The role of endovascular management in the treatment of extracranial carotid and vertebral vascular pathology is extensive and expanding. It is imperative for the neuroendovascular specialist to be proficient with diagnosis and treatment of common extracranial pathologies. For this discussion, we divide extracranial vascular disease processes into atherosclerotic and nonatherosclerotic vascular disease. The atherosclerotic disease discussion includes a review of clinical trials for symptomatic and asymptomatic carotid stenosis and surgical, medical, and endovascular treatments currently available. The nonatherosclerotic injury category encompasses trauma, vascular injury attributable to cancer or cancer treatment, spontaneous dissection and sequelae, and idiopathic epistaxis. Although these underlying pathologies differ, the diagnostic algorithm and therapeutic approaches are similar from the perspective of the neuroendovascular specialist.

ATHEROSCLEROTIC DISEASE
Extracranial Carotid Stenosis

Ischemic stroke is a major cause of morbidity and mortality in the western world. An estimated 8% to 29% of ischemic strokes are caused by extracranial atherosclerotic diseases (most frequently carotid bifurcation stenosis). Current treatment modalities for extracranial cervical carotid atherosclerotic disease include medical management, carotid endarterectomy (CEA), and carotid angioplasty and stenting (CAS).

Carotid endarterectomy
Few surgical procedures have undergone the same rigorous scrutiny as CEA. Ever since its inception in the 1950s, CEA for the treatment of carotid bifurcation stenosis has generated debate with respect to appropriate use. In the 1980s, CEA for the treatment of carotid bifurcation stenosis has generated debate with respect to appropriate use. In the 1980s, CEA was the most common vascular surgical procedure and questions regarding appropriate use led to large randomized trials both in North America and Europe to evaluate its efficacy for treatment of symptomatic and asymptomatic lesions. Carotid stenosis is considered symptomatic if there is a history of ipsilateral stroke, ipsilateral transient ischemic attack, or ipsilateral transient monocular blindness within the preceding 6 months.

Symptomatic carotid stenosis
The North American Symptomatic Carotid Endarterectomy Trial (NASCET) randomized
symptomatic patients from 50 centers to CEA with medical management versus medical management alone. Initial results were reported in 1991 and demonstrated a significant benefit to patients with high-grade carotid stenosis (70% to 99%) who underwent CEA. The risk of an ipsilateral stroke over a 2-year period was 26% in the medical group but 9% in the CEA group, representing an absolute risk reduction for the surgical arm of the study of 17% decreased risk of any ipsilateral stroke over a 2-year period. These results were essentially corroborated in the European Carotid Surgery Trial (ECST) in 1998. Accounting for differences in angiographic quantitation of carotid stenosis, the results of NASCET and ECST were in agreement that CEA was of clear benefit to symptomatic patients with carotid stenosis between 70% and 99% (when measured using the NASCET criteria). Further results of the NASCET study published in 1998 showed that symptomatic patients with moderate stenosis (50% to 69% by NASCET criteria) also benefit from CEA, although that benefit was less striking and more apparent in men than women. These studies established the legitimacy of CEA for treatment of symptomatic carotid stenosis greater than 70%.

Asymptomatic carotid stenosis

The NASCET and ECST studies did not address the increasingly large, asymptomatic population diagnosed with carotid stenosis by imaging. The Asymptomatic Carotid Atherosclerosis Study group (ACAS) trial, published in 1995, randomized 1662 patients with greater than 60% carotid stenosis to CEA with medical management versus medical management alone and found an estimated absolute risk reduction in the surgically treated group of 5.9% in the risk of ipsilateral stroke over a 5-year period as compared with medical management alone. These results were further corroborated by the Asymptomatic Carotid Surgery Trial (ACST) published in 2004 that also showed a 6% absolute risk reduction of ipsilateral stroke over a 5-year period in the surgically treated group. Bear in mind that the patients randomized in the ACAS trial were highly selected, with multiple exclusion criteria (age older than 80, significant medical morbidities, and more), and in addition, the surgeons performing the CEA had to show a perioperative complication rate of less than 3% to be allowed to participate in the study. Previous trials had failed to show a benefit for CEA in asymptomatic patients (Veterans Affairs trial and the Mayo Clinic trial). The American Heart Association guidelines for treatment recommend CEA for symptomatic extracranial carotid stenosis (>70%) if the periprocedural rate of major stroke or death was under 6% and treating asymptomatic extracranial carotid stenosis (>70%) if the periprocedural rate of major stroke and death was under 3% and the life expectancy of the patient was at least 5 years. Also of significance, best medical management has improved significantly since these trials were performed, with a more defined role for statin therapy, angiotensin-converting enzyme (ACE) inhibitors, and added antiplatelet therapy.

