [44].

Endovascular Treatment

See Chap. 4.

Iatrogenic Carotid Injury

Persistent Stapedial Artery and Aberrant Course of the ICA

A persistent stapedial artery is seen in 0.5% of temporal bone specimens at autopsy and is associated with an aberrant course of the internal carotid artery [45].

Illustrative Case 3: Aberrant ICA

An 88-year-old woman with what was thought to represent recurrent otitis media underwent elective myringotomy tube placement in an outpatient setting. Upon tube insertion, perfuse bleeding was encountered. CT angiography revealed a persistent stapedial artery with an aberrant course of the

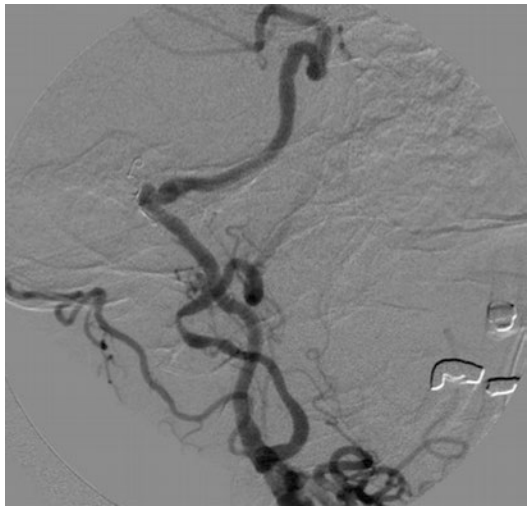

Fig. 16.3 Persistent stapedial artery

ICA and erosion into the middle ear cavity. The bleeding was controlled through navigation of an over-the-wire compliant balloon across the arteriotomy and occlusion of the middle ear cavity with Onyx 34 liquid embolic (Fig. 16.3).

Injury During Sinus Surgery

There is bulging of the internal carotid artery into the lateral wall of the sphenoid sinus in almost all patients with an up to 22% incidence of dehiscence in the bony wall covering the artery. This makes the artery vulnerable to inadvertent laceration during sphenoidotomy.

A clear understanding of the anatomy of the sphenoid sinus in relationship to the internal carotid artery (ICA) and proper training in the principles of endoscopic sinus surgery are fundamental to prevention of such a catastrophic injury. A careful review of preoperative CT scans before the procedure is of utmost importance to detect any variation in the sinus anatomy or vascular anomaly of the carotid artery. The carotid artery may bulge far into the lumen of the sphenoid sinus with a dehiscent bony wall. The sphenoidotomy should be made as medially toward the midline and inferiorly as possible. In addition, instruments like a microdebrider should be avoided in sphenoid sinus surgery to prevent any accidental injury. The ICA is at higher risk of injury during transsphenoidal and expanded endonasal approaches to the skull base. Use of micro-Doppler probe for carotid localization has been found to be a useful adjunct to prevent ICA injury in such surgeries.

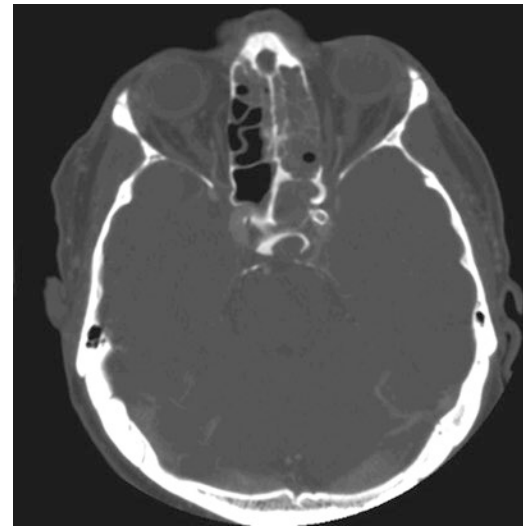

Fig. 16.4 Iatrogenic laceration of the left internal carotid artery. *Large arrow* shows damage to lateral wall of the sphenoid sinus. *Small arrow* shows covered stent deployment in the cavernous ICA

Illustrative Case 4

A 59-year-old man with a past medical history significant for recurrent sinusitis underwent debridement of the ethmoid and sphenoid sinuses for a fungal infection. During sphenoidotomy, perfuse bleeding was encountered. Angiography revealed a cavernous carotid laceration. 4 mm × 12 mm Graftmaster stent was placed across the lesion with immediate cessation of bleeding (Fig. 16.4).

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