Carotid angioplasty and stenting

Initial endovascular treatment of extracranial carotid artery stenosis involved percutaneous balloon angioplasty with isolated case reports and small series published in the early 1980s. The touted benefits of endovascular treatment included minimally invasive approach allowing treatment of patients with severe cardiac and pulmonary disease, decreased cranial nerve injury, and suitability for patients with anatomically difficult lesions for a surgical approach and in patients with a prior history of neck radiation. Although CAS does avoid some of the complications associated with CEA, such as cranial nerve injury, there are specific procedural risks that are unique, such as groin or retroperitoneal hematoma, vessel dissection related to catheterization and contrast reaction, as well as the risk of stroke. There are anatomic configurations unfavorable for CAS such as severe tortuosity of the carotid vessels, or severe angulation between the aortic arch and origin of the brachiocephalic or carotid arteries (Fig. 1). It very quickly became apparent that the major limitation of endovascular treatment was the risk of embolic stroke that initially seemed significantly higher for patients treated with percutaneous transluminal angioplasty (PTA) with or without stenting versus those treated with surgery.

Randomized controlled trials of carotid endarterectomy versus carotid angioplasty and stenting

When evaluating the literature comparing CEA and CAS, several issues must be considered:

1. Case series registries versus randomized controlled trials (RCTs)
2. Symptomatic versus asymptomatic patients
3. Low-risk surgical candidates versus high-risk surgical candidates (and thus would have been excluded from the relevant surgical trials)
4. Periprocedural medical management
5. Use of cerebral protection device during CAS
6. Operator experience
The first RCT comparing CEA and CAS was published in 1998 and was a small trial that was stopped prematurely because of a very high incidence (70%) of stroke in the CAS arm of the trial. The Carotid And Vertebral Transluminal Angioplasty Study (CAVATAS) was published in 2001 and showed no significant difference in 30-day morbidity and mortality rates when comparing CEA and CAS. However, complication rates in both groups were high by conventional standards, with a 30-day death or disabling stroke rate of 10.0% and 9.9% for CAS and CEA respectively. Only about a quarter of endovascularly treated patients received a stent and restenosis rates were higher in the endovascular group.

The first RCT in high-risk patients was the Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy (SAPPHIRE) trial published in 2004. Patients randomized for the trial would likely have been excluded from the large surgical trials because of age and/or comorbidity. Only experienced surgeons and interventionalists were allowed to participate in the trial. Most of the patients were asymptomatic (about 70% in both arms); embolic protection devices and stents were used in the endovascular arm of the study.

**Fig. 1.** Unfavorable anatomy for CAS. (A) Symptomatic right internal carotid artery (ICA) stenosis accessed via high left brachial approach owing to occlusive vascular disease in the femoral arteries. (B) Although diagnostic angiography was successful, the shuttle sheath could not be advanced into a position suitable for stent placement. (C) Symptomatic right ICA stenosis with tortuosity of the common carotid artery. (D) The tortuosity at the arch and the angle of the right ICA origin prevented advancement of the protection device beyond the origin of the guiding catheter. The procedure was aborted and an awake endarterectomy performed. (E, F) Distal patch graft stenosis involves both internal and external carotid arteries, however there is a marked disparity in size between the graft and the internal carotid artery precluding stent placement. (G) The external carotid artery is occluded in this patient with oral bleeding and recurrent tonsilar cancer. The size disparity between the common and internal carotid arteries and the acuteness of the angled precluded stent placement and the vessel was permanently occluded with coils.
the study. The results showed a significantly lower 30-day mortality/major stroke/myocardial infarction (MI) rate in the CAS group (4.8%) versus the CEA group (9.8%) and this benefit was still present at 1 year after intervention. However, it is important to realize that the vast majority of the difference between the two arms was because of a difference in the rate of MI (most MIs were non–Q-wave infarctions detected on routine postoperative tests). If MI is excluded as an end point, there was no statistically significant difference between groups.

The Stent Protected Angioplasty versus Carotid Endarterectomy (SPACE) trial was published in 2006. A total of 1183 symptomatic patients with over 70% stenosis were randomized to CAS or CEA. Use of embolic protection devices was left to the discretion of the treating physician. Results showed a similar rate of complications between the groups. The 30-day mortality/major stroke rate was 6.8% in the CAS arm and 6.3% in the CEA arm. The authors concluded that CEA was still the gold standard because evidence of the equivalence or superiority of endovascular treatment is lacking. The Endarterectomy Versus Stenting in Patients with Symptomatic Severe Carotid Stenosis (EVA-3S) was also published in 2006. After randomizing 527 patients to CAS or CEA, the trial was stopped because of concerns regarding the safety of CAS. Periprocedural 30-day mortality and major stroke rates were 9.6% in the CAS arm compared with 3.9% in the CEA arm. The authors concluded that CEA was still the gold standard because evidence of the equivalence or superiority of endovascular treatment is lacking. The Endarterectomy Versus Stenting in Patients with Symptomatic Severe Carotid Stenosis (EVA-3S) was also published in 2006. After randomizing 527 patients to CAS or CEA, the trial was stopped because of concerns regarding the safety of CAS. Periprocedural 30-day mortality and major stroke rates were 9.6% in the CAS arm compared with 3.9% in the CEA arm. The authors concluded that CEA was still the gold standard because evidence of the equivalence or superiority of endovascular treatment is lacking.

Ongoing studies

Several large prospective RCTs are currently enrolling patients. Perhaps the most eagerly awaited study is the Carotid Revascularization Endarterectomy versus Stenting (CREST) trial, which is funded by the National Institutes of Health (NIH). This trial plans to randomize 2500 patients to CEA or CAS and follow-up for 4 years. Optimal current medical management is provided to both arms and intervention is performed in symptomatic patients with 50% stenosis by angiography (70% by ultrasound) and in asymptomatic patients with 60% stenosis by angiography (70% by ultrasound). Some data from this trial has been published indicating an increased risk for octogenarians undergoing CAS. Other ongoing trials comparing CEA and CAS are the International Carotid Stenting Study (ICSS) evaluating low-risk symptomatic patients and the ACT 1 trial, which is evaluating low-risk asymptomatic patients. The Transatlantic Asymptomatic Carotid Intervention Trial (TACIT) will enroll asymptomatic patients to undergo CEA, CAS, or best medical management only. This trial is necessary because the best medical management represented in the landmark surgical trials was not remotely similar to what has been established as best medical management today.

Carotid angioplasty and stenting technique

Our routine procedure for CAS is as follows. The procedure is performed with moderate sedation allowing for continuous neurologic monitoring. Before the procedure, antiplatelet therapy is initiated with aspirin and clopidogrel for at least 5 days and is continued after the procedure. Preprocedural blood pressure control and initiation of statin therapy is also of paramount importance. All patients who have not undergone prior ipsilateral CEA have external pacemaker pads applied before the start of the procedure as a precaution, if extreme bradycardia is encountered and we also ensure that intravenous (IV) atropine is readily available for rapid administration. Access is usually obtained with a common femoral artery puncture and a thoracic angiogram frequently performed. Anticoagulation with heparin is initiated and care taken to maintain an ACT above 250 seconds for the remainder of the procedure. Selective angiography of the nontarget carotid is usually performed to assess potential collateral circulation. Selective angiography of the target vessel is performed in multiple projections to both confirm the diagnosis of significant carotid artery stenosis and to ascertain the best working projection for the planned intervention (Fig. 2). Anteroposterior (AP) and lateral angiograms of the head are also performed as a baseline examination. Next, an exchange length wire is manipulated into a branch of the internal carotid artery (ICA) and the diagnostic catheter exchanged over the wire for a 6-French 80- to 90-cm guiding sheath. An embolic protection device is then manipulated across the stenosis and the protection device deployed in the internal carotid artery (ICA) distal to the segment to be treated. Predilatation is then performed if needed with a compliant angioplasty balloon. We tend to be more aggressive in predilatation, as we feel this reduces the need for poststent deployment dilatation by improving the stent approximation to the vessel wall. Next, an appropriately sized carotid stent is deployed.
and postdeployment angiography performed. If possible, we avoid poststent deployment balloon angioplasty because it has been our anecdotal experience that a significant number of intraprocedural embolic complications occur during or directly after poststenting balloon angioplasty. This observation may be attributable to a grating effect of the stent on the atheromatous plaque and subsequent emboli during poststenting balloon angioplasty. As a rough guideline, we will accept a 30% or less residual stenosis after stent deployment. Next, the embolic protection device is retrieved and AP and lateral angiograms of the head performed along with a brief on-table neurologic examination. If no complications are encountered, the guiding sheath is exchanged over a wire for a closure device to achieve hemostasis. The patient is then monitored in an ICU setting until the next morning with frequent neurologic examinations and close monitoring of blood pressure.

Long-term efficacy of carotid stenting in terms of stroke prevention appears to be equivalent to carotid endarterectomy but preprocedural morbidity may be increased for patients undergoing carotid stenting. CAS is reserved for selected symptomatic patients with such comorbidities as post CEA re-stenosis, a history of neck irradiation, contralateral laryngeal nerve palsy, anatomically surgically difficult lesions, or poor surgical candidates because of major comorbidities (Fig. 3). Low-risk patients should undergo CAS only in the setting of a RCT. Although currently the standard of care for patients with extracranial carotid stenosis is carotid endarterectomy, it is likely that CAS will have an increasing role to play in the treatment of carotid stenosis in the future. We eagerly await the results of large RCTs comparing the treatment modalities, and to technological improvements to protection devices and stents to further define the role of CAS in the treatment of carotid artery stenosis.

EXTRACRANIAL VERTEBRAL ARTERY STENOSIS

About 25% of all strokes involve the posterior circulation.19 The association between posterior circulation strokes and vertebral artery stenosis is less well defined than the relationship between anterior circulation stroke and carotid stenosis. The vertebral artery origin is the most common location for vertebral artery stenosis and tends to be associated with adjacent atherosclerosis in the subclavian artery (similar to renal artery stenosis and aortic disease). One registry20 found 20% of patients with symptoms of vertibrobasilar ischemia to have a proximal vertebral artery stenosis (defined as at least 50% stenosis) and about half of these patients also had contralateral lesions. The risk of stroke after posterior circulation transient ischemic attack has been reported between 25% and 29% during 5- to 6-year follow-up.21 Despite medical therapy with warfarin or aspirin, these patients have a high recurrence and death rate, reported as 16% to 18%.22
A number of case series have been published describing the primary deployment of balloon expandable stents to treat lesions greater than 50% with high technical success rates. These series also report a low incidence of major procedure-related complications (0% to 3.4%) and low incidence of recurrence (1.7% to 3.0%) on short- and intermediate-term follow-up (3 to 37 months). Periprocedural posterior circulation transient ischemic attacks (TIAs) were reported at rates of 0% to 4.8%.23 Recently, use of an embolic protection device has been advocated during vertebral artery stenting to minimize risk of intra procedural stroke.24 The only randomized

Fig. 3. Accelerated atherosclerosis post radiation (multiple vessels). (A) Arch arteriogram demonstrates marked irregularity of both common carotid arteries as well as the origins of the vertebral arteries bilaterally in a patient with laryngeal carcinoma 5 years post radiation therapy. (B) Selective right vertebral artery arteriogram demonstrates diffuse irregularity in the proximal vertebral segment. (B) Selective left common carotid artery arteriography demonstrates marked irregularity and a focal 1-cm stenosis with ulceration. (D, E) AP and lateral views from a right common carotid artery (RCCA) arteriogram in a 63-year-old with history of mantle therapy for lymphoma demonstrates occlusion of the internal carotid artery and a marked stenosis of the common carotid artery with ulceration (arrow). The external carotid artery provides collateral flow to the brain via ethmoidal collaterals. (F) Following stent placement into the RCCA there was significant improvement in intracranial flow.
controlled evaluation of vertebral artery PTA and stenting compared with medical management was a small subgroup of patients within the CAVATAS trial where 16 patients were randomized. Results were nonrevealing owing to the small sample size. A large RCT comparing extracranial vertebral artery stenting with best medical management has yet to be performed. Vertebral artery angioplasty and stenting can be considered as an emerging therapy for patients with symptomatic vertebral artery stenoses that have failed best medical management.

NONATHEROSCLEROTIC ARTERIAL INJURY

Patients with nonatherosclerotic vascular injuries usually present with symptoms, often hemorrhage and/or stroke. This etiologically diverse category encompasses multiple pathologies including trauma (blunt, penetrating, and iatrogenic), vascular injury attributable to cancer or cancer treatment, spontaneous dissection and sequelae, and idiopathic epistaxis. Although these underlying pathologies differ, the diagnostic algorithm and therapeutic approaches are similar allowing us to consider them together.

Management of these patients varies with the acuity of presentation, for example a patient with oral or nasal bleeding, or severe blunt or penetrating trauma, would receive general supportive measures such as airway and hemodynamic resuscitation, before imaging including diagnostic angiogram. CT scan and in many cases CT angiography can be helpful for triage and evaluation of the site of bleeding or vascular compromise. Once the lesion has been diagnosed and defined, diagnostic angiography can be targeted specifically to the injury including distal embolization with particulates, or liquid embolic agents. With blunt traumatic injury leading to pseudoaneurysm or traumatic dissection seen on CTA, without hemorrhage or stroke, treatment decisions are less clear cut. Many advocate close observation in these patients, and if no contraindications exist, treatment is with an anticoagulation/antiplatelet regimen needed to place and sustain covered stent graft placement for major vessel injury. With penetrating traumatic vascular injury, diagnostic angiogram followed by endovascular treatment targeted specifically to the injury including distal vessel embolization with particulates, or liquid embolic agents. With blunt traumatic injury leading to pseudoaneurysm or traumatic dissection seen on CTA, without hemorrhage or stroke, treatment decisions are less clear cut. Many advocate close observation in these patients, and if no contraindications exist, treatment is with an anticoagulation/antiplatelet regimen, as complications from these injuries are usually embolic. However, if serial imaging demonstrates interval worsening of imaging findings, or the patient becomes symptomatic from the lesion, intervention may be necessary. Vertebral artery dissection and/or occlusion of extracranial vessels often involves the cervical ICA 1 to 3 cm above the carotid bifurcation or the vertebral artery at the level of cervical fracture. Intramural hematoma and dissection can result from blunt or penetrating trauma with variable compromise of the involved vessel lumen. Contained rupture of the injured major vessel (pseudoaneurysm) is most often initially irregular in shape but has a tendency to take on a more smooth fusiform or saccular shape over time, depending on the vessel of origin.

VASCULAR TRAUMA OF THE FACE AND NECK

Trauma to the extracranial carotid or vertebral arteries may be either blunt or penetrating. Penetrating trauma is by far the more common, with gunshot wounds accounting for most penetrating traumatic injuries in the United States (Fig. 4). Although less common, blunt injury to the major arteries of the head and neck can be devastating with major neurologic morbidity and mortality rates estimated around 60% and 30% respectively. Of all comers to a level 1 trauma center with blunt trauma, 0.24% (37 of 15,331 patients) sustained blunt carotid injury in a study published in 1999. This same study maintained that screening for blunt carotid injury identified a number of asymptomatic patients with serious injuries. Twenty-five of 2902 (0.86%) patients screened were diagnosed with blunt carotid injury and 13 of those (52%) were asymptomatic. Prompt diagnosis and treatment are critical to patient outcome. CT angiography has become the imaging modality of choice in the emergency room setting. To our knowledge, no comprehensive comparison studies between CTA and angiography exist for screening of trauma patients. CT/CTA findings of vascular injury can vary widely (Fig. 5). Frank extravasation of contrast is rarely encountered, but when present, is easily identified as nonvascular contrast. Complete traumatic occlusion of extracranial vessels often involves the cervical ICA 1 to 3 cm above the carotid bifurcation or the vertebral artery at the level of cervical fracture. Intramural hematoma and dissection can result from blunt or penetrating trauma with variable compromise of the involved vessel lumen. Contained rupture of the injured major vessel (pseudoaneurysm) is most often initially irregular in shape but has a tendency to take on a more smooth fusiform or saccular shape over time, depending on the vessel of origin.
distal embolization and stroke, particularly in patients requiring cervical manipulation or other surgery (Fig. 6). A recent review of the literature found favorable results for placement of covered stents in the setting of ICA pseudoaneurysm (Fig. 7).28 If major vessel occlusion is seen on the CTA, endovascular options are limited. Although there have been some good outcomes with opening occluded carotids in the setting of atherosclerotic disease, there are few data on recanalizing traumatically occluded vessels. Emergent surgical bypass and/or thrombectomy are seldom used in this setting.

**CAROTID BLOWOUT SYNDROME**

Carotid blowout syndrome (CBS) is defined as the rupture of the extracranial carotid arteries or one of its major branches, with oral, nasal, or peritracheal bleeding and has been coined in the setting of head and neck neoplasm, most often squamous cell carcinoma. Carotid blowout is the most feared sequela of head and neck cancer. Prior radiation therapy or brachytherapy are believed to play a key role in the pathogenesis of carotid blowout, possibly because of the obliterating effects on the vasa vasorum caused by radiation and the associated weakening of the arterial wall. Prior
Fig. 5. CTA findings of vascular injury. (A) Oblique reformat CTA image demonstrates a pseudoaneurysm originating from the distal common carotid artery at the level of the bulb with an expanding left cervical mass. (B) Lateral left common carotid arteriogram demonstrates the opacification of the pseudoaneurysm limit with draping of the superior thyroid artery around partially thrombosed portion of the mass. (C) Axial CTA following endarterectomy in a patient with expanding left cervical hematoma, demonstrates pseudoaneurysm at the level of the anastomosis, best illustrated in continuity on the oblique reformatted image (D). (E) Axial CTA demonstrates typical appearance of a distal cervical ICA dissection with pseudo aneurysmal dilatation.
radiation treatment has been associated with a sevenfold increase of CBS in patients with head and neck cancer. Although not confined within the classic definition of carotid blowout syndrome, it is important to keep in mind that significant hemorrhage can also occur from erosion of tumor into the internal jugular vein and from the tumor itself. In the past few years there has been significant improvement in both diagnosis and treatment of CBS.

The reported incidence of CBS in patients with a history of neck dissection is reported to range from 3% to 4%. In the past, carotid blowout has resulted in very significant mortality and morbidity. Recent improvement in care has markedly reduced both mortality and morbidity. Carotid blowout is now approached as a syndrome, with manifestations that can range from acute life-threatening hemorrhage to asymptomatic encasement of a carotid artery. CBS can be categorized into one of three categories: threatened, impending, and acute carotid blowout. Threatened carotid blowout is defined as physical examination or imaging results that suggest inevitable hemorrhage from one of the carotid arteries or its branches if no action is taken. Impending carotid blowout (also called sentinel hemorrhage) is defined as transient hemorrhage that resolves spontaneously or with packing/pressure. Acute carotid blowout represents hemorrhage that cannot be controlled by packing or pressure.

Once carotid blowout syndrome is clinically suspected, the conventional gold standard for diagnosis has been digital subtraction angiography (DSA). However, we believe that noncontrast CT and/or CT angiography have an important role in diagnosis in clinically stable patients. It is useful to understand the extent of tumor, prior surgery, or region treated with brachytherapy seeds before angiography. Unfortunately, there is a lack of literature on the sensitivity and specificity of angiography, and to our knowledge, there is no direct comparison between DSA and CTA for evaluation of carotid blowout syndrome.

**Management and Angiographic Approach to Carotid Blowout Syndrome**

Initial management of these complex patients revolves around the airway. Controlling the airway is paramount and intubation to prevent aspiration is necessary. Oral, nasal, or wound packing is adjunctive to fluid resuscitation measures and transfusion. Manipulation of the neck wound should be minimized. Once the patient has been stabilized, direct diagnostic and therapeutic strategies can be used.

It is critically important to discuss treatment options and expectations with the patient and/or family representatives with regard to decision making regarding stroke/death risk versus
bleeding/death risk in the treatment of CBS. Patient and family wishes and preferences, including resuscitation status, should be taken into account if bleeding cannot be stopped without significant stroke risk. There are some patients who prefer not to accept the risk of carotid occlusion in spite of the grave prognostic implications of untreated CBS. Diagnostic angiography varies slightly based on the nature and location of the hemorrhage. Bilateral imaging of the common carotids, the internal carotids, the external carotids, and the vertebral arteries will establish the bleeding site as well as the integrity of the Circle of Willis. This is important in patients who may require carotid artery sacrifice. Selective external carotid branch arteriography is often required to identify a bleeding site not demonstrable on more global injections. Internal maxillary, facial, lingual, ascending pharyngeal, and superior thyroid arteries are the vessels most commonly affected. In patients with peritracheal bleeding, the thyrocervical trunks must also be examined bilaterally.

MAJOR VESSEL LESIONS

During the initial angiographic assessment, catheter placement should be proximal to the suspected carotid injury. The location of surgical clips and brachytherapy seeds are local indicators of the potential site of vascular compromise. For frank rupture, sacrifice of the carotid artery above and below the point of rupture will prevent the

**Fig. 7.** (A) MRA multiple intensity projection (MIP) image in a patient with chronic neck pain demonstrates pseudoaneurysm of the internal carotid artery near the skull base. Following angiographic confirmation (B), overlapping stents were placed using a protection device to exclude the pseudoaneurysm from the circulation (C).
Fig. 8. (A–D) A 72-year-old status post salvage laryngectomy for squamous cell carcinoma of larynx with intractable oral bleeding. Axial (A) and coronal (B) CTA demonstrates air (white arrows) dissecting within the carotid space directly adjacent to the left internal carotid artery (ICA) (black arrow) and a local soft tissue mass. Note the absence of the internal jugular vein (IJV) on the left. (C) AP LCCA arteriogram demonstrates marked irregularity of the distal CCA, stenosis at the origin of the ICA with marked irregularity to the level of the skull base. A stump (white arrow) is seen at the origin of the external carotid artery. (D) AP RCCA angiogram demonstrates robust collaterals filling the left intracranial vasculature from the right ICA and the right vertebral arteries. Note the coil mass on the left. (E–G) A 60-year-old male with nasopharyngeal carcinoma and oral bleeding. (E) Narrowing of the internal carotid artery with a small angular contrast collection consistent with pseudoaneurysm within a soft tissue tumor mass (white arrows). The IJV is occluded. (F) Angiography confirms the PSA and the adjacent ICA stenosis. (G) The ICA and the PSA were permanently occluded using detachable platinum coils. Control arteriography demonstrates prompt filling of distal ethmoidal branches of the IMA to collateralize with the ophthalmic artery (OPH) that is filling retrogradely to reconstitute the supraclinoid ICA.
creation of a pattern of unreachable collateral flow. Often both the internal and external carotid branches must be occluded (see Fig. 8). Carotid sacrifice was formerly performed with detachable silicone or latex balloons; however, these are not currently available in the United States. Currently we perform carotid sacrifice with detachable coils, with variable adjunctive use of the Amplatzer vascular plug (AGA Medical Corporation, Plymouth, MN) for occlusion of the proximal aspect of the vessel (Fig. 9).

In CBS patients with a lesion of the common or internal carotid, such as pseudoaneurysm, ulceration, or long-segment irregularity, sacrifice of the involved carotid may be the only life-saving option. Ideally, when time permits, a temporary balloon occlusion test (BOT) is performed to ascertain tolerance of carotid artery occlusion before the procedure. The three components of a temporary balloon occlusion test include diagnostic arteriography for collateral assessment, clinical testing during temporary occlusion, and TC-hexamethylpropyleneamine oxime–single-photon emission computed tomography (HMPAO-SPECT) or xenon CT evaluation of cerebral blood flow during temporary occlusion. Balloon occlusion testing is further described elsewhere in this issue.

Covered stent (stent graft) placement has been used for salvage of major vessels in carotid blowout. This technology is limited in the setting of open wounds, active infection, and unfavorable anatomy. The use of covered stents may be an option in some patients; however, one must be prepared to convert a stent procedure into carotid occlusion if hemostasis cannot be maintained (Fig. 10).^{34,35} When carotid sacrifice cannot be safely performed because of lack of collateral flow or a lack of tolerance of temporary balloon occlusion testing, and stent placement is not an option, surgical extracranial-to-intracranial bypass techniques may be considered, in association with carotid sacrifice, to augment cerebral blood flow.^{36}

When CBS manifests as uncontrollable hemorrhage, carotid sacrifice must then be performed without clinical preocclusion testing (as a lifesaving procedure). In this situation, HMPAO-SPECT is sometimes performed following occlusion to assist in postocclusion ICU hypertensive and hypervolemic management. The occlusion balloon is withdrawn and the carotid sacrificed using retrievable and/or detachable coils. Alternatively, the occlusion balloon catheter can be left in place with the balloon inflated and a microcatheter advanced through the lumen of the occlusion balloon and embolization of the distal ICA performed during balloon occlusion of the proximal ICA. This allows immediate control of bleeding and the ability to raise blood pressure to enhance cerebral perfusion during the permanent occlusion. Clinical management in the ICU includes neurologic monitoring and hemodynamic measures to maintain cerebral perfusion pressure. CT scan of the head can be adjunctive following patient stabilization.

**NON–MAJOR VESSEL SOURCES OF HEMORRHAGE**

Significant oral, nasal, or peritracheal bleeding may result from tumoral neovascularity or erosion
through the wall of a small external carotid branch vessel, rather than major vessel erosion. Selective external carotid branch arteriography is often required to demonstrate the bleeding site. Microcatheterization both permits identification of the bleeding site and allows for more selective placement of embolic material. Selection of embolic material depends on the site and the nature of the hemorrhage source. In our experience, in the trauma setting, embolization is most often performed with particulate such as polyvinyl alcohol foam (PVA) or gelfoam, with or without adjunctive placement of coils. In the setting of head and neck cancer, radiation, and infection, liquid embolic agents are more durable in these fragile, diffusely diseased vessels (Fig. 11).

TUMORAL HEMORRHAGE

Hemorrhage derived from neovascularity of the tumor or local granulation tissue can be treated with medium-sized, 250-micron to 350-micron particles of PVA suspended in dilute contrast media. We introduce the particles into the tumor under roadmap guidance using a microcatheter and 1-cc syringes until the neovascularity is sufficiently reduced. Proximal gelfoam pledgets may be adjunctive. This is a temporizing measure that may allow for healing of friable tumor. One should avoid using small 50-micron to 150-micron particles if additional surgery is not planned, to avoid frank tumor necrosis and subsequent secondary infection.

VASCULAR LACERATION OR PSEUDOANEURYSM

Vascular laceration or pseudoaneurysm (PSA) may be treated directly with microcatheter embolization to address the specific bleeding site in the setting of trauma or head and neck cancer/tumor/radiation (CBS). Embolic materials vary with the setting. Gelfoam with or without complex helical coils may be used for a discrete vessel laceration in the setting of trauma. This will control bleeding in an otherwise intrinsically normal lacerated vessel; we use Trufill n-BCA Liquid Embolic System (Cordis Corporation, a Johnson & Johnson company, Miami, FL), where possible, for pseudoaneurysm or focal extravasation (Fig. 12). Proximal vessel coil embolization should be avoided, because it does not directly address the site of pathology. In most cases, when attempts at proximal occlusion have failed to control hemorrhage, small microcatheters can navigate beyond proximally placed clips or coils for more distal embolization. When choosing an embolic agent, pay attention to the site of the lesion, the nature of adjacent collaterals, the distal...
parenchymal territory, and the role of embolization (ie, presurgical or palliative).

EXTRACRANIAL CAROTID AND VERTEBRAL ARTERY SPONTANEOUS DISSECTION

Arterial dissection is an intimal tear in the wall of an artery allowing intrusion of blood products within the layers of the arterial wall. As this hematoma in the false lumen between the intima and media expands, it may compress the true lumen, resulting in stenosis, occlusion, and/or pseudoaneurysm formation with weakening of the media and adventitia. Spontaneous or nontraumatic dissection of the extracranial carotid and vertebral dissection were once thought to be rare, but may account for as many as 20% of strokes in patients younger than 45 years.37

The pathogenesis of spontaneous dissection is incompletely understood. A history of minor neck trauma is often present although not universal. Patients with underlying connective tissue disorders such as fibromuscular dysplasia, Ehlers-Danlos syndrome, Marfan’s syndrome, and alfa 1 antitrypsin deficiency syndrome may be more vulnerable to cervical vascular dissection. There has been speculation that recent infection may play a role in the pathogenesis of dissection, in part supported by some seasonal variance.38 It has also been speculated that migraine history, hypertension, and smoking may contribute to increased risk of dissection.

Carotid and vertebral dissection are classified into extradural (extracranial) and intradural (intracranial) dissections. Extradural/extracranial dissection is far more common although intradural extension of dissection is usually more significant and carries a poorer prognosis and a risk of subarachnoid hemorrhage. Spontaneous dissection is more common in the carotid arteries (80%) than in the vertebral arteries.39 The arterial segments affected differ between dissection and atherosclerosis. Dissection often involves the distal part of the ICA near the point of fixation at the entry into the carotid canal at the skull base, whereas atherosclerosis most often affects the carotid bulb and the origin of the ICA. Vertebral dissection frequently occurs near points of fixation such as the entry or exit from the foramen transversarium and where the vertebral artery pierces the dura at the

Fig. 11. (A) Lateral common carotid artery angiogram in a patient with a base of tongue cancer demonstrates marked irregularity of the facial artery (black arrow) and marked narrowing and irregularity with pseudoaneurysm of the lingual artery. (B) Selective lingual arteriogram demonstrates marked narrowing and irregularity and a pseudoaneurysm of the lingual artery at the tongue base. (C) The lingual artery and pseudoaneurysm were embolized with NBCA (see the radio-opaque glue cast in the lingual artery after embolization).
Foramen magnum. Atherosclerosis tends to involve the origin of the vertebral artery. Classic clinical features of spontaneous dissection are often not present, but include neck pain, Horner’s syndrome, headache, cranial nerve palsies, and neurologic deficits secondary to distal embolization or ischemia from flow compromise. Embolic phenomena are most common because of platelet aggregation at the site of injury and distal embolization. A study from 2004 demonstrated territorial infarcts (presumed embolic in origin) in all patients with dissection contrasted with watershed infarcts (secondary to hemodynamic phenomena) in only 5% of these patients.40 Extracranial dissection is readily diagnosed with noninvasive methods such as Doppler ultrasound, MRI/MR angiography (MRA), and most recently with CT angiography (CTA). Identification of the false lumen filled with thrombus on the T1 fat saturation image is diagnostic. Irregularity of the lumen or sharp edges rather than curves on the axial CTA images are equally diagnostic when the studies are technically of good quality (Fig. 13). Catheter angiography is usually performed only for problematic cases or when intervention is considered. Treatment is essentially medical and rarely do these patients require endovascular or operative management. Once diagnosed, thromboembolism is the most feared possible sequelae. Management

Fig. 12. (A) Recurrent epistaxis following left internal carotid artery occlusion demonstrates a PSA of the second portion of the IMA. (B) Acrylic glue is identified within the PSA. (C) Oral bleeding 7 days post resection and graft placement for treatment of floor of mouth cancer demonstrates a PSA of the graft at the anastomosis (white arrows). (D) Acrylic glue cast demonstrated within the PSA. The graft remained viable.
revolves around minimizing the risk of embolic phenomenon using antiplatelet agents as the mainstay of treatment. In cases with severe luminal compromise, short-term heparin and/or warfarin administration is sometimes used.

The prognosis depends largely on the severity of neurologic deficit incurred before diagnosis, but is generally good with vessel wall healing with extracranial dissection. Recurrence rates are very low in the absence of underlying connective tissue disease.

IDIOPATHIC EPISTAXIS

A majority of the adult population will experience epistaxis during their lifetime although only a small minority will experience bleeding requiring medical attention. There is a small group of patients who do not respond to conservative therapies such as local packing. Epistaxis is deemed idiopathic if there is no known underlying etiology such as trauma or tumor. These patients are treated with various forms of therapy including posterior and anterior packing, vasoconstrictors, endoscopic cautery, surgical ligation and/or endovascular embolization. Patients with posterior epistaxis are said to be particularly difficult to manage conservatively and often require a more aggressive treatment regimen including endovascular embolization. Endovascular embolization of idiopathic epistaxis was first described in 1974 by Sokoloff and colleagues. Since that time, numerous series have been published detailing results and technique. A recent review of the literature showed an average technical success rate of 88% and average postprocedural complication rate to be 12%. The most common agent used in these series was PVA, with particle size varying from 50 to 750 micrometers although many different distal particulates were used such as embospheres and gelfoam. Endovascular embolization has a high success rate and low morbidity rate. Primary success rates range from 79% to 96% with recurrence after embolization ranging from 0% to 24%. Major complications secondary to epistaxis embolization were rare, 3%, consisting of stroke (most common), monocular blindness, skin sloughing, ICA dissection and postprocedural MI. Minor complications such as self-limiting facial pain and numbness have also been described. Randomized comparison of embolization to other forms of treatment, such as surgery, have not been performed.

An important caveat is worth mentioning. Epistaxis in the setting of hereditary hemorrhagic telangiectasia (HHT or Osler-Weber-Rendu disease) is, in our experience, refractory to effective treatment with embolization. It has been our experience and confirmed in the literature that patients with HHT do not have a durable result after embolization and fare much better with operative septal dermoplasty as the initial therapeutic strategy to control severe epistaxis.

When performing endovascular embolization for epistaxis, a complete diagnostic angiogram is absolutely essential. This includes detailed evaluation of the external and internal carotid arteries. The primary vascular supply to the nasal mucosa includes the internal maxillary and distal facial arteries. Evaluation of the ICA is also critical to minimize the risk of nontarget embolization and to delineate anastomoses between the ECA and ICA circulations. It has been our experience that a definite bleeding site is very infrequently seen during angiography for idiopathic epistaxis. Traditionally,

![Fig. 13. (A) Axial CTA demonstrates a spontaneous dissection of the internal carotid artery with surrounding thrombus (*). Spontaneous dissection is similarly well demonstrated on fat-saturation T1-weighted images (B).](image-url)
embolization for idiopathic epistaxis has involved angiography of the distal nasal branches of the internal maxillary and facial arteries followed by particulate (PVA) embolization of three of the four branches (usually both distal internal maxillary artery [IMA] and the dominant facial artery) to reduce the opportunity for collateral flow to friable nasal vessels before they have time to heal. A key component to decision making is laterality. If the side of bleeding has been determined before angiography, some authors feel that single-vessel embolization, usually the IMA is sufficient for control. The ipsilateral facial nasal branches may then be embolized if there is significant vascular contribution to the nasal mucosa.46

**SUMMARY**

It is imperative for the neuroendovascular specialist to be proficient with diagnosis and treatment of common extracranial pathologies. For atherosclerotic vascular disease, technologies continue to improve and potentially expand the role of endovascular techniques for treatment while study trials are ongoing and results are anxiously awaited to determine the ultimate role of CAS in disease management. For traumatic vascular injury, embolization for vessel closure and vessel preservation using stents and stent grafts are a routine part of patient management. Although carotid blowout remains a challenge for
rapid triage and treatment, our expanding tools and aggressive ICU management help many patients to survive potentially catastrophic hemorrhage. The neuroendovascular techniques are uniquely suited to treat a wide variety of extracranial vascular diseases.

REFERENCES


